Psychological well being and Mental Disorders:

Concept of health-ill health; Positive health, well being; Causal factors in mental disorders (Anxiety disorders, mood disorders, schizophrenia and delusional disorders; personality disorders, substance abuse disorders); Factors influencing positive health, well being, life style and quality of life; Happiness disposition.
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History of mental disorders

Prehistoric times

There is limited evidence by which to judge the existence or nature of mental disorder prior to written records. Evolutionary psychology suggests that some of the underlying genetic dispositions, psychological mechanisms and social demands were present, although some disorders may have developed from a mismatch between ancestral environments and modern conditions. Some related behavioral abnormalities have been found in non-human great apes.

There is evidence from Neolithic times of the practice of trepanation (cutting large holes into the skull), possibly as an attempt to cure ailments which may have included mental disorders.

Ancient civilizations

Egyptian and Mesopotamian

Limited notes in an ancient Egyptian document known as the Ebers papyrus appear to describe disordered states of concentration and attention, and emotional distress in the heart or mind. Some of these have been interpreted as indicating what would later be termed hysteria and melancholy. Somatic treatments typically included applying bodily fluids while reciting magical spells. Hallucinogens may have been used as part of healing rituals. Religious temples may have been used as therapeutic retreats, possibly for the induction of receptive states to facilitate sleep and the interpreting of dreams.

Indian

Ancient Hindu scriptures known as Ramayana and Mahabharata contain fictional descriptions of depression and anxiety states. Mental disorders were generally thought to reflect abstract metaphysical entities, supernatural agents, sorcery or witchcraft. A work known as the Charaka Samhita from circa 600 BC, part of the Hindu Ayurveda ("knowledge of life"), saw ill health as resulting from an imbalance among three kinds of bodily fluids or forces called (Dosha). Different personality types were also described, with different propensities to worries or difficulties. Suggested causes included inappropriate diet; disrespect towards the gods, teachers or others; mental shock due to excessive fear or joy; and faulty bodily activity. Treatments included the use of herbs and ointments, charms and prayers, moral or emotional persuasion, and shocking the person.

Chinese

Mental disorders were treated mainly under Traditional Chinese Medicine by herbs, acupuncture or "emotional therapy". The Inner Canon of the Yellow Emperor described symptoms, mechanisms and therapies for mental illness, emphasizing connections between
bodily organs and emotions. Conditions were thought to comprise five stages or elements and imbalance between Yin and yang.

**Hebrew and Israelite**

The ancient nation of Israel was formed by people with origins in Mesopotamia and Egypt. The concept of a single God, as gradually articulated in Judaism, led to the view that mental disorder was not a problem like any other, caused by one of the gods, but rather caused by problems in the relationship between the individual and God. Passages of the Hebrew Bible/Old Testament have been interpreted as describing mood disorders in figures such as Job, King Saul and in the psalms of David.

**Greek and Roman**

Some ancient Greek scholars proposed that disease was caused by an imbalance in four humours of the body. Hippocrates (460-377 BC), influenced by humoral theory, proposed a triad of mental disorders termed melancholia, mania and phrenitis (an acute mental disorder accompanied by fever). He also spoke of other disorders such as phobia, and is credited with being the first physician to reject supernatural or divine explanations of illness. He believed that disease was the product of environmental factors, diet and living habits, not a punishment inflicted by the gods, and that the appropriate treatment depended on which bodily fluid, or humour, had caused the problem. However, he also objected to speculation about the etiology of madness (for example that it was seated in the heart and diaphragm or "phren") and favoured instead close behavioural observation. Plato (427-347 BC) argued that there were two types of mental illness: "divinely inspired" mental illness that gave the person prophetic powers, and a second type that was caused by a physical disease. Aristotle (384-322 BC), who studied under Plato, abandoned the divinely caused mental illness theory, and proposed instead that all mental illness was caused by physical problems.

In ancient Greece and Rome, madness was associated stereotypically with aimless wandering and violence. However, Socrates considered positive aspects including prophesying (a 'manic art'); mystical initiations and rituals; poetic inspiration; and the madness of lovers. Now often seen as the very epitome of rational thought and as the founder of philosophy, Socrates freely admitted to experiencing what are now called "command hallucinations" (then called his 'daemon'). Pythagoras also heard voices.

Through long contact with Greek culture, and their eventual conquest of Greece, the Romans absorbed many Greek (and other) ideas on medicine. The humoral theory fell out of favor in some quarters. The Greek physician Asclepiades (c. 124 – 40 BC), who practiced in Rome, discarded it and advocated humane treatments, and had insane persons freed from confinement and treated them with natural therapy, such as diet and massages. Arateus (ca AD 30-90) argued that it is hard to pinpoint where a mental illness comes from. However, Galen (AD 129 –ca. 200), practicing in Greece and Rome, revived humoral theory. Galen, however, adopted a single symptom approach rather than broad diagnostic
categories, for example studying separate states of sadness, excitement, confusion and memory loss.

Playwrights such as Homer, Sophocles and Euripides described madmen driven insane by the Gods, imbalanced humors or circumstances. As well as the triad (of which mania was often used as an overarching term for insanity) there were a variable and overlapping range of terms for such things as delusion, eccentricity, frenzy, and lunacy. Physician Celsus argued that insanity is really present when a continuous dementia begins due to the mind being at the mercy of imaginings. He suggested that people must heal their own souls through philosophy and personal strength. He described common practices of dietetics, bloodletting, drugs, talking therapy, incubation in temples, exorcism, incantations and amulets, as well as restraints and "tortures" to restore rationality, including starvation, being terrified suddenly, agitation of the spirit, and stoning and beating. Most, however, did not receive medical treatment but stayed with family or wandered the streets, vulnerable to assault and derision. Accounts of delusions from the time included people who thought themselves to be famous actors or speakers, animals, inanimate objects, or one of the gods. Some were arrested for political reasons, such as Jesus ben Ananias who was eventually released as a madman after showing no concern for his own fate during torture. It has been argued that Jesus of Nazareth was widely considered a dangerous madman, due partly to antisocial and disruptive outbursts including physical aggression, grandiose and nonsensical claims, and terse responses to official questioning - and may have been mocked as a king and crucified for that reason.

Middle Ages

Persia, Arabia and the Muslim Empire

Persian and Arabic scholars were heavily involved in translating, analysing and synthesising Greek texts and concepts. As the Muslim world expanded, these were integrated with religious thought. Over time, new ideas and concepts were developed. Arab texts contained full discussions of melancholia. Mania and various other disorders and phenomena including hallucinations and delusions were also described. Mental disorder was generally thought to be due to reason having gone astray or been lost entirely, and links were made to the brain in various ways, as well as to spiritual/mystical meaning. Al-Balkhi wrote about fear and anxiety, anger and aggression, sadness and depression, and obsessions. Al-Tabari wrote about the need for wise counselling, smartness and gaining trust. Al-Razi (Rhazes) suggested the benefits of hopeful comments and sudden emotional shocks, and addressed psychological, moral and religious problems of the spirit. Al-Farabi (Alpharabius) wrote about the therapeutic effect of music on the soul. Al-Ghazali argued that spiritual diseases were dangerous and result from ignorance and deviation from God. Ibn-Sina (Avicenna) took a combined physiological and psychological approach, addressing conditions such as hallucinations, insomnia, vertigo, melancholia and mania. He speculated about physiological influences on the brain and mental disorders, as well as about psychological interventions. Al-Majusi (Haly Abbas) described diseases in terms of the brain, including sleeping sickness, loss of memory, hypochondria and love sickness. Abu al-Qasim al-Zahrawi (Abulcasis) may have addressed mental disorder related to injury in his
pioneering work in neurosurgery, and Averroes described Parkinson’s disease. Unhammed proposed nine categories of mental disorder.

Under Islam, the mentally disordered were considered incapable yet deserving of humane treatment and protection. For example, Sura 4:5 of the Qur’an states "Do not give your property which God assigned you to manage to the insane: but feed and cloth the insane with this property and tell splendid words to him". Some thought mental disorder could be caused by possession by a djinn (genie), which could be either good or demon-like. There were sometimes beatings to exorcise djinn, or alternatively over-zealous attempts at cures. Islamic views often merged with local traditions. In Morocco the traditional Berber people were animists and the concept of sorcery was integral to the understanding of mental disorder; it was mixed with the Islamic concepts of djinn and often treated by religious scholars combining the roles of holy man, sage, seer and sorcerer.

The first psychiatric hospital ward was founded in Baghdad in 705, and insane asylums were built in Fes in the early 8th century, Cairo in 800 and in Damascus and Aleppo in 1270. Insane patients were benevolently treated using baths, drugs, music and activities. In the centuries to come, The Muslim world would eventually serve as a critical way station of knowledge for Renaissance Europe, through the Latin translations of many scientific Islamic texts. Ibn-Sina’s (Avicenna’s) Canon of Medicine became the standard of medical science in Europe for centuries, together with works of Hippocrates and Galen.

Christian Europe

Conceptions of madness in the Middle Ages in Europe were a mixture of the divine, diabolical, magical and transcendental. Theories of the four humors (black bile, yellow bile, phlegm, and blood) were applied, sometimes separately (a matter of "physic") and sometimes combined with theories of evil spirits (a matter of "faith"). Arnaldus de Villanova (1235–1313) combined "evil spirit" and Galen-oriented "four humours" theories and promoted trepanning as a cure to let demons and excess humours escape. Other bodily remedies in general use included purges, bloodletting and whipping. Madness was often seen as a moral issue, either a punishment for sin or a test of faith and character. Christian theology endorsed various therapies, including fasting and prayer for those estranged from God and exorcism of those possessed by the devil. Thus, although mental disorder was often thought to be due to sin, other more mundane causes were also explored, including intemperate diet and alcohol, overwork, and grief. The Franciscan monk Bartholomeus Anglicus (ca. 1203 - 1272) described a condition which resembles depression in his encyclopedia, De Proprietatibus Rerum, and he suggested that music would help. A semi-official tract called the Prae rogativa regis distinguished between the "natural born idiot" and the "lunatic". The latter term was applied to those with periods of mental disorder; deriving from either Roman mythology describing people "moonstruck" by the goddess Luna or theories of an influence of the moon.

Episodes of mass dancing mania are reported from the Middle Ages, "which gave to the individuals affected all the appearance of insanity". This was one kind of mass delusion or mass hysteria/panic that has occurred around the world through the millennia.
The care of lunatics was primarily the responsibility of the family. In England, if the family were unable or unwilling, an assessment was made by crown representatives in consultation with a local jury and all interested parties, including the subject himself or herself. The process was confined to those with real estate or personal estate, but it encompassed poor as well as rich and took into account psychological and social issues. Most of those considered lunatics at the time probably had more support and involvement from the community than people diagnosed with mental disorders today. As in other eras, visions were generally interpreted as meaningful spiritual and visionary insights; some may have been causally related to mental disorders, but since hallucinations were culturally supported they may not have had the same connections as today.

Modern period

16th to 18th centuries

Some mentally disturbed people may have been victims of the witch-hunts that spread in waves in early modern Europe. However, those judged insane were increasingly admitted to local workhouses, poorhouses and jails (particularly the "pauper insane") or sometimes to the new private madhouses. Restraints and forcible confinement were used for those thought dangerously disturbed or potentially violent to themselves, others or property. The latter likely grew out of lodging arrangements for single individuals (who, in workhouses, were considered disruptive or ungovernable) then there were a few catering each for only a handful of people, then they gradually expanded (e.g. 16 in London in 1774, and 40 by 1819). By the mid-19th century there would be 100 to 500 inmates in each. The development of this network of madhouses has been linked to new capitalist social relations and a service economy, that meant families were no longer able or willing to look after disturbed relatives.

Madness was commonly depicted in literary works, such as the plays of Shakespeare.

By the end of the 17th century and into the Enlightenment, madness was increasingly seen as an organic physical phenomenon, no longer involving the soul or moral responsibility. The mentally ill were typically viewed as insensitive wild animals. Harsh treatment and restraint in chains was seen as therapeutic, helping suppress the animal passions. There was sometimes a focus on the management of the environment of madhouses, from diet to exercise regimes to number of visitors. Severe somatic treatments were used, similar to those in medieval times. Madhouse owners sometimes boasted of their ability with the whip. Treatment in the few public asylums was also barbaric, often secondary to prisons. The most notorious was Bedlam where at one time spectators could pay a penny to watch the inmates as a form of entertainment.

Concepts based in humoral theory gradually gave way to metaphors and terminology from mechanics and other developing physical sciences. Complex new schemes were developed for the classification of mental disorders, influenced by emerging systems for the biological classification of organisms and medical classification of diseases.
The term "crazy" (from Middle English meaning cracked) and insane (from Latin insanus meaning unhealthy) came to mean mental disorder in this period. The term "lunacy", long used to refer to periodic disturbance or epilepsy, came to be synonymous with insanity. "Madness", long in use in root form since at least the early centuries AD, and originally meaning crippled, hurt or foolish, came to mean loss of reason or self-restraint. "Psychosis", from Greek "principle of life/animation", had varied usage referring to a condition of the mind/soul. "Nervous", from an Indo-European root meaning to wind or twist, meant muscle or vigor, was adopted by physiologists to refer to the body's electrochemical signalling process (thus called the nervous system), and was then used to refer to nervous disorders and neurosis. "Obsession", from a Latin root meaning to sit on or sit against, originally meant to besiege or be possessed by an evil spirit, came to mean a fixed idea that could decompose the mind.

With the rise of madhouses and the professionalization and specialization of medicine, there was considerable incentive for medics to become involved. In the 18th century, they began to stake a claim to a monopoly over madhouses and treatments. Madhouses could be a lucrative business, and many made a fortune from them. There were some bourgeois ex-patient reformers who opposed the often brutal regimes, blaming both the madhouse owners and the medics, who in turn resisted the reforms.

Towards the end of the 18th century, a moral treatment movement developed, that implemented more humane, psychosocial and personalized approaches. Notable figures included the medic Vincenzo Chiarugi in Italy under Enlightenment leadership; the ex-patient superintendent Pussin and the psychologically inclined medic Phillipe Pinel in revolutionary France; the Quakers in England, led by businessman William Tuke; and later, in the United States, campaigner Dorothea Dix.

19th century

The 19th century, in the context of industrialization and population growth, saw a massive expansion of the number and size of insane asylums in every Western country, a process called "the great confinement" or the "asylum era". Laws were introduced to compel authorities to deal with those judged insane by family members and hospital superintendents. Although originally based on the concepts and structures of moral treatment, they became large impersonal institutions overburdened with large numbers of people with a complex mix of mental and social-economic problems. The success of moral treatment had cast doubt on the approach of medics, and many had opposed it, but by the mid-19th century many became advocates of it but argued that the mad also often had physical/organic problems, so that both approaches were necessary. This argument has been described as an important step in the profession's eventual success in securing a monopoly on the treatment of lunacy. However, it is well documented that very little therapeutic activity occurred in the new asylum system, that medics were little more than administrators who seldom attended to patients, and then mainly for other physical problems.
Although reports of numerous mental disorders and irrational, unintelligible, or uncontrolled behavior are common in the historical record back to ancient times, clear descriptions of some syndromes, such as the condition that would later be termed schizophrenia, have been identified as relatively rare prior to the 19th century, although interpretations of the evidence and its implications are inconsistent.

Numerous different classification schemes and diagnostic terms were developed by different authorities, taking an increasingly anatomical-clinical descriptive approach. The term "psychiatry" was coined as the medical specialty became more academically established. Asylum superintendents, later to be psychiatrists, were generally called "alienists" because they were thought to deal with people alienated from society; they adopted largely isolated and managerial roles in the asylums while milder "neurotic" conditions were dealt with by neurologists and general physicians, although there was overlap for conditions such as neurasthenia.

In the United States it was proposed that black slaves who tried to escape were suffering from a mental disorder termed drapetomania. It was then argued in scientific journals that mental disorders were rare under conditions of slavery but became more common following emancipation, and later that mental illness in African Americans was due to evolutionary factors or various negative characteristics, and that they were not suitable for therapeutic intervention.

By the 1870s in North America, officials who ran Lunatic Asylums renamed them Insane Asylums. By the late century, the term "asylum" had lost its original meaning as a place of refuge, retreat or safety, and was associated with abuses that had been widely publicized in the media, including by ex-patient organization the Alleged Lunatics’ Friend Society and ex-patients like Elizabeth Packard.

The relative proportion of the public officially diagnosed with mental disorders was increasing, however. This has been linked to various factors, including possibly humanitarian concern; incentives for professional status/money; a lowered tolerance of communities for unusual behavior due to the existence of asylums to place them in (this affected the poor the most); and the strain placed on families by industrialization.

**20th century**

The turn of the 20th century saw the development of psychoanalysis, which came to the fore later. Kraepelin’s classification gained popularity, including the separation of mood disorders from what would later be termed schizophrenia.

Asylum superintendents sought to improve the image and medical status of their profession. Asylum "inmates" were increasingly referred to as "patients" and asylums renamed as hospitals. Referring to people as having a "mental illness" dates from this period in the early 20th century.
In the United States, a "mental hygiene" movement, originally defined in the 19th century, gained momentum and aimed to "prevent the disease of insanity" through public health methods and clinics. The term mental health became more popular, however. Clinical psychology and social work developed as professions alongside psychiatry. Theories of eugenics led to compulsory sterilization movements in many countries around the world for several decades, often encompassing patients in public mental institutions. World War I saw a massive increase of conditions that came to be termed "shell shock".

In Nazi Germany, the institutionalized mentally ill were among the earliest targets of sterilization campaigns and covert "euthanasia" programs. It has been estimated that over 200,000 individuals with mental disorders of all kinds were put to death, although their mass murder has received relatively little historical attention. Despite not being formally ordered to take part, psychiatrists and psychiatric institutions were at the center of justifying, planning and carrying out the atrocities at every stage, and "constituted the connection" to the later annihilation of Jews and other "undesirables" such as homosexuals in the Holocaust.

In other areas of the world, funding was often cut for asylums, especially during periods of economic decline, and during wartime in particular many patients starved to death. Soldiers received increased psychiatric attention, and World War II saw the development in the US of a new psychiatric manual for categorizing mental disorders, which along with existing systems for collecting census and hospital statistics led to the first Diagnostic and Statistical Manual of Mental Disorders (DSM). The International Classification of Diseases (ICD) followed suit with a section on mental disorders.

Previously restricted to the treatment of severely disturbed people in asylums, psychiatrists cultivated clients with a broader range of problems, and between 1917 and 1970 the number practicing outside institutions swelled from 8 percent to 66 percent. The term stress, having emerged out of endocrinology work in the 1930s, was popularized with an increasingly broad biopsychosocial meaning, and was increasingly linked to mental disorders."Outpatient commitment" laws were gradually expanded or introduced in some countries.

Lobotomies, Insulin shock therapy, Electro convulsive therapy, and the "neuroleptic" chlorpromazine came in to use mid-century.

An antipsychiatry movement came to the fore in the 1960s. Deinstitutionalization gradually occurred in the West, with isolated psychiatric hospitals being closed down in favor of community mental health services. However, inadequate services and continued social exclusion often led to many being homeless or in prison. A consumer/survivor movement gained momentum.

Other kinds of psychiatric medication gradually came into use, such as "psychic energizers" and lithium. Benzodiazepines gained widespread use in the 1970s for anxiety and depression, until dependency problems curtailed their popularity. Advances in neuroscience and genetics led to new research agendas. Cognitive behavioral therapy was
developed. Through the 1990s, new SSRI antidepressants became some of the most widely prescribed drugs in the world.

The DSM and then ICD adopted new criteria-based classification, representing a return to a Kraepelin-like descriptive system. The number of "official" diagnoses saw a large expansion, although homosexuality was gradually downgraded and dropped in the face of human rights protests. Different regions sometimes developed alternatives such as the Chinese Classification of Mental Disorders or Latin American Guide for Psychiatric Diagnosis.

21st century

Starting from 2002 DSM-5 Research Agenda researchers were invited to contribute with their publication to the literature basis for the DSM-5, whose draft criteria are now available to the scientific community. In the meanwhile, serious limits of the current version of the DSM extremely high comorbidity, diagnostic heterogeneity of the categories, unclear boundaries have been interpreted as intrinsic anomalies of the criterial, neopositivistic approach leading the system to a state of scientific crisis. Accordingly, a radical rethinking of the concept of mental disorder and the need of a radical scientific revolution in psychiatric taxonomy was proposed.

Mental disorder

Eight women representing prominent mental diagnoses in the 19th century. (Armand Gautier)

A mental disorder or mental illness is a psychological or behavioral pattern generally associated with subjective distress or disability that occurs in an individual, and which is not a part of normal development or culture. Such a disorder may consist of a combination of affective, behavioural, cognitive and perceptual components. The recognition and understanding of mental health conditions have changed over time and across cultures,
and there are still variations in the definition, assessment, and classification of mental disorders, although standard guideline criteria are widely accepted. A few mental disorders are diagnosed based on the harm to others, regardless of the subject's perception of distress. Over a third of people in most countries report meeting criteria for the major categories at some point in their lives. The causes are often explained in terms of a diathesis-stress model or biopsychosocial model. In biological psychiatry, mental disorders are conceptualized as disorders of brain circuits likely caused by developmental processes shaped by a complex interplay of genetics and experience.

Services are based in psychiatric hospitals or in the community. Diagnoses are made by psychiatrists or clinical psychologists using various methods, often relying on observation and questioning in interviews. Treatments are provided by various mental health professionals. Psychotherapy and psychiatric medication are two major treatment options, as are social interventions, peer support and self-help. In some cases there may be involuntary detention and involuntary treatment where legislation allows.

Stigma and discrimination add to the suffering associated with the disorders, and have led to various social movements attempting to increase acceptance.

**Classifications**

The definition and classification of mental disorders is a key issue for mental health and for users and providers of mental health services. Most international clinical documents use the term "mental disorder". There are currently two widely established systems that classify mental disorders—ICD-10 Chapter V: Mental and behavioural disorders, part of the International Classification of Diseases produced by the World Health Organization (WHO), and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) produced by the American Psychiatric Association (APA).

Both list categories of disorder and provide standardized criteria for diagnosis. They have deliberately converged their codes in recent revisions so that the manuals are often broadly comparable, although significant differences remain. Other classification schemes may be used in non-western cultures (see, for example, the Chinese Classification of Mental Disorders), and other manuals may be used by those of alternative theoretical persuasions, for example the Psychodynamic Diagnostic Manual. In general, mental disorders are classified separately to neurological disorders, learning disabilities or mental retardation.

Unlike most of the above systems, some approaches to classification do not employ distinct categories of disorder or dichotomous cut-offs intended to separate the abnormal from the normal. There is significant scientific debate about the different kinds of categorization and the relative merits of categorical versus non-categorical (or hybrid) schemes, with the latter including spectrum, continuum or dimensional systems.

**Disorders**
There are many different categories of mental disorder, and many different facets of human behavior and personality that can become disordered.

Anxiety or fear that interferes with normal functioning may be classified as an anxiety disorder. Commonly recognized categories include specific phobias, generalized anxiety disorder, social anxiety disorder, panic disorder, agoraphobia, obsessive-compulsive disorder and post-traumatic stress disorder.

Other affective (emotion/mood) processes can also become disordered. Mood disorder involving unusually intense and sustained sadness, melancholia or despair is known as Major depression or Clinical depression (milder but still prolonged depression can be diagnosed as dysthymia). Bipolar disorder (also known as manic depression) involves abnormally "high" or pressured mood states, known as mania or hypomania, alternating with normal or depressed mood. Whether unipolar and bipolar mood phenomena represent distinct categories of disorder, or whether they usually mix and merge together along a dimension or spectrum of mood, is under debate in the scientific literature.

Patterns of belief, language use and perception can become disordered (e.g. delusions, thought disorder, hallucinations). Psychotic disorders in this domain include schizophrenia, and delusional disorder. Schizoaffective disorder is a category used for individuals showing aspects of both schizophrenia and affective disorders. Schizotypy is a category used for individuals showing some of the characteristics associated with schizophrenia but without meeting cut-off criteria.

Personality—the fundamental characteristics of a person that influence his or her thoughts and behaviors across situations and time—may be considered disordered if judged to be abnormally rigid and maladaptive. Categorical schemes list a number of different such personality disorders, including those sometimes classed as eccentric (e.g. paranoid, schizoid and schizotypal personality disorders), to those sometimes classed as dramatic or emotional (antisocial, borderline, histrionic or narcissistic personality disorders) or those seen as fear-related (avoidant, dependent, or obsessive-compulsive personality disorders). If an inability to sufficiently adjust to life circumstances begins within three months of a particular event or situation, and ends within six months after the stressor stops or is eliminated, it may instead be classed as an adjustment disorder. There is an emerging consensus that so-called "personality disorders", like personality traits in general, actually incorporate a mixture of acute dysfunctional behaviors that resolve in short periods, and maladaptive temperamental traits that are more stable. Furthermore, there are also non-categorical schemes that rate all individuals via a profile of different dimensions of personality rather than using a cut-off from normal personality variation, for example through schemes based on the Big Five personality traits.

Eating disorders involve disproportionate concern in matters of food and weight. Categories of disorder in this area include anorexia nervosa, bulimia nervosa, exercise bulimia or binge eating disorder.
Sleep disorders such as insomnia involve disruption to normal sleep patterns, or a feeling of tiredness despite sleep appearing normal.

Sexual and gender identity disorders may be diagnosed, including dyspareunia, gender identity disorder and ego-dystonic homosexuality. Various kinds of paraphilia are considered mental disorders (sexual arousal to objects, situations, or individuals that are considered abnormal or harmful to the person or others).

People who are abnormally unable to resist certain urges or impulses that could be harmful to themselves or others, may be classed as having an impulse control disorder, including various kinds of tic disorders such as Tourette’s syndrome, and disorders such as kleptomania (stealing) or pyromania (fire-setting). Various behavioral addictions, such as gambling addiction, may be classed as a disorder. Obsessive-compulsive disorder can sometimes involve an inability to resist certain acts but is classed separately as being primarily an anxiety disorder.

The use of drugs (legal or illegal), when it persists despite significant problems related to the use, may be defined as a mental disorder termed substance dependence or substance abuse (a broader category than drug abuse). The DSM does not currently use the common term drug addiction and the ICD simply talks about "harmful use". Disordered substance use may be due to a pattern of compulsive and repetitive use of the drug that results in tolerance to its effects and withdrawal symptoms when use is reduced or stopped.

People who suffer severe disturbances of their self-identity, memory and general awareness of themselves and their surroundings may be classed as having a dissociative identity disorder, such as depersonalization disorder or Dissociative Identity Disorder itself (which has also been called multiple personality disorder, or "split personality"). Other memory or cognitive disorders include amnesia or various kinds of old age dementia.

A range of developmental disorders that initially occur in childhood may be diagnosed, for example autism spectrum disorders, oppositional defiant disorder and conduct disorder, and attention deficit hyperactivity disorder (ADHD), which may continue into adulthood.

Conduct disorder, if continuing into adulthood, may be diagnosed as antisocial personality disorder (dissocial personality disorder in the ICD). Popularist labels such as psychopath (or sociopath) do not appear in the DSM or ICD but are linked by some to these diagnoses.

Disorders appearing to originate in the body, but thought to be mental, are known as somatoform disorders, including somatization disorder and conversion disorder. There are also disorders of the perception of the body, including body dysmorphic disorder. Neurasthenia is an old diagnosis involving somatic complaints as well as fatigue and low spirits/depression, which is officially recognized by the ICD-10 but no longer by the DSM-IV.
Factitious disorders, such as Munchausen syndrome, are diagnosed where symptoms are thought to be experienced (deliberately produced) and/or reported (feigned) for personal gain.

There are attempts to introduce a category of relational disorder, where the diagnosis is of a relationship rather than on any one individual in that relationship. The relationship may be between children and their parents, between couples, or others. There already exists, under the category of psychosis, a diagnosis of shared psychotic disorder where two or more individuals share a particular delusion because of their close relationship with each other.

Various new types of mental disorder diagnosis are occasionally proposed. Among those controversially considered by the official committees of the diagnostic manuals include self-defeating personality disorder, sadistic personality disorder, passive-aggressive personality disorder and premenstrual dysphoric disorder.

Two recent unique isolated proposals are solastalgia by Glenn Albrecht and hubris syndrome by David Owen. The application of the concept of mental illness to the phenomena described by these authors has in turn been critiqued by Seamus Mac Suibhne.

Causes

Mental disorders can arise from a combination of sources. In many cases there is no single accepted or consistent cause currently established. A common belief even to this day is that disorders result from genetic vulnerabilities exposed by environmental stressors. (see Diathesis–stress model). However, it is clear enough from a simple statistical analysis across the whole spectrum of mental health disorders at least in western cultures that there is a strong relationship between the various forms of severe and complex mental disorder in adulthood and the abuse (physical, sexual or emotional) or neglect of children during the developmental years.

An eclectic or pluralistic mix of models may be used to explain particular disorders, and the primary paradigm of contemporary mainstream Western psychiatry is said to be the biopsychosocial (BPS) model, incorporating biological, psychological and social factors, although this may not always be applied in practice. Biopsychiatry has tended to follow a biomedical model, focusing on "organic" or "hardware" pathology of the brain. Psychoanalytic theories have continued to evolve alongside cognitive-behavioural and systemic-family approaches. Evolutionary psychology may be used as an overall explanatory theory, and attachment theory is another kind of evolutionary-psychological approach sometimes applied in the context of mental disorders. A distinction is sometimes made between a "medical model" or a "social model" of disorder and disability.

Studies have indicated that genes often play an important role in the development of mental disorders, although the reliable identification of connections between specific genes and specific categories of disorder has proven more difficult. Environmental events surrounding pregnancy and birth have also been implicated. Traumatic brain injury may
increase the risk of developing certain mental disorders. There have been some tentative inconsistent links found to certain viral infections, to substance misuse, and to general physical health.

Abnormal functioning of neurotransmitter systems has been implicated, including serotonin, norepinephrine, dopamine and glutamate systems. Differences have also been found in the size or activity of certain brain regions in some cases. Psychological mechanisms have also been implicated, such as cognitive (e.g. reasoning), emotional processes, personality, temperament and coping style.

Social influences have been found to be important, including abuse, bullying and other negative or stressful life experiences. The specific risks and pathways to particular disorders are less clear, however. Aspects of the wider community have also been implicated, including employment problems, socioeconomic inequality, lack of social cohesion, problems linked to migration, and features of particular societies and cultures.

**Causes of mental disorders**

The causes of mental disorders are complex, and interact and vary according to the particular disorder and individual. Genetics, early development, drugs, a loss of a family member, disease or injury, neurocognitive and psychological mechanisms, and life experiences, society and culture, can all contribute to the development or progression of different mental disorders.

**General theories**

There are a number of theories or models seeking to explain the causes (etiology) of mental disorders. They may be based on different foundations, including their basic classification of mental disorders.

The most common view is that disorders tend to result from genetic vulnerabilities and environmental stressors combining to cause patterns of dysfunction or trigger disorders (Diathesis-stress model). A practical mixture of models may often be used to explain particular issues and disorders, although there may be difficulty defining boundaries for indistinct psychiatric syndromes.

The primary model of contemporary mainstream Western psychiatry is the biopsychosocial model (BPS), which merges together biological, psychological and social factors. It may be commonly neglected or misapplied in practice due to being too broad or relativistic, however, and biopsychiatry has tended to follow a biomedical model focused on organic or "hardware" pathology of the brain.

Psychoanalytic theories, focused on unresolved internal and relational conflicts, have been posited as overall explanations of mental disorder, although today most psychoanalytic groups are said to adhere to the biopsychosocial model and to accept an eclectic mix of subtypes of psychoanalysis.
Evolutionary psychology (or more specifically evolutionary psychopathology or psychiatry) has also been proposed as an overall theory, positing that many mental disorders involve the dysfunctional operation of mental modules adapted to ancestral physical or social environments but not necessarily to modern ones. Attachment theory is another kind of evolutionary-psychological approach sometimes applied in the context for mental disorders, which focuses on the role of early caregiver-child relationships, responses to danger, and the search for a satisfying reproductive relationship in adulthood.

An overall distinction is also commonly made between a "medical model" (also known as a biomedical or disease model), and a "social model" (also known as an empowerment or recovery model) of mental disorder and disability, with the former focusing on hypothesized disease processes and symptoms, and the latter focusing on hypothesized social constructionism and social contexts.

**Genes**

Family-linkage and twin studies have indicated that genetic factors often play an important role in the development of mental disorders. The reliable identification of specific genetic susceptibility to particular disorders, through linkage or association studies, has proven difficult. This has been reported to be likely due to the complexity of interactions between genes, environmental events, and early development or to the need for new research strategies. The heritability of behavioral traits associated with mental disorder may be greater in permissive than in restrictive environments, and susceptibility genes probably work through both "within-the-skin" (physiological) pathways and "outside-the-skin" (behavioral and social) pathways. Investigations increasingly focus on links between genes and endophenotypes—more specific traits (including neurophysiological, biochemical, endocrinological, neuroanatomical, cognitive, or neuropsychological)—rather than disease categories. With regard to a prominent mental disorder, Schizophrenia, genetics have been shown to have an increasingly complicated role in the disease. For a long time consensus among scientists was that certain alleles were responsible for schizophrenia, but recent research has shown that this is not necessarily the case. In a study done in 2008 by Walsh et al., the genetics of schizophrenics were compared to those of non-affected individuals. The group did micro-arrays of each individuals’ genome looking for structural variants greater than 100kbp. Individuals with schizophrenia were three times as likely to harbor structural variants that duplicated or deleted one or more genes. This was even more prevalent in children as schizophrenics under age 18 were four times as likely to harbor these mutations. More importantly 53 previously unreported microduplications/deletions were discovered and virtually every rare structural mutation was different.

**Pregnancy and birth**

Environmental events surrounding pregnancy and birth have been linked to an increased development of mental illness in the offspring. This includes maternal exposure to serious psychological stress or trauma, conditions of famine, obstetric birth complications, infections, and gestational exposure to alcohol or cocaine. Such factors have been
hypothesized to affect specific areas of neurodevelopment within the general developmental context and to restrict neuroplasticity.

People with developmental disabilities, such as mental retardation, are more likely to experience mental illness than those in the general community.

**Disease, injury and infection**

Higher rates of mood, psychotic, and substance abuse disorders have been found following traumatic brain injury (TBI). Findings on the relationship between TBI severity and prevalence of subsequent psychiatric disorders have been inconsistent, and occurrence has been linked to prior mental health problems as well as direct neurophysiological effects, in a complex interaction with personality and attitude and social influences.

A number of psychiatric disorders have often been tentatively linked with microbial pathogens, particularly viruses; however while there have been some suggestions of links from animal studies, and some inconsistent evidence for infectious and immune mechanisms (including prenatally) in some human disorders, infectious disease models in psychiatry are reported to have not yet shown significant promise except in isolated cases. There have been some inconsistent findings of links between infection by the parasite Toxoplasma gondii and human mental disorders such as schizophrenia, with the direction of causality unclear. A number of diseases of the white matter can cause symptoms of mental disorder.

Poorer general health has been found among individuals with severe mental illnesses, thought to be due to direct and indirect factors including diet, bacterial infections, substance use, exercise levels, effects of medications, socioeconomic disadvantages, lowered help-seeking or treatment adherence, or poorer healthcare provision. Some chronic general medical conditions have been linked to some aspects of mental disorder, such as AIDS-related psychosis.

The current research on Lyme's disease caused by a deer tick, and related toxins, is expanding the link between bacterial infections and mental illness.

**Individual characteristics**

Mental characteristics of individuals, as assessed by both neurological and psychological studies, have been linked to the development and maintenance of mental disorders. This includes cognitive or neurocognitive factors, such as the way a person perceives, thinks or feels about certain things; or an individual's overall personality, temperament or coping style or the extent of protective factors or "positive illusions" such as optimism, personal control and a sense of meaning.

Abnormal levels of dopamine activity have been implicated in a number of disorders (e.g., reduced in ADHD, increased in schizophrenia), thought to be part of the complex encoding of the importance of events in the external world. Dysfunction in serotonin and other
monoamine neurotransmitters such as norepinephrine and dopamine has also been centrally implicated in mental disorders, including major depression as well as obsessive compulsive disorder, phobias, posttraumatic stress disorder, and generalized anxiety disorder, although the limitations of a simple "monoamine hypothesis" have been highlighted and studies of depleted levels of monoamine neurotransmitters have tended to indicate no simple or directly causal relation with mood or major depression, although features of these pathways may form trait vulnerabilities to depression. Dysfunction of the central gamma-aminobutyric (GABA) system following stress has also been associated with anxiety spectrum disorders and there is now a body of clinical and preclinical literature also indicating an overlapping role in mood disorder.

Findings have indicated abnormal functioning of brainstem structures in disorders such as schizophrenia, related to impairments in maintaining sustained attention. Some abnormalities in the average size or shape of some regions of the brain have been found in some disorders, reflecting genes and/or experience. Studies of schizophrenia have tended to find enlarged ventricles and sometimes reduced volume of the cerebrum and hippocampus, while studies of (psychotic) bipolar disorder have sometimes found increased amygdala volume. Findings differ over whether volumetric abnormalities are risk factors or are only found alongside the course of mental health problems, possibly reflecting neurocognitive or emotional stress processes and/or medication use or substance use. Some studies have also found reduced hippocampal volumes in major depression, possibly worsening with time depressed.

**Life events, emotional stress and relationships**

It is reported that there is good evidence on the importance of psychosocial influences on psychopathology in general, although less known about the specific risk and protective mechanisms. Maltreatment in childhood and in adulthood, including sexual abuse, physical abuse, emotional abuse, domestic violence and bullying, has been linked to the development of mental disorders, through a complex interaction of societal, family, psychological and biological factors. Negative or stressful life events more generally have been implicated in the development of a range of disorders, including mood and anxiety disorders. The main risks appear to be from a cumulative combination of such experiences over time, although exposure to a single major trauma can sometimes lead to psychopathology, including PTSD. Resilience to such experiences varies, and a person may be resistant to some forms of experience but susceptible to others. Features associated with variations in resilience include genetic vulnerability, temperamental characteristics, cognitive set, coping patterns, and other experiences.

Relationship issues have been consistently linked to the development of mental disorders, with continuing debate on the relative importance of the home environment or work/school and peer group. Issues with parenting skills or parental depression or other problems may be a risk factor. Parental divorce appears to increase risk, perhaps only if there is family discord or disorganization, although a warm supportive relationship with one parent may compensate. Details of infant feeding, weaning, toilet training etc. do not appear to be importantly linked to psychopathology. Early social privation, or lack of
ongoing, harmonious, secure, committed relationships, have been implicated in the
development of mental disorders.

Some approaches, such as certain theories of co-counseling, may see all non-neurological
mental disorders as the result of the self-regulating mechanisms of the mind (which
accompany the physical expression of emotions) not being allowed to operate.

**Neighborhoods, society and culture**

Problems in communities or cultures, including poverty, unemployment or
underemployment, lack of social cohesion, and migration, have been implicated in the
development of mental disorders. Stresses and strains related to socioeconomic position
(socioeconomic status (SES) or social class) have been linked to the occurrence of major
mental disorders, with a lower or more insecure educational, occupational, economic or
social position generally linked to more mental disorders. There have been mixed findings
on the nature of the links and on the extent to which pre-existing personal characteristics
influence the links. Both personal resources and community factors have been implicated,
as well as interactions between individual-level and regional-level income levels. The
causal role of different socioeconomic factors may vary by country. Socioeconomic
deprivation in neighborhoods can cause worse mental health, even after accounting for
genetic factors. In addition, minority ethnic groups, including first or second-generation
immigrants, have been found to be at greater risk for developing mental disorders, which
has been attributed to various kinds of life insecurities and disadvantages, including
racism. The direction of causality is sometimes unclear, and alternative hypotheses such as
the Drift Hypothesis sometimes need to be discounted.

Mental disorders have also been linked to the overarching social, economic and cultural
system. A value system that promotes individualism, weakens social ties, and creates
ambivalence towards children, is being spread or imposed via globalization, yet could
adversely affect children’s mental health.

**Gender-specific influences**

Female-specific indicators of mental illness incorporate high progesterone oral
contraceptives.

**Diagnosis**

Many mental health professionals, particularly psychiatrists, seek to diagnose individuals
by ascertaining their particular mental disorder. Some professionals, for example some
clinical psychologists, may avoid diagnosis in favor of other assessment methods such as
formulation of a client’s difficulties and circumstances. The majority of mental health
problems are actually assessed and treated by family physicians during consultations, who
may refer on for more specialist diagnosis in acute or chronic cases. Routine diagnostic
practice in mental health services typically involves an interview (which may be referred to
as a mental status examination), where judgments are made of the interviewee’s
appearance and behavior, self-reported symptoms, mental health history, and current life circumstances. The views of relatives or other third parties may be taken into account. A physical examination to check for ill health or the effects of medications or other drugs may be conducted. Psychological testing is sometimes used via paper-and-pen or computerized questionnaires, which may include algorithms based on ticking off standardized diagnostic criteria, and in rare specialist cases neuroimaging tests may be requested, but these methods are more commonly found in research studies than routine clinical practice.

Time and budgetary constraints often limit practicing psychiatrists from conducting more thorough diagnostic evaluations. It has been found that most clinicians evaluate patients using an unstructured, open-ended approach, with limited training in evidence-based assessment methods, and that inaccurate diagnosis may be common in routine practice. Mental illness involving hallucinations or delusions (especially schizophrenia) are prone to misdiagnosis in developing countries due to the presence of psychotic symptoms instigated by nutritional deficiencies. Comorbidity is very common in psychiatric diagnoses, i.e. the same person given a diagnosis in more than one category of disorder.

**Management**

Treatment and support for mental disorders is provided in psychiatric hospitals, clinics or any of a diverse range of community mental health services. In many countries services are increasingly based on a recovery model that is meant to support each individual’s independence, choice and personal journey to regain a meaningful life, although individuals may be treated against their will in a minority of cases. There are a range of different types of treatment and what is most suitable depends on the disorder and on the individual. Many things have been found to help at least some people, and a placebo effect may play a role in any intervention or medication.

**Psychotherapy**

A major option for many mental disorders is psychotherapy. There are several main types. Cognitive behavioral therapy (CBT) is widely used and is based on modifying the patterns of thought and behavior associated with a particular disorder. Psychoanalysis, addressing underlying psychic conflicts and defenses, has been a dominant school of psychotherapy and is still in use. Systemic therapy or family therapy is sometimes used, addressing a network of significant others as well as an individual.

Some psychotherapies are based on a humanistic approach. There are a number of specific therapies used for particular disorders, which may be offshoots or hybrids of the above types. Mental health professionals often employ an eclectic or integrative approach. Much may depend on the therapeutic relationship, and there may be problems with trust, confidentiality and engagement.

**Medication**
A major option for many mental disorders is psychiatric medication and there are several main groups. Antidepressants are used for the treatment of clinical depression as well as often for anxiety and other disorders. Anxiolytics are used for anxiety disorders and related problems such as insomnia. Mood stabilizers are used primarily in bipolar disorder. Antipsychotics are mainly used for psychotic disorders, notably for positive symptoms in schizophrenia. Stimulants are commonly used, notably for ADHD.

Despite the different conventional names of the drug groups, there may be considerable overlap in the disorders for which they are actually indicated, and there may also be off-label use of medications. There can be problems with adverse effects of medication and adherence to them, and there is also criticism of pharmaceutical marketing and professional conflicts of interest.

Other

Electroconvulsive therapy (ECT) is sometimes used in severe cases when other interventions for severe intractable depression have failed. Psychosurgery is considered experimental but is advocated by certain neurologists in certain rare cases.

Counseling (professional) and co-counseling (between peers) may be used. Psychoeducation programs may provide people with the information to understand and manage their problems. Creative therapies are sometimes used, including music therapy, art therapy or drama therapy. Lifestyle adjustments and supportive measures are often used, including peer support, self-help groups for mental health and supported housing or supported employment (including social firms). Some advocate dietary supplements.

Prognosis

Prognosis depends on the disorder, the individual and numerous related factors. Some disorders are transient, while others may last a lifetime. Some disorders may be very limited in their functional effects, while others may involve substantial disability and support needs. The degree of ability or disability may vary across different life domains. Continued disability has been linked to institutionalization, discrimination and social exclusion as well as to the inherent properties of disorders.

Even those disorders often considered the most serious and intractable have varied courses. Long-term international studies of schizophrenia have found that over a half of individuals recover in terms of symptoms, and around a fifth to a third in terms of symptoms and functioning, with some requiring no medication. At the same time, many have serious difficulties and support needs for many years, although "late" recovery is still possible. The World Health Organization concluded that the long-term studies' findings converged with others in "relieving patients, carers and clinicians of the chronicity paradigm which dominated thinking throughout much of the 20th century."

Around half of people initially diagnosed with bipolar disorder achieve syndromal recovery (no longer meeting criteria for the diagnosis) within six weeks, and nearly all achieve it
within two years, with nearly half regaining their prior occupational and residential status in that period. However, nearly half go on to experience a new episode of mania or major depression within the next two years. Functioning has been found to vary, being poor during periods of major depression or mania but otherwise fair to good, and possibly superior during periods of hypomania in Bipolar II.

Suicide, which is often attributed to some underlying mental disorder, is a leading cause of death among teenagers and adults under 35. There are an estimated 10 to 20 million non-fatal attempted suicides every year worldwide.

Despite often being characterized in purely negative terms, some mental states labeled as disorders can also involve above-average creativity, non-conformity, goal-striving, meticulousness, or empathy. In addition, the public perception of the level of disability associated with mental disorders can change.

**Epidemiology**

Mental disorders are common. World wide more than one in three people in most countries report sufficient criteria for at least one at some point in their life. In the United States 46% qualifies for a mental illness at some point. An ongoing survey indicates that anxiety disorders are the most common in all but one country, followed by mood disorders in all but two countries, while substance disorders and impulse-control disorders were consistently less prevalent. Rates varied by region.

A review of anxiety disorder surveys in different countries found average lifetime prevalence estimates of 16.6%, with women having higher rates on average. A review of mood disorder surveys in different countries found lifetime rates of 6.7% for major depressive disorder (higher in some studies, and in women) and 0.8% for Bipolar I disorder.

In the United States the frequency of disorder is: anxiety disorder (28.8%), mood disorder (20.8%), impulse-control disorder (24.8%) or substance use disorder (14.6%).

A 2004 cross-Europe study found that approximately one in four people reported meeting criteria at some point in their life for at least one of the DSM-IV disorders assessed, which included mood disorders (13.9%), anxiety disorders (13.6%) or alcohol disorder (5.2%). Approximately one in ten met criteria within a 12-month period. Women and younger people of either gender showed more cases of disorder. A 2005 review of surveys in 16 European countries found that 27% of adult Europeans are affected by at least one mental disorder in a 12 month period.

An international review of studies on the prevalence of schizophrenia found an average (median) figure of 0.4% for lifetime prevalence; it was consistently lower in poorer countries.
Studies of the prevalence of personality disorders (PDs) have been fewer and smaller-scale, but one broad Norwegian survey found a five-year prevalence of almost 1 in 7 (13.4%). Rates for specific disorders ranged from 0.8% to 2.8%, differing across countries, and by gender, educational level and other factors. A US survey that incidentally screened for personality disorder found a rate of 14.79%.

Approximately 7% of a preschool pediatric sample were given a psychiatric diagnosis in one clinical study, and approximately 10% of 1- and 2-year-olds receiving developmental screening have been assessed as having significant emotional/behavioral problems based on parent and pediatrician reports.

While rates of psychological disorders are the same for men and women, women have twice the rate of depression than men. Each year 73 million women are afflicted with major depression, and suicide is ranked 7th as the cause of death for women between the ages of 20-59. Depressive disorders account for close to 41.9% of the disability from neuropsychiatric disorders among women compared to 29.3% among men.

**History**

![Early color illustration of psychiatric treatment methods](image)

**Ancient civilizations**

Ancient civilizations described and treated a number of mental disorders. The Greeks coined terms for melancholy, hysteria and phobia and developed the humorism theory. Mental disorders were described, and treatments developed, in Persia, Arabia and in the medieval Islamic world.

**Europe**
Middle Ages

Conceptions of madness in the Middle Ages in Christian Europe were a mixture of the divine, diabolical, magical and humoral, as well as more down to earth considerations. In the early modern period, some people with mental disorders may have been victims of the witch-hunts but were increasingly admitted to local workhouses and jails or sometimes to private madhouses. Many terms for mental disorder that found their way into everyday use first became popular in the 16th and 17th centuries.

Eighteenth century

By the end of the 17th century and into the Enlightenment, madness was increasingly seen as an organic physical phenomenon with no connection to the soul or moral responsibility. Asylum care was often harsh and treated people like wild animals, but towards the end of the 18th century a moral treatment movement gradually developed. Clear descriptions of some syndromes may be rare prior to the 19th century.

Nineteenth century

Industrialization and population growth led to a massive expansion of the number and size of insane asylums in every Western country in the 19th century. Numerous different classification schemes and diagnostic terms were developed by different authorities, and the term psychiatry was coined, though medical superintendents were still known as alienists.

Twentieth century

The turn of the 20th century saw the development of psychoanalysis, which would later come to the fore, along with Kraepelin’s classification scheme. Asylum "inmates" were increasingly referred to as "patients", and asylums renamed as hospitals.

Europe and the U.S.

In the 20th century in the United States, a mental hygiene movement developed, aiming to prevent mental disorders. Clinical psychology and social work developed as professions. World War I saw a massive increase of conditions that came to be termed "shell shock".

World War II saw the development in the U.S. of a new psychiatric manual for categorizing mental disorders, which along with existing systems for collecting census and hospital statistics led to the first Diagnostic and Statistical Manual of Mental Disorders (DSM). The International Classification of Diseases (ICD) followed suit with a section on mental disorders. The term stress, having emerged out of endocrinology work in the 1930s, was increasingly applied to mental disorders.
Electroconvulsive therapy, insulin shock therapy, lobotomies and the "neuroleptic" chlorpromazine came to be used by mid-century. An antipsychiatry movement came to the fore in the 1960s. Deinstitutionalization gradually occurred in the West, with isolated psychiatric hospitals being closed down in favor of community mental health services. A consumer/survivor movement gained momentum. Other kinds of psychiatric medication gradually came into use, such as "psychic energizers" and lithium. Benzodiazepines gained widespread use in the 1970s for anxiety and depression, until dependency problems curtailed their popularity.

Advances in neuroscience and genetics led to new research agendas. Cognitive behavioral therapy was developed. The DSM and then ICD adopted new criteria-based classifications, and the number of "official" diagnoses saw a large expansion. Through the 1990s, new SSRI antidepressants became some of the most widely prescribed drugs in the world. Also during the 1990s, a recovery model developed.

Society and culture

Different societies or cultures and even different individuals in a culture can disagree as to what constitutes optimal versus pathological biological and psychological functioning. Research has demonstrated that cultures vary in the relative importance placed on, for example, happiness, autonomy, or social relationships for pleasure. Likewise, the fact that a
behavior pattern is valued, accepted, encouraged, or even statistically normative in a
culture does not necessarily mean that it is conducive to optimal psychological functioning.

People in all cultures find some behaviors bizarre or even incomprehensible. But just what
they feel is bizarre or incomprehensible is ambiguous and subjective. These differences in
determination can become highly contentious.

The process by which conditions and difficulties come to be defined and treated as medical
conditions and problems, and thus come under the authority of doctors and other health
professionals, is known as medicalization or pathologization.

In the scientific and academic literature on the definition or classification of mental
disorder, one extreme argues that it is entirely a matter of value judgements (including of
what is normal) while another proposes that it is or could be entirely objective and
scientific (including by reference to statistical norms). Common hybrid views argue that
the concept of mental disorder is objective but a "fuzzy prototype" that can never be
precisely defined, or alternatively that it inevitably involves a mix of scientific facts and
subjective value judgments.

**Professions and fields**

A number of professions have developed that specialize in the treatment of mental
disorders, including the medical speciality of psychiatry (including psychiatric nursing), a
subset of psychology known as clinical psychology, social work, as well as mental health
counselors, marriage and family therapists, psychotherapists, counselors and public health
professionals. Those with personal experience of using mental health services are also
increasingly involved in researching and delivering mental health services and working as
mental health professionals. The different clinical and scientific perspectives draw on
diverse fields of research and theory, and different disciplines may favor differing models,
explanations and goals.

**Movements**

The consumer/survivor movement (also known as user/survivor movement) is made up of
individuals (and organizations representing them) who are clients of mental health
services or who consider themselves "survivors" of mental health services. The movement
campaigns for improved mental health services and for more involvement and
empowerment within mental health services, policies and wider society. Patient advocacy
organizations have expanded with increasing deinstitutionalization in developed countries,
working to challenge the stereotypes, stigma and exclusion associated with psychiatric
conditions. An antipsychiatry movement fundamentally challenges mainstream psychiatric
theory and practice, including asserting that psychiatric diagnoses of mental illnesses are
neither real nor useful.

**Intangible experiences**
Religious, spiritual, or transpersonal experiences and beliefs are typically not defined as disordered, especially if widely shared, despite meeting many criteria of delusional or psychotic disorders. Even when a belief or experience can be shown to produce distress or disability—the ordinary standard for judging mental disorders—the presence of a strong cultural basis for that belief, experience, or interpretation of experience, generally disqualifies it from counting as evidence of mental disorder.

**Western bias**

Current diagnostic guidelines have been criticized as having a fundamentally Euro-American outlook. They have been widely implemented, but opponents argue that even when diagnostic criteria are accepted across different cultures, it does not mean that the underlying constructs have any validity within those cultures; even reliable application can prove only consistency, not legitimacy.

Advocating a more culturally sensitive approach, critics such as Carl Bell and Marcello Maviglia contend that the cultural and ethnic diversity of individuals is often discounted by researchers and service providers.

Cross-cultural psychiatrist Arthur Kleinman contends that the Western bias is ironically illustrated in the introduction of cultural factors to the DSM-IV: that disorders or concepts from non-Western or non-mainstream cultures are described as "culture-bound", whereas standard psychiatric diagnoses are given no cultural qualification whatsoever, reveals to Kleinman an underlying assumption that Western cultural phenomena are universal. Kleinman's negative view towards the culture-bound syndrome is largely shared by other cross-cultural critics, common responses included both disappointment over the large number of documented non-Western mental disorders still left out and frustration that even those included were often misinterpreted or misrepresented.

Many mainstream psychiatrists are dissatisfied with the new culture-bound diagnoses, although for different reasons. Robert Spitzer, a lead architect of the DSM-III, has hypothesized that adding cultural formulations was an attempt to appease cultural critics and stated that the formulations lack any scientific motivation or support. Spitzer also posits that the new culture-bound diagnoses are rarely used, maintaining that the standard diagnoses apply regardless of the culture involved. In general, mainstream psychiatric opinion remains that if a diagnostic category is valid, cross-cultural factors are either irrelevant or are significant only to specific symptom presentations.

**Relationships and morality**

Clinical conceptions of mental illness also overlap with personal and cultural values in the domain of morality, so much so that it is sometimes argued that separating the two is impossible without fundamentally redefining the essence of being a particular person in a society. In clinical psychiatry, persistent distress and disability indicate an internal disorder requiring treatment; but in another context, that same distress and disability can be seen as an indicator of emotional struggle and the need to address social and structural
problems. This dichotomy has led some academics and clinicians to advocate a postmodernist conceptualization of mental distress and well-being.

Such approaches, along with cross-cultural and "heretical" psychologies centered on alternative cultural and ethnic and race-based identities and experiences, stand in contrast to the mainstream psychiatric community’s active avoidance of any involvement with either morality or culture. In many countries there are attempts to challenge perceived prejudice against minority groups, including alleged institutional racism within psychiatric services.

**Laws and policies**

Three quarters of countries around the world have mental health legislation. Compulsory admission to mental health facilities (also known as involuntary commitment or sectioning), is a controversial topic. From some points of view it can impinge on personal liberty and the right to choose, and carry the risk of abuse for political, social and other reasons; from other points of view, it can potentially prevent harm to self and others, and assist some people in attaining their right to healthcare when unable to decide in their own interests.

All human-rights oriented mental health laws require proof of the presence of a mental disorder as defined by internationally accepted standards, but the type and severity of disorder that counts can vary in different jurisdictions. The two most often utilized grounds for involuntary admission are said to be serious likelihood of immediate or imminent danger to self or others, and the need for treatment. Applications for someone to be involuntarily admitted may usually come from a mental health practitioner, a family member, a close relative, or a guardian. Human-rights-oriented laws usually stipulate that independent medical practitioners or other accredited mental health practitioners must examine the patient separately and that there should be regular, time-bound review by an independent review body. An individual must be shown to lack the capacity to give or withhold informed consent (i.e. to understand treatment information and its implications). Legal challenges in some areas have resulted in supreme court decisions that a person does not have to agree with a psychiatrist’s characterization of their issues as an "illness", nor with a psychiatrist's conviction in medication, but only recognize the issues and the information about treatment options.

Proxy consent (also known as substituted decision-making) may be given to a personal representative, a family member or a legally appointed guardian, or patients may have been able to enact an advance directive as to how they wish to be treated. The right to supported decision-making may also be included in legislation. Involuntary treatment laws are increasingly extended to those living in the community, for example outpatient commitment laws (known by different names) are used in New Zealand, Australia, the United Kingdom and most of the United States.

The World Health Organization reports that in many instances national mental health legislation takes away the rights of persons with mental disorders rather than protecting
rights, and is often outdated. In 1991, the United Nations adopted the Principles for the Protection of Persons with Mental Illness and the Improvement of Mental Health Care, which established minimum human rights standards of practice in the mental health field. In 2006, the UN formally agreed the Convention on the Rights of Persons with Disabilities to protect and enhance the rights and opportunities of disabled people, including those with psychosocial disabilities.

The term insanity, sometimes used colloquially as a synonym for mental illness, is often used technically as a legal term. The insanity defense may be used in a legal trial (known as the mental disorder defence in some countries).

**Perception and discrimination**

**Stigma**

The social stigma associated with mental disorders is a widespread problem. Some people believe those with serious mental illnesses cannot recover, or are to blame for problems. The US Surgeon General stated in 1999 that: "Powerful and pervasive, stigma prevents people from acknowledging their own mental health problems, much less disclosing them to others." Employment discrimination is reported to play a significant part in the high rate of unemployment among those with a diagnosis of mental illness.

Efforts are being undertaken worldwide to eliminate the stigma of mental illness, although their methods and outcomes have sometimes been criticized.

A 2008 study by Baylor University researchers found that clergy in the US often deny or dismiss the existence of a mental illness. Of 293 Christian church members, more than 32 percent were told by their church pastor that they or their loved one did not really have a mental illness, and that the cause of their problem was solely spiritual in nature, such as a personal sin, lack of faith or demonic involvement. The researchers also found that women were more likely than men to get this response. All participants in both studies were previously diagnosed by a licensed mental health provider as having a serious mental illness. However, there is also research suggesting that people are often helped by extended families and supportive religious leaders who listen with kindness and respect, which can often contrast with usual practice in psychiatric diagnosis and medication.

**Media and general public**

Media coverage of mental illness comprises predominantly negative depictions, for example, of incompetence, violence or criminality, with far less coverage of positive issues such as accomplishments or human rights issues. Such negative depictions, including in children’s cartoons, are thought to contribute to stigma and negative attitudes in the public and in those with mental health problems themselves, although more sensitive or serious cinematic portrayals have increased in prevalence.
In the United States, the Carter Center has created fellowships for journalists in South Africa, the U.S., and Romania, to enable reporters to research and write stories on mental health topics. Former US First Lady Rosalynn Carter began the fellowships not only to train reporters in how to sensitively and accurately discuss mental health and mental illness, but also to increase the number of stories on these topics in the news media. There is a World Mental Health Day, which the US and Canada subsume under a Mental Illness Awareness Week.

The general public have been found to hold a strong stereotype of dangerousness and desire for social distance from individuals described as mentally ill. A US national survey found that a higher percentage of people rate individuals described as displaying the characteristics of a mental disorder as "likely to do something violent to others", compared to the percentage of people who are rating individuals described as being "troubled".

**Violence**

Despite public or media opinion, national studies have indicated that severe mental illness does not independently predict future violent behavior, on average, and is not a leading cause of violence in society. There is a statistical association with various factors that do relate to violence (in anyone), such as substance abuse and various personal, social and economic factors.

In fact, findings consistently indicate that it is many times more likely that people diagnosed with a serious mental illness living in the community will be the victims rather than the perpetrators of violence. In a study of individuals diagnosed with "severe mental illness" living in a US inner-city area, a quarter were found to have been victims of at least one violent crime over the course of a year, a proportion eleven times higher than the inner-city average, and higher in every category of crime including violent assaults and theft. People with a diagnosis may find it more difficult to secure prosecutions, however, due in part to prejudice and being seen as less credible.

However, there are some specific diagnoses, such as childhood conduct disorder or adult antisocial personality disorder or psychopathy, which are defined by or inherently associated with conduct problems and violence. There are conflicting findings about the extent to which certain specific symptoms, notably some kinds of psychosis (hallucinations or delusions) that can occur in disorders such as schizophrenia, delusional disorder or mood disorder, are linked to an increased risk of serious violence on average. The mediating factors of violent acts, however, are most consistently found to be mainly socio-demographic and socio-economic factors such as being young, male, of lower socioeconomic status and, in particular, substance abuse (including alcoholism) to which some people may be particularly vulnerable.

High-profile cases have led to fears that serious crimes, such as homicide, have increased due to deinstitutionalization, but the evidence does not support this conclusion. Violence that does occur in relation to mental disorder (against the mentally ill or by the mentally ill) typically occurs in the context of complex social interactions, often in a family setting.
rather than between strangers. It is also an issue in health care settings and the wider community.

**In animals**

Psychopathology in non-human primates has been studied since the mid-20th century. Over 20 behavioral patterns in captive chimpanzees have been documented as (statistically) abnormal for their frequency, severity or oddness—some of which have also been observed in the wild. Captive great apes show gross behavioral abnormalities such as stereotypy of movements, self-mutilation, disturbed emotional reactions (mainly fear or aggression) towards companions, lack of species-typical communications, and generalized learned helplessness. In some cases such behaviors are hypothesized to be equivalent to symptoms associated with psychiatric disorders in humans such as depression, anxiety disorders, eating disorders and post-traumatic stress disorder. Concepts of antisocial, borderline and schizoid personality disorders have also been applied to non-human great apes.

The risk of anthropomorphism is often raised with regard to such comparisons, and assessment of non-human animals cannot incorporate evidence from linguistic communication. However, available evidence may range from nonverbal behaviors—including physiological responses and homologous facial displays and acoustic utterances—to neurochemical studies. It is pointed out that human psychiatric classification is often based on statistical description and judgement of behaviors (especially when speech or language is impaired) and that the use of verbal self-report is itself problematic and unreliable.

Psychopathology has generally been traced, at least in captivity, to adverse rearing conditions such as early separation of infants from mothers; early sensory deprivation; and extended periods of social isolation. Studies have also indicated individual variation in temperament, such as sociability or impulsiveness. Particular causes of problems in captivity have included integration of strangers into existing groups and a lack of individual space, in which context some pathological behaviors have also been seen as coping mechanisms. Remedial interventions have included careful individually tailored re-socialization programs, behavior therapy, environment enrichment, and on rare occasions psychiatric drugs. Socialization has been found to work 90% of the time in disturbed chimpanzees, although restoration of functional sexuality and care-giving is often not achieved.

Laboratory researchers sometimes try to develop animal models of human mental disorders, including by inducing or treating symptoms in animals through genetic, neurological, chemical or behavioral manipulation, but this has been criticized on empirical grounds and opposed on animal rights grounds.
Classification of mental disorders

The classification of mental disorders, also known as psychiatric nosology or taxonomy, is a key aspect of psychiatry and other mental health professions and an important issue for consumers and providers of mental health services. There are currently two widely established systems for classifying mental disorders—Chapter V of the International Classification of Diseases (ICD-10) produced by the World Health Organization (WHO) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) produced by the American Psychiatric Association (APA). Both list categories of disorders thought to be distinct types, and have deliberately converged their codes in recent revisions so that the manuals are often broadly comparable, although significant differences remain. Other classification schemes may be in use more locally, for example the Chinese Classification of Mental Disorders. Other manuals have some limited use by those of alternative theoretical persuasions, such as the Psychodynamic Diagnostic Manual.

The widely used DSM and ICD classifications employ operational definitions. There is a significant scientific debate about the relative validity of a "categorical" versus a "dimensional" system of classification, as well as significant controversy about the role of science and values in classification schemes and the professional, legal and social uses to which they are put.

Definitions

In the scientific and academic literature on the definition or categorization of mental disorders, one extreme argues that it is entirely a matter of value judgements (including of what is normal) while another proposes that it is or could be entirely objective and scientific (including by reference to statistical norms); other views argue that the concept refers to a "fuzzy prototype" that can never be precisely defined, or that the definition will always involve a mixture of scientific facts (e.g. that a natural or evolved function isn't working properly) and value judgements (e.g. that it is harmful or undesired). Lay concepts of mental disorder vary considerably across different cultures and countries, and may refer to different sorts of individual and social problems.

The WHO and national surveys report that there is no single consensus on the definition of mental disorder/illness, and that the phrasing used depends on the social, cultural, economic and legal context in different contexts and in different societies. The WHO reports that there is intense debate about which conditions should be included under the concept of mental disorder; a broad definition can cover mental illness, mental retardation, personality disorder and substance dependence, but inclusion varies by country and is reported to be a complex and debated issue. There may be a criterion that a condition should not be expected to occur as part of a person's usual culture or religion. However, despite the term "mental", there is not necessarily a clear distinction drawn between mental (dys)functioning and brain (dys)functioning, or indeed between the brain and the rest of the body.
Most international clinical documents avoid the term "mental illness", preferring the term "mental disorder". However, some use "mental illness" as the main over-arching term to encompass mental disorders. Some consumer/survivor movement organizations oppose use of the term "mental illness" on the grounds that it supports the dominance of a medical model. The term "serious mental illness" (SMI) is sometimes used to refer to more severe and long-lasting disorders while "mental health problems" may be used as a broader term, or to refer only to milder or more transient issues. Confusion often surrounds the ways and contexts in which these terms are used.

Mental disorders are generally classified separately to neurological disorders, learning disabilities or mental retardation.

**ICD-10**

The International Classification of Diseases (ICD) is an international standard diagnostic classification for a wide variety of health conditions. Chapter V focuses on "mental and behavioural disorders" and consists of 10 main groups:

- F0: Organic, including symptomatic, mental disorders
- F1: Mental and behavioural disorders due to use of psychoactive substances
- F2: Schizophrenia, schizotypal and delusional disorders
- F3: Mood [affective] disorders
- F4: Neurotic, stress-related and somatoform disorders
- F5: Behavioural syndromes associated with physiological disturbances and physical factors
- F6: Disorders of personality and behaviour in adult persons
- F7: Mental retardation
- F8: Disorders of psychological development
- F9: Behavioural and emotional disorders with onset usually occurring in childhood and adolescence

In addition, a group of "unspecified mental disorders".

Within each group there are more specific subcategories. The ICD includes personality disorders on the same domain as other mental disorders, unlike the DSM. The ICD-10 states that mental disorder is "not an exact term", although is generally used "...to imply the existence of a clinically recognisable set of symptoms or behaviours associated in most cases with distress and with interference with personal functions." (WHO, 1992).

The WHO is revising their classifications in this section as part of the development of the ICD-11 (scheduled for 2014) and an "International Advisory Group" has been established to guide this.

**DSM-IV**
The DSM-IV, produced by the American Psychiatric Association, characterizes mental disorder as "a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual,...is associated with present distress...or disability...or with a significant increased risk of suffering" but that "...no definition adequately specifies precise boundaries for the concept of 'mental disorder'...different situations call for different definitions" (APA, 1994 and 2000). The DSM also states that "there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder."

The DSM-IV-TR (Text Revision, 2000) consists of five axes (domains) on which disorder can be assessed. The five axes are:

- **Axis I: Clinical Disorders (all mental disorders except Personality Disorders and Mental Retardation)**
- **Axis II: Personality Disorders and Mental Retardation**
- **Axis III: General Medical Conditions (must be connected to a Mental Disorder)**
- **Axis IV: Psychosocial and Environmental Problems (for example limited social support network)**
- **Axis V: Global Assessment of Functioning (Psychological, social and job-related functions are evaluated on a continuum between mental health and extreme mental disorder)**

**The main categories of disorder in the DSM are:**

<table>
<thead>
<tr>
<th>DSM Group</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Disorders usually first diagnosed in infancy, childhood or adolescence. <em>&quot;Disorders such as ADHD and epilepsy have also been referred to as developmental disorders and developmentally disabilities.</em></td>
<td>Mental retardation, ADHD</td>
</tr>
<tr>
<td>Delirium, dementia, and amnesia and other cognitive disorders</td>
<td>Alzheimer's disease</td>
</tr>
<tr>
<td>Mental disorders due to a general medical condition</td>
<td>AIDS-related psychosis</td>
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<tr>
<td>Substance-related disorders</td>
<td>Alcohol abuse</td>
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<tr>
<td>Schizophrenia and other psychotic disorders</td>
<td>Delusional disorder</td>
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<tr>
<td>Mood disorders</td>
<td>Major depressive disorder, Bipolar disorder</td>
</tr>
<tr>
<td>Anxiety disorders</td>
<td>General anxiety disorder</td>
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<tr>
<td>Somatoform disorders</td>
<td>Somatization disorder</td>
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<td>Factitious disorders</td>
<td>Münchausen syndrome</td>
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<tr>
<td>Dissociative disorders</td>
<td>Dissociative identity disorder</td>
</tr>
<tr>
<td>Sexual and gender identity disorders</td>
<td>Dyspareunia, Gender identity disorder</td>
</tr>
<tr>
<td>Eating disorders</td>
<td>Anorexia nervosa,</td>
</tr>
</tbody>
</table>
Other schemes

- The Chinese Society of Psychiatry's Chinese Classification of Mental Disorders (currently CCMD-3)
- The Latin American Guide for Psychiatric Diagnosis (GLDP).

Childhood diagnosis

Child and adolescent psychiatry sometimes uses specific manuals in addition to the DSM and ICD. The Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood (DC:0-3) was first published in 1994 by Zero to Three to classify mental health and developmental disorders in the first four years of life. It has been published in 9 languages. The Research Diagnostic criteria-Preschool Age (RDC-PA) was developed between 2000 and 2002 by a task force of independent investigators with the goal of developing clearly specified diagnostic criteria to facilitate research on psychopathology in this age group. The French Classification of Child and Adolescent Mental Disorders (CFTMEA), operational since 1983, is the classification of reference for French child psychiatrists.

Usage

The ICD and DSM classification schemes have achieved widespread acceptance in psychiatry. A survey of 205 psychiatrists, from 66 different countries across all continents, found that ICD-10 was more frequently used and more valued in clinical practice and training, while the DSM-IV was more valued for research, with accessibility to either being limited, and usage by other mental health professionals, policy makers, patients and families less clear. A primary care (e.g. general or family physician) version of the mental disorder section of ICD-10 has been developed (ICD-10-PHC) which has also been used quite extensively internationally. A survey of journal articles indexed in various biomedical databases between 1980 and 2005 indicated that 15,743 referred to the DSM and 3,106 to the ICD.

In Japan, most university hospitals use either the ICD or DSM. ICD appears to be the somewhat more used for research or academic purposes, while both were used equally for clinical purposes. Other traditional psychiatric schemes may also be used.
Types of classification schemes

Categorical schemes

The classification schemes in common usage are based on separate (but may be overlapping) categories of disorder schemes sometimes termed "neo-Kraepelinian" (after the psychiatrist Kraepelin) which is intended to be atheoretical with regard to etiology (causation). These classification schemes have achieved some widespread acceptance in psychiatry and other fields, and have generally been found to have improved inter-rater reliability, although routine clinical usage is less clear. Questions of validity and utility have been raised, both scientifically and in terms of social, economic and political factors—notably over the inclusion of certain controversial categories, the influence of the pharmaceutical industry, or the stigmatizing effect of being categorized or labelled.

Non-categorical schemes

Some approaches to classification do not use categories with single cut-offs separating the ill from the healthy or the abnormal from the normal (a practice sometimes termed "threshold psychiatry" or "dichotomous classification").

Classification may instead be based on broader underlying "spectra", where each spectrum links together a range of related categorical diagnoses and nonthreshold symptom patterns.

Some approaches go further and propose continuously-varying dimensions that are not grouped into spectra or categories; each individual simply has a profile of scores across different dimensions. DSM-5 planning committees are currently seeking to establish a research basis for a hybrid dimensional classification of personality disorders. However, the problem with entirely dimensional classifications is they are said to be of limited practical value in clinical practice where yes/no decisions often need to be made, for example whether a person requires treatment, and moreover the rest of medicine is firmly committed to categories, which are assumed to reflect discrete disease entities. While the Psychodynamic Diagnostic Manual has an emphasis on dimensionality and the context of mental problems, it has been structured largely as an adjunct to the categories of the DSM.

Nevertheless, non-categorical clinical formulation approaches are commonly employed in clinical psychology and some areas of psychiatry, where there may be limited or no reference to diagnostic categories. One such approach advocates taking each specific complaint reported by an individual on its own merits, treated as a phenomenon with its own causes.

Descriptive vs Somatic

Descriptive classifications are based almost exclusively on either descriptions of behavior as reported by various observers, such as parents, teachers, and medical personnel; or
symptoms as reported by individuals themselves. As such, they are quite subjective, not amenable to verification by third parties, and not readily transferable across chronologic and/or cultural barriers.

Somatic nosology, on the other hand, is based almost exclusively on the objective histologic and chemical abnormalities which are characteristic of various diseases and can be identified by appropriately trained pathologists. While not all pathologists will agree in all cases, the degree of uniformity allowed is orders of magnitude greater than that enabled by the constantly changing classification embraced by the DSM system.

**Cultural differences**

Classification schemes may not apply to all cultures. The DSM is based on predominantly American research studies and has been said to have a decidedly American outlook, meaning that differing disorders or concepts of illness from other cultures (including personalistic rather than naturalistic explanations) may be neglected or misrepresented, while Western cultural phenomena may be taken as universal. Culture-bound syndromes are those hypothesized to be specific to certain cultures (typically taken to mean non-Western or non-mainstream cultures); while some are listed in an appendix of the DSM-IV they are not detailed and there remain open questions about the relationship between Western and non-Western diagnostic categories and sociocultural factors, which are addressed from different directions by, for example, cross-cultural psychiatry or anthropology.

**Historical development**

**Antiquity**

In Ancient Greece, Hippocrates and his followers are generally credited with the first classification system for mental illnesses, including mania, melancholia, paranoia, phobias and Scythian disease (transvestism). They held that they were due to different kinds of imbalance in four humors.

**Middle ages to Renaissance**

An elaborate classification of mental disorders was developed in the 10th century by Arabian psychologist Najab ud-din Unhammad. His nosology included nine major categories of mental disorders, with 30 different mental illnesses in total. Some of the categories he described resembled obsessive-compulsive disorders, delusional disorders, degenerative diseases, involutional melancholia, and states of abnormal excitement. Avicenna (980–1037 CE) in the Canon of Medicine listed a number of mental disorders, including "passive male homosexuality".

Laws generally distinguished between "idiots" and "lunatics".
Thomas Sydenham (1624–1689), the "English Hippocrates", emphasized careful clinical observation and diagnosis and developed the concept of a syndrome, a group of associated symptoms having a common course, which would later influence psychiatric classification.

18th century

Evolution in the scientific concepts of psychopathology (literally referring to diseases of the mind) took hold in the late 18th and 19th centuries following the Renaissance and Enlightenment. Individual behaviors that had long been recognized came to be grouped into syndromes.

Boissier de Sauvages developed an extremely extensive psychiatric classification in the mid-18th century, influenced by the medical nosology of Thomas Sydenham and the biological taxonomy of Carl Linnaeus. It was only part of his classification of 2400 medical diseases. These were divided into 10 "classes", one of which comprised the bulk of the mental diseases, divided into four "orders" and 23 "genera". One genus, melancholia, was subdivided into 14 "species".

William Cullen advanced an influential medical nosology which included four classes of neuroses: coma, adynamias, spasms, and vesanias. The vesanias included amentia, melancholia, mania, and oneirodynia.

Towards the end of the 18th century Pinel, influenced by Cullen's scheme, developed his own, again employing the terminology of genera and species. His simplified revision of this reduced all mental illnesses to four basic types. He argued that mental disorders are not separate entities but stem from a single disease that he called "mental alienation".

Attempts were made to merge the ancient concept of delirium with that of insanity, the latter sometimes described as delirium without fever.

The concept of partial insanity developed, and attempts were made to distinguish it from total insanity by criteria such as intensity, content or generalization of delusions.

His successor, Esquirol, extended Pinel's categories to five. Both made a clear distinction between insanity (including mania and dementia) as opposed to mental retardation (including idiocy and imbecility). Esquirol developed a concept of monomania—a periodic delusional fixation or undesirable disposition on one theme—that became a broad and common diagnosis and a part of popular culture for much of the 19th century.

19th century

The botanical taxonomic approach was abandoned in the 19th century, in favor of an anatomical-clinical approach that became increasingly descriptive. There was a focus on identifying the particular psychological faculty involved in particular forms of insanity, although some argued for a more central "unitary" cause. French and German psychiatric nosology was in the ascendancy. The term "psychiatry" ("Psychiatrie") was coined by
German physician Johann Christian Reil in 1808, from the Greek "ψυχή" (psychē: "soul or mind") and "ἰατρός" (iatros: "healer or doctor"). The term "alienation" took on a psychiatric meaning in France, later adopted into medical English. The terms psychosis and neurosis came into use, the former viewed psychologically and the latter neurologically.

In the second half of the century, Karl Kahlbaum and Ewald Hecker developed a descriptive categorization of syndromes, employing terms such as dysthymia, cyclothymia, catatonia, paranoia and hebephrenia. Wilhelm Griesinger (1817–1869) advanced a unitary scheme based on a concept of brain pathology. French psychiatrists Jules Baillarger described "folie à double forme" and Jean-Pierre Falret described "la folie circulaire"—alternating mania and depression.

The concept of adolescent insanity or developmental insanity was advanced by Scottish psychiatrist Thomas Coulston in 1873, describing a psychotic condition which generally afflicted those aged 18–24 years, particularly males, and in 30% of cases proceeded to "a secondary dementia".

The concept of hysteria (wandering womb) had long been used, perhaps since ancient Egyptian times, and was later adopted by Freud. Descriptions of a specific syndrome now known as somatization disorder were first developed by the French physician, Briquet in 1859.

Early 19th century psychiatrists also began to categorize personality disorders. The diagnosis of "moral insanity" became popular, those with the condition did not seem psychotic but seemed to have no ability to comprehend moral principles. In the late 19th century, Koch referred to "psychopathic inferiority", and in the 20th century the disorder became known as "psychopathy" or "sociopathy". Related studies led to the DSM-III category of antisocial personality disorder.

An American physician, Beard, described "neurasthenia" in 1869. German neurologist Westphal, coined the term "obsessional neurosis" now termed obsessive-compulsive disorder, and agoraphobia. Alienists created a whole new series of diagnoses that highlighted single, impulsive behavior, such as kleptomania, dipsomania, pyromania, and nymphomania. The diagnosis of drapetomania was also developed in the Southern United States to explain the perceived irrationality of black slaves trying to escape what was thought to be a suitable role.

The scientific study of homosexuality began in the 19th century, informally viewed either as natural or as a disorder. Kraepelin included it as a disorder in his Compendium der Psychiatrie that he published in successive editions from 1883.

20th century

Influenced by the approach of Kahlbaum and others, and developing his concepts in publications spanning the turn of the century, German psychiatrist Emil Kraepelin
advanced a new system. He grouped together a number of existing diagnoses that appeared to all have a deteriorating course over time—such as catatonia, hebephrenia and dementia paranoides—under another existing term "dementia praecox" (meaning "early senility", later renamed schizophrenia). Another set of diagnoses that appeared to have a periodic course and better outcome were grouped together under the category of manic-depressive insanity (mood disorder). He also proposed a third category of psychosis, called paranoia, involving delusions but not the more general deficits and poor course attributed to dementia praecox. In all he proposed 15 categories, also including psychogenic neurosis, psychopathic personality, and syndromes of defective mental development (mental retardation). He eventually included homosexuality in the category of "mental conditions of constitutional origin".

The neuroses were later split into anxiety disorders and other disorders.

Freud wrote extensively on hysteria and also coined the term, "anxiety neurosis", which appeared in DSM-I and DSM-II. Checklist criteria for this led to studies that were to define panic disorder for DSM-III.

Early 20th century schemes in Europe and the US reflected a brain disease model that had emerged during the 19th century, as well as some ideas from Darwin's theory of evolution and/or Freud's psychoanalytic theories.

Psychoanalytic theory did not rest on classification of distinct disorders, but pursued analyses of unconscious conflicts and their manifestations within an individual's life. The concept of borderline personality disorder developed from psychoanalytic theories.

The philosopher and psychiatrist Karl Jaspers made influential use of a "biographical method" and suggested ways to diagnose based on the form rather than content of beliefs or perceptions. In regard to classification in general he prophetically remarked that: "When we design a diagnostic schema, we can only do so if we forego something at the outset ... and in the face of facts we have to draw the line where none exists... A classification therefore has only provisional value. It is a fiction which will discharge its function if it proves to be the most apt for the time".

Adolph Meyer advanced a mixed biosocial scheme that emphasized the reactions and adaptations of the whole organism to life experiences.

In 1945, William C. Menninger advanced a classification scheme for the US army, called Medical 203, synthesizing ideas of the time into five major groups. This system was adopted by the Veterans Administration in the US and strongly influenced the DSM.

The term stress, having emerged out of endocrinology work in the 1930s, was popularized with an increasingly broad biopsychosocial meaning, and was increasingly linked to mental disorders. The diagnosis of post-traumatic stress disorder was later created.
The Feighner Criteria group described fourteen major psychiatric disorders for which careful research studies were available, including homosexuality. These developed as the Research Diagnostic Criteria, adopted and further developed by the DSM-III.

The DSM and ICD developed, partly in sync, in the context of mainstream psychiatric research and theory. Debates continued and developed about the definition of mental illness, the medical model, categorical vs dimensional approaches, and whether and how to include suffering and impairment criteria. There is some attempt to construct novel schemes, for example from an attachment perspective where patterns of symptoms are construed as evidence of specific patterns of disrupted attachment, coupled with specific types of subsequent trauma.

21st century

The ICD-11 and DSM-5 are being developed at the start of the 21st century. Any radical new developments in classification are said to be more likely to be introduced by the APA than by the WHO, mainly because the former only has to persuade its own board of trustees whereas the latter has to persuade the representatives of over 200 different countries at a formal revision conference. In addition, while the DSM is a bestselling publication that makes huge profits for APA, the WHO incurs major expense in determining international consensus for revisions to the ICD. Although there is an ongoing attempt to reduce trivial or accidental differences between the DSM and ICD, it is thought that the APA and the WHO are likely to continue to produce new versions of their manuals and, in some respects, to compete with one another.

Criticism

There is some ongoing scientific doubt concerning the construct validity and reliability of psychiatric diagnostic categories and criteria even though they have been increasingly standardized to improve inter-rater agreement in controlled research. In the United States, there have been calls and endorsements for a congressional hearing to explore the nature and extent of harm potentially caused by this "minimally investigated enterprise".

Other specific criticisms of the current schemes include: attempts to demonstrate natural boundaries between related syndromes, or between a common syndrome and normality, have failed; the disorders of current classification are probably surface phenomena that can have many different interacting causes, yet "the mere fact that a diagnostic concept is listed in an official nomenclature and provided with a precise operational definition tends to encourage us to assume that it is a "quasi-disease entity" that can be invoked to explain the patient's symptoms"; and that the diagnostic manuals have led to an unintended decline in careful evaluation of each individual person's experiences and social context. Psychodynamic schemes give this latter phenomenological aspect more consideration, but in psychoanalytic terms that have been long criticized on numerous grounds.

Reliance on operational definition demands that intuitive concepts, such as depression need to be operationally defined before they become amenable to scientific investigation.
However, John Stuart Mill pointed out the dangers of believing that anything that could be given a name must refer to a thing and Stephen Jay Gould and others have criticized psychologists for doing just that. One critic states that "Instead of replacing 'metaphysical' terms such as 'desire' and 'purpose', they used it to legitimize them by giving them operational definitions. Thus in psychology, as in economics, the initial, quite radical operationalist ideas eventually came to serve as little more than a 'reassurance fetish' (Koch 1992, 275) for mainstream methodological practice."

Psychiatrist Joel Paris argues that psychiatry is sometimes susceptible to diagnostic fads. Some have been based on theory (overdiagnosis of schizophrenia), some based on etiological (causation) concepts (overdiagnosis of post-traumatic stress disorder), and some based on the development of treatments. Paris points out that psychiatrists like to diagnose conditions they can treat, and gives examples of what he sees as prescribing patterns paralleling diagnostic trends, for example an increase in bipolar diagnosis once lithium came into use, and similar scenarios with the use of electroconvulsive therapy, neuroleptics, tricyclic antidepressants, and SSRIs. He notes that there was a time when every patient seemed to have "latent schizophrenia" and another time when everything in psychiatry seemed to be "masked depression", and he fears that the boundaries of the bipolar spectrum concept, including in application to children, are similarly expanding.

**DSM-IV Codes**

Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision, also known as DSM-IV-TR, is a manual published by the American Psychiatric Association (APA) that includes all currently recognized mental health disorders. The coding system utilized by the DSM-IV is designed to correspond with codes from the International Classification of Diseases, commonly referred to as the ICD. Since early versions of the DSM did not correlate with ICD codes and updates of the publications for the ICD and the DSM are not simultaneous, some distinctions in the coding systems may still be present. For this reason, it is recommended that users of these manuals consult the appropriate reference when accessing diagnostic codes.

To see these codes listed alphabetically, rather than by category, click here.

**NOS = Not Otherwise Specified.**

**Pervasive developmental disorders**

- 299.00 Autistic Disorder
- 299.80 Rett's Disorder
- 299.10 Childhood Disintegrative Disorder
- 299.80 Asperger's Disorder
- 299.80 Pervasive Developmental Disorder NOS
Attention-deficit and disruptive behavior disorders

Attention-Deficit Hyperactivity Disorder

- 314.01 Combined subtype
- 314.01 Predominantly hyperactive-impulsive subtype
- 314.00 Predominantly inattentive subtype
- 314.9 Attention-Deficit Hyperactivity Disorder NOS

Conduct disorder

- 312.81 Childhood onset
- 312.82 Adolescent onset
- 312.89 Unspecified onset
- 313.81 Oppositional Defiant Disorder
- 312.9 Disruptive Behavior Disorder NOS

Feeding and eating disorders of infancy or early childhood

- 307.52 Pica
- 307.59 Feeding disorder of infancy or early childhood

Tic disorders

- 307.23 Tourette’s Disorder
- 307.22 Chronic motor or vocal tic disorder
- 307.21 Transient tic disorder
- 307.20 Tic disorder NOS

Elimination disorders

Encopresis

- 787.6 Encopresis, with constipation and overflow incontinence
- 307.7 Encopresis, without constipation and overflow incontinence
- 307.6 Enuresis (not due to a general medical condition)

Other disorders of infancy, childhood, or adolescence

- 309.21 Separation anxiety disorder
- 313.23 Selective mutism
- 313.89 Reactive attachment disorder of infancy or early childhood
- 307.3 Stereotypic movement disorder
- 313.9 Disorder of infancy, childhood, or adolescence NOS
Delirium, dementia, and amnestic and other cognitive disorders

Delirium

- 293.0 Delirium due to...
- 780.09 Delirium NOS

Dementia

- 290.10 Dementia due to Creutzfeldt-Jakob Disease
- 294.1 Dementia due to head trauma
- 294.9 Dementia due to HIV disease
- 294.1 Dementia due to Huntington’s disease
- 294.1 Dementia due to Parkinson’s disease
- 290.10 Dementia due to Pick’s disease
- 294.1 Dementia due to... [indicate other general medical condition]
- 294.8 Dementia NOS

Dementia of the Alzheimer’s Type, with early onset

- 290.10 Uncomplicated
- 290.11 With delirium
- 290.12 With delusions
- 290.13 With depressed mood

Dementia of the Alzheimer’s Type, with late onset

- 290.0 Uncomplicated
- 290.3 With delirium
- 290.20 With delusions
- 290.21 With depressed mood

Vascular dementia

- 290.40 Uncomplicated
- 290.41 With delirium
- 290.42 With delusions
- 290.43 With depressed mood

Amnestic disorders

- 294.0 Amnestic disorder due to...
- 294.8 Amnestic disorder NOS

Other cognitive disorders
- 294.9 Cognitive disorder NOS

**Mental disorders due to a general medical condition not elsewhere classified**

- 293.89 Catatonic disorder due to...
- 310.1 Personality change due to... (Subtypes: Labile, Disinhibited, Aggressive, Apathetic, Paranoid, Other, Combined, Unspecified)
- 293.9 Mental disorder NOS due to...

**Substance-related disorders**

**Alcohol-related disorders**

**Alcohol**

- 305.00 Abuse
- 303.90 Dependence
- 291.8 -Induced anxiety disorder
- 291.8 -Induced mood disorder
- 291.1 -Induced persisting amnestic disorder
- 291.2 -Induced persisting dementia
- 291.5 -Induced psychotic disorder, with delusions
- 291.3 -Induced psychotic disorder, with hallucinations
- 291.8 -Induced sexual dysfunction
- 291.8 -Induced sleep disorder
- 303.00 Intoxication
- 291.0 Intoxication delirium
- 291.9 -Related disorder NOS
- 291.8 Withdrawal
- 291.0 Withdrawal delirium

**Amphetamine (or amphetamine-like) related disorders**

**Amphetamine (or amphetamine-like)**

- 305.70 Abuse
- 304.40 Dependence
- 292.89 -Induced anxiety disorder
- 292.84 -Induced mood disorder
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 -Induced sexual dysfunction
- 292.89 -Induced sleep disorder
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS
- 292.0 Withdrawal

**Caffeine-related disorders**

**Caffeine**

- 292.89 -Induced anxiety disorder
- 292.89 -Induced sleep disorder
- 305.90 Intoxication
- 292.9 -Related disorder NOS

**Cannabis-related disorders**

**Cannabis**

- 305.20 Abuse
- 304.30 Dependence
- 292.89 -Induced anxiety disorder
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS

**Cocaine-related disorders**

**Cocaine**

- 305.60 Abuse
- 304.20 Dependence
- 292.89 -Induced anxiety disorder
- 292.84 -Induced mood disorder
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 -Induced sexual dysfunction
- 292.89 -Induced sleep disorder
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS
- 292.0 Withdrawal

**Hallucinogen-related disorders**

**Hallucinogen**

- 305.30 Abuse
- 304.50 Dependence
- 292.89 -Induced anxiety disorder
- 292.84 -Induced mood disorder
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.89 -Persisting perception disorder
- 292.9 -Related disorder NOS

Inhalant-related disorders

Inhalant

- 305.90 Abuse
- 304.60 Dependence
- 292.89 -Induced anxiety disorder
- 292.84 -Induced mood disorder
- 292.82 -Induced persisting dementia
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS

Nicotine-related disorders

Nicotine

- 305.1 Dependence
- 292.9 -Related disorder NOS
- 292.0 Withdrawal

Opioid-related disorders

Opioid

- 305.50 Abuse
- 304.00 Dependence
- 292.84 -Induced mood disorder
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 -Induced sexual dysfunction
- 292.89 -Induced sleep disorder
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS
- 292.0 Withdrawal

Phencyclidine (or phencyclidine-like) related disorders

Phencyclidine (or phencyclidine-like)

- 305.90 Abuse
- 304.90 Dependence
- 292.89 -Induced anxiety disorder
- 292.84 -Induced mood disorder
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS

Sedative-, hypnotic-, or anxiolytic-related disorders

Sedative, hypnotic, or anxiolytic

- 305.40 Abuse
- 304.10 Dependence
- 292.89 -Induced anxiety disorder
- 292.84 -Induced mood disorder
- 292.83 -Induced persisting amnestic disorder
- 292.82 -Induced persisting dementia
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 -Induced sexual dysfunction
- 292.89 -Induced sleep disorder
- 292.89 Intoxication
- 292.81 Intoxication delirium
- 292.9 -Related disorder NOS
- 292.0 Withdrawal
- 292.81 Withdrawal delirium

Polysubstance-related disorder

- 304.80 Polysubstance dependence

Other (or unknown) substance-related disorder

Other (or unknown) substance

- 305.90 Abuse
- 304.90 Dependence
- 292.89 -Induced anxiety disorder
- 292.81 -Induced delirium
- 292.84 -Induced mood disorder
- 292.83 -Induced persisting amnestic disorder
- 292.82 -Induced persisting dementia
- 292.11 -Induced psychotic disorder, with delusions
- 292.12 -Induced psychotic disorder, with hallucinations
- 292.89 -Induced sexual dysfunction
- 292.89 -Induced sleep disorder
- 292.89 Intoxication
- 292.9 -Related disorder NOS
- 292.0 Withdrawal

**Schizophrenia and other psychotic disorders**

**Schizophrenia**

- 295.2 Catatonic type
- 295.1 Disorganized type
- 295.3 Paranoid type
- 295.6 Residual type
- 295.9 Undifferentiated type
- 295.4 Schizophreniform disorder
- 295.7 Schizoaffective disorder
- 297.1 Delusional disorder
  - Erotomanic subtype
  - Grandiose subtype
  - Jealous subtype
  - Persecutory subtype
  - Somatic subtype
  - Mixed type
- 298.8 Brief psychotic disorder
- 297.3 Shared psychotic disorder

**Psychotic disorder due to...**

- 293.81 With delusions
- 293.82 With hallucinations
- 298.9 Psychotic disorder NOS

**Mood disorders**

**Depressive disorders**

- 300.4 Dysthymic disorder
Major depressive disorder

Major depressive disorder, recurrent

- 296.36 In full remission
- 296.35 In partial remission
- 296.31 Mild
- 296.32 Moderate
- 296.33 Severe without psychotic features
- 296.34 Severe with psychotic features
- 296.30 Unspecified

Major depressive disorder, single episode

- 296.26 In full remission
- 296.25 In partial remission
- 296.21 Mild
- 296.22 Moderate
- 296.23 Severe without psychotic features
- 296.24 Severe with psychotic features
- 296.20 Unspecified
- 311 Depressive disorder NOS

Bipolar disorders

Bipolar disorders

- 296.80 Bipolar disorder NOS

Bipolar I disorder, most recent episode depressed

- 296.56 In full remission
- 296.55 In partial remission
- 296.51 Mild
- 296.52 Moderate
- 296.53 Severe without psychotic features
- 296.54 Severe with psychotic features
- 296.50 Unspecified
- 296.40 Bipolar I disorder, most recent episode hypomanic

Bipolar I disorder, most recent episode manic

- 296.46 In full remission
- 296.45 In partial remission
- 296.41 Mild
• 296.42 Moderate
• 296.43 Severe without psychotic features
• 296.44 Severe with psychotic features
• 296.40 Unspecified

**Bipolar I disorder, most recent episode mixed**

• 296.66 In full remission
• 296.65 In partial remission
• 296.61 Mild
• 296.62 Moderate
• 296.63 Severe without psychotic features
• 296.64 Severe with psychotic features
• 296.60 Unspecified
• 296.7 Bipolar I disorder, most recent episode unspecified

**Bipolar I disorder, single manic episode**

• 296.06 In full remission
• 296.05 In partial remission
• 296.01 Mild
• 296.02 Moderate
• 296.03 Severe without psychotic features
• 296.04 Severe with psychotic features
• 296.00 Unspecified
• 296.89 Bipolar II disorder
• 301.13 Cyclothymic disorder

**Mood disorder**

• 293.83 Mood disorder due to...
• 296.90 Mood disorder NOS

**Anxiety disorders**

• 300.02 Generalized anxiety disorder

**Panic disorder**

• 300.21 With agoraphobia
• 300.01 Without agoraphobia
• 300.22 Agoraphobia without history of panic disorder
• 300.29 Specific phobia
• 300.23 Social phobia
• 300.3 Obsessive-compulsive disorder
• 309.81 Posttraumatic stress disorder
308.3 Acute stress disorder

**Anxiety disorder**

- 293.84 Anxiety disorder due to a general medical condition
- 293.89 Anxiety disorder due to...
- 300.00 Anxiety disorder NOS

**Somatoform disorders**

- 300.81 Somatization disorder
- 300.81 Undifferentiated somatoform disorder
- 300.11 Conversion disorder

**Pain disorder**

- 307.89 Associated with both psychological factors and a general medical condition
- 307.80 Associated with psychological factors
- 300.7 Hypochondriasis
- 300.7 Body dysmorphic disorder
- 300.81 Somatoform disorder NOS

**Factitious disorders**

**Factitious disorder**

- 300.19 With combined psychological and physical signs and symptoms
- 300.19 With predominantly physical signs and symptoms
- 300.16 With predominantly psychological signs and symptoms
- 300.19 Factitious disorder NOS

**Dissociative disorders**

- 300.6 Depersonalization disorder
- 300.12 Dissociative amnesia
- 300.13 Dissociative fugue
- 300.14 Dissociative identity disorder
- 300.15 Dissociative disorder NOS

**Sexual and gender identity disorders**

**Sexual dysfunctions**

- 625.8 Female hypoactive sexual desire disorder due to...
- 608.89 Male hypoactive sexual desire disorder due to...
- 302.71 Hypoactive sexual desire disorder
- 302.79 Sexual aversion disorder
- 302.72 Female sexual arousal disorder
- 302.72 Male erectile disorder
- 607.84 Male erectile disorder due to...
- 302.73 Female orgasmic disorder
- 302.74 Male orgasmic disorder
- 302.75 Premature ejaculation
- 302.76 Dyspareunia (not due to a general medical condition)
- 625.0 Female dyspareunia due to...
- 608.89 Male dyspareunia due to...
- 306.51 Vaginismus (not due to a general medical condition)
- 625.8 Other female sexual dysfunction due to...
- 608.89 Other male sexual dysfunction due to...

### Sexual Abuse

- V61.1 Sexual abuse of adult
- 995.81 Sexual abuse of adult (if focus of attention is on victim)
- V61.21 Sexual abuse of child
- 995.5 Sexual abuse of child (if focus of attention is on victim)
- 302.9 Sexual disorder NOS
- 302.70 Sexual dysfunction NOS

### Paraphilias

- 302.4 Exhibitionism
- 302.81 Fetishism
- 302.89 Frotteurism
- 302.2 Pedophilia
- 302.83 Sexual masochism
- 302.84 Sexual sadism
- 302.3 Transvestic fetishism
- 302.82 Voyeurism
- 302.9 Paraphilia NOS (not otherwise specified)

### Gender identity disorders

#### Gender identity disorder

- 302.85 In adolescents or adults
- 302.6 In children
- 302.6 Gender identity disorder NOS

### Eating disorders

- 307.1 Anorexia nervosa


- 307.51 Bulimia nervosa
- 307.53 Rumination syndrome
- 307.50 Eating disorder not otherwise specified (EDNOS)

Sleep disorders

Primary sleep disorders

- 307.44 Primary hypersomnia
- 307.42 Primary insomnia
- 347 Narcolepsy
- 780.59 Breathing-related sleep disorder
- 307.45 Circadian rhythm sleep disorder
- 307.47 Dyssomnia NOS

Parasomnias

- 307.47 Nightmare disorder
- 307.46 Sleep terror disorder
- 307.46 Sleepwalking disorder
- 307.47 Parasomnia NOS

Other sleep disorders

Sleep disorder

Sleep disorder due to...

- 780.54 Hypersomnia type
- 780.52 Insomnia type
- 780.59 Mixed type
- 780.59 Parasomnia type
- 307.42 Insomnia related to...
- 307.44 Hypersomnia related to...

Impulse-control disorders not elsewhere classified

- 312.34 Intermittent explosive disorder
- 312.32 Kleptomania
- 312.33 Pyromania
- 312.31 Pathological gambling
- 312.39 Trichotillomania
- 312.30 Impulse-control disorder NOS
- 312.30 Mavvers The Spazzer Knee and Brain Impairment (MTSKABI)
Adjustment disorders

309.9 Unspecified
309.24 With anxiety
309.0 With depressed mood
309.3 With disturbance of conduct
309.28 With mixed anxiety and depressed mood
309.4 With mixed disturbance of emotions and conduct

Personality disorders (Axis II)

Cluster A (odd or eccentric)

301.0 Paranoid personality disorder
301.20 Schizoid personality disorder
301.22 Schizotypal personality disorder

Cluster B (dramatic, emotional, or erratic)

301.7 Antisocial personality disorder
301.83 Borderline personality disorder
301.50 Histrionic personality disorder
301.81 Narcissistic personality disorder

Cluster C (anxious or fearful)

301.82 Avoidant personality disorder
301.6 Dependent personality disorder
301.4 Obsessive-compulsive personality disorder

NOS

301.9 Personality disorder not otherwise specified

Additional codes

V62.3 Academic problem
V62.4 Acculturation problem
995.2 Adverse effects of medication NOS
780.9 Age-related cognitive decline

Antisocial behavior

V71.01 Adult antisocial behavior
- V71.02 Child or adolescent antisocial behavior
- V62.82 Bereavement
- V62.89 Borderline intellectual functioning
- 313.82 Identity problem

**Medication-induced**

**Movement disorder**
- 333.90 Movement disorder NOS
- 333.1 Postural tremor

**Neglect of child**
- V61.21 Neglect of child
- 995.5 Neglect of child (if focus of attention is on victim)

**Neuroleptic-induced**
- 333.99 Acute akathisia
- 333.7 Acute dystonia
- 332.1 Parkinsonism
- 333.82 Tardive dyskinesia
- 333.92 Neuroleptic malignant syndrome
- V71.09 No diagnosis on Axis II
- V71.09 No diagnosis or condition on Axis I
- V15.81 Noncompliance with treatment
- V62.2 Occupational problem
- V61.20 Parent-child relational problem
- V61.1 Partner relational problem
- V62.89 Phase of life problem

**Physical abuse**
- V61.1 Physical abuse of adult
- 995.81 Physical abuse of adult (if focus of attention is on victim)
- V61.21 Physical abuse of child
- 995.5 Physical abuse of child (if focus of attention is on victim)
- 316 Psychological factors affecting medical condition

**Relational problem**
- V62.81 Relational problem NOS
- V61.9 Relational problem related to a mental disorder or general medical condition
- V62.89 Religious or spiritual problem
- V61.8 Sibling relational problem
- 300.9 Unspecified mental disorder (nonpsychotic)
- 799.9 Diagnosis deferred on Axis II
- 799.9 Diagnosis or condition deferred on Axis I
- V65.2 Malingering

Prevalence of mental disorders

The prevalence of mental disorders has been studied around the world, providing estimates on how common mental disorders are. Different criteria or thresholds of severity have sometimes been used. National and international figures are typically estimated by large-scale surveys of self-reported symptoms up to the time of assessment; sometimes a figure is calculated for the occurrence of disorder in the week, month or year prior to assessment—a point or period prevalence; sometimes the figure is for a person’s lifetime prior to assessment—the so-called lifetime prevalence.

Numerous large-scale surveys of the prevalence of mental disorders in adults in the general population have been carried out since the 1980s based on self-reported symptoms assessed by standardized structured interviews, usually carried out over the phone.

Mental disorders have been found to be common, with over a third of people in most countries reporting sufficient criteria to be diagnosed at some point in their life. The World Health Organization (WHO) reported in 2001 that about 450 million people worldwide suffer from some form of mental disorder or brain condition, and that one in four people meet criteria at some point in their life.

The WHO is currently undertaking a global survey of 26 countries in all regions of the world, based on ICD and DSM criteria. The first published figures on the 14 country surveys completed to date, indicate that, of those disorders assessed, anxiety disorders are the most common in all but 1 country (prevalence in the prior 12-month period of 2.4% to 18.2%) and mood disorders next most common in all but 2 countries (12-month prevalence of 0.8% to 9.6%), while substance disorders (0.1%–6.4%) and impulse-control disorders (0.0%–6.8%) were consistently less prevalent. The United States, Colombia, the Netherlands and Ukraine tended to have higher prevalence estimates across most classes of disorder, while Nigeria, Shanghai and Italy were consistently low, and prevalence was lower in Asian countries in general. Cases of disorder were rated as mild (prevalence of 1.8%–9.7%), moderate (prevalence of 0.5%–9.4%) and serious (prevalence of 0.4%–7.7%). However, these are widely believed to be underestimates, due to poor diagnosis (especially in countries without affordable access to mental health services) and low reporting rates, in part because of the predominant use of self-report data, rather than semi-structured instruments such as the Structured Clinical Interview for DSM-IV (SCID); actual lifetime prevalence rates for mental disorders are estimated to be between 65% and 85%.

A review that pooled surveys in different countries up to 2004 found overall average prevalence estimates for any anxiety disorder of 10.6% (in the 12 months prior to assessment) and 16.6% (in lifetime prior to assessment), but that rates for individual
disorders varied widely. Women had generally higher prevalence rates than men, but the magnitude of the difference varied. A review that pooled surveys of mood disorders in different countries up to 2000 found 12-month prevalence rates of 4.1% for major depressive disorder (MDD), 2% for dysthymic disorder and 0.72% for bipolar 1 disorder. The average lifetime prevalence found was 6.7% for MDD (with a relatively low lifetime prevalence rate in higher-quality studies, compared to the rates typically highlighted of 5%–12% for men and 10%–25% for women), and rates of 3.6% for dysthymia and 0.8% for Bipolar 1.

Previous widely cited large-scale surveys in the US were the Epidemiological Catchment Area (ECA) survey and subsequent National Comorbidity Survey (NCS). The NCS was replicated and updated between 2000 and 2003 and indicated that, of those groups of disorders assessed, nearly half of Americans (46.4%) reported meeting criteria at some point in their life for either a DSM-IV anxiety disorder (28.8%), mood disorder (20.8%), impulse-control disorder (24.8%) or substance use disorders (14.6%). Half of all lifetime cases had started by age 14 and 3/4 by age 24. In the prior 12-month period only, around a quarter (26.2%) met criteria for any disorder—anxiety disorders 18.1%; mood disorders 9.5%; impulse control disorders 8.9%; and substance use disorders 3.8%. A substantial minority (23%) met criteria for more than two disorders. A minority (22.3%) of cases were classed as serious, 37.3% as moderate and 40.4% as mild.

A 2004 cross-European study found that approximately one in four people reported meeting criteria at some point in their life for one of the DSM-IV disorders assessed, which included mood disorders (13.9%), anxiety disorders (13.6%) or alcohol disorder (5.2%). Approximately one in ten met criteria within a 12-month period. Women and younger people of either gender showed more cases of disorder.

A 2005 review of 27 studies have found that 27% of adult Europeans is or has been affected by at least one mental disorder in the past 12 months. It was also found that the most frequent disorders were anxiety disorders, depressive, somatoform and substance dependence disorders.

A 2005 review of prior surveys in 46 countries on the prevalence of schizophrenic disorders, including a prior 10-country WHO survey, found an average (median) figure of 0.4% for lifetime prevalence up to the point of assessment and 0.3% in the 12-month period prior to assessment. A related figure not given in other studies (known as lifetime morbid risk), reported to be an accurate statement of how many people would theoretically develop schizophrenia at any point in life regardless of time of assessment, was found to be "about seven to eight individuals per 1,000" (0.7/0.8%). The prevalence of schizophrenia was consistently lower in poorer countries than in richer countries (though not the incidence), but the prevalence did not differ between urban/rural areas or men/women (although incidence did).

Studies of the prevalence of personality disorders (PDs) have been fewer and smaller-scale, but a broader Norwegian survey found a similar overall prevalence of almost 1 in 7 (13.4%), based on meeting personality criteria over the prior five year period. Rates for
specific disorders ranged from 0.8% to 2.8%, with rates differing across countries, and by
gender, educational level and other factors. A US survey that incidentally screened for
personality disorder found an overall rate of 14.79%.

Approximately 7% of a preschool pediatric sample were given a psychiatric diagnosis in
one clinical study, and approximately 10% of 1- and 2-year-olds receiving developmental
screening have been assessed as having significant emotional/behavioral problems based
on parent and pediatrician reports.

Anxiety disorder

The Scream (Norwegian: Skrik) an Expressionist painting by Norwegian artist Edvard Munch

Anxiety disorders are blanket terms covering several different forms of abnormal and
pathological fear and anxiety which only came under the aegis of psychiatry at the very end
of the 19th century. Gelder, Mayou & Geddes (2005) explains that anxiety disorders are
classified in two groups: continuous symptoms and episodic symptoms. Current psychiatric
diagnostic criteria recognize a wide variety of anxiety disorders. Recent surveys have found
that as many as 18% of Americans may be affected by one or more of them.

The term anxiety covers four aspects of experiences an individual may have: mental
apprehension, physical tension, physical symptoms and dissociative anxiety (symptoms
associated with hyperventilation). Anxiety disorder is divided into generalized anxiety, phobic, and panic disorders; each has its own characteristics and symptoms and they require different treatment (Gelder et al. 2005). The emotions present in anxiety disorders range from simple nervousness to bouts of terror (Barker 2003). Standardized screening clinical questionnaires such as Zung Self-Rating Anxiety Scale can be used to detect anxiety symptoms, and suggest the need for a formal diagnostic assessment of anxiety disorder.

**Classification**

**Generalized anxiety disorder**

Generalized anxiety disorder (GAD) is a common chronic disorder characterized by long-lasting anxiety that is not focused on any one object or situation. Those suffering from generalized anxiety experience non-specific persistent fear and worry and become overly concerned with everyday matters. Generalized anxiety disorder is the most common anxiety disorder to affect older adults. Anxiety can be a symptom of a medical or substance abuse problem, and medical professionals must be aware of this. A diagnosis of GAD is made when a person has been excessively worried about an everyday problem for six months or more. A person may find they have problems making daily decisions and remembering commitments as a result of lack of concentration/preoccupation with worry. Appearance looks strained, skin is pale with increased sweating from the hands, feet and axillae. May be tearful which can suggest depression. Before a diagnosis of anxiety disorder is made, nurses and physicians must rule out drug-induced anxiety and medical causes.

**Panic disorder**

In panic disorder, a person suffers from brief attacks of intense terror and apprehension, often marked by trembling, shaking, confusion, dizziness, nausea, difficulty breathing. These panic attacks, defined by the APA as fear or discomfort that abruptly arises and peaks in less than ten minutes, can last for several hours and can be triggered by stress, fear, or even exercise; the specific cause is not always apparent.

In addition to recurrent unexpected panic attacks, a diagnosis of panic disorder requires that said attacks have chronic consequences: either worry over the attacks' potential implications, persistent fear of future attacks, or significant changes in behavior related to the attacks. Accordingly, those suffering from panic disorder experience symptoms even outside specific panic episodes. Often, normal changes in heartbeat are noticed by a panic sufferer, leading them to think something is wrong with their heart or they are about to have another panic attack. In some cases, a heightened awareness (hypervigilance) of body functioning occurs during panic attacks, wherein any perceived physiological change is interpreted as a possible life-threatening illness (i.e., extreme hypochondriasis). However, with the correct professional help 70%-90% of those suffering from panic disorder are helped in 6-8 weeks.
Panic disorder with agoraphobia

A person experiences an unexpected panic attack, then has substantial anxiety over the possibility of having another attack. The person fears and avoids whatever situation might induce a panic attack. The person may never or rarely leave their home to prevent a panic attack they believe to be inescapable, extreme terror.

Phobias

The single largest category of anxiety disorders is that of phobic disorders, which includes all cases in which fear and anxiety is triggered by a specific stimulus or situation. Between 5% and 12% of the population worldwide suffer from phobic disorders. Sufferers typically anticipate terrifying consequences from encountering the object of their fear, which can be anything from an animal to a location to a bodily fluid to a particular situation. Sufferers understand that their fear is not proportional to the actual potential danger but still are overwhelmed by the fear.

Agoraphobia

Agoraphobia is the specific anxiety about being in a place or situation where escape is difficult or embarrassing or where help may be unavailable. Agoraphobia is strongly linked with panic disorder and is often precipitated by the fear of having a panic attack. A common manifestation involves needing to be in constant view of a door or other escape route. In addition to the fears themselves, the term agoraphobia is often used to refer to avoidance behaviors that sufferers often develop. For example, following a panic attack while driving, someone suffering from agoraphobia may develop anxiety over driving and will therefore avoid driving. These avoidance behaviors can often have serious consequences; in severe cases, one can be confined to one’s home.

Social anxiety disorder

Social anxiety disorder (SAD; also known as social phobia) describes an intense fear and avoidance of negative public scrutiny, public embarrassment, humiliation, or social interaction. This fear can be specific to particular social situations (such as public speaking) or, more typically, is experienced in most (or all) social interactions. Social anxiety often manifests specific physical symptoms, including blushing, sweating, and difficulty speaking. Like with all phobic disorders, those suffering from social anxiety often will attempt to avoid the source of their anxiety; in the case of social anxiety this is particularly problematic, and in severe cases can lead to complete social isolation.

Obsessive–compulsive disorder

Obsessive–compulsive disorder (OCD) is a type of anxiety disorder primarily characterized by repetitive obsessions (distressing, persistent, and intrusive thoughts or images) and compulsions (urges to perform specific acts or rituals). It affects roughly around 3% of the population worldwide. The OCD thought pattern may be likened to superstitions insofar as
it involves a belief in a causative relationship where, in reality, one does not exist. Often the process is entirely illogical; for example, the compulsion of walking in a certain pattern may be employed to alleviate the obsession of impending harm. And in many cases, the compulsion is entirely inexplicable, simply an urge to complete a ritual triggered by nervousness.

In a slight minority of cases, sufferers of OCD may only experience obsessions, with no overt compulsions; a much smaller number of sufferers experience only compulsions.

**Post-traumatic stress disorder**

Post-traumatic stress disorder (PTSD) is an anxiety disorder which results from a traumatic experience. Post-traumatic stress can result from an extreme situation, such as combat, natural disaster, rape, hostage situations, more serious kinds of child abuse, or even a serious accident. It can also result from long term (chronic) exposure to a severe stressor, for example soldiers who endure individual battles but cannot cope with continuous combat. Common symptoms include hypervigilance, flashbacks, avoidant behaviors, anxiety, anger and depression. There are a number of treatments which form the basis of the care plan for those suffering with PTSD. Such treatments include cognitive behavioral therapy (CBT), psychotherapy and support from family and friends. These are all examples of treatments used to help people suffering from PTSD.

**Separation anxiety**

Separation anxiety disorder (SepAD) is the feeling of excessive and inappropriate levels of anxiety over being separated from a person or place. Separation anxiety is a normal part of development in babies or children, and it is only when this feeling is excessive or inappropriate that it can be considered a disorder. Separation anxiety disorder affects roughly 7% of adults and 4% of children, but the childhood cases tend to be more severe, in some instances even a brief separation can produce panic.

**Childhood anxiety disorders**

Children as well as adults experience feelings of anxiousness, worry and fear when facing different situations, especially those involving a new experience. However, if anxiety is no longer temporary and begins to interfere with the child's normal functioning or do harm to their learning, the problem may be more than just an ordinary anxiousness and fear common to the age.

When children suffer from a severe anxiety disorder their thinking, decision-making ability, perceptions of the environment, learning and concentration get affected. They not only experience fear, nervousness, and shyness but also start avoiding places and activities. Anxiety also raises blood pressure and heart rate and can cause nausea, vomiting, stomach pain, ulcers, diarrhea, tingling, weakness, and shortness of breath. Some other symptoms are frequent self-doubt and self-criticism, irritability, sleep problems and, in extreme cases, thoughts of not wanting to be alive.
If these children are left untreated, they face risks such as poor results at school, avoidance of important social activities, and substance abuse. Children who suffer from an anxiety disorder are likely to suffer other disorders such as depression, eating disorders, and attention deficit disorders, both hyperactive and inattentive.

About 13 of every 100 children and adolescents between 9 to 17 years experience some kind of anxiety disorder, and girls are more affected than boys. The basic temperament of children may be key in some of their childhood and adolescent disorders.

Research in this area is very difficult to perform because as children grow their fears change, making it difficult for researchers to obtain enough data and thus more reliable results. For instance, between the ages of 6 and 8, children's fear of the dark and imaginary creatures decreases, but they become more anxious about school performance and social relationships. If children experience an excessive amount of anxiety during this stage, this could lead to development of anxiety disorders later in life.

According to research, childhood anxiety disorders are caused by biological and psychological factors. Also, it is suggested that when children have a parent with anxiety disorders, they are more likely to have an anxiety disorder, too. Stress can trigger anxiety disorders, and children and adolescents with anxiety disorders seem to have an increased physical and psychological reaction to stress. Their reaction to danger, even if it is a small one, is quicker and stronger.

**Causes**

**Biological**

Low levels of GABA, a neurotransmitter that reduces activity in the central nervous system, contribute to anxiety. A number of anxiolytics achieve their effect by modulating the GABA receptors.

Selective serotonin reuptake inhibitors, the drugs most commonly used to treat depression, are frequently considered as a first line treatment for anxiety disorders. A 2004 study using functional brain imaging techniques suggests that the effects of SSRIs in alleviating anxiety may result from a direct action on GABA neurons rather than as a secondary consequence of mood improvement.

Severe anxiety and depression can be induced by sustained alcohol abuse which in most cases abates with prolonged abstinence. Even moderate, sustained alcohol use may increase anxiety and depression levels in some individuals. Caffeine, alcohol and benzodiazepine dependence can worsen or cause anxiety and panic attacks. In one study in 1988–1990, illness in approximately half of patients attending mental health services at one British hospital psychiatric clinic, for conditions including anxiety disorders such as panic disorder or social phobia, was determined to be the result of alcohol or...
benzodiazepine dependence. In these patients, an initial increase in anxiety occurred during the withdrawal period followed by a cessation of their anxiety symptoms.

Intoxication from stimulants is likely to be associated with repetitive panic attacks.

There is evidence that chronic exposure to organic solvents in the work environment can be associated with anxiety disorders. Painting, varnishing and carpet-laying are some of the jobs in which significant exposure to organic solvents may occur.

People with obsessive-compulsive disorder (sometimes considered an anxiety disorder), evince increased grey matter volumes in bilateral lenticular nuclei, extending to the caudate nuclei, while decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri. These findings contrast with those in people with other anxiety disorders, who evince decreased (rather than increased) grey matter volumes in bilateral lenticular/caudate nuclei, while also decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri.

**Amygdala**

The amygdala is central to the processing of fear and anxiety, and its function may be disrupted in anxiety disorders. Sensory information enters the amygdala through the nuclei of the basolateral complex (consisting of lateral, basal, and accessory basal nuclei). The basolateral complex processes sensory-related fear memories and communicates their threat importance to memory and sensory processing elsewhere in the brain, such as the medial prefrontal cortex and sensory cortices.

Another important area is the adjacent central nucleus of the amygdala, which controls species-specific fear responses, via connections to the brainstem, hypothalamus, and cerebellum areas. In those with general anxiety disorder, these connections functionally seem to be less distinct, with greater gray matter in the central nucleus. Another difference is that the amygdala areas have decreased connectivity with the insula and cingulate areas that control general stimulus salience, while having greater connectivity with the parietal cortex and prefrontal cortex circuits that underlie executive functions.

The latter suggests a compensation strategy for dysfunctional amygdala processing of anxiety. Researchers have noted "Amygdalo-frontoparietal coupling in generalized anxiety disorder patients may ... reflect the habitual engagement of a cognitive control system to regulate excessive anxiety." This is consistent with cognitive theories that suggest the use in this disorder of attempts to reduce the involvement of emotions with compensatory cognitive strategies.

Clinical and animal studies suggest a correlation between anxiety disorders and difficulty in maintaining balance. A possible mechanism is malfunction in the parabrachial nucleus, a brain structure that, among other functions, coordinates signals from the amygdala with input concerning balance.
Anxiety processing in the basolateral amygdala has been implicated with dendritic arborization of the amygdaloid neurons. SK2 potassium channels mediate inhibitory influence on action potentials and reduce arborization. By overexpressing SK2 in the basolateral amygdala, anxiety in experimental animals can be reduced together with general levels of stress-induced corticosterone secretion.

**Stress**

Anxiety disorder can arise in response to life stresses such as financial worries or chronic physical illness. Somewhere between 4% and 10% of older adults are diagnosed with anxiety disorder, a figure that is probably an underestimate due to the tendency of adults to minimize psychiatric problems or to focus on their physical manifestations. Anxiety is also common among older people who have dementia. On the other hand, anxiety disorder is sometimes misdiagnosed among older adults when doctors misinterpret symptoms of a physical ailment (for instance, racing heartbeat due to cardiac arrhythmia) as signs of anxiety.

**Diagnosis**

Anxiety disorders are often debilitating chronic conditions, which can be present from an early age or begin suddenly after a triggering event. They are prone to flare up at times of high stress and are frequently accompanied by physiological symptoms such as headache, sweating, muscle spasms, palpitations, and hypertension, which in some cases lead to fatigue or even exhaustion.

In casual discourse the words "anxiety" and "fear" are often used interchangeably; in clinical usage, they have distinct meanings: "anxiety" is defined as an unpleasant emotional state for which the cause is either not readily identified or perceived to be uncontrollable or unavoidable, whereas "fear" is an emotional and physiological response to a recognized external threat. The term "anxiety disorder" includes fears (phobias) as well as anxieties.

Anxiety disorders are often comorbid with other mental disorders, particularly clinical depression, which may occur in as many as 60% of people with anxiety disorders. The fact that there is considerable overlap between symptoms of anxiety and depression, and that the same environmental triggers can provoke symptoms in either condition, may help to explain this high rate of comorbidity.

Studies have also indicated that anxiety disorders are more likely among those with family history of anxiety disorders, especially certain types.

Sexual dysfunction often accompanies anxiety disorders although it is difficult to determine whether anxiety causes the sexual dysfunction or whether they arise from a common cause. The most common manifestations in individuals with anxiety disorder are avoidance of intercourse, premature ejaculation or erectile dysfunction among men and pain during intercourse among women. Sexual dysfunction is particularly common among
people affected by panic disorder (who may fear that a panic attack will occur during sexual arousal) and posttraumatic stress disorder.

**Treatment**

The most important clinical point to emerge from studies of social anxiety disorder is the benefit of early diagnosis and treatment. Social anxiety disorder remains under-recognized in primary care practice, with patients often presenting for treatment only after the onset of complications such as clinical depression or substance abuse disorders.

Treatment options available include lifestyle changes; psychotherapy, especially cognitive behavioral therapy; and pharmaceutical therapy. Education, reassurance and some form of cognitive-behavioral therapy should almost always be used in treatment. Research has provided evidence for the efficacy of two forms of treatment available for social phobia: certain medications and a specific form of short-term psychotherapy called cognitive-behavioral therapy (CBT), the central component being gradual exposure therapy.

**Psychotherapy**

Research has shown that cognitive-behavioral therapy (CBT) can be highly effective for several anxiety disorders, particularly panic disorder and social phobia. CBT, as its name suggests, has two main components: cognitive and behavioral. In cases of social anxiety, the cognitive component can help the patient question how they can be so sure that others are continually watching and harshly judging him or her. The behavioral component seeks to change people's reactions to anxiety-provoking situations.

As such it serves as a logical extension of cognitive therapy, whereby people are shown proof in the real world that their dysfunctional thought processes are unrealistic. A key element of this component is gradual exposure, in which the patient is confronted by the things they fear in a structured, sensitive manner. Gradual exposure is an inherently unpleasant technique; ideally it involves exposure to a feared social situation that is anxiety provoking but bearable, for as long as possible, two to three times a week. Often, a hierarchy of feared steps is constructed and the patient is exposed to each step sequentially.

The aim is to learn from acting differently and observing reactions. This is intended to be done with support and guidance, and when the therapist and patient feel they are ready. Cognitive-behavioral therapy for social phobia also includes anxiety management training, which may include techniques such as deep breathing and muscle relaxation exercises, which may be practiced 'in-situ'. CBT can also be conducted partly in group sessions, facilitating the sharing of experiences, a sense of acceptance by others and undertaking behavioral challenges in a trusted environment (Heimberg).

Some studies have suggested social skills training can help with social anxiety. However, it is not clear whether specific social skills techniques and training are required, rather than just support with general social functioning and exposure to social situations.
Additionally, a recent study has suggested that interpersonal therapy, a form of psychotherapy primarily used to treat depression, may also be effective in the treatment of social phobia.

Extensive research supports the neural plasticity of the brain in reaction to stressful experiences. A treatment and prevention method called Adaptive Behavioral therapy is based on understanding the adaptations which occur in developmental years due to stressful experiences and the brain's ability to create new reactions to the same stressor. In this treatment a time gap between when information is received by the brain (stress trigger) and the decision for behavior is widened to allow a re-evaluation to occur as a stressful experience is taking place. This new time to reprocess information is referred to as a Pivotal Moment when new behavior can be consciously created. The development of new adaptive behavior is promoted through the use of interruption techniques and specific tools.

Treatment is structured around a self report notebook which is used to collect historic and current stress patterns of response. Treatment is guided by the therapist in a direct manner in sessions and experiences between sessions are used to provide new experimental behavior. Adaptive behavioral therapy is also used as a preventive treatment. Practice with smaller stressors creates familiarity with healthy adaptive responses for future use.

**Medications**

When medication is indicated, SSRIs are generally recommended as first line agents. SNRIs such as venlafaxine (Effexor) are also effective. Benzodiazepines are also sometimes indicated for short-term or PRN use. They are usually considered as a second-line treatment due to disadvantages such as cognitive impairment and due to their risks of dependence and withdrawal problems. MAOIs such as phenelzine (Nardil) and tranylcypromine (Parnate) are considered an effective treatment and are especially useful in treatment-resistant cases, however, dietary restrictions and medical interactions may limit their use. There is evidence that certain newer medications including the GABA analogue pregabalin (Lyrica) and the novel antidepressant mirtazapine (Remeron) are effective treatments for anxiety disorders. TCAs such as imipramine, as well as atypical antipsychotics such as quetiapine, and piperazines such as hydroxyzine are occasionally prescribed.

These medications need to be used with extreme care among older adults, who are more likely to suffer side effects because of coexisting physical disorders. Adherence problems are more likely among elderly patients, who may have difficulty understanding, seeing, or remembering instructions.

**SSRIs**
Selective serotonin reuptake inhibitors (SSRIs), a class of antidepressants, are considered by many to be the first choice medication for generalised social phobia. These drugs elevate the level of the neurotransmitter serotonin, among other effects. The first drug formally approved by the Food and Drug Administration was paroxetine, sold as Paxil in the U.S. or Seroxat in the UK. Compared to older forms of medication, there is less risk of tolerability and drug dependency. However, their efficacy and increased suicide risk has been subject to controversy.

In a 1995 double-blind, placebo-controlled trial, the SSRI paroxetine was shown to result in clinically meaningful improvement in 55% of patients with generalized social anxiety disorder, compared with 23.9% of those taking placebo. An October 2004 study yielded similar results. Patients were treated with either fluoxetine, psychotherapy, fluoxetine and psychotherapy, placebo and psychotherapy, or a placebo. The first four sets saw improvement in 50.8% to 54.2% of the patients. Of those assigned to receive only a placebo, 31.7% achieved a rating of 1 or 2 on the Clinical Global Impression-Improvement scale. Those who sought both therapy and medication did not see a boost in improvement.

General side-effects are common during the first weeks while the body adjusts to the drug. Symptoms may include headaches, nausea, insomnia and changes in sexual behavior. Treatment safety during pregnancy has not been established. In late 2004 much media attention was given to a proposed link between SSRI use and juvenile suicide. For this reason, the use of SSRIs in pediatric cases of depression is now recognized by the Food and Drug Administration as warranting a cautionary statement to the parents of children who may be prescribed SSRIs by a family doctor. Recent studies have shown no increase in rates of suicide. These tests, however, represent those diagnosed with depression, not necessarily with social anxiety disorder. However, due to the nature of the conditions, those taking SSRIs for social phobias are far less likely to have suicidal ideation than those with depression.

**Other drugs**

Although SSRIs are often the first choice for treatment, other prescription drugs are used, sometimes only if SSRIs fail to produce any clinically significant improvement.

In 1985, before the introduction of SSRIs, anti-depressants such as monoamine oxidase inhibitors (MAOIs) were frequently used in the treatment of social anxiety. Their efficacy appears to be comparable or sometimes superior to SSRIs or benzodiazepines. However, because of the dietary restrictions required, high toxicity in overdose, and incompatibilities with other drugs, its usefulness as a treatment for social phobics is now limited. Some argue for their continued use, however, or that a special diet does not need to be strictly adhered to. A newer type of this medication, Reversible inhibitors of monoamine oxidase subtype A (RIMAs) inhibit the MAO enzyme only temporarily, improving the adverse-effect profile but possibly reducing their efficacy.

Benzodiazepines such as alprazolam and clonazepam are an alternative to SSRIs. These drugs are often used for short-term relief of severe, disabling anxiety. Although
benzodiazepines are still sometimes prescribed for long-term everyday use in some countries, there is much concern over the development of drug tolerance, dependency and recreational abuse. It has been recommended that benzodiazepines are only considered for individuals who fail to respond to safer medications. Benzodiazepines augment the action of GABA, the major inhibitory neurotransmitter in the brain; effects usually begin to appear within minutes or hours.

The novel antidepressant mirtazapine has been proven effective in treatment of social anxiety disorder. This is especially significant due to mirtazapine’s fast onset and lack of many unpleasant side-effects associated with SSRIs (particularly, sexual dysfunction).

In Japan, the serotonin-norepinephrine reuptake inhibitor (SNRI) Milnacipran is used in the treatment of Taijin kyofusho a Japanese variant of social anxiety disorder.

Some people with a form of social phobia called performance phobia have been helped by beta-blockers, which are more commonly used to control high blood pressure. Taken in low doses, they control the physical manifestation of anxiety and can be taken before a public performance.

A novel treatment approach has recently been developed as a result of translational research. It has been shown that a combination of acute dosing of d-cycloserine (DCS) with exposure therapy facilitates the effects of exposure therapy of social phobia (Hofmann, Meuret, Smits, et al., 2006). DCS is an old antibiotic medication used for treating tuberculosis and does not have any anxiolytic properties per se. However, it acts as an agonist at the glutamatergic N-methyl-D-aspartate (NMDA) receptor site, which is important for learning and memory (Hofmann, Pollack, & Otto, 2006). It has been shown that administering a small dose acutely 1 hour before exposure therapy can facilitate extinction learning that occurs during therapy.

Treatment controversy arises because while some studies indicate that a combination of medication and psychotherapy can be more effective than either one alone, others suggest pharmacological interventions are largely palliative, and can actually interfere with the mechanisms of successful therapy. Meta-analysis indicates that psychotherapeutic interventions have better long-term efficacy compared to pharmacotherapy. However, the right treatment may very much depend on the individual patient’s genetics and environmental factors.

**Alternative medicine**

Regular aerobic exercise, improving sleep hygiene and reducing caffeine are often useful in treating anxiety.

Herbal drugs are often used in patients with somatoform disorders. In one clinical trial, butterbur in a fixed herbal drug combination (Ze 185 = 4-combination versus 3-combination without butterbur and placebo) was used in patients with somatoform disorders. For a 2-week treatment in patients with somatization disorder (F45.0) and
undifferentiated somatoform disorder (F45.1), 182 patients were randomized for a 3-arm trial (butterbur root, valerian root, passionflower herb, lemon balm leaf versus valerian root, passionflower herb, lemon balm leaf versus placebo). Anxiety (visual analogue scale - VAS) and depression (Beck’s Depression Inventory - BDI) were used as primary parameters, and Clinical Global Impression (CGI) was used a secondary parameter. The 4-combination was significantly superior to the 3-combination and placebo in all the primary and secondary parameters (PP-population), without serious adverse events.

Many other natural remedies have been used for anxiety disorder. These include kava, where the potential for benefit seems greater than that for harm with short-term use in patients with mild to moderate anxiety. Based on Cochrane’s systematic review of seven RCTs (n = 380), with findings supported by five lower-quality trials (n = 320), the American Academy of Family Physicians (AAFP) recommends use of kava for patients with mild to moderate anxiety disorders who are not using alcohol or taking other medicines metabolized by the liver, but who wish to use “natural” remedies. Side effects of kava in the clinical trials were rare and mild.

Inositol has been found to have modest effects in patients with panic disorder or obsessive-compulsive disorder. St. John’s wort and Sympateryl have also been used to treat anxiety, but with little scientific evidence as to their effectiveness.

**Epidemiology**

In the United States the lifetime prevalence of anxiety disorders is about 29%.

**Prognosis**

It is the most common cause of disability in the workplace in the United States.

**Generalized anxiety disorder**

Generalized anxiety disorder (GAD) is an anxiety disorder that is characterized by excessive, uncontrollable and often irrational worry about everyday things that is disproportionate to the actual source of worry. This excessive worry often interferes with daily functioning, as individuals suffering GAD typically anticipate disaster, and are overly concerned about everyday matters such as health issues, money, death, family problems, friend problems, relationship problems or work difficulties. Individuals often exhibit a variety of physical symptoms, including fatigue, fidgeting, headaches, nausea, numbness in hands and feet, muscle tension, muscle aches, difficulty swallowing, bouts of difficulty breathing, difficulty concentrating, trembling, twitching, irritability, agitation, sweating, restlessness, insomnia, hot flashes, and rashes and inability to fully control the anxiety (ICD-10). These symptoms must be consistent and on-going, persisting at least six months, for a formal diagnosis of GAD to be introduced. Approximately 6.8 million American adults experience GAD, and 2 percent of adult Europeans, in any given year, experience GAD.
Standardized rating scales such as GAD-7 can be used to assess severity of generalized anxiety disorder symptoms. It is the most common cause of disability in the workplace in the United States.

**Prevalence**

The World Health Organization’s Global Burden of Disease project did not include generalized anxiety disorders. In lieu of global statistics, here are some prevalence rates from around the world:

- Australia: 3 percent of adults
- Canada: Between 3 and 5 percent of adults
- Italy: 2.9 percent
- Taiwan: 0.4 percent
- United States: approx. 3.1 percent of people age 18 and over in a given year (9.5 million)

55 to 60 percent of people diagnosed in clinical settings are women.

**Epidemiology**

The usual age of onset is variable - from childhood to late adulthood, with the median age of onset being approximately 31 (Kessler, Berglund, et al., 2005). Most studies find that GAD is associated with an earlier and more gradual onset than the other anxiety disorders.

Women are two to three times more likely to suffer from generalized anxiety disorder than men, although this finding appears to be restricted to only developed countries, the spread of GAD is somewhat equal in developing nations. GAD is also common in the elderly population.

**Potential causes**

Some research suggests that GAD may run in families, and it may also grow worse during stress. GAD usually begins at an earlier age and symptoms may manifest themselves more slowly than in most other anxiety disorders. Some people with GAD report onset in early adulthood, usually in response to a life stressor. Once GAD develops, it can be chronic, but can be managed, if not all-but-alleviated, with proper treatment.

**Substance induced**

Long-term use of benzodiazepines can worsen underlying anxiety, with evidence that reduction of benzodiazepines can lead to a lessening of anxiety symptoms. Similarly, long-term alcohol use is associated with anxiety disorders, with evidence that prolonged abstinence can result in a disappearance of anxiety symptoms.

In one study in 1988–90, illness in approximately half of patients attending mental health services at British hospital psychiatric clinic, for conditions including anxiety disorders
such as panic disorder or social phobia, was determined to be the result of alcohol or benzodiazepine dependence. In these patients, anxiety symptoms, while worsening initially during the withdrawal phase, disappeared with abstinence from benzodiazepines or alcohol. Sometimes anxiety pre-existed alcohol or benzodiazepine dependence but the dependence was acting to keep the anxiety disorders going and often progressively making them worse. Recovery from benzodiazepines tends to take a lot longer than recovery from alcohol but people can regain their previous good health.

**Neurology**

Generalized anxiety disorder has been linked to disrupted functional connectivity of the amygdala and its processing of fear and anxiety. Sensory information enters the amygdala through the nuclei of the basolateral complex (consisting of lateral, basal, and accessory basal nuclei). The basolateral complex processes sensory-related fear memories and communicate their threat importance to memory and sensory processing elsewhere in the brain such as the medial prefrontal cortex and sensory cortices. Another area the adjacent central nucleus of the amygdala that controls species-specific fear responses its connections brainstem, hypothalamus, and cerebellum areas. In those with generalized anxiety disorder these connections functionally seem to be less distinct and there is greater gray matter in the central nucleus. Another difference is that the amygdala areas have decreased connectivity with the insula and cingulate areas that control general stimulus salience while having greater connectivity with the parietal cortex and prefrontal cortex circuits that underlie executive functions. The latter suggests a compensation strategy for dysfunctional amygdala processing of anxiety. This is consistent with cognitive theories that suggest the use in this disorder of attempts to reduce the involvement of emotions with compensatory cognitive strategies.

**Diagnosis**

**DSM-IV-TR criteria**

DSM-IV-TR diagnostic criteria for generalized anxiety disorder are as follows:

A. Excessive anxiety and worry (apprehensive expectation), occurring more-days-than-not for at least 6 months, about a number of events or activities (such as work or school performance).

B. The person finds it difficult to control the worry.

C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms present for more-days-than-not for the past 6 months).

- restlessness or feeling keyed up or on edge
- being easily fatigued
- difficulty concentrating or mind going blank
- irritability
- muscle tension
- sleep disturbance (difficulty falling or staying asleep, or restless unsatisfying sleep)

D. The focus of the anxiety and worry is not confined to features of other Axis I disorder (such as social phobia, OCD, PTSD etc.)

E. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hyperthyroidism), and does not occur exclusively during a mood disorder, psychotic disorder, or a pervasive developmental disorder.

ICD-10 criteria

F41.1 Generalized anxiety disorder
Note: For children different criteria may be applied (see F93.80).

A. A period of at least six months with prominent tension, worry and feelings of apprehension, about every-day events and problems.
B. At least four symptoms out of the following list of items must be present, of which at least one from items (1) to (4).

Autonomic arousal symptoms

(1) Palpitations or pounding heart, or accelerated heart rate.
(2) Sweating.
(3) Trembling or shaking.
(4) Dry mouth (not due to medication or dehydration).

Symptoms concerning chest and abdomen

(5) Difficulty breathing.
(6) Feeling of choking.
(7) Chest pain or discomfort.
(8) Nausea or abdominal distress (e.g. churning in stomach).

Symptoms concerning brain and mind

(9) Feeling dizzy, unsteady, faint or light-headed.
(10) Feelings that objects are unreal (derealization), or that one’s self is distant or "not really here" (depersonalization).
(11) Fear of losing control, going crazy, or passing out.
(12) Fear of dying.
General symptoms

(13) Hot flushes or cold chills.
(14) Numbness or tingling sensations.

Symptoms of tension

(15) Muscle tension or aches and pains.
(16) Restlessness and inability to relax.
(17) Feeling keyed up, or on edge, or of mental tension.
(18) A sensation of a lump in the throat, or difficulty with swallowing.

Other non-specific symptoms

(19) Exaggerated response to minor surprises or being startled.
(20) Difficulty in concentrating, or mind going blank, because of worrying or anxiety.
(21) Persistent irritability.
(22) Difficulty getting to sleep because of worrying.

C. The disorder does not meet the criteria for panic disorder (F41.0), phobic anxiety disorders (F40.-), obsessive-compulsive disorder (F42.-) or hypochondriacal disorder (F45.2).

D. Most commonly used exclusion criteria: not sustained by a physical disorder, such as hyperthyroidism, an organic mental disorder (F0) or psychoactive substance-related disorder (F1), such as excess consumption of amphetamine-like substances, or withdrawal from benzodiazepines.

Treatment

Psychotherapy

Psychotherapy for GAD should be oriented toward combating the individual's low-level, ever-present anxiety. Such anxiety is often accompanied by poor planning skills, high stress levels, and difficulty in relaxing. This last point is important because it is the easiest one in which the therapist can play an especially effective teaching role.

Relaxation skills can be taught either alone or with the use of biofeedback. Education about relaxation and simple relaxation exercises, such as deep breathing, are excellent places to begin therapy. While biofeedback (the ability to allow the patient to hear or see feedback of their body’s physiological state) is beneficial, it is not required for effective relaxation to be taught to most people. Progressive muscle relaxation and more general imagery techniques can be used as therapy progresses. Teaching an individual how to relax, and the ability to do it in any place or situation is vital to reducing the low-level anxiety levels. Individuals who learn these skills, which can be taught in a brief-therapy framework, go on to lead
productive, generally anxiety-free lives once therapy is complete. A common reason for failure to make any gains with relaxation skills is simply because the client does not practice them outside of the therapy session. From the onset of therapy, the individual who suffers from GAD should be encouraged to set a regular schedule in which to practice relaxation skills learned in session, at least twice a day for a minimum of 20 minutes (although more often and for longer periods of time is better). Lack of treatment progress can often be traced to a failure to follow through with homework assignments of practicing relaxation.

Reducing stress and increasing overall coping skills may also be beneficial in helping the client. Many people who have GAD also lead very active (some would say, "hectic") lives. Helping the individual find a better balance in their lives between self-enrichment, family, significant other, and work may be important. People who have GAD have lived with their anxiety for such a long time they may not recognize a life without constant worrying and activity. Helping the individual realize that life doesn't have to be boring just because one isn't always worrying or doing things may also help.

Individual therapy is usually the recommended treatment modality. Many times people who present with GAD feel a bit awkward discussing their anxiety in front of others, especially if they are less than accepting. A clear distinction should be made at the onset of the evaluation to differentiate GAD from social phobia, however, and the appropriate diagnosis should be made. It would be unwise to recommend group therapy to someone who had social phobia or GAD early on, because of the social component to either disorder. Placing a person into a group setting without minimal interpersonal and relaxation skills being taught first in individual therapy is a recipe for disaster and early treatment termination.

Non-specific factors in therapy are important to these patients, as they will make the most beneficial gains in a supportive and accepting therapeutic environment. Simply listening to the individual and offering objective feedback about their experiences is likely helpful. Examining stressors in the client's life and helping the individual find better ways of handling these stressors is likely to be beneficial. Modeling techniques of appropriate social behaviors within therapy session may help. Clinicians should not confuse GAD with specific phobias, which have much more acute and traumatic symptoms. In the same respect, treatments for specific phobias generally are not appropriate nor effective with GAD. Some clinicians easily confuse this important distinction.

Hypnotherapy is also an appropriate treatment modality for those individuals who are highly suggestible. Often hypnotherapy is combined with other relaxation techniques.

If an individual finds themselves hyperventilating then they are breathing in too much oxygen. One of the correct things to do is to direct them to breath into a paper bag. This does increase the percent of CO2 in the inhalation, which thereby helps keep the O2/CO2 balance. While this technique is valid, the better technique is to slow down respiration rate and volume with slow deep breaths (without the paper bag).

**Medications**

Medication should be prescribed if the anxiety symptoms are serious and interfering with normal daily functioning. Psychotherapy and relaxation techniques can't be worked on effectively if the individual is overwhelmed by anxiety or cannot concentrate.

The most commonly prescribed anti-anxiety agent for this disorder has historically been benzodiazepines, despite a dearth of clinical research that shows this particular class of drugs is any more effective than others. Diazepam (Valium) and lorazepam (Ativan) are the two most prescribed benzodiazepines. Lorazepam will produce a more lengthy sedating effect than diazepam, although it will take longer to appear. Individuals on these medications should always be advised about the medications' side effects, especially their sedative properties and impairment on performance.

Tricyclic antidepressants often are an effective treatment alternative to benzodiazepines and may be a better choice over a longer treatment period.

Medication for this disorder should only be used to treat acute symptoms of anxiety. Medication should be tapered off when it is discontinued.

Phillip W. Long, M.D. also notes:

"Buspirone, a new nonbenzodiazepine antianxiety drug, is non-addictive and does not impair mechanical performance such as driving. Response to buspirone occurs approximately in two weeks, as compared to the more rapid onset associated with benzodiazepines. Schwiezer et al. (1986) studied patients who previously had taken benzodiazepines for the treatment of anxiety and who were later placed on buspirone. These patients were found to have a poor response to buspirone."

**Self-Help**

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. Many support groups exist within communities throughout the world which are devoted to helping individuals with this disorder share their commons experiences and feelings of anxiety. Individuals should first be able to tolerate and effectively handle a social group interaction. Pushing an individual into a group setting, whether it be self-help or a regular group therapy experience, is counterproductive and may lead to a worsening of symptoms.

A meta-analysis of 35 studies shows cognitive behavioral therapy to be more effective in the long term than pharmacologic treatment (drugs such as SSRIs), and while both treatments reduce anxiety, CBT is more effective in reducing depression.
Cognitive behavioral therapy

Cognitive behavioral therapy (CBT) is a psychological method of treatment for GAD that involves a therapist working with the patient to understand how thoughts and feelings influence behavior. The goal of the therapy is to change negative thought patterns that lead to the patient’s anxiety, replacing them with positive, more realistic ones. Elements of the therapy include exposure strategies to allow the patient to gradually confront their anxieties and feel more comfortable in anxiety-provoking situations, as well as to practice the skills they have learned. CBT can be used alone or in conjunction with medication.

CBT usually helps one third of the patients substantially, whilst another third does not respond at all to treatment.

SSRIs

Pharmaceutical treatments for GAD include selective serotonin reuptake inhibitors (SSRIs), which are antidepressants that influence brain chemistry to block the reabsorption of serotonin in the brain. SSRIs are mainly indicated for clinical depression, but are also very effective in treating anxiety disorders. Common side effects include nausea, sexual dysfunction, headache, diarrhea, constipation, among others. Common SSRIs prescribed for GAD include:

- fluoxetine (Prozac, Sarafem)
- paroxetine (Paxil, Aropax)
- escitalopram (Lexapro, Cipralex)
- sertraline (Zoloft)

Pregabalin

Pregabalin (Lyrica) acts on the voltage-dependent calcium channel in order to decrease the release of neurotransmitters such as glutamate, noradrenaline and substance P. Its therapeutic effect appears after 1 week of use and is similar in effectiveness to lorazepam, alprazolam and venlafaxine but pregabalin has demonstrated superiority by producing more consistent therapeutic effects for psychic and somatic anxiety symptoms. Long-term trials have shown continued effectiveness without the development of tolerance and additionally unlike benzodiazepines it does not disrupt sleep architecture and produces less severe cognitive and psychomotor impairment; it also has a low potential for abuse and dependence and may be preferred over the benzodiazepines for these reasons.

Other drugs

- Psychotropic drugs

  - Buspirone (BuSpar) is a serotonin receptor partial agonist, belonging to the azaspirodecaneidione class of compounds.
  - Duloxetine (Cymbalta)- SNRI - type antidepressant
- Imipramine (Tofranil) is a tricyclic antidepressant (TCA).
- Other tricyclic antidepressants - as clomipramine, etc. TCAs are thought to act on serotonin, norepinephrine, and dopamine in the brain.
- Venlafaxine (Effexor XR) is a serotonin-norepinephrine reuptake inhibitor (SNRI). SNRIs alter the chemistries of both norepinephrine and serotonin in the brain.
- Some of MAO inhibitors - such as Moclobemide, rarely Nialamide

- Non-psychotropic drugs
  - Propranolol (Inderal) - Sympatholytic, beta-adrenoblocker
  - Clonidine - Sympatholytic
  - Guanfacine - Sympatholytic
  - Prazosin - Sympatholytic, alpha-adrenoblocker

Benzodiazepines

Benzodiazepines (or "benzos") are fast-acting hypnotic sedative depressants that are also used to treat GAD and other anxiety disorders. Benzodiazepines are prescribed for generalized anxiety disorder and show beneficial effects in the short term. However, they have long term adverse effects and for this reason the FDA has only approved them for short term usage (6–12 weeks). The World Council of Anxiety does not recommend the long-term use of benzodiazepines because they are associated with the development of tolerance, psychomotor impairment, cognitive and memory impairments, physical dependence and a withdrawal syndrome. Side effects include drowsiness, reduced motor coordination and problems with equilibration. Common benzodiazepines used to treat GAD include:

- alprazolam (Xanax, Xanax XR, Niravam)
- chlordiazepoxide (Librium)
- clonazepam (Klonopin)
- clorazepate (Tranxene)
- diazepam (Valium)
- lorazepam (Ativan)

GAD and comorbid depression

In the National Comorbidity Survey (2005), 58 percent of patients diagnosed with major depression were found to have an anxiety disorder; among these patients, the rate of comorbidity with GAD was 17.2 percent, and with panic disorder, 9.9 percent. Patients with a diagnosed anxiety disorder also had high rates of comorbid depression, including 22.4 percent of patients with social phobia, 9.4 percent with agoraphobia, and 2.3 percent with panic disorder. For many, the symptoms of both depression and anxiety are not severe enough (i.e. are subsyndromal) to justify a primary diagnosis of either major depressive disorder (MDD) or an anxiety disorder. However, Dysthymic Disorder is the most prevalent comorbid diagnosis of GAD clients.
Patients can also be categorized as having mixed anxiety-depressive disorder, and they are at significantly increased risk of developing full-blown depression or anxiety.

Accumulating evidence indicates that patients with comorbid depression and anxiety tend to have greater illness severity and a lower treatment response than those with either disorder alone. In addition, social function and quality of life are more greatly impaired.

In addition to coexisting with depression, research shows that GAD often coexists with substance abuse or other conditions associated with stress, such as irritable bowel syndrome. Patients with physical symptoms such as insomnia or headaches should also tell their doctors about their feelings of worry and tension. This will help the patient’s health care provider to recognize whether the person is suffering from GAD.

**Panic disorder**

Panic disorder is an anxiety disorder characterized by recurring severe panic attacks. It may also include significant behavioral change lasting at least a month and of ongoing worry about the implications or concern about having other attacks. The latter are called anticipatory attacks (DSM-IVR). Panic disorder is not the same as agoraphobia (fear of public places), although many with panic disorder also suffer from agoraphobia. Panic attacks cannot be predicted, therefore an individual may become stressed, anxious or worried wondering when the next panic attack will occur. There are other schools of thought that Panic disorder is differentiated as a medical condition, or chemical imbalance. The DSM-IV-TR describes Panic disorder, and Anxiety differently. Panic attacks have a sudden or out-of-blue cause that lasts shorter with more intense symptoms, as opposed to Anxiety attacks having stressors that build to less severe reactions and can last for weeks or months. Panic attacks can occur in children, as well as adults. Panic in young people may be particularly distressing because the child has less insight about what is happening, and his/her parent is also likely to experience distress when attacks occur.

Screening tools like Panic Disorder Severity Scale can be used to detect possible cases of disorder, and suggest the need for a formal diagnostic assessment.

**Background**

Panic disorder is a potentially disabling disorder, but can be controlled and successfully treated. Because of the intense symptoms that accompany panic disorder, it may be mistaken for a life-threatening physical illness such as a heart attack. This misconception often aggravates or triggers future attacks (some are called Anticipatory Attacks). People frequently go to hospital emergency rooms when they are having panic attacks, and extensive medical tests may be performed to rule out these other conditions, thus creating further anxiety. Nonetheless, Coryell et al. found death rates in panic disorder patients exceeded those in the general population. In their study, 20% of deaths in 113 former psychiatric inpatients with panic disorder followed 35 years later were suicides; however, due to the co-morbidity of anxiety disorders, it is unclear whether panic disorder was the
main cause of suicide. This study also found that men with panic disorder had twice the risk of cardiovascular mortality compared to men in the general population. Effective treatment of panic disorder has been shown to offset costs of medical care by as much as 94%. There are three types of panic attacks: unexpected, situationally bounded and situationally predisposed (American psychiatric association 2000).

**Signs and symptoms**

Panic disorder sufferers usually have a series of intense episodes of extreme anxiety during panic attacks. These attacks typically last about ten minutes, but can be as short-lived as 1–5 minutes and last as long as twenty minutes or until medical intervention. However, attacks can wax and wane for a period of hours (panic attacks rolling into one another), and the intensity and specific symptoms of panic may vary over the duration. Common symptoms of an attack include rapid heartbeat, perspiration, dizziness, dyspnea, trembling, uncontrollable fear and hyperventilation. Other symptoms are sweating, shortness of breath, sensation of choking, chest pain, nausea, numbness or tingling, chills or hot flashes, and some sense of altered reality. In addition, the person usually has thoughts of impending doom. Individuals suffering from an episode have often a strong wish of escaping from the situation that provoked the attack. The anxiety of Panic Disorder is particularly severe and noticeably episodic compared to that from Generalized Anxiety Disorder. There are instances when panic attacks are provoked by exposure to certain stimuli e.g. seeing a mouse. Other attacks may emanate out of nowhere or in specific settings e.g. the dentist’s office. Some individuals deal with these events on a regular basis, sometimes daily or weekly. The outward symptoms of a panic attack often cause negative social experiences (e.g. embarrassment, social stigma, social isolation, etc.).

Limited symptom attacks are similar to panic attacks, but have fewer symptoms. Most people with PD experience both panic attacks and limited symptom attacks.

**Causes**

There is no single cause for panic disorder, however, panic disorder has been found to run in families, and suggests that inheritance plays a strong role in determining who will get it. It has also been found to exist as a co-morbid condition with many hereditary disorders, such as bipolar disorder, and a genetic predisposition to alcoholism.

Psychological factors, stressful life events, life transitions, environment, and thinking in a way that exaggerates relatively normal bodily reactions are also believed to play a role in the onset of panic disorder. Often the first attacks are triggered by physical illnesses, major stress, or certain medications. People who tend to take on excessive responsibilities may develop a tendency to suffer panic attacks. Post-traumatic stress disorder (PTSD) patients also show a much higher rate of panic disorder than the general population.

There is some evidence to suggest hypoglycemia, hyperthyroidism, mitral valve prolapse, labyrinthitis, pheochromocytoma and respiratory conditions can cause or aggravate panic disorder.
Prepulse inhibition has been found to be reduced in patients with Panic Disorder.

Stimulants like caffeine, in excess, are a rather common cause for panic attacks. Many SSRIs also have stimulant side-effects during the beginning of treatment which may exacerbate the condition and have actually caused first-time panic attacks in otherwise healthy individuals being treated for depression.

Flöttmann describes the genesis of panic psychodynamically. Panic is a stress symptom. Fear is characteristic of each developmental stage because of feeling of guilt or symbiotic binding. Floating fear or panic stands for the parental stressing call: "Come back to me. You'll panic in your life, you'll have fear of sexuality, fear of separation from me, of being autonomous, and you'll have fear in any situation in your life! You'll feel anxiously, if you do anything that is separating you from mother or father. Don't grow up!" It is the panic that appears in any developmental moment of life.

There are other researchers looking at some individuals with panic disorder as having a chemical imbalance within the limbic system and one of its regulatory chemicals GABA-A. The reduced production of GABA-A sends false information to the amygdala which regulates the body's "fight or flight response" mechanism and in return, produces the physiological symptoms that lead to the disorder. Clonazepam, an anticonvulsant benzodiazepine with a long half-life, has been successful in keeping the condition in check.

Mediators and Moderators of Panic Disorder

Recently, researchers have begun to identify mediators and moderators of aspects of panic disorder. One such mediator is the partial pressure of carbon dioxide, which mediates the relationship between panic disorder patients receiving breathing training and anxiety sensitivity; thus, breathing training affects the partial pressure of carbon dioxide in a patient's arterial blood, which in turn lowers anxiety sensitivity. Another mediator is hypochondriacal concerns, which mediate the relationship between anxiety sensitivity and panic symptomatology; thus, anxiety sensitivity affects hypochondriacal concerns which, in turn, affect panic symptomatology.

Perceived threat control has been identified as a moderator within panic disorder, moderating the relationship between anxiety sensitivity and agoraphobia; thus, the level of perceived threat control dictates the degree to which anxiety sensitivity results in agoraphobia. Another recently-identified moderator of panic disorder is genetic variations in the gene coding for galanin; these genetic variations moderate the relationship between females suffering from panic disorder and the level of severity of panic disorder symptomatology.

Substance abuse and panic disorder

A growing body of evidence exists that shows a link between substance abuse and panic disorder.
Smoking

Several studies have found that cigarette smoking increases the risk of panic attacks and panic disorder in young people. While the mechanism of how smoking increases panic attacks is not fully understood, a few hypotheses have been derived. Smoking cigarettes may lead to panic attacks by causing changes in respiratory function (e.g. feeling short of breath). These respiratory changes in turn can lead to the formation of panic attacks, as respiratory symptoms are a prominent feature of panic. Respiratory abnormalities have been found in children with high levels of anxiety, which suggests that a person with these difficulties may be susceptible to panic attacks, and thus more likely to subsequently develop panic disorder. Nicotine, a stimulant, could contribute to panic attacks. However, nicotine withdrawal may also cause significant anxiety which could contribute to panic attacks.

Alcohol and sedatives

About 30% of people with panic disorder use alcohol and 17% use other psychoactive drugs. This is in comparison with 61% (alcohol) and 7.9% (other psychoactive drugs) of the general population who use alcohol and psychoactive drugs, respectively. Utilization of recreational drugs or alcohol generally make symptoms worse. Most stimulant drugs (caffeine, nicotine, cocaine) would be expected to worsen the condition, since they directly increase the symptoms of panic, such as heart rate.

Deacon and Valentiner (2000) conducted a study that examined co-morbid panic attacks and substance use in a non-clinical sample of young adults who experienced regular panic attacks. The authors found that compared to healthy controls, therapeutic alcohol and sedative use was greater for non-clinical participants who experienced panic attacks. These findings are consistent with the suggestion made by Cox, Norton, Dorward, and Fergusson (1989) that panic disorder patients self-medicate if they believe that certain substances will be successful in alleviating their symptoms. If panic disorder patients are indeed self-medicating, there may be a portion of the population with undiagnosed panic disorder who will not seek professional help as a result of their own self-medication. In fact, for some patients panic disorder is only diagnosed after they seek treatment for their self-medication habit.

While alcohol initially helps ease panic disorder symptoms, medium- or long-term alcohol abuse can cause panic disorder to develop or worsen during alcohol intoxication, especially during alcohol withdrawal syndrome. This effect is not unique to alcohol but can also occur with long term use of drugs which have a similar mechanism of action to alcohol such as the benzodiazepines which are sometimes prescribed as tranquilizers to people with alcohol problems. The reason chronic alcohol misuse worsens panic disorder is due to distortion of the brain chemistry and function.

Approximately 10% of patients will experience notable protracted withdrawal symptoms, which can include panic disorder, after discontinuation of benzodiazepines. Protracted
withdrawal symptoms tend to resemble those seen during the first couple of months of withdrawal but usually are of a subacute level of severity compared to the symptoms seen during the first 2 or 3 months of withdrawal. It is not known definitively whether such symptoms persisting long after withdrawal are related to true pharmacological withdrawal or whether they are due to structural neuronal damage as result of chronic use of benzodiazepines or withdrawal. Nevertheless such symptoms do typically lessen as the months and years go by eventually disappearing altogether.

A significant proportion of patients attending mental health services for conditions including anxiety disorders such as panic disorder or social phobia have developed these conditions as a result of alcohol or sedative abuse. Anxiety may pre-exist alcohol or sedative independence, which then acts to perpetuate or worsen the underlying anxiety disorder. Someone suffering the toxic effects of alcohol abuse or chronic sedative use or abuse will not benefit from other therapies or medications for underlying psychiatric conditions. as they do not address the root cause of the symptoms. Recovery from sedative Symptoms may temporarily worsen during alcohol withdrawal or benzodiazepine withdrawal. The World Council of Anxiety does not recommend benzodiazepines for the long term treatment of anxiety disorders due to a range of problems associated with long term use of benzodiazepines including tolerance, psychomotor impairment, cognitive and memory impairments, physical dependence and a benzodiazepine withdrawal syndrome upon discontinuation of benzodiazepines.

Panic Attack DSM Disorder Criteria Summary include: Palpitations, pounding heart, or accelerated heart rate, sweating, trembling or shaking. Sensations of shortness of breath or smothering, feeling of choking, chest pain or discomfort, Nausea or abdominal distress, Feeling dizzy, unsteady, lightheaded, faint, derealization, fear of losing control or going crazy, fear of dying, paresthesias, chills or hot flushes. This is DSM Disorder Criteria Summary for panic attacks.

Diagnosis

The DSM-IV-TR diagnostic criteria for panic disorder require unexpected, recurrent panic attacks, followed in at least once instance by at least a month of a significant and related behavior change, a persistent concern of more attacks, or a worry about the attack's consequences. There are two types, one with and one without agoraphobia. Diagnosis is excluded by attacks due to a drug or medical condition, or by panic attacks that are better accounted for by other mental disorders.

Treatment

Identification of treatments that engender as full a response as possible, and can minimize relapse, is imperative. Cognitive behavioural therapy is the treatment of choice for panic disorder. When cognitive behavioural therapy is not an option pharmacotherapy can be used. SSRIs are considered a first line pharmacotherapeutic option.
In addition, people with panic disorder may need treatment for other emotional problems. Comorbid clinical depression, personality disorders and alcohol abuse are known risk factors for treatment failure.

As with many disorders, having a support structure of family and friends who understand the condition can help increase the rate of recovery. During an attack, it is not uncommon for the sufferer to develop irrational, immediate fear, which can often be dispelled by a supporter who is familiar with the condition. For more serious or active treatment, there are support groups for anxiety sufferers which can help people understand and deal with the disorder.

Current treatment guidelines American Psychiatric Association and the American Medical Association primarily recommend either cognitive-behavioral therapy or one of a variety of psychopharmacological interventions. Some evidence exists supporting the superiority of combined treatment approaches.

Treatment can bring significant relief to 70 percent to 90 percent of people with panic disorder, and early treatment can help keep the disease from progressing to the later stages where agoraphobia develops.

Before undergoing any treatment for panic disorder, a person should undergo a thorough medical examination to rule out other possible causes of the distressing symptoms. This is necessary because a number of other conditions, such as excessive levels of thyroid hormone, certain types of epilepsy, or cardiac arrhythmias, which are disturbances in the rhythm of the heartbeat, can cause symptoms resembling those of panic disorder.

Several effective treatments have been developed for panic disorder and agoraphobia. In 1991, a conference held at the National Institutes of Health (NIH) under the sponsorship of the National Institute of Mental Health and the Office of Medical Applications of Research, surveyed the available information on panic disorder and its treatment. The conferees concluded that a form of psychotherapy called cognitive-behavioral therapy and medications are both effective for panic disorder. A treatment should be selected according to the individual needs and preferences of the patient, the panel said, and any treatment that fails to produce an effect within six to eight weeks should be reassessed.

**Cognitive-Behavioral Therapy**

This is a combination of cognitive therapy, which can modify or eliminate thought patterns contributing to the patient’s symptoms, and behavioral therapy, which aims to help the patient to change his or her behavior.

Typically the patient undergoing cognitive-behavioral therapy meets with a therapist for one to three hours a week. In the cognitive portion of the therapy, the therapist usually conducts a careful search for the thoughts and feelings that accompany the panic attacks. These mental events are discussed in terms of the “cognitive model” of panic attacks.
The cognitive model states that individuals with panic disorder often have distortions in their thinking, of which they may be unaware, and these may give rise to a cycle of fear. The cycle is believed to operate this way: First the individual feels a potentially worrisome sensation such as an increasing heart rate, tightened chest muscles, or a queasy stomach. This sensation may be triggered by some worry, an unpleasant mental image, a minor illness, or even exercise. The person with panic disorder responds to the sensation by becoming anxious. The initial anxiety triggers still more unpleasant sensations, which in turn heighten anxiety, giving rise to catastrophic thoughts. The person thinks, “I am having a heart attack” or “I am going insane,” or some similar thought. As the vicious cycle continues, a panic attack results. The whole cycle might take only a few seconds, and the individual may not be aware of the initial sensations or thoughts.

Proponents of this theory point out that, with the help of a skilled therapist, people with panic disorder often can learn to recognize the earliest thoughts and feelings in this sequence and modify their responses to them. Patients are taught that typical thoughts such as “That terrible feeling is getting worse!” or “I’m going to have a panic attack” or “I’m going to have a heart attack” can be replaced with substitutes such as “It’s only uneasiness; it will pass” that help to reduce anxiety and ward off a panic attack. Specific procedures for accomplishing this are taught. By modifying thought patterns in this way, the patient gains more control over the problem.

In cognitive therapy, discussions between the patient and the therapist are not usually focused on the patient’s past, as is the case with some forms of psychotherapy. Instead, conversations focus on the difficulties and successes the patient is having at the present time, and on skills the patient needs to learn.

The behavioral portion of cognitive-behavioral therapy may involve systematic training in relaxation techniques. By learning to relax, the patient may acquire the ability to reduce generalized anxiety and stress that often sets the stage for panic attacks.

Breathing exercises often are included in the behavioral therapy. The patient learns to control his or her breathing and avoid hyperventilation—a pattern of rapid, shallow breathing that can trigger or exacerbate some people’s panic attacks.

Another important aspect of behavioral therapy is exposure to internal sensations called interoceptive exposure. During interoceptive exposure the therapist will do an individual assessment of internal sensations associated with panic. Depending on the assessment, the therapist may then encourage the patient to bring on some of the sensations of a panic attack by, for example, exercising to increase heart rate, breathing rapidly to trigger lightheadedness and respiratory symptoms, or spinning around to trigger dizziness. Exercises to produce feelings of unreality may also be used. Then the therapist teaches the patient to cope effectively with these sensations and to replace alarmist thoughts such as “I am going to die,” with more appropriate ones, such as “It’s just a little dizziness; I can handle it.”
Another important aspect of behavioral therapy is “in vivo” or real-life exposure. The therapist and the patient determine whether the patient has been avoiding particular places and situations, and which patterns of avoidance are causing the patient problems. They agree to work on the avoidance behaviors that are most seriously interfering with the patient’s life. For example, fear of driving may be of paramount importance for one patient, while inability to go to the grocery store may be most handicapping for another.

Some therapists will go to an agoraphobic patient’s home to conduct the initial sessions. Often therapists take their patients on excursions to shopping malls and other places the patients have been avoiding. Or they may accompany their patients who are trying to overcome fear of driving a car.

The patient approaches a feared situation gradually, attempting to stay in spite of rising levels of anxiety. In this way the patient sees that as frightening as the feelings are, they are not dangerous, and they do pass. On each attempt, the patient faces as much fear as he or she can stand. Patients find that with this step-by-step approach, aided by encouragement and skilled advice from the therapist, they can gradually master their fears and enter situations that had seemed unapproachable.

Many therapists assign the patient “homework” to do between sessions. Sometimes patients spend only a few sessions in one-on-one contact with a therapist and continue to work on their own with the aid of a printed manual.

Often the patient will join a therapy group with others striving to overcome panic disorder or phobias, meeting with them weekly to discuss progress, exchange encouragement, and receive guidance from the therapist.

Cognitive-behavioral therapy generally requires at least eight to 12 weeks. Some people may need a longer time in treatment to learn and implement the skills. This kind of therapy, which is reported to have a low relapse rate, is effective in eliminating panic attacks or reducing their frequency. It also reduces anticipatory anxiety and the avoidance of feared situations.

**Treatment with Medications**

In this treatment approach, which is also called pharmacotherapy, a prescription medication is used both to prevent panic attacks or reduce their frequency and severity, and to decrease the associated anticipatory anxiety. When patients find that their panic attacks are less frequent and severe, they are increasingly able to venture into situations that had been off-limits to them. In this way, they benefit from exposure to previously feared situations as well as from the medication.

The selective serotonin reuptake inhibitors (SSRIs) are now the first line of medication treatment for panic disorder. Other commonly used medications are the tricyclic antidepressants, the high-potency benzodiazepines, and the monoamine oxidase inhibitors.
(MAOIs). Determination of which drug to use is based on considerations of safety, efficacy, and the personal needs and preferences of the patient.

Scientists supported by NIMH are seeking ways to improve drug treatment for panic disorder. Studies are underway to determine the optimal duration of treatment with medications, who they are most likely to help, and how to moderate problems associated with withdrawal.

Appropriate medication is highly effective for panic disorder. Although there is little evidence that pharmacological interventions can directly alter phobias, few studies have been performed, and medication treatment of panic makes phobia treatment far easier. Medications can include:

**Antidepressants (SSRIs, MAOIs, tricyclic antidepressants):** these are taken regularly every day, and alter neurotransmitter configurations which in turn can help to block symptoms. Although these medications are described as "antidepressants", nearly all of them — especially the tricyclic antidepressants — have anti-anxiety properties, in part, due to their sedative effects. SSRIs have been known to exacerbate symptoms in panic disorder patients, especially in the beginning of treatment and have even provoked panic attacks in otherwise healthy individuals. SSRIs are also known to produce withdrawal symptoms which include rebound anxiety and panic attacks. Comorbid depression has been cited as imparting the worst course, leading to chronic, disabling illness.

**Anti-anxiety drugs (benzodiazepines):** Use of benzodiazepines for panic disorder is controversial with opinion differing in the medical literature. The American Psychiatric Association states that benzodiazepines can be effective for the treatment of panic disorder and recommends that the choice of whether to use benzodiazepines, antidepressants with antipanic properties or psychotherapy should be based on the individual patient’s history and characteristics. They reported that in their view there is insufficient evidence to recommend one treatment over another for panic disorder. The APA noted that while benzodiazepines have the advantage of a rapid onset of action, that this is offset by the risk of developing a benzodiazepine dependence. The National Institute of Clinical Excellence came to a different conclusion, they pointed out the problems of using uncontrolled clinical trials to assess the effectiveness of pharmacotherapy and based on placebo controlled research they concluded that benzodiazepines were not effective in the long-term for panic disorder and recommended that benzodiazepines not be used for longer than 4 weeks for panic disorder. Instead NICE clinical guidelines recommend alternative pharmacotherapeutic or psychotherapeutic interventions. Other experts believe that benzodiazepines are best avoided due to the risks of the development of tolerance and physical dependence. The World Federation of Societies of Biological Psychiatry, say that benzodiazepines should not be used as a first line treatment option but are an option for treatment resistant cases of panic disorder. Despite increasing focus on the use of antidepressants and other agents for the treatment of anxiety as recommended best practice, benzodiazepines have remained a commonly used medication for panic disorder.
Combination Treatments

Many believe that a combination of medication and cognitive-behavioral therapy represents the best alternative for the treatment of panic disorder. The combined approach is said to offer rapid relief, high effectiveness, and a low relapse rate. However, there is a need for more research studies to determine whether this is in fact the case.

Comparing medications and psychological treatments, and determining how well they work in combination, is the goal of several NIMH-supported studies. The largest of these is a 5-year clinical trial that will include 480 patients and involve four centers at the State University of New York at Albany, Cornell University, Hillside Hospital/Columbia University, and Yale University. This study is designed to determine how treatment with imipramine compares with a cognitive-behavioral approach, and whether combining the two yields benefits over either method alone.

Psychodynamic Treatment

This is a form of “talk therapy” in which the therapist and the patient, working together, seek to uncover emotional conflicts that may underlie the patient’s problems.

Although psychodynamic approaches may help to relieve the stress that contributes to panic attacks, they do not seem to stop the attacks directly. In fact, there is no scientific evidence that this form of therapy by itself is effective in helping people to overcome panic disorder or agoraphobia. However, if a patient's panic disorder occurs along with some broader and pre-existing emotional disturbance, psychodynamic treatment may be a helpful addition to the overall treatment program.

Psychotherapy

Panic Disorder is not the same as phobic symptoms, although phobias commonly result from panic disorder. CBT and one tested form of psychodynamic psychotherapy have been shown efficacious in treating panic disorder with and without agoraphobia. A number of randomized clinical trials have shown that CBT achieves reported panic-free status in 70-90% of patients.

For children with panic disorder, CBT is the only scientifically-proven psychotherapeutic treatment. The methods used are similar to those used with adults, but may be modified to be developmentally-appropriate "Evidence-Based Treatment for Anxiety".

Clinically, a combination of psychotherapy and medication can often produce good results, although research evidence of this approach has been less robust. Some improvement may be noticed in a fairly short period of time — about 6 to 8 weeks. Psychotherapy can improve the effectiveness of medication, reduce the likelihood of relapse for someone who has discontinued medication, and offer help for people with panic disorder who do not respond at all to medication.
The goal of cognitive behavior therapy is to help a patient reorganize thinking processes and anxious thoughts regarding an experience that provokes panic. An approach that proved successful for 87% of patients in a controlled trial is interoceptive therapy, which simulates the symptoms of panic to allow patients to experience them in a controlled environment.

Symptom inductions generally occur for one minute and may include:

- Intentional hyperventilation – creates lightheadedness, derealization, blurred vision, dizziness
- Spinning in a chair – creates dizziness, disorientation
- Straw breathing – creates dyspnea, airway constriction
- Breath holding – creates sensation of being out of breath
- Running in place – creates increased heart rate, respiration, perspiration
- Body tensing – creates feelings of being tense and vigilant

The key to the induction is that the exercises should mimic the most frightening symptoms of a panic attack. Symptom inductions should be repeated three to five times per day until the patient has little to no anxiety in relation to the symptoms that were induced. Often it will take a period of weeks for the afflicted to feel no anxiety in relation to the induced symptoms. With repeated trials, a person learns through experience that these internal sensations do not need to be feared and becomes less sensitized or desensitized to the internal sensation. After repeated trials, when nothing catastrophic happens, the brain learns (hippocampus & amygdala) to not fear the sensations, and the sympathetic nervous system activation fades.

For patients whose panic disorder involves agoraphobia, the traditional cognitive therapy approach has been in vivo exposure, in which the affected individual, accompanied by a therapist, is gradually exposed to the actual situation that provokes panic.

Another form of psychotherapy which has shown effectiveness in controlled clinical trials is panic-focused psychodynamic psychotherapy, which focuses on the role of dependency, separation anxiety, and anger in causing panic disorder. The underlying theory posits that due to biochemical vulnerability, traumatic early experiences, or both, people with panic disorder have a fearful dependence on others for their sense of security, which leads to separation anxiety and defensive anger. Therapy involves first exploring the stressors that lead to panic episodes, then probing the psychodynamics of the conflicts underlying panic disorder and the defense mechanisms that contribute to the attacks, with attention to transference and separation anxiety issues implicated in the therapist-patient relationship.

Comparative clinical studies suggest that muscle relaxation techniques and breathing exercises are not efficacious in reducing panic attacks. In fact, breathing exercises may actually increase the risk of relapse.

Appropriate treatment by an experienced professional can prevent panic attacks or at least substantially reduce their severity and frequency — bringing significant relief to percent of
people with panic disorder. Relapses may occur, but they can often be effectively treated just like the initial episode.

**Epidemiology**

Panic disorder is a serious health problem that in many cases can be successfully treated, although there is no known cure. It typically strikes in early adulthood; roughly half of all people who have panic disorder develop the condition before age 24, especially if the person has been subjected to a traumatic experience. However, some sources say that the majority of young people affected for the first time are between the ages of 25 and 30. Women are twice as likely as men to develop panic disorder.

Panic disorder can continue for months or even years, depending on how and when treatment is sought. If left untreated, it may worsen to the point where the person’s life is seriously affected by panic attacks and by attempts to avoid or conceal the condition. In fact, many people have had problems with friends and family or employment while struggling to cope with panic disorder. Some people with panic disorder may begin to lie to conceal their condition, because of the stigma of mental illness. In some individuals, symptoms may occur frequently for a period of months or years, then many years may pass symptom-free. In others, the symptoms persist at the same level indefinitely. There is also some evidence that many individuals (especially those who develop symptoms at an early age) may experience a cessation of symptoms naturally later in life (i.e. past age 50).

**Panic disorder in children**

A retrospective study has shown that 40% of adult panic disorder patients reported that their disorder began before the age of 20. In an article examining the phenomenon of panic disorder in youth, Diler et al. (2004) found that only a few past studies have examined the occurrence of juvenile panic disorder. They report that these studies have found that the symptoms of juvenile panic disorder almost replicate those found in adults (e.g. heart palpitations, sweating, trembling, hot flashes, nausea, abdominal distress, and chills). The anxiety disorders co-exist with staggeringly high numbers of other mental disorders in adults. The same comorbid disorders that are seen in adults are also reported in children with juvenile panic disorder. Last and Strauss (1989) examined a sample of 17 adolescents with panic disorder and found high rates of comorbid anxiety disorders, major depressive disorder, and conduct disorders. Eassau et al. (1999) also found a high number of comorbid disorders in a community-based sample of adolescents with panic attacks or juvenile panic disorder. Within the sample, adolescents were found to have the following comorbid disorders: major depressive disorder (80%), dysthymic disorder (40%), generalized anxiety disorder (40%), somatoform disorders (40%), substance abuse (40%), and specific phobia (20%). Consistent with this previous work, Diler et al. (2004) found similar results in their study in which 42 youths with juvenile panic disorder were examined. Compared to non-panic anxiety disordered youths, children with panic disorder had higher rates of comorbid major depressive disorder and bipolar disorder.
Despite the evidence pointing to the existence of early-onset panic disorder, the DSM-IV-TR currently only recognizes six anxiety disorders in children: separation anxiety disorder, generalized anxiety disorder, specific phobia, obsessive-compulsive disorder, social anxiety disorder (a.k.a. social phobia), and post-traumatic stress disorder. Panic disorder is notably excluded from this list.

**Phobia**

A phobia (from the Greek: φόβος, Phóbos, meaning "fear" or "morbid fear") is a type of anxiety disorder, usually defined as a persistent fear of an object or situation in which the sufferer commits to great lengths in avoiding despite the fear, typically disproportional to the actual danger posed, often being recognized as irrational. In the event, the phobia cannot be avoided entirely, as the sufferer will endure the situation or object with marked distress and significant interference in social or occupational activities. The terms distress and impairment as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR) should also take into account the context of the sufferer's environment if attempting a diagnosis. The DSM-IV-TR states that if a phobic stimulus, whether it be an object or a social situation, is absent entirely in an environment - a diagnosis cannot be made. An example of this situation would be an individual who has a fear of mice (Suriphobia) but lives in an area devoid of mice. Even though the concept of mice causes marked distress and impairment within the individual, because the individual does not encounter mice in the environment no actual distress or impairment is ever experienced. Proximity and the degree to which escape from the phobic stimulus should also be considered. As the sufferer approaches a phobic stimulus, anxiety levels increase (e.g. as one gets closer to a snake, fear increases in Ophidiophobia), and the degree to which escape of the phobic stimulus is limited and has the effect of varying the intensity of fear in instances such as riding an elevator (e.g. anxiety increases at the midway point between floors and decreases when the floor is reached and the doors open). Finally, a point warranting clarification is that the term phobia is an encompassing term and when discussed is usually done in terms of specific phobias and social phobias. Specific phobias are nouns such as arachnophobia or acrophobia which, as the name implies, are specific, and social phobia are phobias within social situations such as public speaking and crowded areas.

**Clinical phobias**

Psychologists and psychiatrists classify most phobias into three categories and, according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), such phobias are considered to be sub-types of anxiety disorder. The three categories are:

- Social phobia- fears involving other people or social situations such as performance anxiety or fears of embarrassment by scrutiny of others, such as eating in public. Overcoming social phobia is often very difficult without the help of therapy or support groups. Social phobia may be further subdivided into
- generalized social phobia (also known as social anxiety disorder or simply social anxiety) and
- specific social phobia, in which anxiety is triggered only in specific situations. The symptoms may extend to psychosomatic manifestation of physical problems. For example, sufferers of paruresis find it difficult or impossible to urinate in reduced levels of privacy. This goes far beyond mere preference: when the condition triggers, the person physically cannot empty their bladder.
- Specific phobias - fear of a single specific panic trigger such as spiders, snakes, dogs, water, heights, flying, catching a specific illness, etc. Many people have these fears but to a lesser degree than those who suffer from specific phobias. People with the phobias specifically avoid the entity they fear.
- Agoraphobia - a generalized fear of leaving home or a small familiar 'safe' area, and of possible panic attacks that might follow. It may also be caused by various specific phobias such as fear of open spaces, social embarrassment (social agoraphobia), fear of contamination (fear of germs, possibly complicated by obsessive-compulsive disorder) or PTSD (post traumatic stress disorder) related to a trauma that occurred out of doors.

Phobias vary in severity among individuals. Some individuals can simply avoid the subject of their fear and suffer relatively mild anxiety over that fear. Others suffer full-fledged panic attacks with all the associated disabling symptoms. Most individuals understand that they are suffering from an irrational fear, but they are powerless to override their initial panic reaction.

**Specific phobias**

As briefly mentioned above, a specific phobia is a marked and persistent fear of an object or situation which brings about an excessive or unreasonable fear when in the presence of, or anticipating, a specific object; furthermore, the specific phobias may also include concerns with losing control, panicking, and fainting which is the direct result of an encounter with the phobia. The important distinction from social phobias are specific phobias are defined in regards to objects or situations whereas social phobias emphasizes more on social fear and the evaluations that might accompany them.

**Diagnosis**

The diagnostic criteria for 300.29 Specific Phobias as outlined by the DSM-IV-TR:

- Marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation (e.g., flying, heights, animals, receiving an injection, seeing blood).
- Exposure to the phobic stimulus almost invariably provokes an immediate anxiety response, which may take the form of a situationally bound or situationally predisposed panic attack. Note: In children, the anxiety may be expressed by crying, tantrums, freezing, or clinging.
The person recognizes that the fear is excessive or unreasonable. Note: In children, this feature may be absent.

- The phobic situation(s) is avoided or else is endured with intense anxiety or distress.
- The avoidance, anxious anticipation or distress in the feared situation(s) interferes significantly with the person’s normal routine, occupational (or academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.
- In individuals under the age of 18, the duration is at least 6 months.

- The anxiety, panic attack, or phobic avoidance associated with the specific object or situation are not better accounted for by another mental disorder, such as Obsessive-Compulsive Disorder (e.g., fear of dirt in someone with an obsession about contamination), Posttraumatic Stress Disorder (e.g., avoidance of stimuli associated with a severe stressor), Separation Anxiety Disorder (e.g., avoidance of school), Social Phobia (e.g., avoidance of social situations because of fear of embarrassment), Panic Disorder With Agoraphobia, or Agoraphobia Without History of Panic Disorder.

### Social phobia

The key difference between specific phobias and social phobias is social phobias include fear of public situations and scrutiny which leads to embarrassment or humiliation in the diagnostic criteria. In social phobias, there is also a generalized category which is included as a specifier below.

### Diagnosis

The diagnostic criteria for 300.23 Social Phobia as outlined by the DSM-IV-TR:

- A marked and persistent fear of one or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. The individual fears that he or she will act in a way (or show anxiety symptoms) that will be humiliating or embarrassing. Note: In children there must be evidence of the capacity for age-appropriate social relationships with familiar people and the anxiety must occur in peer settings, not just in interactions with adults.
- Exposure to the feared social situation almost invariably provokes anxiety, which may take the form of a situationally bound or situationally predisposed Panic Attack. Note: In children the anxiety may be expressed by crying, tantrums, freezing, or shrinking from social situations with unfamiliar people.
- The person recognized that the fear is excessive or unreasonable. Note: In children this feature may be absent.
- The feared social or performance situations are avoided or else are endured with intense anxiety or distress.
- The avoidance, anxious anticipation, or distress in the feared social or performance situation(s) interferes significantly with the person’s normal routine, occupational
(academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.

- In individuals under age 18, the duration is at least 6 months.
- The fear of avoidance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition and is not better accounted for by another mental disorder (e.g., Panic Disorder With or Without Agoraphobia, Separation Anxiety Disorder, Body Dysmorphic Disorder, a Pervasive Developmental Disorder, Schizoid Personality Disorder).
- If a general medical condition or another mental disorder is present, the fear in Criterion A (Exposure to the social or performance situation almost invariably provokes an immediate anxiety response) is unrelated to it, e.g., the fear is not of Stuttering, trembling in Parkinson’s disease, or exhibiting abnormal eating behavior in Anorexia Nervosa or Bulimia Nervosa.

Specify if:

- Generalized: if the fears include most social situations (also consider the additional diagnosis of Avoidant Personality Disorder).

Notice the severe overlap between specific and social phobias which is indicative of the nature between the two. The differences from specific phobias unanimously lay only in the word “social”.

**Etiology**

**Environmental**

Much of the progress in understanding the acquisition of fear responses in phobias can be attributed to the Pavlovian Model which is synonymous with Classical Conditioning. Myers and Davis (2007) describe the acquisition of fear as when a conditioned stimulus (e.g., a distinctive place) is paired with an aversive unconditioned stimulus (e.g., a electric shock) to an end result in which the subject exhibits a conditioned feared response to the distinctive place (CS+UCS=CR). For how this model works in the context of phobias, one simply has to look at the fear of heights, or acrophobia. In this phobia, the CS is heights such as the top floors of a high rise building or a roller coaster. The UCS can be said to originate from an aversive or traumatizing event in the person’s life such as being trapped on a roller coaster as a child or in an elevator at the top floor of a building. The result of combining these two stimuli leads to a new association called the CR (fear of heights) which is simply the CS (heights) transformed by the aversive UCS (being trapped on a roller coaster or elevator) leading to the feared conditioned response. This model does not suggest that once you have a conditioned feared response to an object or situation you have a phobia. As listed above, to meet the criteria for being diagnosed with a phobia one also has to show symptoms of impairment and avoidance. In the example above, for the CR to be classified as a phobia it would have to exhibit signs of impairment due to avoidance. Impairment, which can be considered along the same lines as a disability from a clinician’s standpoint, is defined as being unable to complete tasks in one’s daily life whether it be occupational,
academical, or social. In the recent example, an impairment of occupation could result from not taking on a job solely because its location happens to be at the top floor of a building, or socially not participating in a social event at a theme park. The avoidance aspect is defined as behavior that results in the omission of an aversive event that would otherwise occur with the goal of the preventing anxiety. The above direct conditioning model, though very influential in the theory of fear acquisition, should not suggest the only way to acquire a phobia. Rachman proposed three main pathways to acquire fear conditioning involving direct conditioning, vicarious acquisition and informational/instructional acquisition.

As experimentation with the aforementioned direct conditioning modeling continued, it became increasingly evident that more that just classical conditioning can influence the onset of a phobia. Rachman (1978) proposed that vicarious acquisition was a critical component to the etiology of phobias, so it was decided to include information and instruction from the parent and family members to better understand its onset. Of the research conducted in this area, one of the best examples of how vicarious conditioning, more specifically modeling, effects the acquisition of a phobia can be said to have come from Cook & Mineka’s (1989) work on rhesus monkeys. In this experiment, Cook & Mineka, through the use of video, appraised 22 rhesus monkeys on their fear to evolutionary relevant stimuli (e.g. crocodiles and snakes), and evolutionary irrelevant stimuli (e.g. flowers and artificial rabbits) to see if fear conditioning using the direct conditioning model (Pavlov's model) leads to fear acquisition (or more specifically the conditioned fear response). The results of the research showed that after 12 sessions the rhesus monkeys acquired a fear to the evolutionary relevant stimuli and not to the evolutionary irrelevant stimuli; furthermore, the experiment also revealed that when they exposed monkeys to other monkeys that interacted with snakes without showing fear, this group did not acquire the fear which supports the theory of vicarious conditioning through modeling. According to Pavlov’s theory of classical conditioning, the experimenters should have been able to condition a feared response within the rhesus monkeys to the evolutionary irrelevant stimuli because the Pavlovian model posits that any UCS can elicit a CR. The result show the necessary augmentation of the Pavlov model with the vicarious acquisition model.

**Neurobiology**

Phobias are generally caused by an event recorded by the amygdala and hippocampus and labeled as deadly or dangerous; thus whenever a specific situation is approached again the body reacts as if the event were happening repeatedly afterward. Treatment comes in some way or another as a replacing of the memory and reaction to the previous event perceived as deadly with something more realistic and based more rationally. In reality most phobias are irrational, in that the subconscious association causes far more fear than is warranted based on the actual danger of the stimulus; a person with a phobia of water may admit that their physiological arousal is irrational and over-reactive, but this alone does not cure the phobia.
Phobias are more often than not linked to the amygdala, an area of the brain located behind the pituitary gland in the limbic lobe. The amygdala may trigger secretion of hormones that affect fear and aggression. When the fear or aggression response is initiated, the amygdala may trigger the release of hormones into the body to put the human body into an "alert" state, in which they are ready to move, run, fight, etc. This defensive "alert" state and response is generally referred to in psychology as the fight-or-flight response.

**Treatments**

Various methods are claimed to treat phobias. Their proposed benefits may vary from person to person.

Some therapists use virtual reality or imagery exercise to desensitize patients to the feared entity. These are parts of systematic desensitization therapy.

Cognitive behavioral therapy (CBT) can be beneficial. Cognitive behavioral therapy allows the patient to challenge dysfunctional thoughts or beliefs by being mindful of their own feelings with the aim that the patient will realize their fear is irrational. CBT may be conducted in a group setting. Gradual desensitisation treatment and CBT are often successful, provided the patient is willing to endure some discomfort. In one clinical trial, 90% of patients were observed with no longer having a phobic reaction after successful CBT treatment.
Eye Movement Desensitization and Reprocessing (EMDR) has been demonstrated in peer-reviewed clinical trials to be effective in treating some phobias. Mainly used to treat Post-traumatic stress disorder, EMDR has been demonstrated as effective in easing phobia symptoms following a specific trauma, such as a fear of dogs following a dog bite.

Hypnotherapy coupled with Neuro-linguistic programming can also be used to help remove the associations that trigger a phobic reaction. However, lack of research and scientific testing compromises its status as an effective treatment.

Antidepressant medications such SSRIs, MAOIs may be helpful in some cases of phobia. Benzodiazepines may be useful in acute treatment of severe symptoms but the risk benefit ratio is against their long-term use in phobic disorders.

There are also new pharmacological approaches, which target learning and memory processes that occur during psychotherapy. For example, it has been shown that glucocorticoids can enhance extinction-based psychotherapy.

Emotional Freedom Technique, a psychotherapeutic alternative medicine tool, also considered to be pseudoscience by the mainstream medicine, is allegedly useful.

These treatment options are not mutually exclusive. Often a therapist will suggest multiple treatments.

**Epidemiology**

Phobias are a common form of anxiety disorders. An American study by the National Institute of Mental Health (NIMH) found that between 8.7% and 18.1% of Americans suffer from phobias. Broken down by age and gender, the study found that phobias were the most common mental illness among women in all age groups and the second most common illness among men older than 25.

**Non-psychological conditions**

The word phobia may also signify conditions other than fear. For example, although the term hydrophobia means a fear of water, it may also mean inability to drink water due to an illness, or may be used to describe a chemical compound which repels water. It was also once used as a synonym for rabies, as an aversion to water is one of its symptoms. Likewise, the term photophobia may be used to define a physical complaint (i.e. aversion to light due to inflamed eyes or excessively dilated pupils) and does not necessarily indicate a fear of light.

**Non-clinical uses of the term**

It is possible for an individual to develop a phobia over virtually anything. The name of a phobia generally contains a Greek word for what the patient fears plus the suffix -phobia.
Creating these terms is something of a word game. Few of these terms are found in medical literature. However, this does not necessarily make it a non-psychological condition.

**Terms for prejudice or discrimination**

A number of terms with the suffix -phobia are used non-clinically but have gained public acceptance, though they are often considered buzzwords. Such terms are primarily understood as negative attitudes towards certain categories of people or other things, used in an analogy with the medical usage of the term. Usually these kinds of "phobias" are described as fear, dislike, disapproval, prejudice, hatred, discrimination, or hostility towards the object of the "phobia". Often this attitude is based on prejudices and is a particular case of most xenophobia. These non-clinical phobias are typically used as labels cast on someone by another person or some other group.

Below are some examples:

- **Chemophobia** – prejudice against artificial substances in favour of "natural" substances.
- **Ephebiphobia** – fear or dislike of youth or adolescents.
- **Homophobia** – fear or dislike of homosexuals or homosexuality.
- **Xenophobia** – fear or dislike of strangers or the unknown, sometimes used to describe nationalistic political beliefs and movements. It is also used in fictional work to describe the fear or dislike of space aliens.

**List of phobias**

The English suffixes -phobia, -phobic, -phobe (of Greek origin: φόβος/φοβία ) occur in technical usage in psychiatry to construct words that describe irrational, disabling fear as a mental disorder (e.g., agoraphobia), in chemistry to describe chemical aversions (e.g., hydrophobic), in biology to describe organisms that dislike certain conditions (e.g., acidophobia), and in medicine to describe hypersensitivity to a stimulus, usually sensory (e.g., photophobia). In common usage they also form words that describe dislike or hatred of a particular thing or subject. The suffix is antonymic to -phil-.

The following lists include words ending in -phobia, and include fears that have acquired names. In some cases, the naming of phobias has become a word game, of notable example being a 1998 humorous article published by BBC News.

In some cases a word ending in -phobia may have an antonym with the suffix -phil-, e.g., Germanophobe / Germanophile.

**Phobia lists**

A large number of-phobia lists circulate on the Internet, with words collected from indiscriminate sources, often copying each other. Also, a number of psychiatric websites
exist that at the first glance cover a huge number of phobias, but in fact use a standard text to fit any phobia and reuse it for all unusual phobias by merely changing the name. Sometimes it leads to bizarre results, such as suggestions to cure "prostitute phobia". Such practice is known as content spamming and is used to attract search engines.

**Psychological conditions**

In many cases specialists prefer to avoid the suffix -phobia and use more descriptive terms, see, e.g., personality disorders, anxiety disorders, avoidant personality disorder, love-shyness.

- Ablutophobia – fear of bathing, washing, or cleaning.
- Acrophobia, Altophobia – fear of heights.
- Agoraphobia, Agoraphobia Without History of Panic Disorder – fear of places or events where escape is impossible or when help is unavailable.
- Acousticophobia - fear of sound and voice.
- Agraphobia – fear of sexual abuse.
- Aichmophobia – fear of sharp or pointed objects (such as a needle or knife).
- Agyrophobia – fear of crossing roads.
- Androphobia – fear of men.
- Anthropophobia – fear of people or being in a company, a form of social phobia.
- Anthrophobia – fear of flowers.
- Aquaphobia – fear of water. Distinct from Hydrophobia, a scientific property that makes chemicals averse to interaction with water, as well as an archaic name for rabies.
- Arachnophobia – fear of spiders.
- Astraphobia - fear of thunder and lightning.
- Atychiphobia – fear of failure
- Autophobia – fear of loneliness
- Aviophobia, Aviatophobia – fear of flying.
- Blood-injection-injury type phobia – a DSM-IV subtype of specific phobias.
- Chirotophobia - fear of bats.
- Cibophobia, Sitophobia – aversion to food, synonymous to Anorexia nervosa.
- Claustrophobia – fear of having no escape and being closed in.
- Coulrophobia – fear of clowns (not restricted to evil clowns).
- Decidophobia – fear of making decisions.
- Odontophobia, Dentophobia, Odontophobia – fear of dentists and dental procedures.
- Disposophobia, better known as "compulsive hoarding" – the fear of getting rid of or losing things.
- Dysmorphophobia, or body dysmorphic disorder – a phobic obsession with a real or imaginary body defect.
- Emetophobia – fear of vomiting.
- Ergasiophobia, Ergophobia – fear of work or functioning, or a surgeon’s fear of operating.
- Ergophobia – fear of work or functioning.
- Erotophobia – fear of sexual love or sexual questions.
- Erythrophobia – pathological blushing.
- Friggatriskaidékaphobia - fear of Friday the 13th.
- Gelotophobia – fear of being laughed at.
- Gephyrophobia – fear of bridges.
- Genophobia, Coitophobia – fear of sexual intercourse.
- Gerascophobia – fear of growing old or aging.
- Gerontophobia – fear of growing old, or a hatred or fear of the elderly
- Glossophobia – fear of speaking in public or of trying to speak.
- Gymnophobia – fear of nudity.
- Gynophobia – fear of women.
- Halitophobia - fear of bad breath.
- Haptophobia – fear of being touched.
- Heliophobia – fear of sunlight.
- Hemophobia, Haemophobia – fear of blood.
- Hexakosioihexekontahexaphobia – fear of the number 666.
- Homophobia - fear/hatred of homosexuals
- Hoplophobia – fear of weapons, specifically firearms (Generally a political term but the clinical phobia is also documented).
- Hydrophobia - fear of Rabies (Hydrophobia).
- Hylophobia - fear of trees, forests or wood.
- Ligyrophobia – fear of loud noises.
- Lipophobia – fear/avoidance of fats in food.
- Medication phobia – fear of medications.
- Mysophobia – fear of germs, contamination or dirt.
- Necrophobia – fear of death and, or the dead.
- Neophobia, Cainophobia, Cainotophobia, Cenophobia, Centophobia, Kainophobia, Kinophobia – fear of newness, novelty.
- Nomophobia – fear of being out of mobile phone contact.
- Nosophobia – fear of contracting a disease.
- Nosocomophobia – fear of hospitals.
- Oikophobia – fear of home surroundings and household appliances.
- Osmophobia, Olfactophobia – fear of smells.
- Paraskavedekatriaphobia, Paraskevidekatriaphobia, Friggatriskaidékaphobia – fear of Friday the 13th.
- Panphobia – fear of everything or constant fear of an unknown cause.
- Pediophobia - Fear of dolls
- Phasmophobia – fear of ghosts, spectres or phantasms.
- Phagophobia – fear of swallowing.
- Pharmacophobia – same as medication phobia.
- Philophobia - fear of love
- Phobophobia – fear of having a phobia.
- Phonophobia – fear of loud sounds.
- Porphyrophobia - fear of the color purple.
- Pyrophobia – fear of fire.
- Radiophobia – fear of radioactivity or X-rays.
- Sociophobia – fear of people or social situations.
- Scopophobia – fear of being looked at or stared at.
- Somniphobia – fear of sleep.
- Spectrophobia – fear of mirrors and one’s own reflections.
- Taphophobia – fear of the grave, or fear of being placed in a grave while still alive.
- Technophobia – fear of technology (see also Luddite).
- Telephone phobia – fear or reluctance of making or taking phone calls.
- Tetraphobia – fear of the number 4.
- Thanatophobia – fear of death.
- Tokophobia – fear of childbirth.
- Traumatophobia – a synonym for injury phobia: fear of having an injury.
- Triskaidekaphobia, Terdekaphobia – fear of the number 13.
- Trypanophobia, Belonephobia, Enetophobia – fear of needles or injections.
- Workplace phobia – fear of the workplace.
- Xenophobia – fear of strangers, foreigners, or aliens.
- Xylophobia, Hylophobia, Ylophobia - fear of trees, forests or wood.

**Animal phobias**

- Ailurophobia – fear/dislike of cats.
- Apiphobia – fear/dislike of bees (also known as melissophobia, from the Greek melissa "bee").
- Arachnophobia – fear/dislike of spiders and other arachnids.
- Bovinophobia - fear/dislike of cattle.
- Chiroptophobia – fear/dislike of bats.
- Cynophobia – fear/dislike of dogs.
- Entomophobia – fear/dislike of insects.
- Equinophobia – fear/dislike of horses (also known as hippophobia).
- Herpetophobia - fear/dislike of reptiles and/or amphibians.
- Ichthyophobia – fear/dislike of fish.
- Murophobia – fear/dislike of mice and/or rats.
- Ophidiophobia – fear/dislike of snakes.
- Ornithophobia – fear/dislike of birds.
- Selachophobia - fear of sharks.
- Scoleciphobia – fear of worms.
- Zoophobia - fear of animals.

**Non-psychological conditions**

- Hydrophobia – fear of water (a symptom of rabies).
- Photophobia – hypersensitivity to light causing aversion to light
- Phonophobia – hypersensitivity to sound causing aversion to sounds.
- Osmophobia – hypersensitivity to smells causing aversion to odors.

**Biology, chemistry**
Biologists use a number of -phobia/-phobic terms to describe predispositions by plants and animals against certain conditions. For antonyms, see here.

- Acidophobia/Acidophobic – preference for non-acidic conditions.
- Heliophobia/Heliophobic – aversion to sunlight.
- Hydrophobia/Hydrophobic – a property of being repelled by water.
- Lipophobia/Lipophobic – a property of fat rejection
- Ombrophobia – avoidance of rain
- Photophobia (biology) a negative phototaxis or phototropism response, or a tendency to stay out of the light
- Superhydrophobe – the property given to materials that are extremely difficult to get wet.
- Thermophobia – aversion to heat.

Prejudices and discrimination

The suffix -phobia is used to coin terms that denote a particular anti-ethnic or anti-demographic sentiment, such as Americanophobia, Europhobia, Francophobia, Hispanophobia, and Indophobia. Often a synonym with the prefix "anti-" already exists (e.g., Polonophobia vs. anti-Polonism). Anti-religious sentiments are expressed in terms such as Christianophobia and Islamophobia. Sometimes the terms themselves could even be considered racist, such as with "Negrophobia."

Other prejudices include:

- Biphobia – fear/dislike of bisexuality or bisexuals.
- Christianophobia - fear/dislike of Christians
- Ephebophobia – fear/dislike of youth.
- Gerontophobia, Gerascophobia – fear/dislike of aging or the elderly.
- Heterophobia – fear/dislike of heterosexuals.
- Homophobia – fear/dislike of homosexuality or homosexuals.
- Islamophobia - fear/dislike of Muslims
- Judeophobia – fear/dislike of Jews.
- Lesbophobia – fear/dislike of lesbians.
- Pedophobia, Pediophobia – fear/dislike of children.
- Psychophobia – fear/dislike of mental illness or the mentally ill.
- Sinophobia – fear/dislike of Chinese.
- Transphobia – fear/dislike of transgender or transsexual people.
- Xenophobia – fear/dislike of foreigners.
- Germanophobia - fear/dislike of Germans.

Jocular and fictional phobias

- Aibohphobia – a joke term for the fear of palindromes, which is a palindrome itself. The term is a piece of computer humor entered into the 1981 The Devil’s DP Dictionary
Anachrophobia – fear of temporal displacement, from a Doctor Who novel by Jonathan Morris.

Anatidaephobia – fear that somewhere, somehow, a duck is watching you (Derived from the word Anatidae, which is the family contain ducks, and the suffix -phobia). Comes from Gary Larson’s The Far Side.

Anoraknophobia – a portmanteau of "anorak" and "arachnophobia". Used in the Wallace and Gromit comic book Anoraknophobia. Also the title of an album by Marillion.

Arachibutyrophobia – fear of peanut butter sticking to the roof of the mouth. The word is used by Charles M. Schulz in a 1982 installment of his "Peanuts" comic strip and by Peter O’Donnell in his 1985 Modesty Blaise adventure novel Dead Man’s Handle.

Hippopotomonstrosesquipedaliophobia – fear of long words. Hippopotomo- "big" due to its allusion to the Greek-derived word hippopotamus (though this is derived as hippo- "horse" compounded with potam-os "river", so originally meaning "river horse"; according to the Oxford English, "hippopotamine” has been construed as large since 1847, so this coinage is reasonable); -monstr- is from Latin words meaning "monstrous", -o- is a noun-compounding vowel; -sesquipedali- comes from "sesquipedalian" meaning a long word (literally "a foot and a half long" in Latin), -o- is a noun-compounding vowel, and -phobia means "fear". Note: This was mentioned on the first episode of Brainiac Series Five as one of Tickle’s Teasers.

Keanophobia - fear of Keanu Reeves, portrayed in the Dean Koontz book, False Memory, where a woman has an irrational fear of Keanu Reeves and has to see her psychiatrist, Mark Ahriman, each week. He calls her the "Keanuphobe" in his head. She eventually ends up killing her psychiatrist because she believes that he is one of the Machine agents trying to control her.

Lupopolipophobia - fear of being pursued by timber wolves around a kitchen table while wearing socks on a newly waxed floor, also from Gary Larson’s The Far Side.

Nihilophobia - fear of nothingness (comes from the combination of the Latin word nihil which means nothing, none, and the suffix -phobia), as described by the Doctor in the Star Trek: Voyager episode Night. Voyager’s morale officer and chef Neelix suffers from this condition, having panic attacks while the ship was traversing a dark expanse of space known as the Void. It is also the title of a 2008 album by Neuronium. Also, the animated version of George of the Jungle (2007 TV series) is seen suffering in one episode of the cartoon, where they are telling scary stories.

Venustraphobia – fear of beautiful women, according to a 1998 humorous article published by BBC News. The word is a portmanteau of "Venus trap" and "phobia". Venustraphobia is the title of a 2006 album by Casbah Club.

Monkeyphobia - fear of monkeys, as named by Lord Monkey Fist in the animated series Kim Possible. Due to spending a summer in a cabin with a crazy chimp mascot, Ron Stoppable has a fear of monkeys, which he gets over several times, usually during battles with Monkey Fist, who is essentially Ron’s arch-nemesis.

**Miscellaneous**

- Arachnophobia – "fear/dislike of spiders," a film
Agoraphobia

Agoraphobia (from Greek ἀγορά, "marketplace"; and φόβος/φοβία, -phobia) is an anxiety disorder defined as a morbid fear or having a panic attack or panic-like symptoms in a situation that is perceived to be difficult (or embarrassing) from which to escape. These situations can include, but are not limited to, wide-open spaces, crowds, or uncontrolled social conditions. Alternatively, social anxiety problems may also be an underlying cause. As a result, sufferers of agoraphobia avoid public and/or unfamiliar places, especially large, open spaces such as shopping malls or airports from which they cannot easily escape if they have a panic attack. In severe cases, the sufferer may become confined to his or her home, unable to leave their safe haven.

Although mostly thought to be a fear of public places, it is now believed that agoraphobia develops as a complication of panic attacks. However, there is evidence that the implied one-way causal relationship between spontaneous panic attacks and agoraphobia in DSM-IV may be incorrect. Onset is usually between ages 20 and 40 years and more common in women. Approximately 3.2 million adults in the US between the ages of 18 and 54, or about 2.2%, suffer from agoraphobia. Agoraphobia can account for approximately 60% of phobias. Agoraphobia, as studies have shown, has two age groups at which the first onset generally occurs — early to mid twenties and in the early thirties — thus helping to distinguish between simple phobias in child and adolescent years (Gelder, Mayou & Geddes, 2005).

In response to a traumatic event, anxiety may interrupt the formation of memories and disrupt the learning processes, resulting in dissociation. Depersonalization (a feeling of disconnection from one’s self) and derealisation (a feeling of disconnection from one's surroundings) are other dissociative methods of withdrawing from anxiety.

Definition

Not to be confused with agraphobia, agoraphobia is a condition where the sufferer becomes anxious in environments that are unfamiliar or where he or she perceives that they have little control. Triggers for this anxiety may include wide open spaces, crowds (social anxiety), or traveling (even short distances). Agoraphobia is often, but not always, compounded by a fear of social embarrassment, as the agoraphobic fears the onset of a panic attack and appearing distraught in public. This is also sometimes called 'social agoraphobia' which may be a type of social anxiety disorder also sometimes called "social phobia".
Not all agoraphobia is social in nature, however. Some agoraphobics have a fear of open spaces. Agoraphobia is also a defined as "a fear, sometimes terrifying, by those who have experienced one or more panic attacks". In these cases, the sufferer is fearful of a particular place because they have experienced a panic attack at the same location in a previous time. Fearing the onset of another panic attack, the sufferer is fearful or even avoids the location. Some refuse to leave their home even in medical emergencies because the fear of being outside of their comfort area is too great.

The sufferer can sometimes go to great lengths to avoid the locations where they have experienced the onset of a panic attack. Agoraphobia, as described in this manner, is actually a symptom professionals check for when making a diagnosis of panic disorder. Other syndromes like obsessive compulsive disorder or post traumatic stress disorder can also cause agoraphobia, basically any irrational fear that keeps one from going outside can cause the syndrome.

It is not uncommon for agoraphobics to also suffer from temporary separation anxiety disorder when certain other individuals of the household depart from the residence temporarily, such as a parent or spouse, or when the agoraphobic is left home alone. Such temporary conditions can result in an increase in anxiety or a panic attack.

Another common associative disorder of agoraphobia is necrophobia, the fear of death. The anxiety level of agoraphobics often increases when dwelling upon the idea of eventually dying, which they consciously or unconsciously associate with being the ultimate separation from their mortal emotional comfort and safety zones and loved ones, even for those who may otherwise spiritually believe in some form of divine afterlife existence.

**Gender differences**

Agoraphobia occurs about twice as commonly among women as it does in men. The gender difference may be attributable to several factors: social-cultural traditions that encourage, or permit, the greater expression of avoidant coping strategies by women (including dependent and helpless behaviors); women perhaps being more likely to seek help and therefore be diagnosed; men being more likely to abuse alcohol in reaction to anxiety and be diagnosed as an alcoholic. Research has not yet produced a single clear explanation for the gender difference in agoraphobia.

**Causes and contributing factors**

Although the exact causes of agoraphobia are currently unknown, some clinicians who have treated or attempted to treat agoraphobia offer plausible hypotheses. The condition has been linked to the presence of other anxiety disorders, a stressful environment or substance abuse. Chronic use of tranquilizers and sleeping pills such as benzodiazepines has been linked to onset of agoraphobia. In 10 patients who had developed agoraphobia during benzodiazepine dependence, symptoms abated within the first year of assisted withdrawal.
Research has uncovered a linkage between agoraphobia and difficulties with spatial orientation. Individuals without agoraphobia are able to maintain balance by combining information from their vestibular system, their visual system and their proprioceptive sense. A disproportionate number of agoraphobics have weak vestibular function and consequently rely more on visual or tactile signals. They may become disoriented when visual cues are sparse (as in wide open spaces) or overwhelming (as in crowds). Likewise, they may be confused by sloping or irregular surfaces. In a virtual reality study, agoraphobics showed impaired processing of changing audiovisual data in comparison with healthy subjects.

**Alternate theories**

**Attachment theory**

Some scholars have explained agoraphobia as an attachment deficit, i.e., the temporary loss of the ability to tolerate spatial separations from a secure base. Recent empirical research has also linked attachment and spatial theories of agoraphobia.

**Spatial theory**

In the social sciences there is a perceived clinical bias in agoraphobia research. Branches of the social sciences, especially geography, have increasingly become interested in what may be thought of as a spatial phenomenon. One such approach links the development of agoraphobia with modernity.

**Diagnosis**

Most people who present to mental health specialists develop agoraphobia after the onset of panic disorder (American Psychiatric Association, 1998). Agoraphobia is best understood as an adverse behavioral outcome of repeated panic attacks and subsequent anxiety and preoccupation with these attacks that leads to an avoidance of situations where a panic attack could occur. In rare cases where agoraphobics do not meet the criteria used to diagnose panic disorder, the formal diagnosis of agoraphobia without history of panic disorder is used (primary agoraphobia).

**Association with panic attacks**

Agoraphobia patients can experience sudden panic attacks when traveling to places where they fear they are out of control, help would be difficult to obtain, or they could be embarrassed. During a panic attack, epinephrine is released in large amounts, triggering the body’s natural fight-or-flight response. A panic attack typically has an abrupt onset, building to maximum intensity within 10 to 15 minutes, and rarely lasts longer than 30 minutes. Symptoms of a panic attack include palpitations, a rapid heartbeat, sweating, trembling, nausea, vomiting, dizziness, tightness in the throat and shortness of breath. Many patients report a fear of dying or of losing control of emotions and/or behavior.
Treatments

Cognitive behavioral treatments

Exposure treatment can provide lasting relief to the majority of patients with panic disorder and agoraphobia. Disappearance of residual and subclinical agoraphobic avoidance, and not simply of panic attacks, should be the aim of exposure therapy. Similarly, Systematic desensitization may also be used. Many patients can deal with exposure easier if they are in the company of a friend they can rely on (Gelder, Mayou and Geddes 2005). It is vital that patients remain in the situation until anxiety has abated because if they leave the situation the phobic response will not decrease and it may even rise (Gelder, Mayou and Geddes 2005).

Cognitive restructuring has also proved useful in treating agoraphobia. This treatment involves coaching a participant through a diaoetic discussion, with the intent of substituting irrational, counterproductive beliefs with more factual and beneficial ones.

Relaxation techniques are often useful skills for the agoraphobic to develop, as they can be used to stop or prevent symptoms of anxiety and panic.

Psychopharmaceutical treatments

Anti-depressant medications most commonly used to treat anxiety disorders are mainly in the SSRI (selective serotonin reuptake inhibitor) class and include sertraline, paroxetine and fluoxetine. Benzodiazepine tranquillizers, MAO inhibitors and tricyclic antidepressants are also commonly prescribed for treatment of agoraphobia. Antidepressants are important because some have antipanic effects (Gelder, Mayou and Geddes 2005). Antidepressants should be used in conjunction with exposure as a form of self-help or with cognitive behaviour therapy (Gelder, Mayou and Geddes 2005). Some evidence shows that a combination of medication and cognitive behaviour therapy is the most effective treatment for agoraphobia (Gelder, Mayou and Geddes 2005).

Alternative treatments

Eye movement desensitization and reprogramming (EMDR) has been studied as a possible treatment for agoraphobia, with poor results. As such, EMDR is only recommended in cases where cognitive-behavioral approaches have proven ineffective or in cases where agoraphobia has developed following trauma.

Many people with anxiety disorders benefit from joining a self-help or support group (telephone conference call support groups or online support groups being of particular help for completely housebound individuals). Sharing problems and achievements with others as well as sharing various self-help tools are common activities in these groups. In particular stress management techniques and various kinds of meditation practices as well as visualization techniques can help people with anxiety disorders calm themselves and
may enhance the effects of therapy. So can service to others which can distract from the self-absorption that tends to go with anxiety problems. There is also preliminary evidence that aerobic exercise may have a calming effect. Since caffeine, certain illicit drugs, and even some over-the-counter cold medications can aggravate the symptoms of anxiety disorders, they should be avoided.

**Social anxiety disorder**

Social anxiety disorder (SAD or SANd) (DSM-IV 300.23) is an anxiety disorder characterized by intense fear in social situations causing considerable distress and impaired ability to function in at least some parts of daily life. It is a rather extreme form of social phobia (SP), although the latter is sometimes treated synonymously. The diagnosis of social anxiety disorder can be of a specific disorder (when only some particular situations are feared) or a generalized disorder. Generalized social anxiety disorder typically involves a persistent, intense, chronic fear of being judged by others and of being embarrassed or humiliated by one's own actions. These fears can be triggered by perceived or actual scrutiny from others. While the fear of social interaction may be recognized by the person as excessive or unreasonable, overcoming it can be quite difficult. Physical symptoms often accompanying social anxiety disorder include excessive blushing, sweating (hyperhidrosis), trembling, palpitations, nausea, and stammering often accompanied with rapid speech. Panic attacks may also occur under intense fear and discomfort. An early diagnosis may help minimize the symptoms and the development of additional problems, such as depression. Some sufferers may use alcohol or other drugs to reduce fears and inhibitions at social events. It is common for sufferers of social phobia to self-medicate in this fashion, especially if they are undiagnosed, untreated, or both; this can lead to alcoholism, eating disorders or other kinds of substance abuse.

Standardized rating scales such as Social Phobia Inventory can be used for screening social anxiety disorder and measuring severity of social phobia. A person with the disorder may be treated with psychotherapy, medication, or both. Research has shown cognitive behavior therapy, whether individually or in a group, to be effective in treating social phobia. The cognitive and behavioral components seek to change thought patterns and physical reactions to anxiety-inducing situations. Attention given to social anxiety disorder has significantly increased in the United States since 1999 with the approval and marketing of drugs for its treatment. Prescribed medications include several classes of antidepressants: selective serotonin reuptake inhibitors (SSRIs) such as Zoloft, Prozac, and Paxil; serotonin-norepinephrine reuptake inhibitors (SNRIs); and monoamine oxidase inhibitors (MAOIs). Other commonly used medications include beta-blockers and benzodiazepines, as well as newer antidepressants, such as mirtazapine. A herb called kava has also attracted attention as a possible treatment, although safety concerns exist, especially given the unregulated nature of herbs in the United States.

**History**
Literary descriptions of shyness can be traced back to the days of Hippocrates around 400 B.C. Hippocrates described someone who "through bashfulness, suspicion, and timorousness, will not be seen abroad; loves darkness as life and cannot endure the light or to sit in lightsome places; his hat still in his eyes, he will neither see, nor be seen by his good will. He dare not come in company for fear he should be misused, disgraced, overshoot himself in gesture or speeches, or be sick; he thinks every man observes him."

The first mention of a psychiatric term, social phobia (phobie des situations sociales), was made in the early 1900s. Psychologists used the term "social neurosis" to describe extremely shy patients in the 1930s. After extensive work by Joseph Wolpe on systematic desensitization, research in phobias and their treatment grew. The idea that social phobia was a separate entity from other phobias came from the British psychiatrist Isaac Marks, in the 1960s. This was accepted by the American Psychiatric Association and was first officially included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders. The definition of the phobia was revised in 1989 to allow comorbidity with avoidant personality disorder, and introduced generalized social phobia. Social phobia had been largely ignored prior to 1985.

After a call to action by psychiatrist Michael Liebowitz and clinical psychologist Richard Heimberg, there was an increase in attention to and research on the disorder. The DSM-IV gave social phobia the alternative name social anxiety disorder. Research on the psychology and sociology of everyday social anxiety continued. Cognitive Behavioural models and therapies were developed for social anxiety disorder. In the 1990s, paroxetine became the first prescription drug in the U.S. approved to treat social anxiety disorder, with others following.

Social phobia in many cases can be an extremely debilitating disorder, especially because one who struggles with it often suffers alone.

**Symptoms**

**Cognitive aspects**

In cognitive models of social anxiety disorder, social phobics experience dread over how they will be presented to others. They may be overly self-conscious, pay high self-attention after the activity, or have high performance standards for themselves. According to the social psychology theory of self-presentation, a sufferer attempts to create a well-mannered impression on others but believes he or she is unable to do so. Many times, prior to the potentially anxiety-provoking social situation, sufferers may deliberately go over what could go wrong and how to deal with each unexpected case. After the event, they may have the perception they performed unsatisfactorily. Consequently, they will review anything that may have possibly been abnormal or embarrassing. These thoughts do not just terminate soon after the encounter, but may extend for weeks or longer. Those with social phobia tend to interpret neutral or ambiguous conversations with a negative outlook and many studies suggest that socially anxious individuals remember more negative memories than those less distressed.
An example of an instance may be that of an employee presenting to his co-workers. During the presentation, the person may stutter a word, upon which he or she may worry that other people significantly noticed and think that their perceptions of him or her as a presenter have been tarnished. This cognitive thought propels further anxiety which compounds with further stuttering, sweating, and, potentially, a panic attack.

**Behavioral aspects**

Social anxiety disorder is a persistent fear of one or more situations in which the person is exposed to possible scrutiny by others and fears that he or she may do something or act in a way that will be humiliating or embarrassing. It exceeds normal "shyness" as it leads to excessive social avoidance and substantial social or occupational impairment. Feared activities may include almost any type of social interaction, especially small groups, dating, parties, talking to strangers, restaurants, etc. Possible physical symptoms include "mind going blank", fast heartbeat, blushing, stomach ache, nausea and gagging. Cognitive distortions are a hallmark, and learned about in CBT (cognitive-behavioral therapy). Thoughts are often self-defeating and inaccurate.

According to psychologist B.F. Skinner, phobias are controlled by escape and avoidance behaviors. For instance, a student may leave the room when talking in front of the class (escape) and refrain from doing verbal presentations because of the previously encountered anxiety attack (avoid). Major avoidance behaviors could include an almost pathological/compulsive lying behavior in order to preserve self-image and avoid judgement in front of others. Minor avoidance behaviors are exposed when a person avoids eye contact and crosses arms to avoid recognizable shaking. A fight-or-flight response is then triggered in such events. Preventing these automatic responses is at the core of treatment for social anxiety.

**Physiological aspects**

Physiological effects, similar to those in other anxiety disorders, are present in social phobics. Faced with an uncomfortable situation, children with social anxiety may display tantrums, weeping, clinging to parents, and shutting themselves out. In adults, it may be tears as well as experiencing excessive sweating, nausea, shaking, and palpitations as a result of the fight-or-flight response. The walk disturbance (where a person is so worried about how they walk that they may lose balance and fall) may appear, especially when passing a group of people. Blushing is commonly exhibited by individuals suffering from social phobia. These visible symptoms further reinforce the anxiety in the presence of others. A 2006 study found that the area of the brain called the amygdala, part of the limbic system, is hyperactive when patients are shown threatening faces or confronted with frightening situations. They found that patients with more severe social phobia showed a correlation with the increased response in the amygdala.

**Prevalence**
<table>
<thead>
<tr>
<th>Country</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>United States</td>
<td>2–7%</td>
</tr>
<tr>
<td>England</td>
<td>0.4%</td>
</tr>
<tr>
<td>Scotland</td>
<td>1.8%</td>
</tr>
<tr>
<td>Wales</td>
<td>0.6%</td>
</tr>
<tr>
<td>Australia</td>
<td>1–2.7%</td>
</tr>
<tr>
<td>Brazil</td>
<td>4.7–7.9%</td>
</tr>
</tbody>
</table>

When prevalence estimates were based on the examination of psychiatric clinic samples, social anxiety disorder was thought to be a relatively rare disorder. The opposite was instead true; social anxiety was common but many were afraid to seek psychiatric help, leading to an understatement of the problem. Prevalence rates vary widely because of its vague diagnostic criteria and its overlapping symptoms with other disorders. There has been some debate on how the studies are conducted and whether the illness truly impairs the respondents as laid out in the official criteria. Psychologist Dr. Ray Crozier argues, "it is difficult to ascertain whether the person being interviewed adheres to the DSM-III-R criteria or whether they are merely exhibiting poor social skills or shyness."

The National Comorbidity Survey of over 8,000 American correspondents in 1994 revealed a 12-month and lifetime prevalence rates of 7.9 percent and 13.3 percent making it the third most prevalent psychiatric disorder after depression and alcohol dependence and the most apparent of the anxiety disorders. According to U.S. epidemiological data from the National Institute of Mental Health, social phobia affects 5.3 million adult Americans in any given year. Cross-cultural studies have reached prevalence rates with the conservative rates at 5 percent of the population. However, other estimates vary within 2 percent and 7 percent of the U.S. adult population.

The mean onset of social phobia is 10 to 13 years. Onset after age 25 is rare and is typically preceded by panic disorder or major depression. Social anxiety disorder occurs in females nearly twice as often as males, although men are more likely to seek help. The prevalence of social phobia appears to be increasing among white, married, and well-educated individuals. As a group, those with generalized social phobia are less likely to graduate from high school and are more likely to rely on government financial assistance or have poverty-level salaries. Surveys carried out in 2002 show the youth of England, Scotland, and Wales have a prevalence rate of 0.4 percent, 1.8 percent, and 0.6 percent, respectively. The prevalence of self-reported social anxiety for Nova Scotians older than 14 years was 4.2 percent in June 2004 with women (4.6 percent) reporting more than men (3.8 percent). In Australia, social phobia is the 8th and 5th leading disease or illness for males and females between 15–24 years of age as of 2003. Because of the difficulty in separating
social phobia from poor social skills or shyness, some studies have a large range of prevalence. The table also shows higher prevalence in Brazil.

**Comorbidity**

There is a high degree of comorbidity with other psychiatric disorders. Social phobia often occurs alongside low self-esteem and clinical depression, due to lack of personal relationships and long periods of isolation from avoiding social situations. To try to reduce their anxiety and alleviate depression, people with social phobia may use alcohol or other drugs, which can lead to substance abuse. It is estimated that one-fifth of patients with social anxiety disorder also suffer from alcohol dependence. The most common complementary psychiatric condition is unipolar depression. In a sample of 14,263 people, of the 2.4 percent of persons diagnosed with social phobia, 16.6 percent also met the criteria for clinical depression. Besides depression, the most common disorders diagnosed in patients with social phobia are panic disorder (33 percent), generalized anxiety disorder (19 percent), post-traumatic stress disorder (36 percent), substance abuse disorder (18 percent), and attempted suicide (23 percent). In one study of social anxiety disorder among patients who developed comorbid alcoholism, panic disorder, or depression, social anxiety disorder preceded the onset of alcoholism, panic disorder and depression in 75 percent, 61 percent, and 90 percent of patients, respectively. Avoidant personality disorder is also highly correlated with social phobia. Because of its close relationship and overlapping symptoms with other illnesses, treating social phobics may help understand underlying connection in other psychiatric disorders.

There is research indicating that social anxiety disorder is often correlated with bipolar disorder. Some researchers believe they share an underlying cyclothymic-anxious-sensitive disposition. In addition, studies show that more socially phobic patients treated with antidepressant medication develop hypomania than non-phobic controls. This can be seen as the medication creating a new problem, and also has this adverse effect in a proportion of those without social phobia.

**Causes and perspectives**

Research into the causes of social anxiety and social phobia is wide-ranging, encompassing multiple perspectives from neuroscience to sociology. Scientists have yet to pinpoint the exact causes. Studies suggest that genetics can play a part in combination with environmental factors.

**Genetic and family factors**

It has been shown that there is a two to threefold greater risk of having social phobia if a first-degree relative also has the disorder. This could be due to genetics and/or due to children acquiring social fears and avoidance through processes of observational learning or parental psychosocial education. Studies of identical twins brought up (via adoption) in different families have indicated that, if one twin developed social anxiety disorder, then the other was between 30 percent and 50 percent more likely than average to also develop
the disorder. To some extent this 'heritability' may not be specific – for example, studies have found that if a parent has any kind of anxiety disorder or clinical depression, then a child is somewhat more likely to develop an anxiety disorder or social phobia. Studies suggest that parents of those with social anxiety disorder tend to be more socially isolated themselves (Bruch and Heimberg, 1994; Caster et al., 1999), and shyness in adoptive parents is significantly correlated with shyness in adopted children (Daniels and Plomin, 1985);

Adolescents who were rated as having an insecure (anxious-ambivalent) attachment with their mother as infants were twice as likely to develop anxiety disorders by late adolescence, including social phobia.

A related line of research has investigated 'behavioural inhibition' in infants – early signs of an inhibited and introspective or fearful nature. Studies have shown that around 10–15 percent of individuals show this early temperament, which appears to be partly due to genetics. Some continue to show this trait in to adolescence and adulthood, and appear to be more likely to develop social anxiety disorder.

**Social experiences**

A previous negative social experience can be a trigger to social phobia, perhaps particularly for individuals high in 'interpersonal sensitivity'. For around half of those diagnosed with social anxiety disorder, a specific traumatic or humiliating social event appears to be associated with the onset or worsening of the disorder; this kind of event appears to be particularly related to specific (performance) social phobia, for example regarding public speaking (Stemberg et al., 1995). As well as direct experiences, observing or hearing about the socially negative experiences of others (e.g. a faux pas committed by someone), or verbal warnings of social problems and dangers, may also make the development of a social anxiety disorder more likely. Social anxiety disorder may be caused by the longer-term effects of not fitting in, or being bullied, rejected or ignored (Beidel and Turner, 1998). Shy adolescents or avoidant adults have emphasised unpleasant experiences with peers or childhood bullying or harassment (Gil martin, 1987). In one study, popularity was found to be negatively correlated with social anxiety, and children who were neglected by their peers reported higher social anxiety and fear of negative evaluation than other categories of children. Socially phobic children appear less likely to receive positive reactions from peers and anxious or inhibited children may isolate themselves.

**Social/cultural influences**

Cultural factors that have been related to social anxiety disorder include a society's attitude towards shyness and avoidance, affecting ability to form relationships or access employment or education, and shame. One study found that the effects of parenting are different depending on the culture – American children appear more likely to develop social anxiety disorder if their parents emphasize the importance of others' opinions and use shame as a disciplinary strategy (Leung et al., 1994), but this association was not found for Chinese/Chinese-American children. In China, research has indicated that shy-inhibited
children are more accepted than their peers and more likely to be considered for leadership and considered competent, in contrast to the findings in Western countries. Purely demographic variables may also play a role – for example there are possibly lower rates of social anxiety disorder in Mediterranean countries and higher rates in Scandinavian countries, and it has been hypothesized that hot weather and high density may reduce avoidance and increase interpersonal contact.

Problems in developing social skills, or 'social effectiveness', may be a cause of some social anxiety disorder, through either inability or lack of confidence to interact socially and gain positive reactions and acceptance from others. The studies have been mixed, however, with some studies not finding significant problems in social skills while others have. What does seem clear is that the socially anxious perceive their own social skills to be low. It may be that the increasing need for sophisticated social skills in forming relationships or careers, and an emphasis on assertiveness and competitiveness, is making social anxiety problems more common, at least among the 'middle classes'. An interpersonal or media emphasis on 'normal' or 'attractive' personal characteristics has also been argued to fuel perfectionism and feelings of inferiority or insecurity regarding negative evaluation from others. The need for social acceptance or social standing has been elaborated in other lines of research relating to social anxiety

**Evolutionary context**

A long-accepted evolutionary explanation of anxiety is that it reflects an in-built 'fight or flight' system, which errs on the side of safety. One line of research suggests that specific dispositions to monitor and react to social threats may have evolved, reflecting the vital and complex importance of social living and social rank in human ancestral environments. Charles Darwin originally wrote about the evolutionary basis of shyness and blushing, and modern evolutionary psychology and psychiatry also addresses social phobia in this context. It has been hypothesized that in modern day society these evolved tendencies can become more inappropriately activated and result in some of the cognitive 'distortions' or 'irrationalities' identified in cognitive-behavioral models and therapies

**Substance induced**

While alcohol initially helps social phobia, excessive alcohol misuse can worsen social phobia symptoms and can cause panic disorder to develop or worsen during alcohol intoxication and especially during alcohol withdrawal syndrome. This effect is not unique to alcohol but can also occur with long term use of drugs which have a similar mechanism of action to alcohol such as the benzodiazepines which are sometimes prescribed as tranquilisers. Benzodiazepines possess anti-anxiety properties and can be useful for the short-term treatment of severe anxiety. Like the anticonvulsants, they tend to be mild and well tolerated, although extremely addictive. Benzodiazepines are usually administered orally for the treatment of anxiety; however, occasionally lorazepam or diazepam may be given intravenously for the treatment of panic attacks.
The World Council of Anxiety does not recommend benzodiazepines for the long term treatment of anxiety due to a range of problems associated with long term use of benzodiazepines including tolerance, psychomotor impairment, cognitive and memory impairments, physical dependence and a benzodiazepine withdrawal syndrome upon discontinuation of benzodiazepines. Despite increasing focus on the use of antidepressants and other agents for the treatment of anxiety, benzodiazepines have remained a mainstay of anxiolytic pharmacotherapy due to their robust efficacy, rapid onset of therapeutic effect, and generally favorable side effect profile. Treatment patterns for psychotropic drugs appear to have remained stable over the past decade, with benzodiazepines being the most commonly used medication for panic disorder.

Approximately half of patients attending mental health services for conditions including anxiety disorders such as panic disorder or social phobia are the result of alcohol or benzodiazepine dependence. Sometimes anxiety pre-existed alcohol or benzodiazepine dependence but the alcohol or benzodiazepine dependence act to keep the anxiety disorders going and often progressively making them worse. Many people who are addicted to alcohol or prescribed benzodiazepines when it is explained to them they have a choice between ongoing ill mental health or quitting and recovering from their symptoms decide on quitting alcohol and/or their benzodiazepines. It was noted that every individual has an individual sensitivity level to alcohol or sedative hypnotic drugs and what one person can tolerate without ill health another will suffer very ill health and that even moderate drinking can cause rebound anxiety syndromes and sleep disorders. A person who is suffering the toxic effects of alcohol or benzodiazepines will not benefit from other therapies or medications as they do not address the root cause of the symptoms. Symptoms may temporarily worsen however, during alcohol withdrawal or benzodiazepine withdrawal.

**Psychological factors**

Research has indicated the role of 'core' or 'unconditional' negative beliefs (e.g. I am inept) and 'conditional' beliefs nearer to the surface (e.g. If I show myself, I will be rejected). They are thought to develop based on personality and adverse experiences and to be activated when the person feels under threat. One line of work has focused more specifically on the key role of self-presentational concerns. The resulting anxiety states are seen as interfering with social performance and the ability to concentrate on interaction, which in turn creates more social problems, which strengthens the negative schema. Also highlighted has been a high focus on and worry about anxiety symptoms themselves and how they might appear to others. A similar model emphasizes the development of a distorted mental representation of their self and over-estimates of the likelihood and consequences of negative evaluation, and of the performance standards that others have. Such cognitive-behavioral models consider the role of negatively-biased memories of the past and the processes of rumination after an event, and fearful anticipation before it. Studies have also highlighted the role of subtle avoidance and defensive factors, and shown how attempts to avoid feared negative evaluations or use 'safety behaviors' (Clark & Wells, 1995) can make social interaction more difficult and the anxiety worse in the long run. This work has been
influential in the development of Cognitive Behavioral Therapy for social anxiety disorder, which has been shown to have efficacy.

**Neural mechanisms**

There are many researches investigating neural bases of social anxiety disorder. Although the exact neural mechanisms are not found yet there is evidence relating social anxiety disorder to imbalance in some neurochemicals and hyperactivity in some of brain areas.

**Dopamine**

Sociability is closely tied to dopamine neurotransmission. Misuse of stimulants like amphetamines to increase self-confidence and improve social performance is common. In a recent study a direct relation between social status of volunteers and binding affinity of dopamine D2/3 receptors in the striatum was found. Other research shows that the binding affinity of dopamine D2 receptors in the striatum of social anxiety sufferers is lower than controls. Some other researches show an abnormality in dopamine transporter density in the striatum of social anxiety sufferers. However, some researchers do not replicate previous findings of evidence of dopamine abnormality in social anxiety disorder. Studies have shown high prevalence of social anxiety in Parkinson's disease and schizophrenia. In a recent study, social phobia was diagnosed in 50% of Parkinson's disease patients. Other researchers have found social phobia symptoms in patients treated with dopamine antagonists like haloperidol, emphasizing the role of dopamine neurotransmission in social anxiety disorder. Also, concentration problems, mental and physical fatigue, Anhedonia and decreased self-confidence are seen in those with social anxiety disorder.

**Other neurotransmitters**

Some scientists hypothesize that social phobia is related to an imbalance of the brain chemical serotonin. A recent study report increased Serotonin transporter binding in psychotropic medication-naive patients with generalized social anxiety disorder. Although there are not many evidences of abnormality in Serotonin neurotransmission, the limited efficacy of medications which affect serotonin levels may indicate the role of this pathway. Paroxetine and Sertraline are two SSRIs that have been confirmed by FDA to treat social anxiety disorder. Some researchers believe that SSRIs decrease activity of Amygdala. There is also increasing focus on other candidate transmitters, e.g. Norepinephrine and Glutamate, which may be over-active in social anxiety disorder, and the inhibitory transmitter GABA which is subsequently lacking.

**Brain areas**

The amygdala is part of the limbic system which is related to fear cognition and emotional learning. Individuals with social anxiety disorder have been found to have a hypersensitive amygdala, for example in relation to social threat cues (e.g. someone might be evaluating you negatively), angry or hostile faces, and while just waiting to give a speech. Recent
research has also indicated that another area of the brain, the anterior cingulate cortex, which was already known to be involved in the experience of physical pain, also appears to be involved in the experience of 'social pain', for example perceiving group exclusion.

**Treatment**

The most important clinical point to emerge from studies of social anxiety disorder is the benefit of early diagnosis and treatment. Social anxiety disorder remains under-recognized in primary care practice, with patients often presenting for treatment only after the onset of complications such as clinical depression or substance abuse disorders.

Research has provided evidence for the efficacy of two forms of treatment available for social phobia: certain medications and a specific form of short-term psychotherapy called Cognitive-behavioral therapy (CBT), the central component being gradual exposure therapy.

**Psychosocial Treatment**

**Basic Principles**

In the case of an agoraphobic patient, the clinician might have the patient imagine (perhaps in hypnotic trance, but this does not seem to add to the effectiveness of treatment) taking a fearful trip, remaining in the anxiety-producing fantasy as long as possible, then "returning" to the therapist's office. This is repeated a number of times, and the patient is instructed to perform the same exercise as often as possible between sessions. Family members are frequently engaged to assist in the process and monitor the "homework." Written journals and diaries may also be used.

In most patients, panic attacks can be treated at the same time, in the same way. Sometimes attention to the physiologic cues of panic or mounting anxiety helps the patient to recognize and control panic symptoms.

**Psychotherapy**

Psychotherapy can be a useful part of the treatment of the anxious or phobic patient. The term "psychotherapy" implies a wide variety of kinds of therapist-patient interaction, overlapping considerably with the behavioral treatments. It is almost impossible to work with a patient in any context without providing considerable interest, support, and understanding. Beyond this, the patient who has given up a symptom may suffer feelings of loss for the symptom itself, for the "equilibrium" of life-style which has existed surrounding the symptom, or both. The opportunity for continuing counseling may be valuable.

Research has shown that cognitive behavioral therapy (CBT) can be highly effective for several anxiety disorders, particularly specific phobias, obsessive compulsive disorder, panic disorder and social anxiety disorder. CBT, as its name suggests, has two main components, cognitive and behavioral. In cases of social anxiety, the cognitive component
can help the patient question how they can be so sure that others are continually watching and harshly judging them. The behavioral component seeks to change people's reactions to anxiety-provoking situations. As such it serves as a logical extension of cognitive therapy, whereby people are shown proof in the real world that their dysfunctional thought processes are unrealistic. A key element of this component was gradual exposure, in which the patient was confronted by the things they fear in a structured, sensitive manner. Gradual exposure is an inherently unpleasant technique; and had high drop out rates. Ideally it involves exposure to a feared social situation that is anxiety provoking but bearable, for as long as possible, two to three times a week. Often, a hierarchy of feared steps is constructed and the patient is exposed each step sequentially. Now modern CBT treatments have been enhanced by focusing treatment on cognitive process, e.g., behavioral experiment with attentional focus, video feedback experiments, addressing fears of negative evaluation, identifying & removing safety seeking behaviors, etc. The aim is to learn from acting differently and observing reactions. This is intended to be done with support and guidance, and when the therapist and patient feel they are ready. Cognitive-behavioral therapy for social phobia for few patients now includes anxiety management training, which may include techniques such as deep breathing and muscle relaxation exercises, which may be practiced 'in-situ'. These early interventions, for some, can be useful but for most can become safety seeking behaviors (and thus unhelpful) so need to be suggested in a considered case conceptualization (i.e., individualized understanding of the cognitive and behavioral factors that are maintaining the problem). CBT can also be conducted partly in group sessions, facilitating the sharing of experiences, a sense of acceptance by others and undertaking behavioral challenges in a trusted environment (Heimberg).

Those patients who do not respond to the briefer treatments often benefit from more in depth psychodynamic psychotherapy. The typical patient after such treatment was in better condition than 77% of untreated controls evaluated at the same time. The various modes examined included psychodynamic, cognitive, and humanistic, as well as behavioral and social, therapies.

**Behavior Therapy**

The behavioral technique of "exposure" is an effective treatment, both short- and long-term, for agoraphobics and many other phobias patients. The use of exposure in fantasy, presenting increasingly anxiety-producing situations as discomfort dissipates at each level, is a form of systematic desensitization. Exposure in vivo also involves gradual adaptation to anxiety-producing objects or situations, but the objects or situations are actually present during the treatment. Flooding, rapid exposure to almost overwhelming volumes of phobic material, also known as implosion, may be used either in fantasy or in vivo.

In general, the behavioral treatments, perhaps coupled with appropriate psychotherapy, have the greatest likelihood of effectiveness, and should be tried before medication is prescribed on any chronic basis.
Some studies have suggested social skills training can help with social anxiety. However, it is not clear whether specific social skills techniques and training are required, rather than just support with general social functioning and exposure to social situations.

Additionally, a recent study has suggested that interpersonal therapy, a form of psychotherapy primarily used to treat depression, may also be effective in the treatment of social phobia.

Pharmacological treatments

SSRIs

Selective serotonin reuptake inhibitors (SSRIs), a class of antidepressants, are considered by many to be the first choice medication for generalised social phobia. These drugs elevate the level of the neurotransmitter serotonin, among other effects. The first drug formally approved by the Food and Drug Administration was paroxetine, sold as Paxil in the U.S. or Seroxat in the UK. Compared to older forms of medication, there is less risk of tolerability and drug dependency. However, their efficacy and increased suicide risk has been subject to controversy.

In a 1995 double-blind, placebo-controlled trial, the SSRI paroxetine was shown to result in clinically meaningful improvement in 55 percent of patients with generalized social anxiety disorder, compared with 23.9 percent of those taking placebo. An October 2004 study yielded similar results. Patients were treated with either fluoxetine, psychotherapy, fluoxetine and psychotherapy, placebo and psychotherapy, or a placebo. The first four sets saw improvement in 50.8 to 54.2 percent of the patients. Of those assigned to receive only a placebo, 31.7 percent achieved a rating of 1 or 2 on the Clinical Global Impression-Improvement scale. Those who sought both therapy and medication did not see a boost in improvement.

General side-effects are common during the first weeks while the body adjusts to the drug. Symptoms may include headaches, nausea, insomnia and changes in sexual behavior. Treatment safety during pregnancy has not been established. In late 2004 much media attention was given to a proposed link between SSRI use and juvenile suicide. For this reason, the use of SSRIs in pediatric cases of depression is now recognized by the Food and Drug Administration as warranting a cautionary statement to the parents of children who may be prescribed SSRIs by a family doctor. Recent studies have shown no increase in rates of suicide. These tests, however, represent those diagnosed with depression, not necessarily with social anxiety disorder. However, it should be noted that due to the nature of the conditions, those taking SSRIs for social phobias are far less likely to have suicidal ideation than those with depression.

Other drugs

Although SSRIs are often the first choice for treatment, other prescription drugs are also used, sometimes only if SSRIs fail to produce any clinically significant improvement.
In 1985, before the introduction of SSRIs, anti-depressants such as monoamine oxidase inhibitors (MAOIs) were frequently used in the treatment of social anxiety. Their efficacy appears to be comparable or sometimes superior to SSRIs or benzodiazepines. However, because of the dietary restrictions required, high toxicity in overdose, and incompatibilities with other drugs, its usefulness as a treatment for social phobics is now limited. Some argue for their continued use, however, or that a special diet does not need to be strictly adhered to. A newer type of this medication, Reversible inhibitors of monoamine oxidase subtype A (RIMAs) inhibit the MAO enzyme only temporarily, improving the adverse-effect profile but possibly reducing their efficacy.

Benzodiazepines such as alprazolam and clonazepam are an alternative to SSRIs. These drugs are often used for short-term relief of severe, disabling anxiety. Although benzodiazepines are still sometimes prescribed for long-term everyday use in some countries, there is some concern over the development of drug tolerance, dependency and misuse. It has been recommended that benzodiazepines are only considered for individuals who fail to respond to safer medications. Benzodiazepines augment the action of GABA, the major inhibitory neurotransmitter in the brain; effects usually begin to appear within minutes or hours.

The novel antidepressant mirtazapine has been proven effective in treatment of social anxiety disorder. This is especially significant due to mirtazapine's fast onset and lack of many unpleasant side-effects associated with SSRIs (particularly, sexual dysfunction).

In Japan, the serotonin-norepinephrine reuptake inhibitor (SNRI), Milnacipran is used in the treatment of Taijin kyofusho a Japanese variant of social anxiety disorder.

Some people with a form of social phobia called performance phobia have been helped by beta-blockers, which are more commonly used to control high blood pressure. Taken in low doses, they control the physical manifestation of anxiety and can be taken before a public performance.

A novel treatment approach has recently been developed as a result of translational research. It has been shown that a combination of acute dosing of d-cycloserine (DCS) with exposure therapy facilitates the effects of exposure therapy of social phobia. DCS is an old antibiotic medication used for treating tuberculosis and does not have any anxiolytic properties per se. However, it acts as an agonist at the glutamatergic N-methyl-D-aspartate (NMDA) receptor site, which is important for learning and memory. It has been shown that administering a small dose acutely 1 hour before exposure therapy can facilitate extinction learning that occurs during therapy.
Obsessive-compulsive disorder

Obsessive-compulsive disorder (OCD) is an anxiety disorder characterized by intrusive thoughts that produce uneasiness, apprehension, fear, or worry, by repetitive behaviors aimed at reducing the associated anxiety, or by a combination of such obsessions and compulsions. Symptoms of the disorder include excessive washing or cleaning; repeated checking; extreme hoarding; preoccupation with sexual, violent or religious thoughts; aversion to particular numbers; and nervous rituals, such as opening and closing a door a certain number of times before entering or leaving a room. These symptoms can be alienating and time-consuming, and often cause severe emotional and financial distress. The acts of those who have OCD may appear paranoid and potentially psychotic. However, OCD sufferers generally recognize their obsessions and compulsions as irrational, and may become further distressed by this realization.

OCD is the fourth most common mental disorder, and is diagnosed nearly as often as asthma and diabetes mellitus. In the United States, one in 50 adults suffers from OCD. Obsessive-compulsive disorder affects children and adolescents as well as adults. Roughly one third to one half of adults with OCD report a childhood onset of the disorder, suggesting the continuum of anxiety disorders across the life span. The phrase "obsessive-compulsive" has become part of the English lexicon, and is often used in an informal or caricatured manner to describe someone who is excessively meticulous, perfectionistic, absorbed, or otherwise fixated. Although these signs are present in OCD, a person who exhibits them does not necessarily have OCD, and may instead have obsessive-compulsive personality disorder (OCPD), an autism spectrum disorder, or no clinical condition.
Multiple psychological and biological factors may be involved in causing obsessive-compulsive syndromes. Standardized rating scales such as Yale-Brown Obsessive Compulsive Scale can be used to assess the severity of OCD symptoms.

**Signs and symptoms**

**Obsessions**

Obsessions are thoughts that recur and persist despite efforts to ignore or confront them. People with OCD frequently perform tasks, or compulsions, to seek relief from obsession-related anxiety. Within and among individuals, the initial obsessions, or intrusive thoughts, vary in their clarity and vividness. A relatively vague obsession could involve a general sense of disarray or tension accompanied by a belief that life cannot proceed as normal while the imbalance remains. A more intense obsession could be a preoccupation with the thought or image of someone close to them dying. Other obsessions concern the possibility that someone or something other than oneself—such as God, the Devil, or disease—will harm either the person with OCD or the people or things that the person cares about. Other individuals with OCD may experience the sensation of invisible protrusions emanating from their bodies, or have the feeling that inanimate objects are ensouled.

Some people with OCD experience sexual obsessions that may involve intrusive thoughts or images of "kissing, touching, fondling, oral sex, anal sex, intercourse, incest and rape" with "strangers, acquaintances, parents, children, family members, friends, coworkers, animals and religious figures", and can include "heterosexual or homosexual content" with persons of any age. As with other intrusive, unpleasant thoughts or images, most "normal" people have some disquieting sexual thoughts at times, but people with OCD may attach extraordinary significance to the thoughts. For example, obsessive fears about sexual orientation can appear to the person with OCD, and even to those around them, as a crisis of sexual identity. Furthermore, the doubt that accompanies OCD leads to uncertainty regarding whether one might act on the troubling thoughts, resulting in self-criticism or self-loathing.

People with OCD understand that their notions do not correspond with reality; however, they feel that they must act as though their notions are correct. For example, an individual who engages in compulsive hoarding might be inclined to treat inorganic matter as if it had the sentience or rights of living organisms, while accepting that such behavior is irrational on a more intellectual level. In severe OCD, obsessions can shift into delusions when resistance to the obsession is abandoned and insight into its senselessness is lost. (Insel and Akiskal (1986))

**Compulsions**

Some people with OCD perform compulsive rituals because they inexplicably feel they have to, others act compulsively so as to mitigate the anxiety that stems from particular obsessive thoughts. The person might feel that these actions somehow either will prevent a dreaded event from occurring, or will push the event from their thoughts. In any case, the
individual's reasoning is so idiosyncratic or distorted that it results in significant distress for the individual with OCD or for those around them. Excessive skin picking (i.e., dermatillomania) or hair plucking (i.e., trichotillomania) and nail biting (i.e., onychophagia) are all on the Obsessive-Compulsive Spectrum. Individuals with OCD are aware that their thoughts and behavior are not rational, but they feel bound to comply with them to fend off feelings of panic or dread.

Some common compulsions include counting specific things (such as footsteps) or in specific ways (for instance, by intervals of two) and doing other repetitive actions, often with atypical sensitivity to numbers or patterns. People might repeatedly wash their hands or clear their throats, make sure certain items are in a straight line, repeatedly check that their parked cars have been locked before leaving them, constantly organize in a certain way, turn lights on and off, keep doors closed at all times, touch objects a certain number of times before exiting a room, walk in a certain routine way like only stepping on a certain color of tile, or have a routine for using stairs, such as always finishing a flight on the same foot.

People rely on compulsions as an escape from their obsessive thoughts; however, they are aware that the relief is only temporary, that the intrusive thoughts will soon return. Some people use compulsions to avoid situations that may trigger their obsessions. Although some people do certain things over and over again, they do not necessarily perform these actions compulsively. For example, bedtime routines, learning a new skill, and religious practices are not compulsions. Whether or not behaviors are compulsions or mere habit depends on the context in which the behaviors are performed. For example, arranging and ordering DVDs for eight hours a day would be expected of one who works in a video store, but would seem abnormal in other situations. In other words, habits tend to bring efficiency to one's life, while compulsions tend to disrupt it.

In addition to the anxiety and fear that typically accompanies OCD, sufferers may spend hours performing such compulsions every day. In such situations, it can be hard for the person to fulfill their work, family, or social roles. In some cases, these behaviors can also cause adverse physical symptoms. For example, people who obsessively wash their hands with antibacterial soap and hot water can make their skin red and raw with dermatitis.

People with OCD can use rationalizations to explain their behavior; however, these rationalizations do not apply to the overall behavior but to each instance individually. For example, a person compulsively checking the front door may argue that the time taken and stress caused by one more check of the front door is much less than the time and stress associated with being robbed, and thus checking is the better option. In practice, after that check, the person is still not sure and deems it is still better to perform one more check, and this reasoning can continue as long as necessary.

**Without overt compulsions**

OCD sometimes manifests without overt compulsions. Nicknamed "Pure-O", OCD without overt compulsions could, by one estimate, characterize as many as 50 percent to 60 percent
of OCD cases. Rather than engaging in observable compulsions, the person with this subtype might perform more covert, mental rituals, or might feel driven to avoid the situations in which particular thoughts seem likely to intrude. As a result of this avoidance, people can struggle to fulfill both public and private roles, even if they place great value on these roles and even if they had fulfilled the roles successfully in the past. Moreover, the individual's avoidance can confuse others who do not know its origin or intended purpose, as it did in the case of a man whose wife began to wonder why he would not hold their infant child.

Causes

Scholars generally agree that both psychological and biological factors play a role in causing the disorder, although they differ in their degree of emphasis upon either type of factor.

Psychological

Biological

OCD has been linked to abnormalities with the neurotransmitter serotonin, although it could be either a cause or an effect of these abnormalities. Serotonin is thought to have a role in regulating anxiety. To send chemical messages from one neuron to another, serotonin must bind to the receptor sites located on the neighboring nerve cell. It is hypothesized that the serotonin receptors of OCD sufferers may be relatively understimulated. This suggestion is consistent with the observation that many OCD patients benefit from the use of selective serotonin reuptake inhibitors (SSRIs), a class of antidepressant medications that allow for more serotonin to be readily available to other nerve cells.

A possible genetic mutation may contribute to OCD. A mutation has been found in the human serotonin transporter gene, hSERT, in unrelated families with OCD. Moreover, data from identical twins supports the existence of a "heritable factor for neurotic anxiety". Further, individuals with OCD are more likely to have first-degree family members exhibiting the same disorders than do matched controls. In cases where OCD develops during childhood, there is a much stronger familial link in the disorder than cases in which OCD develops later in adulthood. In general, genetic factors account for 45-65% of OCD symptoms in children diagnosed with the disorder. Environmental factors also play a role in how these anxiety symptoms are expressed; various studies on this topic are in progress and the presence of a genetic link is not yet definitely established.

People with OCD evince increased grey matter volumes in bilateral lenticular nuclei, extending to the caudate nuclei, while decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri. These findings contrast with those in people with other anxiety disorders, who evince decreased (rather than increased) grey matter volumes in bilateral lenticular / caudate nuclei, while also decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri. Orbitofrontal cortex overactivity
is attenuated in patients who have successfully responded to SSRI medication, a result believed to be caused by increased stimulation of serotonin receptors 5-HT2A and 5-HT2C. The striatum, linked to planning and the initiation of appropriate actions, has also been implicated; mice genetically engineered with a striatal abnormality exhibit OCD-like behavior, grooming themselves three times as frequently as ordinary mice. Recent evidence supports the possibility of a heritable predisposition for neurological development favoring OCD.

Rapid onset of OCD in children may be caused by Group A streptococcal infection, a condition hypothesized by its acronym PANDAS.

Neurotransmitters role

Researchers have yet to pinpoint the exact cause of OCD, but brain differences, genetic influences, and environmental factors are being studied. Brain scans of people with OCD have shown that they have different patterns of brain activity than people without OCD and that different functioning of circuitry within a certain part of the brain, the striatum, may cause the disorder. Differences in other parts of the brain and an imbalance of brain chemicals, especially serotonin and dopamine, may also contribute to OCD. Independent studies have consistently found unusual dopamine and serotonin activity in various regions of the brain in individuals with OCD. These can be defined as dopaminergic hyperfunction in the prefrontal cortex and serotonergic hypofunction in the basal ganglia.

Diagnosis

Formal diagnosis may be performed by a psychologist, psychiatrist, clinical social worker, or other licensed mental health professional. To be diagnosed with OCD, a person must have obsessions, compulsions, or both, according to the Diagnostic and Statistical Manual of Mental Disorders (DSM). The Quick Reference to the 2000 edition of the DSM suggests that several features characterize clinically significant obsessions and compulsions. Such obsessions, the DSM says, are recurrent and persistent thoughts, impulses, or images that are experienced as intrusive and that cause marked anxiety or distress. These thoughts, impulses, or images are of a degree or type that lies outside the normal range of worries about conventional problems. A person may attempt to ignore or suppress such obsessions, or to neutralize them with some other thought or action, and will tend to recognize the obsessions as idiosyncratic or irrational.

Compulsions become clinically significant when a person feels driven to perform them in response to an obsession, or according to rules that must be applied rigidly, and when the person consequently feels or causes significant distress. Therefore, while many people who do not suffer from OCD may perform actions often associated with OCD (such as ordering items in a pantry by height), the distinction with clinically significant OCD lies in the fact that the person who suffers from OCD must perform these actions, otherwise they will experience significant psychological distress. These behaviors or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these activities are not logically or practically connected to the issue, or they are excessive.
In addition, at some point during the course of the disorder, the individual must realize that their obsessions or compulsions are unreasonable or excessive. Moreover, the obsessions or compulsions must be time-consuming (taking up more than one hour per day) or cause impairment in social, occupational, or scholastic functioning. It is helpful to quantify the severity of symptoms and impairment before and during treatment for OCD. In addition to the patient’s estimate of the time spent each day harboring obsessive-compulsive thoughts or behaviors, Fenske and Schwenk in their article “Obsessive-Compulsive Disorder: Diagnosis and Management,” argue that more concrete tools should be used to gauge the patient's condition (2009). This may be done with rating scales, such as the most trusted Yale–Brown Obsessive Compulsive Scale (Y-BOCS). With measurements like these, psychiatric consultation can be more appropriately determined because it has been standardized.

**Differential diagnosis**

OCD is often confused with the separate condition obsessive–compulsive personality disorder (OCPD). OCD is ego dystonic, meaning that the disorder is incompatible with the sufferer’s self-concept. Because disorders that are ego dystonic go against a person’s self-concept, they tend to cause much distress. OCPD, on the other hand, is ego syntonic—marked by the person’s acceptance that the characteristics displayed as a result of this disorder are compatible with his or her self-image.

People with OCD are often aware that their behavior is not rational and are unhappy about their obsessions but nevertheless feel compelled by them. People with OCPD are not aware of anything abnormal; they will readily explain why their actions are rational, and it is usually impossible to convince them otherwise.

People with OCD are ridden with anxiety; by contrast, people with OCPD tend to derive pleasure from their obsessions or compulsions.

Some OCD sufferers exhibit what is known as overvalued ideas. In such cases, the person with OCD will truly be uncertain whether the fears that cause them to perform their compulsions are irrational or not. After some discussion, it is possible to convince the individual that their fears may be unfounded. It may be more difficult to do ERP therapy on such patients because they may be unwilling to cooperate, at least initially. For this reason OCD has often been likened to a disease of pathological doubt, in which the sufferer, though not usually delusional, is often unable to realize fully which dreaded events are reasonably possible and which are not. There are severe cases in which the sufferer has an unshakeable belief in the context of OCD that is difficult to differentiate from psychosis.

OCD is different from behaviors such as gambling addiction and overeating. People with these disorders typically experience at least some pleasure from their activity; OCD sufferers do not actively want to perform their compulsive tasks and experience no pleasure from doing so.
OCD can, like many forms of chronic stress, lead to clinical depression over time. The constant stress of the condition can cause sufferers to develop a deadening of spirit, a numbing frustration, or sense of hopelessness. OCD’s effects on day-to-day life, particularly its substantial consumption of time, can produce difficulties with work, finances, and relationships. There is no known cure for OCD, but a number of successful treatment options are available.

Management

According to a team of Duke University-led psychiatrists, behavioral therapy (BT), cognitive behavioral therapy (CBT), and medications should be regarded as first-line treatments for OCD. Psychodynamic psychotherapy may help in managing some aspects of the disorder. The American Psychiatric Association notes a lack of controlled demonstrations that psychoanalysis or dynamic psychotherapy is effective "in dealing with the core symptoms of OCD."

Behavioral therapy

The specific technique used in BT/CBT is called exposure and ritual prevention (also known as "exposure and response prevention") or ERP; this involves gradually learning to tolerate the anxiety associated with not performing the ritual behavior. At first, for example, someone might touch something only very mildly "contaminated" (such as a tissue that has been touched by another tissue that has been touched by the end of a toothpick that has touched a book that came from a "contaminated" location, such as a school.) That is the "exposure". The "ritual prevention" is not washing. Another example might be leaving the house and checking the lock only once (exposure) without going back and checking again (ritual prevention). The person fairly quickly habituates to the anxiety-producing situation and discovers that their anxiety level has dropped considerably; they can then progress to touching something more "contaminated" or not checking the lock at all—again, without performing the ritual behavior of washing or checking.

Exposure ritual/response prevention (ERP) has a strong evidence base. It is generally considered the most effective treatment for OCD.

It has generally been accepted that psychotherapy, in combination with psychotropic medication, is more effective than either option alone. However, more recent studies have shown no difference in outcomes for those treated with the combination of medicine and CBT versus CBT alone.

More recent behavioral work has focused on associative splitting. It is a new technique aimed at reducing obsessive thoughts. The method draws upon the “fan effect” of associative priming: The sprouting of new associations diminishes the strength of existing ones. As OCD patients show marked biases or restrictions in OCD-related semantic networks (e.g., cancer is only associated with “illness” or “death”, fire is only associated with “danger” or “destruction”), they are encouraged to imagine neutral or positive associations to OCD-related cognitions (cancer = zodiac sign, animal, crab; fire = fireflies,
fireworks, candlelight-dinner). First studies tentatively confirm the feasibility and effectiveness of the approach for a subgroup of patients.

**Medication**

Medications as treatment include selective serotonin reuptake inhibitors (SSRIs) such as paroxetine, sertraline, fluoxetine, escitalopram and fluvoxamine and the tricyclic antidepressants, in particular clomipramine. SSRIs prevent excess serotonin from being pumped back into the original neuron that released it. Instead, serotonin can then bind to the receptor sites of nearby neurons and send chemical messages or signals that can help regulate the excessive anxiety and obsessive thoughts. In some treatment-resistant cases, a combination of clomipramine and an SSRI has shown to be effective even when neither drug on its own has been efficacious.

Treatment of OCD is an area needing significant improvement in prescribing regimens. Benzodiazepines are sometimes used, although they are generally believed to be ineffective for treating OCD; however, effectiveness was found in one small study. Serotonergic antidepressants typically take longer to show benefit in OCD than with most other disorders they are used to treat. It is common for 2–3 months to elapse before any tangible improvement is noticed. In addition to this, treatment usually requires high dosages. Fluoxetine, for example, is usually prescribed in dosages of 20 mg per day for clinical depression, whereas with OCD the dosage often ranges from 20 mg to 80 mg or higher, if necessary. In most cases antidepressant therapy alone provides only a partial reduction in symptoms, even in cases that are not deemed treatment resistant. Much current research is devoted to the therapeutic potential of the agents that affect the release of the neurotransmitter glutamate or the binding to its receptors. These include riluzole, memantine, gabapentin, N-Acetylcysteine, and lamotrigine. MDMA, which is a powerful and illicit serotonergic drug, has also been anecdotally reported to temporarily alleviate the symptoms of OCD.

The atypical antipsychotics olanzapine, quetiapine, and risperidone have also been found to be useful as adjuncts to an SSRI in treatment-resistant OCD. However, these drugs are often poorly tolerated, and have significant metabolic side effects that limit their use. None of the atypical antipsychotics have demonstrated efficacy as a monotherapy.

**Electroconvulsive therapy**

Electroconvulsive therapy (ECT) has been found effective in severe and refractory cases.

**Psychosurgery**

For some, medication, support groups and psychological treatments fail to alleviate obsessive-compulsive symptoms. These patients may choose to undergo psychosurgery as a last resort. In this procedure, a surgical lesion is made in an area of the brain (the cingulate cortex). In one study, 30% of participants benefited significantly from this procedure. Deep-brain stimulation and vagus nerve stimulation are possible surgical interventions.
options that do not require destruction of brain tissue. In the US, the Food and Drug Administration approved deep-brain stimulation for the treatment of OCD under a humanitarian device exemption requiring that the procedure be performed only in a hospital with specialist qualifications to do so.

In the US, psychosurgery for OCD is a treatment of last resort and will not be performed until the patient has failed several attempts at medication (at the full dosage) with augmentation, and many months of intensive cognitive-behavioral therapy with exposure and ritual/response prevention. Likewise, in the United Kingdom, psychosurgery cannot be performed unless a course of treatment from a suitably qualified cognitive-behavioral therapist has been carried out.

**In children and adolescents**

Therapeutic treatment may be effective in reducing ritual behaviors of OCD for children and adolescents. Family involvement, in the form of behavioral observations and reports, is a key component to the success of such treatments. Parental intervention also provides positive reinforcement for a child who exhibits appropriate behaviors as alternatives to compulsive responses. After one or two years of therapy, in which a child learns the nature of his or her obsession and acquires strategies for coping, that child may acquire a larger circle of friends, exhibit less shyness, and become less self-critical.

Although the causes of OCD in younger age groups range from brain abnormalities to psychological preoccupations, life stress such as bullying and traumatic familial deaths may also contribute to childhood cases of OCD, and acknowledging these stressors can play an role in treating the disorder.

The mental technique of “thought stopping” can help reduce or eliminate obsessive thoughts. In this procedure, whenever an individual has an obsessive thought, he or she is encouraged to utter “STOP” in mid-thought to interrupt the obsession. A variant of the process avoids making the word “STOP” a stimulus to the obsessive thoughts: in the presence of an obsessive thought, a child counts loudly backward from ten, and then evokes a pleasant scene.

**Experimental**

The naturally occurring sugar inositol has been suggested as a treatment for OCD, as it appears to modulate the actions of serotonin and reverse desensitisation of neurotransmitter receptors.

Nutrition deficiencies may also contribute to OCD and other mental disorders. Vitamin and mineral supplements may aid in such disorders and provide nutrients necessary for proper mental functioning.

μ-Opioids, such as hydrocodone and tramadol, may rapidly ameliorate OCD symptoms. Tramadol is an atypical opioid that appears to provide the anti-OCD effects of an opiate and
inhibit the re-uptake of serotonin (in addition to norepinephrine) Oral morphine, administered once weekly, has been shown to reduce OCD symptoms in some treatment-resistant patients. The mechanism of therapeutic action is unknown. Administration of opiate treatment may be contraindicated in individuals concurrently taking CYP2D6 inhibitors such as fluoxetine and paroxetine.

Psychedelics such as LSD, peyote, and tryptamine alkaloid psilocybin have been proposed as treatment due to their observed effects on OCD symptoms. It has been hypothesised that hallucinogens may stimulate 5-HT2A receptors and, less significantly, 5-HT2C receptors, causing an inhibitory effect on the orbitofrontal cortex, an area of the brain strongly associated with hyperactivity and OCD.

Regular nicotine treatment may ameliorate symptoms of OCD, although the pharmacodynamical mechanism by which this is achieved is not yet known, and more detailed studies are needed to fully confirm this hypothesis.

Since choline's anti-dopaminergic effects often worsen OCD symptoms, anticholinergics are sometimes used as a supplementary treatment for OCD symptoms.

St John's Wort was previously believed to be of benefit due to its (non-selective) serotonin re-uptake inhibiting qualities, but a double-blind study using a flexible-dose schedule (600–1800 mg/day) found no difference between St John's Wort and a placebo.

**Epidemiology**

OCD does not have a higher affinity for a specific gender. In 80% of cases, symptoms present before the age of 18. Studies have placed the prevalence of the disorder at between one and three percent, although the prevalence of clinically recognized OCD is much lower, suggesting that many individuals with the disorder may not be diagnosed. The fact that many individuals do not seek treatment may be due in part to stigma associated with OCD.

In a 1980 study of adults from several U.S. cities, the lifetime prevalence rate of OCD for both sexes was recorded at 2.5 percent. Education also appears to be a factor. The lifetime prevalence of OCD is lower for those who have graduated from high school than for those who have not (1.9 percent versus 3.4 percent). However, in the case of college education, lifetime prevalence is higher for those who graduate with a degree (3.1 percent) than it is for those who have only some college background (2.4 percent). As far as age is concerned, the onset of OCD usually ranges from the late teenage years until the mid-20s in both sexes, but the age of onset tends to be slightly younger in males than in females.

A study suggests that OCD symptoms in Japanese patients are similar to those found in Western countries, suggesting that this disorder transcends culture and geography. The study, published in 2008, appears to contradict previous theories, said the study's lead author, Hisato Matsunaga. Having "hypothesized that symptom structure might be substantially influenced by the sociocultural differences", Hisato said that he was surprised by the results.
It has been proposed that sufferers are generally of above-average intelligence, as the very nature of the disorder necessitates complicated thinking patterns.

**Comorbidity**

People with OCD may be diagnosed with other conditions, such as major depressive disorder, generalized anxiety disorder, anorexia nervosa, social anxiety disorder, bulimia nervosa, Tourette syndrome, Asperger syndrome, compulsive skin picking, body dysmorphic disorder, trichotillomania, and (as already mentioned) obsessive-compulsive personality disorder. There is some research demonstrating a link between drug addiction and OCD as well. Many who suffer from OCD also suffer from panic attacks. There is a higher risk of drug addiction among those with any anxiety disorder (possibly as a way of coping with the heightened levels of anxiety), but drug addiction among OCD patients may serve as a type of compulsive behavior and not just as a coping mechanism. Depression is also extremely prevalent among sufferers of OCD. One explanation for the high depression rate among OCD populations was posited by Mineka, Watson, and Clark (1998), who explained that people with OCD (or any other anxiety disorder) may feel depressed because of an "out of control" type of feeling. In further consideration of OCD comorbidities, the research of Fenske and Schwenk reports that studies have shown that depression among those with OCD is particularly alarming because their risk of suicide is high; more than 50 percent of patients experience suicidal tendencies, and 15 percent have attempted suicide. Individuals with OCD have also been found to be affected by delayed sleep phase syndrome at a substantially higher rate than the general public.

**Prognosis**

Psychological interventions such as behavioral and cognitive-behavioral therapy as well as pharmacological treatment can lead to substantial reduction of OCD symptoms for the average patient. However, OCD symptoms persist at moderate levels even following adequate treatment course and a completely symptom-free period is uncommon.

**Cognitive performance**

OCD is associated with higher IQ.

A 2009 study that conducted "a battery of neuropsychological tasks to assess nine cognitive domains with a special focus on executive functions concluded that "few neuropsychological differences emerged between the OCD and healthy participants when concomitant factors were controlled."

**History**

From the 14th to the 16th century in Europe, it was believed that people who experienced blasphemous, sexual, or other obsessive thoughts were possessed by the Devil. Based on this reasoning, treatment involved banishing the "evil" from the "possessed" person
through exorcism. In the early 1910s, Sigmund Freud attributed obsessive-compulsive behavior to unconscious conflicts that manifest as symptoms. Freud describes the clinical history of a typical case of "touching phobia" as starting in early childhood, when the person has a strong desire to touch an item. In response, the person develops an "external prohibition" against this type of touching. However, this "prohibition does not succeed in abolishing" the desire to touch; all it can do is repress the desire and "force it into the unconscious".

**Society and culture**

- British poet, essayist, and lexicographer Samuel Johnson is an example of a historical figure with a retrospective diagnosis of OCD. He had elaborate rituals for crossing the thresholds of doorways, and repeatedly walked up and down staircases counting the steps.
- American aviator and filmmaker Howard Hughes is known to have suffered from OCD. Friends of Hughes have mentioned his obsession with minor flaws in clothing and he is reported to have had a great fear of germs, common among OCD patients.
- English footballer David Beckham has been outspoken regarding his struggle with OCD. He has told media that he has to count all of his clothes, and his magazines have to lie in a straight line. He has expressed a desire to get help for his problems.
- American game show host Marc Summers has written a book about how OCD has affected his life. The book is titled Everything in Its Place: My Trials and Triumphs with Obsessive Compulsive Disorder.
- Movies and television often portray idealized representations of disorders such as OCD. These depictions may lead to increased public awareness, understanding, and sympathy for such disorders.
- Melvin Udall played by Jack Nicholson in As Good As It Gets repeatedly lock and unlock his door, wouldn’t step on any cracks, and brought his own plastic cutlery to a restaurant. There were other indications that he had OCD.

**Intrusive thoughts**

Intrusive thoughts are unwelcome involuntary thoughts, images, or unpleasant ideas that may become obsessions, are upsetting or distressing, and can be difficult to manage or eliminate. Most people experience these thoughts. When they are associated with obsessive-compulsive disorder (OCD), depression, and sometimes attention-deficit hyperactive disorder (ADHD), they may become paralyzing, anxiety-provoking, or persistent. Intrusive thoughts may also be associated with episodic memory, unwanted worries or memories from OCD, posttraumatic stress disorder, other anxiety disorders, eating disorders, or psychosis. According to Lee Baer (a specialist at the OCD clinic of Massachusetts General Hospital), intrusive thoughts, urges, and images are of inappropriate things at inappropriate times, usually falling into three categories: "inappropriate aggressive thoughts, inappropriate sexual thoughts, or blasphemous religious thoughts".
Description

Many people experience the type of bad or unwanted thoughts that people with more troubling intrusive thoughts have, but most people are able to dismiss these thoughts. For most people, intrusive thoughts are a "fleeting annoyance." London psychologist Stanley Rachman presented a questionnaire to healthy college students and found that virtually all said they had these thoughts from time to time, including thoughts of sexual violence, sexual punishment, "unnatural" sex acts, painful sexual practices, blasphemous or obscene images, thoughts of harming elderly people or someone close to them, violence against animals or toward children, and impulsive or abusive outbursts or utterances. Such bad thoughts are universal among humans, and have "almost certainly always been a part of the human condition".

When intrusive thoughts occur with obsessive-compulsive disorder (OCD), patients are less able to ignore the unpleasant thoughts and may pay undue attention to them, causing the thoughts to become more frequent and distressing. The thoughts may become obsessions which are paralyzing, severe, and constantly present, and can range from thoughts of violence or sex to religious blasphemy. Distinguishing them from normal intrusive thoughts experienced by many people, the intrusive thoughts associated with OCD may be anxiety provoking, irrepensible, and persistent.

How people react to intrusive thoughts may determine whether these thoughts will become severe, turn into obsessions, or require treatment. Intrusive thoughts can occur with or without compulsions. Carrying out the compulsion reduces the anxiety, but makes the urge to perform the compulsion stronger each time it recurs, reinforcing the intrusive thoughts. According to Baer, suppressing the thoughts only makes them stronger, and recognizing that bad thoughts do not signify that one is truly evil is one of the steps to overcoming them. There is evidence of the benefit of acceptance as an alternative to suppression of intrusive thoughts. A study showed that those instructed to suppress intrusive thoughts experienced more distress after suppression, while patients instructed to accept the bad thoughts experienced decreased discomfort. These results may be related to underlying cognitive processes involved in OCD. But, accepting the thoughts can be more difficult for persons with OCD. In the 19th century, OCD was known as "the doubting sickness"; the "pathological doubt" that accompanies OCD can make it harder for a person with OCD to distinguish "normal" intrusive thoughts as experienced by most people, causing them to "suffer in silence, feeling too embarrassed or worried that they will be thought crazy".

The possibility that most patients suffering from intrusive thoughts will ever act on those thoughts is low. Patients who are experiencing intense guilt, anxiety, shame, and upset over these thoughts are different from those who actually act on them. The history of violent crime is dominated by those who feel no guilt or remorse; the very fact that someone is tormented by intrusive thoughts and has never acted on them before is an excellent predictor that they will not act upon the thoughts. Patients who are not troubled or shamed by their thoughts, do not find them distasteful, or who have actually taken action, might need to have more serious conditions such as psychosis or potentially criminal behaviors...
ruled out. According to Baer, a patient should be concerned that intrusive thoughts are
dangerous if the person does not feel upset by the thoughts, or rather finds them
pleasurable; has ever acted on violent or sexual thoughts or urges; hears voices or sees
things that others do not see; or feels uncontrollable irresistible anger.

**Inappropriate aggressive thoughts**

Intrusive thoughts may involve violent obsessions about hurting others or themselves.
They can include such thoughts as harming an innocent child, jumping from a bridge,
mountain or the top of a tall building, urges to jump in front of a train or automobile, and
urges to push another in front of a train or automobile. Rachman’s survey of healthy college
students found that virtually all of them had intrusive thoughts from time to time, including:

- Causing harm to elderly people
- Imagining or wishing harm upon someone close to one’s self
- Impulses to violently attack, hit, harm or kill a person, small child, or animal
- Impulses to shout at or abuse someone, or attack and violently punish someone, or
  say something rude, inappropriate, nasty or violent to someone.

These thoughts are part of being human, and need not ruin the quality of life. Treatment is
available when the thoughts are associated with OCD and become persistent, severe, or
distressing.

**Inappropriate sexual thoughts**

Sexual obsessions involve intrusive thoughts or images of "kissing, touching, fondling, oral
sex, anal sex, intercourse, and rape" with "strangers, acquaintances, parents, children,
family members, friends, coworkers, animals and religious figures", involving
"heterosexual or homosexual content" with persons of any age.

Like other unwanted intrusive thoughts or images, everyone has some inappropriate
sexual thoughts at times, but people with OCD may attach significance to the unwanted
sexual thoughts, generating anxiety and distress. The doubt that accompanies OCD leads to
uncertainty regarding whether one might act on the intrusive thoughts, resulting in self-
criticism or loathing.

One of the more common sexual intrusive thoughts occurs when an obsessive person
doubts his or her sexual identity. As in the case of most sexual obsessions, sufferers may
feel shame and live in isolation, finding it hard to discuss their fears, doubts, and concerns
about their sexual identity.

A person experiencing sexual intrusive thoughts may feel shame, "embarrassment, guilt,
distress, torment, fear that you may act on the thought or perceived impulse, and doubt
about whether you have already acted in such a way." Depression may be a result of the
self-loathing that can occur, depending on how much the OCD interferes with daily
functioning or causes distress. Their concern over these thoughts may cause them to scrutinize their bodies to determine if the thoughts result in feelings of arousal. But, focusing attention of any part of the body can result in feelings in that part of the body, hence doing so may decrease confidence and increase fear about acting on the urges. Part of treatment of sexual intrusive thoughts involves therapy to help sufferers accept intrusive thoughts and stop trying to reassure themselves by checking their bodies.

**Blasphemous religious thoughts**

Blasphemous thoughts are a common component of OCD, documented throughout history; notable religious figures such as Martin Luther and St. Ignatius were known to be tormented by intrusive, blasphemous or religious thoughts and urges. Martin Luther had urges to curse God and Jesus, and was obsessed with images of "the Devil's behind". St. Ignatius had numerous obsessions, including the fear of stepping on pieces of straw forming a cross, fearing that it showed disrespect to Christ. A study of 50 patients with a primary diagnosis of obsessive-compulsive disorder found that 40% had religious and blasphemous thoughts and doubts—a higher number than the 38% who had the obsessional thoughts related to dirt and contamination more commonly associated with OCD. One study suggests that content of intrusive thoughts may vary depending on culture, and that blasphemous thoughts may be more common in men than in women.

According to Fred Penzel, a New York psychologist, some common religious obsessions and intrusive thoughts are:

- sexual thoughts about God, saints, and religious figures such as Mary
- bad thoughts or images during prayer or meditation
- thoughts of being possessed
- fears of sinning or breaking a religious law or performing a ritual incorrectly
- fears of omitting prayers or reciting them incorrectly
- repetitive and intrusive blasphemous thoughts
- urges or impulses to say blasphemous words or commit blasphemous acts during religious services.

Suffering can be greater and treatment complicated when intrusive thoughts involve religious implications; patients may believe the thoughts are inspired by Satan, and may fear punishment from God or have magnified shame because they perceive themselves as sinful. Symptoms can be more distressful for sufferers with strong religious convictions or beliefs.

Baer believes that blasphemous thoughts are more common in Catholics and evangelical Protestants than in other religions, whereas Jews or Muslims tend to have obsessions related more to complying with the laws and rituals of their faith, and performing the rituals perfectly. He hypothesizes that this is because what is considered inappropriate varies among cultures and religions, and intrusive thoughts torment their sufferers with whatever is considered most inappropriate in the surrounding culture.
Associated conditions

Intrusive thoughts are associated with OCD or obsessive-compulsive personality disorder, but may also occur with other conditions such as post-traumatic stress disorder, clinical depression, postpartum depression, and anxiety. One of these conditions is almost always present in people whose intrusive thoughts reach a clinical level of severity. A large study published in 2005 found that aggressive, sexual, and religious obsessions were broadly associated with comorbid anxiety disorders and depression. The intrusive thoughts that occur in schizophrenia differ from the obsessional thoughts that occur with OCD or depression in that they are false or delusional beliefs.

Post-traumatic stress disorder

The key difference between OCD and post-traumatic stress disorder (PTSD) is that the intrusive thoughts of PTSD sufferers are of traumatic events that actually happened to them, whereas OCD sufferers have thoughts of imagined catastrophes. PTSD patients with intrusive thoughts have to sort out violent, sexual, or blasphemous thoughts from memories of traumatic experiences. When patients with intrusive thoughts do not respond to treatment, physicians may suspect past physical, emotional, or sexual abuse.

Depression

People who are clinically depressed may experience intrusive thoughts more intensely, and view them as evidence that they are worthless or sinful people. The suicidal thoughts that are common in depression must be distinguished from intrusive thoughts, because suicidal thoughts—unlike harmless sexual, aggressive, or religious thoughts—can be dangerous.

Postpartum depression

Unwanted thoughts by mothers about harming their newborn infants are common in postpartum depression. A 1999 study of 65 women with postpartum major depression by Katherine Wisner et al. found the most frequent aggressive thought for women with postpartum depression was causing harm to their newborn infants. A study of 85 new parents found that 89% experienced intrusive images, for example, of the baby suffocating, having an accident, being harmed, or being kidnapped.

Some women may develop symptoms of OCD during pregnancy or the postpartum period. Postpartum OCD occurs mainly in women who may already have OCD, perhaps in a mild or undiagnosed form. Postpartum depression and OCD may be comorbid (often occurring together). And though physicians may focus more on the depressive symptoms, one study found that obsessive thoughts did accompany postpartum depression in 57% of new mothers.

Wisner found common obsessions about harming babies in mothers experiencing postpartum depression include images of the baby lying dead in a casket or being eaten by sharks; drowning, stabbing or throwing the baby down stairs; or putting the baby in the
microwave. Baer estimates that up to 200,000 new mothers with postpartum depression each year may develop these obsessional thoughts about their babies; and because they may be reluctant to share these thoughts with a physician or family member, or suffer in silence and fear they are "crazy", their depression can worsen.

Intrusive fears of harming immediate children can last longer than the postpartum period. A study of 100 clinically depressed women found that 41% had obsessive fears that they might harm their child, and some were afraid to care for their children. Among non-depressed mothers, the study found 7% had thoughts of harming their child—a rate that yields an additional 280,000 non-depressed mothers in the United States with intrusive thoughts about harming their children.

**Frequency**

According to Baer, most people who suffer bad or unacceptable thoughts have not identified themselves as having OCD, because they may not have what they believe to be classic symptoms of OCD, such as handwashing. Yet, he says, epidemiological studies suggest that intrusive thoughts are the most common kind of OCD worldwide; if people in the United States with intrusive thoughts gathered together, they would form the fourth-largest city in the US, following New York City, Los Angeles and Chicago. A 2007 study found that 78% of a clinical sample of OCD patients had intrusive images.

The prevalence of OCD in every culture studied is at least 2% of the population, and the majority of those have obsessions, or bad thoughts, only; this results in a conservative estimate of more than 2 million sufferers in the United States alone (as of 2000). One author estimates that one in 50 adults has OCD and about 10–20% of these have sexual obsessions. A recent study found that 25% of 293 patients with a primary diagnosis of OCD had a history of sexual obsessions.

**Treatment**

Treatment for intrusive thoughts is similar to treatment for OCD. Exposure and response prevention therapy—also referred to as habituation or desensitization—is useful in treating intrusive thoughts. Mild cases can also be treated with cognitive behavioral therapy, which helps patients identify and manage the unwanted thoughts.

**Exposure therapy**

Exposure therapy is the treatment of choice for intrusive thoughts. According to Deborah Osgood-Hynes, Psy.D. Director of Psychological Services and Training at the MGH/McLean OCD Institute, "In order to reduce a fear, you have to face a fear. This is true of all types of anxiety and fear reactions, not just OCD."

Because it is uncomfortable to experience bad thoughts and urges, shame, doubt or fear, the initial reaction is usually to do something to make the feelings diminish. By engaging in a ritual or compulsion to diminish the anxiety or bad feeling, the action is strengthened via
a process called negative reinforcement—the mind learns that the way to avoid the bad feeling is by engaging in a ritual or compulsions. When OCD becomes severe, this leads to more interference in life and continues the frequency and severity of the thoughts the person sought to avoid.

Exposure therapy (or exposure and response prevention) is the practice of staying in an anxiety-provoking or feared situation until the distress or anxiety diminishes. The goal is to reduce the fear reaction, learning to not react to the bad thoughts. This is the most effective way to reduce the frequency and severity of the intrusive thoughts. The goal is to be able to "expose yourself to the thing that most triggers your fear or discomfort for one to two hours at a time, without leaving the situation, or doing anything else to distract or comfort you." Exposure therapy will not completely eliminate intrusive thoughts—everyone has bad thoughts—but most patients find that it can decrease their thoughts sufficiently that intrusive thoughts no longer interfere with their lives.

**Cognitive behavioral therapy**

Cognitive behavioral therapy (CBT) is a newer therapy than exposure therapy, available for those unable or unwilling to undergo exposure therapy. Cognitive therapy has been shown to be useful in reducing intrusive thoughts, but developing a conceptualization of the obsessions and compulsions with the patient is important.

**Pharmaceutical**

Antidepressants or antipsychotic medications may be used for more severe cases, if intrusive thoughts do not respond to cognitive behavioral or exposure therapy alone. Whether the cause of intrusive thoughts is OCD, depression, or post-traumatic stress disorder, the selective serotonin reuptake inhibitor (SSRI) drugs (a class of antidepressants) are the most commonly prescribed. Intrusive thoughts may occur in persons with Tourette syndrome (TS) who also have OCD; the obsessions in TS-related OCD are thought to respond to SSRI drugs as well.

Antidepressants which have been shown to be effective in treating OCD include fluvoxamine (trade name Luvox), fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil), citalopram (Celexa), and clomipramine (Anafranil). Although SSRIs are known to be effective for OCD in general, there have been fewer studies on their effectiveness for intrusive thoughts. A retrospective chart review of patients with sexual symptoms treated with SSRIs showed the greatest improvement was in those with intrusive sexual obsessions typical of OCD. A study of ten patients with religious or blasphemous obsessions found that most patients responded to treatment with fluoxetine or clomipramine. Women with postpartum depression often have anxiety as well, and may need lower starting doses of SSRIs; they may not respond fully to the medication, and may benefit from adding cognitive behavioral or response prevention therapy.
Patients with intense intrusive thoughts that do not respond to SSRIs or other antidepressants may be prescribed typical and atypical neuroleptics including risperidone (trade name Risperdal), ziprasidone (Geodon), haloperidol (Haldol), and pimozide (Ora).n

Studies suggest that therapeutic doses of inositol may be useful in the treatment of obsessive thoughts.

**Purely Obsessional OCD**

Purely Obsessional Obsessive-Compulsive Disorder (also called Pure Obsessional OCD, Pure-O, OCD without overt compulsions or Primarily Obsessional OCD) is a lesser-known form or manifestation of OCD. For people with Purely Obsessional OCD, there are usually no observable compulsions, such as those commonly seen in those with the typical form of OCD (checking, counting, hand-washing, etc.). While ritualizing and neutralizing behaviors do take place, they are almost entirely in the form of excessive mental rumination.

**Common themes**

The nature and type of Purely Obsessional OCD varies greatly, but the central theme for all sufferers is the emergence of a disturbing intrusive thought or question, an unwanted/inappropriate mental image, or a frightening impulse that causes the person extreme anxiety because it is antithetical to closely-held religious beliefs, morals, or societal mores. While those without Purely Obsessional OCD might instinctively respond to bizarre intrusive thoughts or impulses, in the form of repeated checking or hand-washing, etc., someone with Purely Obsessional OCD will respond with profound alarm followed by an intense attempt to neutralize the thought or avoid having the thought again. The person begins to ask themselves constantly "Am I really capable of something like that?" or "Could that really happen?" or "Is that really me?" (even though they usually realize that their fear is irrational, which causes them further distress) and puts tremendous effort into escaping or resolving the unwanted thought. They then end up in a vicious cycle of mentally searching for reassurance and trying to get a definitive answer.

**Common intrusive thoughts/obsessions include themes of:**

- **Responsibility**: with an excessive concern over someone’s well-being marked specifically by guilt over believing they have harmed or might harm (either on purpose or inadvertently) someone.
- **Sexuality**: including recurrent doubt over one’s sexual orientation (also called HOCD or "homosexual OCD"). People with this theme display a very different set of symptoms than those actually experiencing an actual crisis in sexuality. The question "Am I gay" takes on a pathological form. Many people with this type of obsession are in healthy and fulfilling romantic relationships, either with members of the opposite sex, or the same sex (in which case their fear would be "Am I straight?").
- **Violence**: which involves a constant fear of violently harming oneself or loved ones or persistent worry that one is a pedophile and might harm a child.
- **Religiosity**: manifesting as intrusive thoughts or impulses revolving around blasphemous and sacrilegious themes.
- **Health**: including consistent fears of having or contracting a disease (different from hypochondriasis) through seemingly impossible means (for example, touching an object that has just been touched by someone with a disease) or mistrust of a diagnostic test.
- **Relationship Substantiation**: in which someone in a romantic relationship endlessly tried to ascertain the justification for being or remaining in that relationship. It includes obsessive thoughts to the tune of "How do I know this is real love?" "How do I know he/she is the one?" "Am I attracted enough to this person?" or "Am I in love with this person, or is it just love?" The agony of attempting to arrive at certainty leads to an intense and endless cycle of anxiety because it is impossible to arrive at a definite answer.

**Diagnosis and treatment**

Those suffering from Purely Obsessional OCD might appear normal and high-functioning, yet spend a great deal of time ruminating, trying to solve or answer any of the questions that cause them distress.

For example, an intrusive thought "I could just kill Bill with this steak knife" is followed by a catastrophic misinterpretation of the thought, i.e. "How could I have such a thought? Deep down, I must be a psychopath." This might lead a person to continually surf the web, reading numerous articles on defining psychopathy. This reassurance-seeking ritual will, ironically, provide no further clarification and could exacerbate the intensity of the search for the answer. There are numerous corresponding cognitive biases present, including thought-action fusion, over-importance of thoughts, and need for control over thoughts.

The disorder is particularly easy to miss by many well-trained clinicians, as it closely resembles markers of generalized anxiety disorder and does not include observable, compulsive behaviors. Clinical "success" is reached when the Purely Obsessional OCD sufferer becomes indifferent to the need to answer the question. While many clinicians will mistakenly offer reassurance and try to help their patient achieve a definitive answer (an unfortunate consequence of therapists treating Purely Obsessional OCD as generalized anxiety disorder), this method only contributes to the intensity or length of the patient’s rumination, as the neuropathways of the OCD brain will predictably come up with creative ways to "trick" the person out of reassurance, negating any temporary relief and perpetuating the cycle of obsessing.

The most effective treatment for Purely Obsessional OCD appears to be Cognitive-Behavioral Therapy. More specifically exposure and response prevention (ERP) as well as Cognitive Therapy (CT) which may or may not be combined with the use of medication, such as SSRIs. People suffering from OCD without overt compulsions are considered by
some researchers more refractory towards ERP compared to other OCD sufferers and therefore ERP can prove less successful than CT.

Exposure and response prevention (ERP) of the "Pure-O" is theoretically based on the principles of classical conditioning and extinction. The spike often presents itself as a paramount question or disastrous scenario. A response that answers the spike in a way that leaves ambiguity is sometimes warranted. "If I don't remember what I had for breakfast yesterday my mother will die of cancer!" Using the antidote procedure, a cognitive response would be one in which the subject accepts this possibility and is willing to take the risk of his mother dying of cancer or the question recurring for eternity. No effort is expended in directly answering the question in an effort to find resolution. In another example, the spike would be, "Maybe I said something offensive to my boss yesterday." A recommended response would be, "Maybe I did. I'll live with the possibility and take the risk he'll fire me tomorrow." Using this procedure, it is imperative that the distinction be made between the therapeutic response and rumination. The therapeutic response does not seek to answer the question but to accept the uncertainty of the unsolved dilemma.

**Posttraumatic stress disorder**

Posttraumatic stress disorder (also known as post-traumatic stress disorder or PTSD) is a severe anxiety disorder that can develop after exposure to any event that results in psychological trauma. This event may involve the threat of death to oneself or to someone else, or to one's own or someone else's physical, sexual, or psychological integrity, overwhelming the individual's ability to cope. As an effect of psychological trauma, PTSD is less frequent and more enduring than the more commonly seen acute stress response.

Diagnostic symptoms for PTSD include re-experiencing the original trauma(s) through flashbacks or nightmares, avoidance of stimuli associated with the trauma, and increased arousal – such as difficulty falling or staying asleep, anger, and hypervigilance. Formal diagnostic criteria (both DSM-IV-TR and ICD-10) require that the symptoms last more than one month and cause significant impairment in social, occupational, or other important areas of functioning.

**Classification**

Posttraumatic stress disorder is classified as an anxiety disorder, characterized by aversive anxiety-related experiences, behaviors, and physiological responses that develop after exposure to a psychologically traumatic event (sometimes months after). Its features persist for longer than 30 days, which distinguishes it from the briefer acute stress disorder. These persisting posttraumatic stress symptoms cause significant disruptions of one or more important areas of life function. It has three sub-forms: acute, chronic, and delayed-onset.
Causes

Psychological trauma

PTSD is believed to be caused by either physical trauma or psychological trauma, or more frequently a combination of both. According to Atkinson et al. (2000) PTSD is more likely to be caused by physical or psychological trauma caused by humans such as rape, war, or terrorist attack than trauma caused by natural disasters. Possible sources of trauma include experiencing or witnessing childhood or adult physical, emotional or sexual abuse. In addition, experiencing or witnessing an event perceived as life-threatening such as physical assault, adult experiences of sexual assault, accidents, drug addiction, illnesses, medical complications, or employment in occupations exposed to war (such as soldiers) or disaster (such as emergency service workers).

Traumatic events that may cause PTSD symptoms to develop include violent assault, kidnapping, sexual assault, torture, being a hostage, prisoner of war or concentration camp victim, experiencing a disaster, violent automobile accidents or getting a diagnosis of a life-threatening illness. Children or adults may develop PTSD symptoms by experiencing bullying or mobbing. Preliminary research suggests that child abuse may interact with mutations in a stress-related gene to increase the risk of PTSD in adults.

Multiple studies show that parental PTSD and other posttraumatic disturbances in parental psychological functioning can, despite a traumatized parent’s best efforts, interfere with their response to their child as well as their child's response to trauma. Parents with violence-related PTSD may, for example, inadvertently expose their children to developmentally inappropriate violent media due to their need to manage their own emotional dysregulation. Clinical findings indicate that a failure to provide adequate treatment to children after they suffer a traumatic experience, depending on their vulnerability and the severity of the trauma, will ultimately lead to PTSD symptoms in adulthood.

Neuroendocrinology

PTSD symptoms may result when a traumatic event causes an overactive adrenaline response, which creates deep neurological patterns in the brain. These patterns can persist long after the event that triggered the fear, making an individual hyper-responsive to future fearful situations.

PTSD displays biochemical changes in the brain and body that differ from other psychiatric disorders such as major depression. Individuals diagnosed with PTSD respond more strongly to a dexamethasone suppression test than individuals diagnosed with clinical depression.

In addition, most people with PTSD also show a low secretion of cortisol and high secretion of catecholamines in urine, with a norepinephrine/cortisol ratio consequently higher than comparable non-diagnosed individuals. This is in contrast to the normative fight-or-flight
response, in which both catecholamine and cortisol levels are elevated after exposure to a stressor.

Brain catecholamine levels are low, and corticotropin-releasing factor (CRF) concentrations are high. Together, these findings suggest abnormality in the hypothalamic-pituitary-adrenal (HPA) axis.

Given the strong cortisol suppression to dexamethasone in PTSD, HPA axis abnormalities are likely predicated on strong negative feedback inhibition of cortisol, itself likely due to an increased sensitivity of glucocorticoid receptors. Some researchers have associated the response to stress in PTSD with long-term exposure to high levels of norepinephrine and low levels of cortisol, a pattern associated with improved learning in animals.

Translating this reaction to human conditions gives a pathophysiological explanation for PTSD by a maladaptive learning pathway to fear response through a hypersensitive, hyperreactive and hyperresponsive HPA axis.

Low cortisol levels may predispose individuals to PTSD: Following war trauma, Swedish soldiers serving in Bosnia and Herzegovina with low pre-service salivary cortisol levels had a higher risk of reacting with PTSD symptoms, following war trauma, than soldiers with normal pre-service levels. Because cortisol is normally important in restoring homeostasis after the stress response, it is thought that trauma survivors with low cortisol experience a poorly contained—that is, longer and more distressing—response, setting the stage for PTSD.

However, there is considerable controversy within the medical community regarding the neurobiology of PTSD. A review of existing studies on this subject showed no clear relationship between cortisol levels and PTSD. Only a slight majority have found a decrease in cortisol levels while others have found no effect or even an increase.

**Neuroanatomy**

[Regions of the brain associated with stress and posttraumatic stress disorder]
Three areas of the brain whose function may be altered in PTSD have been identified: the prefrontal cortex, amygdala and hippocampus. Much of this research has utilised PTSD victims from the Vietnam War. For example, a prospective study using the Vietnam Head Injury Study showed that damage to the prefrontal cortex may actually be protective against later development of PTSD. In a study by Gurvits et al., combat veterans of the Vietnam War with PTSD showed a 20% reduction in the volume of their hippocampus compared with veterans who suffered no such symptoms. This finding could not be replicated in chronic PTSD patients traumatized at an air show plane crash in 1988 (Ramstein, Germany).

In human studies, the amygdala has been shown to be strongly involved in the formation of emotional memories, especially fear-related memories. Neuroimaging studies in humans have revealed both morphological and functional aspects of PTSD.

The amygdalocentric model of PTSD proposes that it is associated with hyperarousal of the amygdala and insufficient top-down control by the medial prefrontal cortex and the hippocampus particularly during extinction. This is consistent with an interpretation of PTSD as a syndrome of deficient extinction ability. Further animal and clinical research into the amygdala and fear conditioning may suggest additional treatments for the condition.

**Genetics**

There is evidence that susceptibility to PTSD is hereditary. For twin pairs exposed to combat in Vietnam, having a monozygotic (identical) twin with PTSD was associated with an increased risk of the co-twin having PTSD compared to twins that were dizygotic (non-identical twins).

Recently, it has been found that several single-nucleotide polymorphisms (SNPs) in FK506 binding protein 5 (FKBP5) interact with childhood trauma to predict severity of adult PTSD. These findings suggest that individuals with these SNPs who are abused as children are more susceptible to PTSD as adults.

This is particularly interesting given that FKBP5 SNPs have previously been associated with peritraumatic dissociation (that is, dissociation at the time of the trauma), which has itself been shown to be predictive of PTSD. Furthermore, FKBP5 may be less expressed in those with current PTSD. Another recent study found a single SNP in a putative estrogen response element on ADCYAP1R1 (encodes pituitary adenylate cyclase-activating polypeptide type I receptor or PAC1) to predict PTSD diagnosis and symptoms in females. Incidentally, this SNP is also associated with fear discrimination. The study suggests that perturbations in the PACAP-PAC1 pathway are involved in abnormal stress responses underlying PTSD.

**Risk factors**
Although most people (50-90%) encounter trauma over a lifetime, only about 8% develop full PTSD. Vulnerability to PTSD presumably stems from an interaction of biological diathesis, early childhood developmental experiences, and trauma severity.

Predictor models have consistently found that childhood trauma, chronic adversity, and familial stressors increase risk for PTSD as well as risk for biological markers of risk for PTSD after a traumatic event in adulthood. This effect of childhood trauma, which is not well understood, may be a marker for both traumatic experiences and attachment problems. Proximity to, duration of, and severity of the trauma also make an impact; and interpersonal traumas cause more problems than impersonal ones.

**Military experience**

Schnurr, Lunney, and Sengupta identified risk factors for the development of PTSD in Vietnam veterans. Among those are:

- Hispanic ethnicity, coming from an unstable family, being punished severely during childhood, childhood asocial behavior and depression as pre-military factors
- War-zone exposure, peritraumatic dissociation, depression as military factors
- Recent stressful life events, post-Vietnam trauma and depression as post-military factors

They also identified certain protective factors, such as:

- Japanese-American ethnicity, high school degree or college education, older age at entry to war, higher socioeconomic status and a more positive paternal relationship as pre-military protective factors
- Social support at homecoming and current social support as post-military factors.

Other research also indicates the protective effects of social support in averting PTSD or facilitating recovery if it develops.

There may also be an attitudinal component; for example, a soldier who believes that they will not sustain injuries may be more likely to develop symptoms of PTSD than one who anticipates the possibility, should either be wounded. Likewise, the later incidence of suicide among those injured in home fires above those injured in fires in the workplace suggests this possibility.

**Foster care**

In the Casey Family Northwest Alumni Study, conducted in conjunction with researchers from the Harvard Medical School in Oregon and Washington state, the rate of PTSD in adults who were in foster care for one year between the ages of 14-18 was found to be higher than that of combat veterans. Up to 25 percent of those in the study meet the diagnostic criteria for PTSD as compared to 12-13 percent of Iraq war veterans and 15 percent of Vietnam War veterans, and a rate of 4 percent in the general population. The
recovery rate for foster home alumni was 28.2% as opposed to 47% in the general population.

In one study (Dubner and Motta, 1999), 60% of children in foster care who had experienced sexual abuse had PTSD, and 42% of those who had been physically abused fulfilled the PTSD criteria. PTSD was also found in 18% of the children who were not abused. These children may have developed PTSD due to witnessing violence in the home, or as a result of real or perceived parental abandonment.

**Diagnosis**

**Criteria**

The diagnostic criteria for PTSD, stipulated in the Diagnostic and Statistical Manual of Mental Disorders IV (Text Revision) (DSM-IV-TR), may be summarized as:

**A: Exposure to a traumatic event**

This must have involved both (a) loss of "physical integrity", or risk of serious injury or death, to self or others, and (b) a response to the event that involved intense fear, horror or helplessness (or in children, the response must involve disorganized or agitated behavior). (The DSM-IV-TR criterion differs substantially from the previous DSM-III-R stressor criterion, which specified the traumatic event should be of a type that would cause "significant symptoms of distress in almost anyone," and that the event was "outside the range of usual human experience.")

**B: Persistent re-experiencing**

One or more of these must be present in the victim: flashback memories, recurring distressing dreams, subjective re-experiencing of the traumatic event(s), or intense negative psychological or physiological response to any objective or subjective reminder of the traumatic event(s).

**C: Persistent avoidance and emotional numbing**

This involves a sufficient level of:

- avoidance of stimuli associated with the trauma, such as certain thoughts or feelings, or talking about the event(s);
- avoidance of behaviors, places, or people that might lead to distressing memories;
- inability to recall major parts of the trauma(s), or decreased involvement in significant life activities;
- decreased capacity (down to complete inability) to feel certain feelings;
- an expectation that one's future will be somehow constrained in ways not normal to other people.
D: Persistent symptoms of increased arousal not present before

These are all physiological response issues, such as difficulty falling or staying asleep, or problems with anger, concentration, or hypervigilance.

E: Duration of symptoms for more than 1 month

If all other criteria are present, but 30 days have not elapsed, the individual is diagnosed with Acute stress disorder.

F: Significant impairment

The symptoms reported must lead to "clinically significant distress or impairment" of major domains of life activity, such as social relations, occupational activities, or other "important areas of functioning".

Assessment

Since the introduction of DSM-IV, the number of possible events which might be used to diagnose PTSD has increased; one study suggests that the increase is around 50%. Various scales exist to measure the severity and frequency of PTSD symptoms. Standardized screening tools such as Trauma Screening Questionnaire and PTSD Symptom Scale can be used to detect possible symptoms of posttraumatic stress disorder, and suggest the need for a formal diagnostic assessment.

Research-based alternative symptom groups

Emerging factor analytic research suggests that PTSD symptoms group empirically into four clusters, not the three currently described in the Diagnostic and Statistical Manual of Mental Disorders. One model supported by this research divides the traditional avoidance symptoms into a cluster of numbing symptoms (such as loss of interest and feeling emotionally numb) and a cluster of behavioral avoidance symptoms (such as avoiding reminders of the trauma). An alternative model adds a fourth cluster of dysphoric symptoms. These include symptoms of emotional numbing, as well as anger, sleep disturbance, and difficulty concentrating (traditionally grouped under the hyperarousal cluster).

DSM-5 proposed diagnostic criteria changes

In preparation for the May 2013 release of the DSM-5, the fifth version of the American Psychiatric Association's diagnostic manual, draft diagnostic criteria was released for public comment, followed by a two-year period of field testing. Proposed changes to the criteria include:
- Criterion A (prior exposure to traumatic events) is more specifically stated, and evaluation of an individual’s emotional response at the time (current criterion A2) is dropped.
- Several items in Criterion B (intrusion symptoms) are rewritten to add or augment certain distinctions now considered important.
- Special consideration is given to developmentally appropriate criteria for use with children and adolescents. This is especially evident in the restated Criterion B - intrusion symptoms. Development of age-specific criteria for diagnosis of PTSD is ongoing at this time.
- Criterion C (avoidance and numbing) has been split into "C" and "D":
- Criterion C (new version) now focuses solely on avoidance of behaviors or physical or temporal reminders of the traumatic experience(s). What were formerly two symptoms are now three, due to slight changes in descriptions.
- New Criterion D focuses on negative alterations in cognition and mood associated with the traumatic event(s), and contains two new symptoms, one expanded symptom, and four largely unchanged symptoms specified in the previous criteria.
- Criterion E (formerly "D"), which focuses on increased arousal and reactivity, contains one modestly revised, one entirely new, and four unchanged symptoms.
- Criterion F (formerly "E") still requires duration of symptoms to have been at least one month.
- Criterion G (formerly "F") stipulates symptom impact ("disturbance") in the same way as before.
- The "acute" vs "delayed" distinction is dropped; the "delayed" specifier is considered appropriate if clinical symptom onset is no sooner than 6 months after the traumatic event(s).

"Developmental trauma disorder", a proposed new diagnosis, was still under discussion at the time of the draft publication.

**Public policy response**

In recent history, catastrophes (by human means or not) such as the 2004 Indian Ocean tsunami may have caused PTSD in many survivors and rescue workers. Today relief workers from organizations such as the Red Cross and the Salvation Army provide counseling after major disasters as part of their standard procedures to curb severe cases of posttraumatic stress disorder.

**United States**

A review of the provision of compensation to veterans for PTSD by the United States Department of Veterans Affairs began in 2005 after the VA had noted a 30% increase in PTSD claims in recent years. This led to a backlash from veterans’-rights groups, and to some highly publicized suicides by veterans who feared losing their benefits, which in some cases constituted their only income. In response, on November 10, 2005, the Secretary of Veterans Affairs announced that “the Department of Veterans Affairs (VA) will not review
the files of 72,000 veterans currently receiving disability compensation for posttraumatic stress disorder...

The diagnosis of PTSD in U.S. military veterans has been a subject of some controversy due to uncertainties in objectively diagnosing PTSD in those who may have been exposed to trauma, and due to this diagnosis' association with some incidence of compensation-seeking behavior.

Many veterans of the wars in Iraq and Afghanistan returning home have faced significant physical, emotional and relational disruptions. In response, the United States Marine Corps has instituted programs to assist them in re-adjusting to civilian life, especially in their relationships with spouses and loved ones, to help them communicate better and understand what the other has gone through. Walter Reed Army Institute of Research (WRAIR) developed the Battlemind program to assist service members avoid or ameliorate PTSD and related problems.

Other countries

In the UK, there has been some controversy that National Health Service is dumping veterans on service charities like Combat Stress.

Veterans Affairs Canada offers a new program that includes rehabilitation, financial benefits, job placement, health benefits program, disability awards and family support.

Management

Prevention and early intervention strategies

Modest benefits have been seen from early access to cognitive behavioral therapy, as well as from some medications such as propranolol. Critical incident stress management has been suggested as a means of preventing PTSD but subsequent studies suggest the likelihood of its producing iatrogenic outcomes. A review of multiple studies confirmed the finding of no benefit to trauma survivors from single-session early-response interventions, as well as a failure of blanket multiple-session prevention interventions to yield a benefit to all participants (some were even harmed).

Early detection

The ability to prescreen individuals would be of great help in getting treatment to those who are at risk of PTSD prior to development of the syndrome. Several biological indicators have been identified that are related to later PTSD development. First, Delhanty found that higher response times and a smaller hippocampal volume were identified as linked to later PTSD development. However, both of these indicators are relatively difficult to test for and need specialized tests and or equipment to identify. A blood biomarker is much easier to test for. Van Zuiden et al. found just such a biomarker when testing U.S. Army soldiers prior to deployment. They found that soldiers with more glucocorticoid receptors (GR) were more likely to be diagnosed with PTSD six months after deployment. However, higher GR
levels have not been identified as a cause of PTSD, and may instead be an intermediary, or even an indicator that the individual has previously experienced traumatic events. There is a great deal of overlap between high GR levels and those who later are diagnosed with and without PTSD. Thus, the identification of high GR is simply a vulnerability indicator at this time.

Delaney found that biological precursors existed directly following traumatic exposure in those who later developed chronic PTSD and were significantly different from those who did not. Directly following the traumatic event later sufferers often have significantly lower levels of hypothalamic pituitary-adrenocortical activity and a corresponding decrease in Cortisol. Other methods of early detection include the identification of specific risk factors associated with later PTSD symptoms. Resnick, Acierno, Holmes, Kilpatrick, and Jager for example were able to identify that the forensic exam given to victims after a rape was associated with PTSD. Finally, global treatments attempt to avoid the problems of early detection by simply treating everyone involved. However, many studies have found this to be often ineffective and for global treatments to at times increase prevalence rates of PTSD.

**Preventive Treatments**

**Psychological debriefing**

The first form of preventive treatment is that of a psychological debriefing. Psychological debriefing is the most often used preventive measure. One of the main reasons for this is the relative ease with which this treatment can be given to individuals directly following an event. It consists of interviews that are meant to allow individuals to directly confront the event and share their feelings with the counselor and to help structure their memories of the event. However, while this form of therapy is the most often used it is actually the least effective. Studies have had mixed findings concerning psychological debriefings and have ranged from being of significant help to helping in the formation of PTSD in individuals who would otherwise have not developed PTSD. The greater number of studies tends to simply find that it is neither overly beneficial nor harmful.

**Risk Targeted Interventions**

Risk targeted interventions are those that attempt to mitigate specific formative information or events. It can target modeling normal behaviors, instruction on a task or giving information on the event. For example, rape victims were given an instruction video on the procedures for a forensic exam. Also included in the video was advice on how to identify and stop avoidance behavior and control anxiety. Finally, the individuals modeling the forensic exam were shown to be calm and relaxed. PTSD diagnosis for those who saw the video were thirty three percent less than for those who went through the standard forensic procedure.

**Psychobiological Treatments**
Psychobiological treatments have also found success, especially with cortisol. Psychobiological treatments target biological changes that occur after a traumatic event. They also attempt to chemically alter learning or memory formation. Cortisol treatments after a traumatic event have found success in mitigating later diagnosis of PTSD. As discussed earlier Cortisol is often lower in individuals who are at risk of PTSD after a traumatic event than their counterparts. By increasing cortisol levels to normal levels this has been shown to reduce arousal post event as well prevent GR upregulation.

**Stepped Collaborative Care**

Stepped collaborative care is where individuals who are at risk are monitored for symptoms. As symptoms of PTSD appear the level of care is increased to treat those symptoms.

**Psychotherapeutic interventions**

Many forms of psychotherapy have been advocated for trauma-related problems such as PTSD. Basic counseling practices common to many treatment responses for PTSD include education about the condition and provision of safety and support.

The psychotherapy programs with the strongest demonstrated efficacy include cognitive behavioral programs, variants of exposure therapy, stress inoculation training (SIT), variants of cognitive therapy (CT), eye movement desensitization and reprocessing (EMDR), and many combinations of these procedures. A 2010 review disagrees that these treatments have proven efficacy, and points out methodological flaws in the studies and previous meta-analyses.

EMDR or trauma-focused cognitive behavioral therapy (TFCBT) was recommended as first-line treatments for trauma victims in a 2007 review; however, "the evidence base [for EMDR] was not as strong as that for TFCBT ... Furthermore, there was limited evidence that TFCBT and EMDR were superior to supportive/non-directive treatments, hence it is highly unlikely that their effectiveness is due to non-specific factors such as attention." A meta-analytic comparison of EMDR and cognitive behavioral therapy found both protocols indistinguishable in terms of effectiveness in treating PTSD; however "the contribution of the eye movement component in EMDR to treatment outcome" is unclear.

**Behavioral and Cognitive Behavioral therapy**

Cognitive Behavioral Therapy (CBT) seeks to change the way a trauma victim feels and acts by changing the patterns of thinking and/or behavior responsible for negative emotions. CBT have been proven to be an effective treatment for PTSD, and is currently considered the standard of care for PTSD by the United States Department of Defense In CBT, individuals learn to identify thoughts that make them feel afraid or upset, and replace them with less distressing thoughts. The goal is to understand how certain thoughts about cause PTSD-related stress.
Recent research on contextually based third-generation behavior therapies suggests that they may produce results comparable to some of the better validated therapies. Many of these therapy methods have a significant element of exposure, and have demonstrated success in treating the primary problems of PTSD and co-occurring depressive symptoms.

Exposure therapy is a type of cognitive behavioral therapy that involves assisting trauma survivors to re-experience distressing trauma-related memories and reminders in order to facilitate habituation and successful emotional processing of the trauma memory. Most exposure therapy programs include both imaginal confrontation with the traumatic memories and real-life exposure to trauma reminders; this therapy modality is well supported by clinical evidence. Indeed, the success of exposure-based therapies has raised the question of whether exposure is a necessary ingredient in the treatment of PTSD. Some organizations have endorsed the need for exposure. The US Department of Veterans Affairs has been actively training mental health treatment staff in Prolonged Exposure Therapy and Cognitive Processing Therapy in an effort to better treat US Veterans with PTSD.

**Eye movement desensitization and reprocessing**

Eye movement desensitization and reprocessing (EMDR) is specifically targeted as a treatment for PTSD. Based on the evidence of controlled research, the American Psychiatric Association and the United States Department of Veterans Affairs and Department of Defense have placed EMDR in the highest category of effectiveness and research support in the treatment of trauma. Several international bodies have made similar recommendations. However, some reviewers no longer believe that the eye movements assist in recovery, proposing instead that the review of and engagement with memories, processing of cognitions, and rehearsal of coping skills are the psychotherapeutically effective components of the procedure.

**Interpersonal psychotherapy**

Other approaches, particularly involving social supports, may also be important. An open trial of interpersonal psychotherapy reported high rates of remission from PTSD symptoms without using exposure. A current, NIMH-funded trial in New York City is now (and into 2013) comparing interpersonal psychotherapy, prolonged exposure therapy, and relaxation therapy.

**Medication**

A variety of medications has shown adjunctive benefit in reducing PTSD symptoms, but "there is no clear drug treatment for PTSD". Positive symptoms (re-experiencing, hypervigilance, increased arousal) generally respond better to medication than negative symptoms (avoidance, withdrawal), and it is recommended that any drug trial last for at least 6–8 weeks.

**Symptom management: potentially useful medication classes**
SSRIs (selective serotonin reuptake inhibitors). SSRIs are considered to be a first-line drug treatment. SSRIs for which there are data to support use include: citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, and sertraline.

Among the anti-depressants described in this section, bupropion and venlafaxine have the lowest patient drop-out rates. Sertraline, fluoxetine, and nefazodone have a modestly higher drop-out rate (~15%), and the heterocyclics and paroxetine have the highest rates (~20%+). Where drop-out is caused or feared because of medication side-effects, it should be remembered that most patients do not experience such side-effects.

Alpha-adrenergic antagonists. Prazosin ("Minipress"), in a small study of combat veterans, has shown substantial benefit in relieving or reducing nightmares. Clonidine ("Catapres") can be helpful with startle, hyperarousal, and general autonomic hyperexcitability.

Anti-convulsants, mood stabilizers, anti-aggression agents. Carbamazepine ("Tegretol") has likely benefit in reducing arousal symptoms involving noxious affect, as well as mood or aggression. Topiramate ("Topamax") has been effective in achieving major reductions in flashbacks and nightmares, and no reduction of effect was seen over time. Zolpidem ("Ambien") has also proven useful in treating sleep disturbances.

Lamotrigine ("Lamictal") may be useful in reducing reexperiencing symptoms, as well as avoidance and emotional numbing. Valproic acid ("Depakene") and has shown reduction of symptoms of irritability, aggression, and impulsiveness, and in reducing flashbacks. Similarly, lithium carbonate has worked to control mood and aggressions (but not anxiety) symptoms. Buspirone ("BuSpar") has an effect similar to that of lithium, with the additional benefit of working to reduce hyperarousal symptoms.

Antipsychotics. Risperidone can be used to help with dissociation, mood issues, and aggression.

Atypical antidepressants. Nefazodone ("Serzone") can be effective with sleep disturbance symptoms, and with secondary depression, anxiety, and sexual dysfunction symptoms. Trazodone ("Desyrel") can also reduce or eliminate problems with disturbed sleep, and with anger and anxiety.

Beta blockers. Propranolol ("Inderal") has demonstrated possibilities in reducing hyperarousal symptoms, including sleep disturbances.

Benzodiazepines. These can be used with caution for short-term anxiety relief, hyperarousal, and sleep disturbance. While benzodiazepines can alleviate acute anxiety, there is no consistent evidence that they can stop the development of PTSD, or are at all effective in the treatment of posttraumatic stress disorder. Additionally benzodiazepines may reduce the effectiveness of psychotherapeutic interventions and there is some evidence that benzodiazepines may contribute to the development and chronification of PTSD. Other drawbacks include the risk of developing a benzodiazepine dependence and
withdrawal syndrome; additionally individuals with PTSD are at an increased risk of abusing benzodiazepines.

Glucocorticoids. Additionally, post-stress high dose corticosterone administration was recently found to reduce 'PTSD-like' behaviors in a rat model of PTSD. In this study, corticosterone impaired memory performance, suggesting that it may reduce risk for PTSD by interfering with consolidation of traumatic memories. The neurodegenerative effects of the glucocorticoids, however, may prove this treatment counterproductive.

Heterocyclic / Tricyclic anti-depressants anti-depressants. Amitriptyline ("Elavil") has shown benefit for positive distress symptoms, and for avoidance, and Imipramine ("Tofranil") has shown benefit for intrusive symptoms.

Monoamine-oxidase inhibitors (MAOs). Phenelzine ("Nardil") has for some time been observed to be effective with hyperarousal and depression, and is especially effective with nightmares.

Miscellaneous other medications. Clinical trials evaluating methylenedioxymethamphetamine (MDMA, "Ecstasy") in conjunction with psychotherapy are being conducted in Switzerland and Israel.

**Symptom prevention: potentially useful medication classes**

Some medications have shown benefit in preventing PTSD or reducing its incidence, when given in close proximity to a traumatic event. These medications include:

Alpha-adrenergic antagonists. Anecdotal report of success in using clonidine ("Catapres") to reduce traumatic stress symptoms suggests that it may have benefit in preventing PTSD.

Beta blockers. Propranolol ("Inderal"), similarly to clonidine, may be useful if there are significant symptoms of "over-arousal". These may inhibit the formation of traumatic memories by blocking adrenaline's effects on the amygdala.

Glucocorticoids. There is some evidence suggesting that administering glucocorticoids immediately after a traumatic experience may help prevent PTSD. Several studies have shown that individuals who receive high doses of hydrocortisone for treatment of septic shock, or following surgery, have a lower incidence and fewer symptoms of PTSD.

Opiates. In a retrospective analysis of combat injury field data for US troops in Iraq, it was found that those who received morphine in the early stages of their treatment had a significantly lower rate of subsequent PTSD, when compared with those who did not receive morphine at that time.

**Medications by symptom group affected**
Medications can affect one or more of the symptoms, in one or more of the three major symptom classes involved in diagnosing PTSD, which can be summarized in the following table:

**Symptom class Symptom Medication**

<table>
<thead>
<tr>
<th>Symptom class</th>
<th>Symptom</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reexperiencing</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>intrusive recall</td>
<td></td>
<td>amitriptyline; fluoxetine; imipramine; lamotrigine; sertraline</td>
</tr>
<tr>
<td>intrusive reexperiencing</td>
<td></td>
<td>amitriptyline; fluoxetine; imipramine; nefazodone; sertraline (women only); topiramate;</td>
</tr>
<tr>
<td>sleep disturbance, nightmares</td>
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<td>benzodiazepines; carbamazepine; clonidine; nefazodone; phenelzine; prazosin; topiramate; trazodone; zolpidem</td>
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<tr>
<td>dissociative recall</td>
<td></td>
<td>risperidone</td>
</tr>
<tr>
<td>intense psychological distress (anger, anxiety) when exposed to reminders of traumatic event(s)</td>
<td></td>
<td>benzodiazepines; buspirone; carbamazepine; lithium (not for anxiety); nefazodone; trazodone</td>
</tr>
<tr>
<td><strong>Avoidance</strong></td>
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<td></td>
</tr>
<tr>
<td>avoidance</td>
<td></td>
<td>amitriptyline; fluoxetine; lamotrigine; nefazodone; sertraline</td>
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<tr>
<td>feelings of detachment or estrangement from others</td>
<td></td>
<td>amitriptyline; risperidone</td>
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<tr>
<td>restricted range of affect (numbing)</td>
<td></td>
<td>amitriptyline; lamotrigine; sertraline (women only)</td>
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<tr>
<td><strong>Hyperarousal</strong></td>
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<tr>
<td>general hyperarousal</td>
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<tr>
<td>sleep disturbance, nightmares</td>
<td></td>
<td>benzodiazepines; carbamazepine; clonidine; nefazodone; phenelzine; trazodone; zolpidem</td>
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<tr>
<td>irritability, anger (and impulsiveness)</td>
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<td>carbamazepine; nefazodone; valproic acid</td>
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<td>anger</td>
<td></td>
<td>buspirone; fluoxetine; lithium; trazodone</td>
</tr>
<tr>
<td>aggression</td>
<td></td>
<td>risperidone</td>
</tr>
<tr>
<td>exaggerated startle response; general autonomic hyperexcitability</td>
<td></td>
<td>benzodiazepines; buspirone; carbamazepine; clonidine; propranolol; valproic acid</td>
</tr>
</tbody>
</table>
Some medications can also help with symptoms which may occur secondary to PTSD.

<table>
<thead>
<tr>
<th>Secondary symptom</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>depression</td>
<td>nefazodone; phenelzine</td>
</tr>
<tr>
<td>dream content distortions</td>
<td>nefazodone</td>
</tr>
<tr>
<td>relapse of symptoms</td>
<td>carbamazepine; clonidine; buprenorphine</td>
</tr>
<tr>
<td>self-mutilation</td>
<td>clonidine</td>
</tr>
<tr>
<td>sexual function reduction</td>
<td>nefazodone</td>
</tr>
<tr>
<td>sleep hours reduction</td>
<td>nefazodone</td>
</tr>
</tbody>
</table>

**Medication and self-medication issues and risks with PTSD**

Alcohol abuse and drug abuse commonly co-occur with PTSD. Recovery from posttraumatic stress disorder or other anxiety disorders may be hindered, or the condition worsened, by medication or substance overuse, abuse, or dependence; resolving these problems can bring about a marked improvement in an individual’s mental health status and anxiety levels.

Benzodiazepines are risky in several ways. They can be especially addictive when PTSD is present, and this is especially true with the fast-acting ones. Dis-inhibition upon initiation of treatment is another risk with this medication class. Finally, termination of the drug can be especially difficult. Recovery from benzodiazepine abuse or dependence tends to take a lot longer than recovery from alcohol abuse or dependence, but people can regain their previous good health. PTSD symptoms may temporarily worsen however, during alcohol withdrawal or benzodiazepine withdrawal.

Yohimbine (not considered specifically appropriate for PTSD) increases arousal by increasing release of endogenous norepinephrine, and can worsen PTSD symptoms.

**Epidemiology**

There is debate over the rates of PTSD found in populations, but despite changes in diagnosis and the criteria used to define PTSD between 1997 and 2007, epidemiological rates have not changed significantly.

**International PTSD rates**

The United Nations' World Health Organization publishes estimates of PTSD impact for each of its member states; the latest data available are for 2004. Considering only the 25 most populated countries, ranked by overall age-standardized Disability-Adjusted Life Year (DALY) rate, the top half of the ranked list is dominated by Asian/Pacific countries, the USA, and Egypt. Ranking the countries by the male-only or female-only rates produces much the same result, but with less meaningfulness, as the score range in the single sex rankings is much reduced (4 for women, 3 for men, as compared with 14 for the overall score range),
suggesting that the differences between female and male rates, within each country, is what drives the distinctions between the countries.

**United States**

The National Comorbidity Survey has estimated that the lifetime prevalence of PTSD among adult Americans is 7.8%, with women (10.4%) twice as likely as men (5%) to have PTSD at some point in their lives.

The United States Department of Veterans Affairs estimates that 830,000 Vietnam War veterans suffered symptoms of PTSD. The National Vietnam Veterans’ Readjustment Study (NVVRS) found 15.2% of male and 8.5% of female Vietnam Vets to suffer from current PTSD at the time of the study. Life-Time prevalence of PTSD was 30.9% for males and 26.9% for females. In a reanalysis of the NVVRS data, along with analysis of the data from the Matsunaga Vietnam Veterans Project, Schnurr, Lunney, Sengupta, and Waelde found that, contrary to the initial analysis of the NVVRS data, a large majority of Vietnam veterans suffered from PTSD symptoms (but not the disorder itself). Four out of five reported recent symptoms when interviewed 20–25 years after Vietnam.

**In other species**

There have been reports of captive and wild elephants suffering from posttraumatic stress reactions, the latter from seeing members of their herd shot by hunters. Service dogs used overseas in the military have been said to develop posttraumatic stress after witnessing war.

**History**

**Earliest reports**

Reports of battle-associated stress reactions appear as early as the 6th century BC/BCE. One of the first descriptions of PTSD was made by the Greek historian Herodotus. In 490 BC/BCE he described, during the Battle of Marathon, an Athenian soldier who suffered no injury from war but became permanently blind after witnessing the death of a fellow soldier.

**Modern recognition in military settings**

In the early 19th century military medical doctors started diagnosing soldiers with "exhaustion" after the stress of battle. This "exhaustion" was characterized by mental shutdown due to individual or group trauma. Soldiers during the 19th century were not supposed to be scared or show any fear in the midst of battle. The only treatment for this "exhaustion" was to bring the afflicted to the back for a bit then send them back into battle. During the intense and frequently repeated stress, the soldiers became fatigued as a part of their body’s natural shock reaction.
According to Stéphane Audoin-Rouzeau and Annette Becker, "One-tenth of mobilized American men were hospitalized for mental disturbances between 1942 and 1945, and after thirty-five days of uninterrupted combat, 98% of them manifested psychiatric disturbances in varying degrees."

Although PTSD-like symptoms have also been recognized in combat veterans of many military conflicts since, the modern understanding of PTSD dates from the 1970s, largely as a result of the problems that were still being experienced by US military veterans of the war in Vietnam.

Previous diagnoses now considered historical equivalents of PTSD include railway spine, stress syndrome, shell shock, battle fatigue, or traumatic war neurosis.

**Terminology**

The term post-traumatic stress disorder (PTSD) was coined in the mid 1970s, in part through the efforts of anti-Vietnam War activists and the anti war group Vietnam Veterans Against the War and Chaim F. Shatan, who worked with them and coined the term post-Vietnam Syndrome; the condition was added to the DSM-III as posttraumatic stress disorder.

Early in 1978, the term was used in a working group finding presented to the Committee of Reactive Disorders. The term was formally recognized in 1980. (In the authoritative DSM-IV, the spelling "posttraumatic stress disorder" is used. Elsewhere, "posttraumatic" is often rendered as two words — "post-traumatic stress disorder" or "post traumatic stress disorder" — especially in less formal writing on the subject.)

**Separation anxiety disorder**

Separation anxiety disorder is a psychological condition in which an individual experiences excessive anxiety regarding separation from home or from people to whom the individual has a strong emotional attachment (like a father, mother, grandparents, and brothers or sisters). Separation Anxiety Disorder (SAD), is characterized by significant and recurrent amounts of worry upon (or anticipation of) separation from a child or adolescent’s home or from those to whom the child or adolescent is attached.

Those suffering from SAD may worry about losing their parents and/or getting lost or kidnapped. They often refuse to go to certain places (e.g., school) because of fears of separation, or become extremely fearful when they are left alone without their parents. These children and adolescents may also refuse to sleep alone, experience nightmares about separation, or experience various physical complaints (e.g., body-aches, nausea) when separated from their parents. Separation anxiety may cause significant impairment in important areas of functioning, (e.g., academic and social). The duration of this problem must last for at least four weeks and must present itself before the child is 18 years of age.
Background

Present in all age groups, adult separation anxiety disorder (affecting roughly 7% of adults) is more common than childhood separation anxiety disorder (affecting approximately 4% of children). Separation Anxiety can also occur in dogs, which can lead to chewing for relieving stress. Separation anxiety disorder is often characterized by some of the following symptoms:

- Recurring distress when separated from the subject of attachment (such as significant other, the father or the mother, or home)
- Persistent, excessive worrying about losing the subject of attachment, and/or that some event will lead to separation from a major attachment
- Excessive fear about being alone without subject of attachment
- Persistent reluctance or refusal to go to sleep without being near a major attachment figure, like a significant other or mother
- Recurrent nightmares about separation
- Crying

Often, separation anxiety disorder is a symptom of a co-morbid condition. Studies show that children suffering from separation anxiety disorder are much more likely to have ADHD, bipolar disorder, panic disorder, and other disorders later in life.

Separation anxiety disorder versus separation anxiety

Separation anxiety disorder should not be confused with separation anxiety, which occurs as "a normal stage of development for healthy, secure babies." Separation anxiety occurs as babies begin to understand their own selfhood—or understand that they are a separate person from their primary caregiver. At the same time, the concept of object permanence emerges—which is when children learn that something still exists when it is not seen or heard. As babies begin to understand that they can be separated from their primary caregiver, they do not understand that their caregiver will return, nor do they have a concept of time. This, in turn, causes a normal and healthy anxious reaction.

Some sources state that separation anxiety typically onsets around 8 months of age and increases until 13–15 months, when it begins to decline. Other sources report a peak from 18–36 months.

Compared to separation anxiety, separation anxiety disorder is when the symptoms of separation anxiety becomes problematic for day-to-day living.

Psychosocial Treatment

Cognitive Behavioral Therapy is scientifically proven to help treat separation anxiety disorder. Cognitive and Behavioral Therapies for children and adolescents usually are short-term treatments (i.e., often between 6-20 sessions) that focus on teaching young people and their parents specific skills. CBT is different from many other therapy methods because it involves the active participation of the client in the therapy process.
approaches by focusing on the ways that a person’s cognitions (i.e., thoughts), emotions, and behaviors are connected and how they affect one another. Because emotions, thoughts, and behaviors are all linked, CBT approaches allow for therapists to intervene at different points in the cycle. Learn more from the Society of Clinical Child and Adolescent Psychology

Separation anxiety in dogs

Separation Anxiety in Dogs is a condition where dogs, when left alone, exhibit distress and behavior problems. It is similar to Separation Anxiety Disorder that people experience when they are away from someone they feel strongly emotionally attached to (i.e. a father or mother). In pets’ case, they become distressed and anxious when they are separated from their owner.

Since dogs are "pack" animals, they naturally want to always be with their owners (who they consider their pack). Most normal dogs are able to handle being alone for about 8 hours a day; as long as they have a yard to defecate. For dogs with Separation Anxiety, they become nervous when owners go to leave and often will try to leave with them. As they are leaving, one can hear them barking, wishing that they would come back. And, upon return, they find their home has been destroyed in some way or the dog has, to some extreme, hurt themselves.

Separation Anxiety affects even those pets who receive the most love and attention; it does not only affect neglected pets.

Typical Behaviors
Often owners confuse their pets behavior with discipline problems rather than with a disorder. Some behavior characteristics to look out for in a pet one might think is suffering from separation anxiety are:

- Destructive chewing
- Howling, barking, whining
- Urination, defecation in the house
- Self mutilation
- Digging and scratching at doors or windows in an attempt to reunite with their owner

**Causes**

The cause of dog separation anxiety is unknown. Some pets retain their early fear of being left alone. Causes can include:

- Being left alone early in life
- Rejection as puppies
- Lack of stimulation
- Poor health.

Older pets have difficult times with moving from place to place and accepting new people or pets into their lives.

**Mood disorder**

Mood disorder is the term designating a group of diagnoses in the Diagnostic and Statistical Manual of Mental Disorders (DSM IV TR) classification system where a disturbance in the person’s mood is hypothesized to be the main underlying feature. The classification is known as mood (affective) disorders in ICD 10.

English psychiatrist Henry Maudsley proposed an overarching category of affective disorder. The term was then replaced by mood disorder, as the latter term refers to the underlying or longitudinal emotional state, whereas the former refers to the external expression observed by others.

Two groups of mood disorders are broadly recognized; the division is based on whether the person has ever had a manic or hypomanic episode. Thus, there are depressive disorders, of which the best known and most researched is major depressive disorder (MDD) commonly called clinical depression or major depression, and bipolar disorder (BD), formerly known as manic depression and characterized by intermittent episodes of mania or hypomania, usually interlaced with depressive episodes.

**Classification**
Depressive disorders

Major depressive disorder (MDD), commonly called major depression, unipolar depression, or clinical depression, where a person has one or more major depressive episodes. After a single episode, Major Depressive Disorder (single episode) would be diagnosed. After more than one episode, the diagnosis becomes Major Depressive Disorder (Recurrent). Depression without periods of mania is sometimes referred to as unipolar depression because the mood remains at one emotional state or "pole".

Individuals with a major depressive episode or major depressive disorder are at increased risk for suicide. Seeking help and treatment from a health professional dramatically reduces the individual’s risk for suicide. Studies have demonstrated that asking if a depressed friend or family member has thought of committing suicide is an effective way of identifying those at risk, and it does not "plant" the idea or increase an individual's risk for suicide in any way. Epidemiological studies carried out in Europe suggest that at this moment, roughly 8.5 percent of the world's population are suffering from a depressive disorder. No age group seems to be exempt from depression and studies have found that depression appears in infants as young as 6 months old who have been separated from their mothers.

Diagnosticians recognize several subtypes or course specifiers:

Atypical depression (AD) is characterized by mood reactivity (paradoxical anhedonia) and positivity, significant weight gain or increased appetite ("comfort eating"), excessive sleep or somnolence (hypersomnia), a sensation of heaviness in limbs known as leaden paralysis, and significant social impairment as a consequence of hypersensitivity to perceived interpersonal rejection. Difficulties in measuring this subtype have led to questions of its validity and prevalence.

Melancholic depression is characterized by a loss of pleasure (anhedonia) in most or all activities, a failure of reactivity to pleasurable stimuli, a quality of depressed mood more pronounced than that of grief or loss, a worsening of symptoms in the morning hours, early morning waking, psychomotor retardation, excessive weight loss (not to be confused with anorexia nervosa), or excessive guilt.

Psychotic major depression (PMD), or simply psychotic depression, is the term for a major depressive episode, particularly of melancholic nature, where the patient experiences psychotic symptoms such as delusions or, less commonly, hallucinations. These are most commonly mood-congruent (content coincident with depressive themes).

Catatonic depression is a rare and severe form of major depression involving disturbances of motor behavior and other symptoms. Here the person is mute and almost stuporose, and either immobile or exhibits purposeless or even bizarre movements. Catatonic symptoms can also occur in schizophrenia, a manic episode, or be due to neuroleptic malignant syndrome.
Postpartum depression (PPD) is listed as a course specifier in DSM-IV-TR; it refers to the intense, sustained and sometimes disabling depression experienced by women after giving birth. Postpartum depression, which has incidence rate of 10–15%, typically sets in within three months of labor, and lasts as long as three months. It is quite common for women to experience a short term feeling of tiredness and sadness in the first few weeks after giving birth; however, postpartum depression is different because it can cause significant hardship and impaired functioning at home, work, or school as well as possibly difficulty in relationships with family members, spouses, friends, or even problems bonding with the newborn. In the treatment of postpartum major depressive disorders and other unipolar depressions in women who are breastfeeding, nortriptyline, paroxetine (Paxil), and sertraline (Zoloft) are generally considered to be the preferred medications.

Seasonal affective disorder (SAD), also known as "winter depression" or "winter blues", is a specifier. Some people have a seasonal pattern, with depressive episodes coming on in the autumn or winter, and resolving in spring. The diagnosis is made if at least two episodes have occurred in colder months with none at other times over a two-year period or longer. It is commonly hypothesised that people who live at higher latitudes tend to have less sunlight exposure in the winter and therefore experience higher rates of SAD, but the epidemiological support for this proposition is not strong (and latitude is not the only determinant of the amount of sunlight reaching the eyes in winter). SAD is also more prevalent in people who are younger and typically affects more females than males.

Dysthymia, which is a chronic, different mood disturbance where a person reports a low mood almost daily over a span of at least two years. The symptoms are not as severe as those for major depression, although people with dysthymia are vulnerable to secondary episodes of major depression (sometimes referred to as double depression). The treatment of dysthymia is largely the same as for major depression, including antidepressant medications and psychotherapy.

Depressive Disorder Not Otherwise Specified (DD-NOS) is designated by the code 311 for depressive disorders that are impairing but do not fit any of the officially specified diagnoses. According to the DSM-IV, DD-NOS encompasses "any depressive disorder that does not meet the criteria for a specific disorder." It includes the research diagnoses of recurrent brief depression, and minor depressive disorder listed below.

Recurrent brief depression (RBD), distinguished from major depressive disorder primarily by differences in duration. People with RBD have depressive episodes about once per month, with individual episodes lasting less than two weeks and typically less than 2–3 days. Diagnosis of RBD requires that the episodes occur over the span of at least one year and, in female patients, independently of the menstrual cycle. People with clinical depression can develop RBD, and vice versa, and both illnesses have similar risks.

Minor depressive disorder, or simply minor depression, which refers to a depression that does not meet full criteria for major depression but in which at least two symptoms are present for two weeks.
Bipolar disorders

Bipolar disorder (BD), a mood disorder formerly known as "manic depression" and described by alternating periods of mania and depression (and in some cases rapid cycling, mixed states, and psychotic symptoms). Subtypes include:

- Bipolar I is distinguished by the presence or history of one or more manic episodes or mixed episodes with or without major depressive episodes. A depressive episode is not required for the diagnosis of Bipolar I disorder, but depressive episodes are often part of the course of the illness.
- Bipolar II consisting of recurrent intermittent hypomanic and depressive episodes.
- Cyclothymia is a form of bipolar disorder, consisting of recurrent hypomanic and dysthymic episodes, but no full manic episodes or full major depressive episodes.
- Bipolar Disorder Not Otherwise Specified (BD-NOS), sometimes called "sub-threshold" bipolar, indicates that the patient suffers from some symptoms in the bipolar spectrum (e.g. manic and depressive symptoms) but does not fully qualify for any of the three formal bipolar DSM-IV diagnoses mentioned above.

It is estimated that roughly one percent of the adult population suffers from bipolar I, roughly one percent of the adult population suffers from bipolar II or cyclothymia, and somewhere between two and five percent suffer from "sub-threshold" forms of bipolar disorder.

Substance induced mood disorders

A mood disorder can be classified as substance-induced if its etiology can be traced to the direct physiologic effects of a psychoactive drug or other chemical substance, or if the development of the mood disorder occurred contemporaneously with substance intoxication or withdrawal. Alternatively, an individual may have a mood disorder coexisting with a substance abuse disorder. Substance-induced mood disorders can have features of a manic, hypomanic, mixed, or depressive episode. Most substances can induce a variety of mood disorders. For example, stimulants such as amphetamine, methamphetamine, and cocaine can cause manic, hypomanic, mixed, and depressive episodes.

Alcohol induced mood disorders

High rates of major depressive disorder occur in heavy drinkers and those with alcoholism. Controversy has previously surrounded whether those who abused alcohol and developed depression were self-medicating their pre-existing depression, but recent research has concluded that, while this may be true in some cases, alcohol misuse directly causes the development of depression in a significant number of heavy drinkers. High rates of suicide also occur in those who have alcohol-related problems. It is usually possible to differentiate between alcohol-related depression and depression which is not related to alcohol intake by taking a careful history of the patient. Depression and other mental health problems
associated with alcohol misuse may be due to distortion of brain chemistry, as they tend to improve on their own after a period of abstinence.

**Benzodiazepine induced mood disorders**

The long-term use of benzodiazepines, such as Valium and Librium, may have a similar effect on the brain as alcohol, and are also implicated in depression. Major depressive disorder can also develop as a result of chronic use of benzodiazepines or as part of a protracted withdrawal syndrome. Benzodiazepines are a class of medication which are commonly used to treat insomnia, anxiety and muscular spasms. As with alcohol, the effects of benzodiazepine on neurochemistry, such as decreased levels of serotonin and norepinephrine, are believed to be responsible for the increased depression. Major depressive disorder may also occur as part of the benzodiazepine withdrawal syndrome. In a long-term follow-up study of patients dependent on benzodiazepines, it was found that 10 people (20%) had taken drug overdoses while on chronic benzodiazepine medication despite only two people ever having had any pre-existing depressive disorder. A year after a gradual withdrawal program, no patients had taken any further overdoses. Depression resulting from withdrawal from benzodiazepines usually subsides after a few months but in some cases may persist for 6–12 months.

**Interferon-alpha induced mood disorders**

Combination therapy with interferon-α and ribavirin for chronic hepatitis C virus (HCV) infection may induce major depression. In the study by Leutscher et al., evaluating 325 chronically HCV infected patients undergoing antiviral therapy, it was observed that (1) depressive symptoms among patients undergoing HCV therapy are commonly overlooked by routine clinical interviews, (2) the emergence of depression compromises the outcome of HCV therapy, and (3) the Major Depression Inventory (MDI) scale may be useful in identifying patients at risk for treatment-induced depression.

**Origin**

A number of authors have suggested that mood disorders are an evolutionary adaptation. A low or depressed mood can increase an individual’s ability to cope with situations in which the effort to pursue a major goal could result in danger, loss, or wasted effort. In such situations, low motivation may give an advantage by inhibiting certain actions. This theory helps to explain why mood disorders are so prevalent, and why they so often strike people during their peak reproductive years. These characteristics would be difficult to understand if depression were a dysfunction.

A depressed mood is a predictable response to certain types of life occurrences, such as loss of status, divorce, or death of a child or spouse. These are events that signal a loss of reproductive ability or potential, or that did so in humans' ancestral environment. A depressed mood can be seen as an adaptive response, in the sense that it causes an individual to turn away from the earlier (and reproductively unsuccessful) modes of behavior.
A depressed mood is common during illnesses, such as influenza. It has been argued that this is an evolved mechanism that assists the individual in recovering by limiting his/her physical activity. The occurrence of low-level depression during the winter months, or seasonal affective disorder, may have been adaptive in the past, by limiting physical activity at times when food was scarce. It is argued that humans have retained the instinct to experience low mood during the winter months, even if the availability of food is no longer determined by the weather.

**Sociocultural aspects**

Kay Redfield Jamison and others have explored the possible links between mood disorders—especially bipolar disorder—and creativity. It has been proposed that a "ruminating personality type may contribute to both [mood disorders] and art." The relationship between depression and creativity appears to be especially strong among poets.

**Epidemiology**

According to a substantial amount of epidemiology studies conducted, women are twice as likely to develop certain mood disorders, such as major depression. There is an equal number of men and women who are diagnosed with bipolar disorder.
Major depressive disorder

Vincent van Gogh's 1890 painting

Major depressive disorder (MDD) (also known as recurrent depressive disorder, clinical depression, major depression, unipolar depression, or unipolar disorder) is a mental disorder characterized by an all-encompassing low mood accompanied by low self-esteem, and by loss of interest or pleasure in normally enjoyable activities. This cluster of symptoms (syndrome) was named, described and classified as one of the mood disorders in the 1980 edition of the American Psychiatric Association’s diagnostic manual. The term "depression" is ambiguous. It is often used to denote this syndrome but may refer to other mood disorders or to lower mood states lacking clinical significance. Major depressive disorder is a disabling condition which adversely affects a person’s family, work or school life, sleeping and eating habits, and general health. In the United States, around 3.4% of people with major depression commit suicide, and up to 60% of people who committed suicide had depression or another mood disorder.

The diagnosis of major depressive disorder is based on the patient’s self-reported experiences, behavior reported by relatives or friends, and a mental status examination. There is no laboratory test for major depression, although physicians generally request tests for physical conditions that may cause similar symptoms. If depressive disorder is not detected in the early stages it may result in a slow recovery and affect or worsen the person’s physical health. Standardized screening tools such as Major Depression Inventory can be used to detect major depressive disorder. The most common time of onset is between the ages of 20 and 30 years, with a later peak between 30 and 40 years.
Typically, patients are treated with antidepressant medication and, in many cases, also receive psychotherapy or counseling, although the effectiveness of medication for mild or moderate cases is questionable. Hospitalization may be necessary in cases with associated self-neglect or a significant risk of harm to self or others. A minority are treated with electroconvulsive therapy (ECT), under a short-acting general anaesthetic. The course of the disorder varies widely, from one episode lasting weeks to a lifelong disorder with recurrent major depressive episodes. Depressed individuals have shorter life expectancies than those without depression, in part because of greater susceptibility to medical illnesses and suicide. It is unclear whether or not medications affect the risk of suicide. Current and former patients may be stigmatized.

The understanding of the nature and causes of depression has evolved over the centuries, though this understanding is incomplete and has left many aspects of depression as the subject of discussion and research. Proposed causes include psychological, psycho-social, hereditary, evolutionary and biological factors. Certain types of long-term drug use can both cause and worsen depressive symptoms. Psychological treatments are based on theories of personality, interpersonal communication, and learning. Most biological theories focus on the monoamine chemicals serotonin, norepinephrine and dopamine, which are naturally present in the brain and assist communication between nerve cells.

**Symptoms and signs**

Major depression significantly affects a person’s family and personal relationships, work or school life, sleeping and eating habits, and general health. Its impact on functioning and well-being has been equated to that of chronic medical conditions such as diabetes.

A person having a major depressive episode usually exhibits a very low mood, which pervades all aspects of life, and an inability to experience pleasure in activities that were formerly enjoyed. Depressed people may be preoccupied with, or ruminate over, thoughts and feelings of worthlessness, inappropriate guilt or regret, helplessness, hopelessness, and self-hatred. In severe cases, depressed people may have symptoms of psychosis. These symptoms include delusions or, less commonly, hallucinations, usually unpleasant. Other symptoms of depression include poor concentration and memory (especially in those with melancholic or psychotic features), withdrawal from social situations and activities, reduced sex drive, and thoughts of death or suicide.

Insomnia is common among the depressed. In the typical pattern, a person wakes very early and cannot get back to sleep, but insomnia can also include difficulty falling asleep. Insomnia affects at least 80% of depressed people. Hypersomnia, or oversleeping, can also happen, affecting 15% of depressed people. Some antidepressants may also cause insomnia due to their stimulating effect.

A depressed person may report multiple physical symptoms such as fatigue, headaches, or digestive problems; physical complaints are the most common presenting problem in developing countries, according to the World Health Organization’s criteria for depression. Appetite often decreases, with resulting weight loss, although increased appetite and
weight gain occasionally occur. Family and friends may notice that the person's behavior is either agitated or lethargic.

**In children**

Although it is common for most children and teenagers to feel down or sad sometimes, a smaller number of youth experience a more severe phenomenon known as depression. Such young people, who are often described as "clinically" depressed, feel sad, hopeless, or irritable for weeks or even months at a time. They may lose interest in activities that they used to enjoy (e.g., playing with friends); their sleeping and eating habits often change (i.e., they may eat or sleep either more or less than usual); and they may have trouble thinking or paying attention, even to TV programs or games. Depressed children may often display an irritable mood rather than a depressed mood, and show varying symptoms depending on age and situation. Most lose interest in school and show a decline in academic performance. They may be described as clingy, demanding, dependent, or insecure. Diagnosis may be delayed or missed when symptoms are interpreted as normal moodiness. Depression may also coexist with attention-deficit hyperactivity disorder (ADHD), complicating the diagnosis and treatment of both.

Of particular concern, youths who are clinically depressed may think or talk a lot about death and some depressed children have more specific thoughts about hurting or killing themselves. Often children and teenagers may have similar symptoms when they are grieving the loss of someone close to them. In clinical depression, however, these thoughts and feelings tend to appear even when the child has not experienced a loss or a sad event.

**In the elderly**

Older depressed people may have cognitive symptoms of recent onset, such as forgetfulness, and a more noticeable slowing of movements. Depression often coexists with physical disorders common among the elderly, such as stroke, other cardiovascular diseases, Parkinson's disease, and chronic obstructive pulmonary disease.

**Causes**

The biopsychosocial model proposes that biological, psychological, and social factors all play a role in causing depression. The diathesis–stress model specifies that depression results when a preexisting vulnerability, or diathesis, is activated by stressful life events. The preexisting vulnerability can be either genetic, implying an interaction between nature and nurture, or schematic, resulting from views of the world learned in childhood.

These interactive models have gained empirical support. For example, researchers in New Zealand took a prospective approach to studying depression, by documenting over time how depression emerged among an initially normal cohort of people. The researchers concluded that variation among the serotonin transporter (5-HTT) gene affects the chances that people who have dealt with very stressful life events will go on to experience depression. Specifically, depression may follow such events, but seems more likely to
appear in people with one or two short alleles of the 5-HTT gene. Additionally, a Swedish study estimated the heritability of depression—the degree to which individual differences in occurrence are associated with genetic differences—to be around 40% for women and 30% for men, and evolutionary psychologists have proposed that the genetic basis for depression lies deep in the history of naturally selected adaptations. A substance-induced mood disorder resembling major depression has been causally linked to long-term drug use or drug abuse, or to withdrawal from certain sedative and hypnotic drugs.

**Biological**

**Monoamine hypothesis**

Of approx. 30 neurotransmitters which have been identified, researchers have discovered associations between clinical depression and the function of three major neurochemicals. These substances are serotonin, norepinephrine, and dopamine. Antidepressants influence the overall balance of these three neurotransmitters within structures of the brain which regulate emotion, reactions to stress, and the physical drives of sleep, appetite, and sexuality.

Most antidepressant medications increase the levels of one or more of the monoamines—the neurotransmitters serotonin, norepinephrine and dopamine—in the synaptic cleft between neurons in the brain. Some medications affect the monoamine receptors directly.

Serotonin is hypothesized to regulate other neurotransmitter systems; decreased serotonin activity may allow these systems to act in unusual and erratic ways. According to this "permissive hypothesis", depression arises when low serotonin levels promote low levels of norepinephrine, another monoamine neurotransmitter. Some antidepressants enhance
the levels of norepinephrine directly, whereas others raise the levels of dopamine, a third monoamine neurotransmitter. These observations gave rise to the monoamine hypothesis of depression. In its contemporary formulation, the monoamine hypothesis postulates that a deficiency of certain neurotransmitters is responsible for the corresponding features of depression: "Norepinephrine may be related to alertness and energy as well as anxiety, attention, and interest in life; [lack of] serotonin to anxiety, obsessions, and compulsions; and dopamine to attention, motivation, pleasure, and reward, as well as interest in life." The proponents of this theory recommend the choice of an antidepressant with mechanism of action that impacts the most prominent symptoms. Anxious and irritable patients should be treated with SSRIs or norepinephrine reuptake inhibitors, and those experiencing a loss of energy and enjoyment of life with norepinephrine- and dopamine-enhancing drugs.

Besides the clinical observations that drugs which increase the amount of available monoamines are effective antidepressants, recent advances in psychiatric genetics indicate that phenotypic variation in central monoamine function may be marginally associated with vulnerability to depression. Despite these findings, the cause of depression is not simply monoamine deficiency. In the past two decades, research has revealed multiple limitations of the monoamine hypothesis, and its explanatory inadequacy has been highlighted within the psychiatric community. A counterargument is that the mood-enhancing effect of MAO inhibitors and SSRIs takes weeks of treatment to develop, even though the boost in available monoamines occurs within hours. Another counterargument is based on experiments with pharmacological agents that cause depletion of monoamines; while deliberate reduction in the concentration of centrally available monoamines may slightly lower the mood of unmedicated depressed patients, this reduction does not affect the mood of healthy people. An intact monoamine system is necessary for antidepressants to achieve therapeutic effectiveness, but some medications like tianeptine and opipramol have antidepressant properties despite the fact that the former is a serotonin reuptake enhancer and the latter has no effect on the monoamine system. The monoamine hypothesis, already limited, has been further oversimplified when presented to the general public as a mass marketing tool, usually phrased as a "chemical imbalance".

In 2003 a gene-environment interaction (GxE) was hypothesized to explain why life stress is a predictor for depressive episodes in some individuals, but not in others, depending on an allelic variation of the serotonin-transporter-linked promoter region (5-HTTLPR); a 2009 meta-analysis showed stressful life events were associated with depression, but found no evidence for an association with the 5-HTTLPR genotype. Another 2009 meta-analysis agreed with the latter finding. A 2010 review of studies in this area found a systematic relationship between the method used to assess environmental adversity and the results of the studies; this review also found that both 2009 meta-analyses were significantly biased toward negative studies, which used self-report measures of adversity.

Other theories

MRI scans of patients with depression have revealed a number of differences in brain structure compared to those who are not depressed. Although there is some inconsistency in the results, meta-analyses have shown there is evidence for smaller hippocampal
volumes and increased numbers of hyperintensive lesions. Hyperintensities have been associated with patients with a late age of onset, and have led to the development of the theory of vascular depression.

There may be a link between depression and neurogenesis of the hippocampus, a center for both mood and memory. Loss of hippocampal neurons is found in some depressed individuals and correlates with impaired memory and dysthymic mood. Drugs may increase serotonin levels in the brain, stimulating neurogenesis and thus increasing the total mass of the hippocampus. This increase may help to restore mood and memory. Similar relationships have been observed between depression and an area of the anterior cingulate cortex implicated in the modulation of emotional behavior. One of the neurotrophins responsible for neurogenesis is brain-derived neurotrophic factor (BDNF). The level of BDNF in the blood plasma of depressed subjects is drastically reduced (more than threefold) as compared to the norm. Antidepressant treatment increases the blood level of BDNF. Although decreased plasma BDNF levels have been found in many other disorders, there is some evidence that BDNF is involved in the cause of depression and the mechanism of action of antidepressants.

There is some evidence that major depression may be caused in part by an overactive hypothalamic-pituitary-adrenal axis (HPA axis) that results in an effect similar to the neuro-endocrine response to stress. Investigations reveal increased levels of the hormone cortisol and enlarged pituitary and adrenal glands, suggesting disturbances of the endocrine system may play a role in some psychiatric disorders, including major depression. Oversecretion of corticotropin-releasing hormone from the hypothalamus is thought to drive this, and is implicated in the cognitive and arousal symptoms.

The hormone estrogen has been implicated in depressive disorders due to the increase in risk of depressive episodes after puberty, the antenatal period, and reduced rates after menopause. Conversely, the premenstrual and postpartum periods of low estrogen levels are also associated with increased risk. Sudden withdrawal of, fluctuations in or periods of sustained low levels of estrogen have been linked to significant mood lowering. Clinical recovery from depression postpartum, perimenopause, and postmenopause was shown to be effective after levels of estrogen were stabilized or restored.

Other research has explored potential roles of molecules necessary for overall cellular functioning: cytokines. The symptoms of major depressive disorder are nearly identical to those of sickness behavior, the response of the body when the immune system is fighting an infection. This raises the possibility that depression can result from a maladaptive manifestation of sickness behavior as a result of abnormalities in circulating cytokines. The involvement of pro-inflammatory cytokines in depression is strongly suggested by a meta-analysis of the clinical literature showing higher blood concentrations of IL-6 and TNF-α in depressed subjects compared to controls.

Finally, some relationships have been reported between specific subtypes of depression and climatic conditions. Thus, the incidence of psychotic depression has been found to
increase when the barometric pressure is low, while the incidence of melancholic depression has been found to increase when the temperature and/or sunlight are low.

Psychological

Various aspects of personality and its development appear to be integral to the occurrence and persistence of depression, with negative emotionality as a common precursor. Although depressive episodes are strongly correlated with adverse events, a person's characteristic style of coping may be correlated with his or her resilience. Additionally, low self-esteem and self-defeating or distorted thinking are related to depression. Depression is less likely to occur, as well as quicker to remit, among those who are religious. It is not always clear which factors are causes or which are effects of depression; however, depressed persons who are able to reflect upon and challenge their thinking patterns often show improved mood and self-esteem.

American psychiatrist Aaron T. Beck, following on from the earlier work of George Kelly and Albert Ellis, developed what is now known as a cognitive model of depression in the early 1960s. He proposed that three concepts underlie depression: a triad of negative thoughts composed of cognitive errors about oneself, one's world, and one's future; recurrent patterns of depressive thinking, or schemas; and distorted information processing. From these principles, he developed the structured technique of cognitive behavioral therapy (CBT). According to American psychologist Martin Seligman, depression in humans is similar to learned helplessness in laboratory animals, who remain in unpleasant situations when they are able to escape, but do not because they initially learned they had no control.

Attachment theory, which was developed by English psychiatrist John Bowlby in the 1960s, predicts a relationship between depressive disorder in adulthood and the quality of the earlier bond between the infant and their adult caregiver. In particular, it is thought that "the experiences of early loss, separation and rejection by the parent or caregiver (conveying the message that the child is unlovable) may all lead to insecure internal working models ... Internal cognitive representations of the self as unlovable and of attachment figures as unloving [or] untrustworthy would be consistent with parts of Beck's cognitive triad". While a wide variety of studies has upheld the basic tenets of attachment theory, research has been inconclusive as to whether self-reported early attachment and later depression are demonstrably related.

Depressed individuals often blame themselves for negative events, and, as shown in a 1993 study of hospitalized adolescents with self-reported depression, those who blame themselves for negative occurrences may not take credit for positive outcomes. This tendency is characteristic of a depressive attributional, or pessimistic explanatory style. According to Albert Bandura, a Canadian social psychologist associated with social cognitive theory, depressed individuals have negative beliefs about themselves, based on experiences of failure, observing the failure of social models, a lack of social persuasion that they can succeed, and their own somatic and emotional states including tension and stress.
These influences may result in a negative self-concept and a lack of self-efficacy; that is, they do not believe they can influence events or achieve personal goals.

An examination of depression in women indicates that vulnerability factors—such as early maternal loss, lack of a confiding relationship, responsibility for the care of several young children at home, and unemployment—can interact with life stressors to increase the risk of depression. For older adults, the factors are often health problems, changes in relationships with a spouse or adult children due to the transition to a care-giving or care-needing role, the death of a significant other, or a change in the availability or quality of social relationships with older friends because of their own health-related life changes.

The understanding of depression has also received contributions from the psychoanalytic and humanistic branches of psychology. From the classical psychoanalytic perspective of Austrian psychiatrist Sigmund Freud, depression, or melancholia, may be related to interpersonal loss and early life experiences. Existential therapists have connected depression to the lack of both meaning in the present and a vision of the future. The founder of humanistic psychology, American psychologist Abraham Maslow, suggested that depression could arise when people are unable to attain their needs or to self-actualize (to realize their full potential).

Social

Poverty and social isolation are associated with increased risk of mental health problems in general. Child abuse (physical, emotional, sexual, or neglect) is also associated with increased risk of developing depressive disorders later in life. Such a link has good face validity given that it is during the years of development that a child is learning how to become a social being. Abuse of the child by the caregiver is bound to distort the developing personality and create a much greater risk for depression and many other debilitating mental and emotional states. Disturbances in family functioning, such as parental (particularly maternal) depression, severe marital conflict or divorce, death of a parent, or other disturbances in parenting are additional risk factors. In adulthood, stressful life events are strongly associated with the onset of major depressive episodes. In this context, life events connected to social rejection appear to be particularly related to depression. Evidence that a first episode of depression is more likely to be immediately preceded by stressful life events than are recurrent ones is consistent with the hypothesis that people may become increasingly sensitized to life stress over successive recurrences of depression.

The relationship between stressful life events and social support has been a matter of some debate; the lack of social support may increase the likelihood that life stress will lead to depression, or the absence of social support may constitute a form of strain that leads to depression directly. There is evidence that neighborhood social disorder, for example, due to crime or illicit drugs, is a risk factor, and that a high neighborhood socioeconomic status, with better amenities, is a protective factor. Adverse conditions at work, particularly demanding jobs with little scope for decision-making, are associated with depression,
although diversity and confounding factors make it difficult to confirm that the relationship is causal.

**Evolutionary**

From the standpoint of evolutionary theory, major depression is hypothesized, in some instances, to increase an individual’s reproductive fitness. Evolutionary approaches to depression and evolutionary psychology posit specific mechanisms by which depression may have been genetically incorporated into the human gene pool, accounting for the high heritability and prevalence of depression by proposing that certain components of depression are adaptations, such as the behaviors relating to attachment and social rank. Current behaviors can be explained as adaptations to regulate relationships or resources, although the result may be maladaptive in modern environments.

From another viewpoint, a counseling therapist may see depression not as a biochemical illness or disorder but as "a species-wide evolved suite of emotional programmes that are mostly activated by a perception, almost always over-negative, of a major decline in personal usefulness, that can sometimes be linked to guilt, shame or perceived rejection". This suite may have manifested in aging hunters in humans’ foraging past, who were marginalized by their declining skills, and may continue to appear in alienated members of today’s society. The feelings of uselessness generated by such marginalization could hypothetically prompt support from friends and kin. Additionally, in a manner analogous to that in which physical pain has evolved to hinder actions that may cause further injury, "psychic misery" may have evolved to prevent hasty and maladaptive reactions to distressing situations.

**Drug and alcohol use**

According to the DSM-IV, a diagnosis of mood disorder cannot be made if the cause is believed to be due to "the direct physiological effects of a substance"; when a syndrome resembling major depression is believed to be caused immediately by substance abuse or by an adverse drug reaction, it is referred to as, "substance-induced mood disturbance". Alcoholism or excessive alcohol consumption significantly increases the risk of developing major depression. Like alcohol, the benzodiazepines are central nervous system depressants; this class of medication is commonly used to treat insomnia, anxiety, and muscular spasms. Similar to alcohol, benzodiazepines increase the risk of developing major depression. This increased risk may be due in part to the effects of drugs on neurochemistry, such as decreased levels of serotonin and norepinephrine. Chronic use of benzodiazepines also can cause or worsen depression, or depression may be part of a protracted withdrawal syndrome.

**Diagnosis**

**Clinical assessment**
A diagnostic assessment may be conducted by a suitably trained general practitioner, or by a psychiatrist or psychologist, who records the person’s current circumstances, biographical history, current symptoms and family history. The broad clinical aim is to formulate the relevant biological, psychological and social factors that may be impacting on the individual’s mood. The assessor may also discuss the person’s current ways of regulating their mood (healthy or otherwise) such as alcohol and drug use. The assessment also includes a mental state examination, which is an assessment of the person’s current mood and thought content, in particular the presence of themes of hopelessness or pessimism, self-harm or suicide, and an absence of positive thoughts or plans. Specialist mental health services are rare in rural areas, and thus diagnosis and management is largely left to primary care clinicians. This issue is even more marked in developing countries. The score on a rating scale alone is insufficient to diagnose depression to the satisfaction of the DSM or ICD, but it provides an indication of the severity of symptoms for a time period, so a person who scores above a given cut-off point can be more thoroughly evaluated for a depressive disorder diagnosis. Several rating scales are used for this purpose. Screening programs have been advocated to improve detection of depression, but there is evidence that they do not improve detection rates, treatment, or outcome.

Primary care physicians and other non-psychiatrist physicians have difficulty diagnosing depression, in part because they are trained to recognize and treat physical symptoms, and depression can cause a myriad of physical (psychosomatic) symptoms. Non-psychiatrists miss two-thirds of cases and unnecessarily treat other patients.

Before diagnosing a major depressive disorder, a doctor generally performs a medical examination and selected investigations to rule out other causes of symptoms. These include blood tests measuring TSH and thyroxine to exclude hypothyroidism; basic electrolytes and serum calcium to rule out a metabolic disturbance; and a full blood count including ESR to rule out a systemic infection or chronic disease. Adverse affective reactions to medications or alcohol misuse are often ruled out, as well. Testosterone levels may be evaluated to diagnose hypogonadism, a cause of depression in men.

Subjective cognitive complaints appear in older depressed people, but they can also be indicative of the onset of a dementing disorder, such as Alzheimer’s disease. Cognitive testing and brain imaging can help distinguish depression from dementia. A CT scan can exclude brain pathology in those with psychotic, rapid-onset or otherwise unusual symptoms. No biological tests confirm major depression. Investigations are not generally repeated for a subsequent episode unless there is a medical indication.

Biomarkers of depression have been sought to provide an objective method of diagnosis. There are several potential biomarkers, including Brain-Derived Neurotrophic Factor and various functional MRI techniques. One study developed a decision tree model of interpreting a series of fMRI scans taken during various activities. In their subjects, the authors of that study were able to achieve a sensitivity of 80% and a specificity of 87%, corresponding to a negative predictive value of 98% and a positive predictive value of 32% (positive and negative likelihood ratios were 6.15, 0.23 respectively). However, much more research is needed before these tests could be used clinically.
**DSM-IV-TR and ICD-10 criteria**

The most widely used criteria for diagnosing depressive conditions are found in the American Psychiatric Association's revised fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), and the World Health Organization's International Statistical Classification of Diseases and Related Health Problems (ICD-10) which uses the name recurrent depressive disorder. The latter system is typically used in European countries, while the former is used in the US and many other non-European nations, and the authors of both have worked towards conforming one with the other.

Both DSM-IV-TR and ICD-10 mark out typical (main) depressive symptoms. ICD-10 defines three typical depressive symptoms (depressed mood, anhedonia, and reduced energy), two of which should be present to determine depressive disorder diagnosis. According DSM-IV-TR there are two main depressive symptoms—depressed mood, anhedonia, at least one of which must be present to determine diagnosis of major depressive episode.

Major depressive disorder is classified as a mood disorder in DSM-IV-TR. The diagnosis hinges on the presence of single or recurrent major depressive episodes. Further qualifiers are used to classify both the episode itself and the course of the disorder. The category Depressive Disorder Not Otherwise Specified is diagnosed if the depressive episode's manifestation does not meet the criteria for a major depressive episode. The ICD-10 system does not use the term major depressive disorder, but lists very similar criteria for the diagnosis of a depressive episode (mild, moderate or severe); the term recurrent may be added if there have been multiple episodes without mania.

**Major depressive episode**

A major depressive episode is characterized by the presence of a severely depressed mood that persists for at least two weeks. Episodes may be isolated or recurrent and are categorized as mild (few symptoms in excess of minimum criteria), moderate, or severe (marked impact on social or occupational functioning). An episode with psychotic features—commonly referred to as psychotic depression—is automatically rated as severe. If the patient has had an episode of mania or markedly elevated mood, a diagnosis of bipolar disorder is made instead. Depression without mania is sometimes referred to as unipolar because the mood remains at one emotional state or "pole".

DSM-IV-TR excludes cases where the symptoms are a result of bereavement, although it is possible for normal bereavement to evolve into a depressive episode if the mood persists and the characteristic features of a major depressive episode develop. The criteria have been criticized because they do not take into account any other aspects of the personal and social context in which depression can occur. In addition, some studies have found little empirical support for the DSM-IV cut-off criteria, indicating they are a diagnostic convention imposed on a continuum of depressive symptoms of varying severity and duration: Excluded are a range of related diagnoses, including dysthymia, which involves a chronic but milder mood disturbance; recurrent brief depression, consisting of briefer
depressive episodes; minor depressive disorder, whereby only some of the symptoms of major depression are present; and adjustment disorder with depressed mood, which denotes low mood resulting from a psychological response to an identifiable event or stressor.

Subtypes

The DSM-IV-TR recognizes five further subtypes of MDD, called specifiers, in addition to noting the length, severity and presence of psychotic features:

- Melancholic depression is characterized by a loss of pleasure in most or all activities, a failure of reactivity to pleasurable stimuli, a quality of depressed mood more pronounced than that of grief or loss, a worsening of symptoms in the morning hours, early morning waking, psychomotor retardation, excessive weight loss (not to be confused with anorexia nervosa), or excessive guilt.
- Atypical depression is characterized by mood reactivity (paradoxical anhedonia) and positivity, significant weight gain or increased appetite (comfort eating), excessive sleep or sleepiness (hypersomnia), a sensation of heaviness in limbs known as leaden paralysis, and significant social impairment as a consequence of hypersensitivity to perceived interpersonal rejection.
- Catatonic depression is a rare and severe form of major depression involving disturbances of motor behavior and other symptoms. Here the person is mute and almost stuporous, and either remains immobile or exhibits purposeless or even bizarre movements. Catatonic symptoms also occur in schizophrenia or in manic episodes, or may be caused by neuroleptic malignant syndrome.
- Postpartum depression, or mental and behavioural disorders associated with the puerperium, not elsewhere classified, refers to the intense, sustained and sometimes disabling depression experienced by women after giving birth. Postpartum depression has an incidence rate of 10–15% among new mothers. The DSM-IV mandates that, in order to qualify as postpartum depression, onset occur within one month of delivery. It has been said that postpartum depression can last as long as three months.
- Seasonal affective disorder (SAD) is a form of depression in which depressive episodes come on in the autumn or winter, and resolve in spring. The diagnosis is made if at least two episodes have occurred in colder months with none at other times, over a two-year period or longer.

Differential diagnoses

To confer major depressive disorder as the most likely diagnosis, other potential diagnoses must be considered, including dysthymia, adjustment disorder with depressed mood or bipolar disorder. Dysthymia is a chronic, milder mood disturbance in which a person reports a low mood almost daily over a span of at least two years. The symptoms are not as severe as those for major depression, although people with dysthymia are vulnerable to secondary episodes of major depression (sometimes referred to as double depression). Adjustment disorder with depressed mood is a mood disturbance appearing as a
psychological response to an identifiable event or stressor, in which the resulting emotional or behavioral symptoms are significant but do not meet the criteria for a major depressive episode. Bipolar disorder, also known as manic-depressive disorder, is a condition in which depressive phases alternate with periods of mania or hypomania. Although depression is currently categorized as a separate disorder, there is ongoing debate because individuals diagnosed with major depression often experience some hypomanic symptoms, indicating a mood disorder continuum.

Other disorders need to be ruled out before diagnosing major depressive disorder. They include depressions due to physical illness, medications, and substance abuse. Depression due to physical illness is diagnosed as a mood disorder due to a general medical condition. This condition is determined based on history, laboratory findings, or physical examination. When the depression is caused by a substance abused including a drug of abuse, a medication, or exposure to a toxin, it is then diagnosed as a substance-induced mood disorder. In such cases, a substance is judged to be etiologically related to the mood disturbance.

Schizoaffective disorder is different from major depressive disorder with psychotic features because in the schizoaffective disorder at least two weeks of delusions or hallucinations must occur in the absence of prominent mood symptoms.

Depressive symptoms may be identified during schizophrenia, delusional disorder, and psychotic disorder not otherwise specified, and in such cases those symptoms are considered associated features of these disorders, therefore, a separate diagnosis is not deemed necessary unless the depressive symptoms meet full criteria for a major depressive episode. In that case, a diagnosis of depressive disorder not otherwise specified may be made as well as a diagnosis of schizophrenia.

Some cognitive symptoms of dementia such as disorientation, apathy, difficulty concentrating and memory loss may get confused with a major depressive episode in major depressive disorder. They are especially difficult to determine in elderly patients. In such cases, the premorbid state of the patient may be helpful to differentiate both disorders. In the case of dementia, there tends to be a premorbid history of declining cognitive function. In the case of a major depressive disorder patients tend to exhibit a relatively normal premorbid state and abrupt cognitive decline associated with the depression.

**Prevention**

Behavioral interventions, such as interpersonal therapy, are effective at preventing new onset depression. Because such interventions appear to be most effective when delivered to individuals or small groups, it has been suggested that they may be able to reach their large target audience most efficiently through the Internet. However, an earlier meta-analysis found preventive programs with a competence-enhancing component to be superior to behaviorally oriented programs overall, and found behavioral programs to be particularly unhelpful for older people, for whom social support programs were uniquely beneficial. Additionally, the programs that best prevented depression comprised more than
eight sessions, each lasting between 60 and 90 minutes; were provided by a combination of lay and professional workers; had a high-quality research design; reported attrition rates; and had a well-defined intervention. The "Coping with Depression" course (CWD) is claimed to be the most successful of psychoeducational interventions for the treatment and prevention of depression (both for its adaptability to various populations and its results), with a risk reduction of 38% in major depression and an efficacy as a treatment comparing favorably to other psychotherapies.

Management

The three most common treatments for depression are psychotherapy, medication, and electroconvulsive therapy. Psychotherapy is the treatment of choice for people under 18, while electroconvulsive therapy is only used as a last resort. Care is usually given on an outpatient basis, while treatment in an inpatient unit is considered if there is a significant risk to self or others.

Treatment options are much more limited in developing countries, where access to mental health staff, medication, and psychotherapy is often difficult. Development of mental health services is minimal in many countries; depression is viewed as a phenomenon of the developed world despite evidence to the contrary, and not as an inherently life-threatening condition. Physical exercise is recommended for management of mild depression, but it has only a moderate, statistically insignificant effect on symptoms in most cases of major depressive disorder.

Psychotherapy

Psychotherapy can be delivered, to individuals, groups, or families by mental health professionals, including psychotherapists, psychiatrists, psychologists, clinical social workers, counselors, and suitably trained psychiatric nurses. With more complex and chronic forms of depression, a combination of medication and psychotherapy may be used.

Cognitive behavioral therapy (CBT) currently has the most research evidence for the treatment of depression in children and adolescents, and CBT and interpersonal psychotherapy (IPT) are preferred therapies for adolescent depression. In people under 18, according to the National Institute for Health and Clinical Excellence, medication should only be offered in conjunction with a psychological therapy, such as CBT, interpersonal therapy, or family therapy.

Psychotherapy has been shown to be effective in older people. Successful psychotherapy appears to reduce the recurrence of depression even after it has been terminated or replaced by occasional booster sessions.

The most-studied form of psychotherapy for depression is CBT, which teaches clients to challenge self-defeating, but enduring ways of thinking (cognitions) and change counterproductive behaviours. Research beginning in the mid-1990s suggested that CBT could perform as well or better than antidepressants in patients with moderate to severe
depression. CBT may be effective in depressed adolescents, although its effects on severe episodes are not definitively known. Combining fluoxetine with CBT appeared to bring no additional benefit, or, at the most, only marginal benefit. Several variables predict success for cognitive behavioral therapy in adolescents: higher levels of rational thoughts, less hopelessness, fewer negative thoughts, and fewer cognitive distortions. CBT is particularly beneficial in preventing relapse. Several variants of cognitive behavior therapy have been used in depressed patients, most notably rational emotive behavior therapy, and more recently mindfulness-based cognitive therapy.

Psychoanalysis is a school of thought, founded by Sigmund Freud, which emphasizes the resolution of unconscious mental conflicts. Psychoanalytic techniques are used by some practitioners to treat clients presenting with major depression. A more widely practiced, eclectic technique, called psychodynamic psychotherapy, is loosely based on psychoanalysis and has an additional social and interpersonal focus. In a meta-analysis of three controlled trials of Short Psychodynamic Supportive Psychotherapy, this modification was found to be as effective as medication for mild to moderate depression.

Logotherapy, a form of existential psychotherapy developed by Austrian psychiatrist Viktor Frankl, addresses the filling of an "existential vacuum" associated with feelings of futility and meaninglessness. It is posited that this type of psychotherapy may be useful for depression in older adolescents.

Koreans use two different coping methods: emotion-focused coping and problem-focused coping. Emotion-focused coping decreases emotional distress through avoidance, distancing, and finding positive values in negative events. Problem-focused coping influences environmental conditions by altering the source of stress or changing one’s self (example, finding alternative ways of gratification).

**Antidepressants**

Zoloft (sertraline) is primarily used to treat major depression in adult outpatients. In 2007, it was the most prescribed antidepressant on the U.S. retail market, with 29,652,000 prescriptions.
The effectiveness of antidepressants is none to minimal in those with mild or moderate depression but significant in those with very severe disease. The effects of antidepressants are somewhat superior to those of psychotherapy, especially in cases of chronic major depression, although in short-term trials more patients—especially those with less serious forms of depression—cease medication than cease psychotherapy, most likely due to adverse effects from the medication and to patients’ preferences for psychological therapies over pharmacological treatments.

To find the most effective antidepressant medication with minimal side effects, the dosages can be adjusted, and if necessary, combinations of different classes of antidepressants can be tried. Response rates to the first antidepressant administered range from 50-75%, and it can take at least six to eight weeks from the start of medication to remission, when the patient is back to their normal self. Antidepressant medication treatment is usually continued for 16 to 20 weeks after remission, to minimize the chance of recurrence, and even up to one year of continuation is recommended. People with chronic depression may need to take medication indefinitely to avoid relapse.

Selective serotonin reuptake inhibitors (SSRIs) are the primary medications prescribed owing to their effectiveness, relatively mild side effects, and because they are less toxic in overdose than other antidepressants. Patients who do not respond to one SSRI can be switched to another antidepressant, and this results in improvement in almost 50% of cases. Another option is to switch to the atypical antidepressant bupropion. Venlafaxine, an antidepressant with a different mechanism of action, may be modestly more effective than SSRIs. However, venlafaxine is not recommended in the UK as a first-line treatment because of evidence suggesting its risks may outweigh benefits and it is specifically discouraged in children and adolescents. For adolescent depression, fluoxetine and escitalopram are the two recommended choices. Antidepressants have not been found to be beneficial in children. There is also insufficient evidence to determine effectiveness in those with depression complicated by dementia. Any antidepressant can cause low serum sodium levels (also called hyponatremia); nevertheless, it has been reported more often with SSRIs. It is not uncommon for SSRIs to cause or worsen insomnia; the sedating antidepressant mirtazapine can be used in such cases.

Monoamine oxidase inhibitors, an older class of antidepressants, have been plagued by potentially life-threatening dietary and drug interactions. They are still used only rarely, although newer and better tolerated agents of this class have been developed.

The terms "refractory depression" and "treatment-resistant depression" are used to describe cases that do not respond to adequate courses of at least two antidepressants. In many major studies, only about 35% of patients respond well to medical treatment. It may be difficult for a doctor to decide when someone has treatment-resistant depression or whether the problem is due to coexisting disorders, which are common among patients with major depression.
A team of psychologists from multiple American universities found that antidepressant drugs hardly have better effects than a placebo in cases of mild or moderate depression. The study focused on paroxetine and imipramine.

For children, adolescents, and probably young adults between 18–24 years old, there is a higher risk of both suicidal ideations and suicidal behavior in those treated with SSRIs. For adults, it is unclear whether or not SSRIs affect the risk of suicidality. One review found no connection; another an increased risk; and a third no risk in those 25–65 years old and a decrease risk in those more than 65. Epidemiological data has found that the widespread use of antidepressants in the new “SSRI-era” is associated with a significant decline in suicide rates in most countries with traditionally high baseline suicide rates. The causality of the relationship is inconclusive. A black box warning was introduced in the United States in 2007 on SSRI and other antidepressant medications due to increased risk of suicide in patients younger than 24 years old. Similar precautionary notice revisions were implemented by the Japanese Ministry of Health.

**Electroconvulsive therapy**

Electroconvulsive therapy (ECT) is a procedure whereby pulses of electricity are sent through the brain via two electrodes, usually one on each temple, to induce a seizure while the patient is under a brief period of general anaesthesia. Hospital psychiatrists may recommend ECT for cases of severe major depression which have not responded to antidepressant medication or, less often, psychotherapy or supportive interventions. ECT can have a quicker effect than antidepressant therapy and thus may be the treatment of choice in emergencies such as catatonic depression where the patient has stopped eating and drinking, or where a patient is severely suicidal. ECT is probably more effective than pharmacotherapy for depression in the immediate short-term, although a landmark community-based study found much lower remission rates in routine practice. When ECT is used on its own, the relapse rate within the first six months is very high; early studies put the rate at around 50%, while a more recent controlled trial found rates of 84% even with placebos. The early relapse rate may be reduced by the use of psychiatric medications or further ECT (although the latter is not recommended by some authorities) but remains high. Common initial adverse effects from ECT include short and long-term memory loss, disorientation and headache. Although memory disturbance after ECT usually resolves within one month, ECT remains a controversial treatment, and debate on its efficacy and safety continues.

**Prognosis**

Major depressive episodes often resolve over time whether or not they are treated. Outpatients on a waiting list show a 10–15% reduction in symptoms within a few months, with approximately 20% no longer meeting the full criteria for a depressive disorder. The median duration of an episode has been estimated to be 23 weeks, with the highest rate of recovery in the first three months.
Studies have shown that 80% of those suffering from their first major depressive episode will suffer from at least 1 more during their life, with a lifetime average of 4 episodes. Other general population studies indicate around half those who have an episode (whether treated or not) recover and remain well, while the other half will have at least one more, and around 15% of those experience chronic recurrence. Studies recruiting from selective inpatient sources suggest lower recovery and higher chronicity, while studies of mostly outpatients show that nearly all recover, with a median episode duration of 11 months. Around 90% of those with severe or psychotic depression, most of whom also meet criteria for other mental disorders, experience recurrence.

Recurrence is more likely if symptoms have not fully resolved with treatment. Current guidelines recommend continuing antidepressants for four to six months after remission to prevent relapse. Evidence from many randomized controlled trials indicates continuing antidepressant medications after recovery can reduce the chance of relapse by 70% (41% on placebo vs. 18% on antidepressant). The preventive effect probably lasts for at least the first 36 months of use.

Those people who experience repeated episodes of depression require ongoing treatment in order to prevent more severe, long-term depression. In some cases, people need to take medications for long periods of time or for the rest of their lives.

Cases when outcome is poor are associated with inappropriate treatment, severe initial symptoms that may include psychosis, early age of onset, more previous episodes, incomplete recovery after 1 year, pre-existing severe mental or medical disorder, and family dysfunction as well.

Depressed individuals have a shorter life expectancy than those without depression, in part because depressed patients are at risk of dying by suicide. However, they also have a higher rate of dying from other causes, being more susceptible to medical conditions such as heart disease. Up to 60% of people who commit suicide have a mood disorder such as major depression, and the risk is especially high if a person has a marked sense of hopelessness or has both depression and borderline personality disorder. The lifetime risk of suicide associated with a diagnosis of major depression in the US is estimated at 3.4%, which averages two highly disparate figures of almost 7% for men and 1% for women (although suicide attempts are more frequent in women). The estimate is substantially lower than a previously accepted figure of 15% which had been derived from older studies of hospitalized patients.

Depression is often associated with unemployment and poverty. Major depression is currently the leading cause of disease burden in North America and other high-income countries, and the fourth-leading cause worldwide. In the year 2030, it is predicted to be the second-leading cause of disease burden worldwide after HIV, according to the World Health Organization. Delay or failure in seeking treatment after relapse, and the failure of health professionals to provide treatment, are two barriers to reducing disability.
Epidemiology

Prevalence

Age-standardised disability-adjusted life year (DALY) rates of unipolar depressive disorders by country (per 100,000 inhabitants) in 2004.

Depression is a major cause of morbidity worldwide. Lifetime prevalence varies widely, from 3% in Japan to 17% in the US. In most countries the number of people who would suffer from depression during their lives falls within an 8–12% range. In North America the probability of having a major depressive episode within a year-long period is 3–5% for males and 8–10% for females. Population studies have consistently shown major depression to be about twice as common in women as in men, although it is unclear why this is so, and whether factors unaccounted for are contributing to this. The relative increase in occurrence is related to pubertal development rather than chronological age, reaches adult ratios between the ages of 15 and 18, and appears associated with psychosocial more than hormonal factors.

People are most likely to suffer their first depressive episode between the ages of 30 and 40, and there is a second, smaller peak of incidence between ages 50 and 60. The risk of major depression is increased with neurological conditions such as stroke, Parkinson's disease, or multiple sclerosis and during the first year after childbirth. It is also more common after cardiovascular illnesses, and is related more to a poor outcome than to a better one. Studies conflict on the prevalence of depression in the elderly, but most data suggest there is a reduction in this age group. Depressive disorder are most common to observe in urban than in rural population and the prevalence is in groups with higher socioeconomic factors i.e. homeless people.

Comorbidity

Major depression frequently co-occurs with other psychiatric problems. The 1990–92 National Comorbidity Survey (US) reports that 51% of those with major depression also suffer from lifetime anxiety. Anxiety symptoms can have a major impact on the course of a depressive illness, with delayed recovery, increased risk of relapse, greater disability and increased suicide attempts. American neuroendocrinologist Robert Sapolsky similarly argues that the relationship between stress, anxiety, and depression could be measured and demonstrated biologically. There are increased rates of alcohol and drug abuse and particularly dependence, and around a third of individuals diagnosed with ADHD develop comorbid depression. Post-traumatic stress disorder and depression often co-occur.

Depression and pain often co-occur, especially if it is chronic or uncontrollable pain. This conforms with Seligman’s theory of learned helplessness. One or more pain symptoms is present in 65% of depressed patients, and anywhere from five to 85% of patients with pain will be suffering from depression, depending on the setting; there is a lower prevalence in general practice, and higher in specialty clinics. The diagnosis of depression is often
delayed or missed, and the outcome worsens. The outcome can also obviously worsen if the depression is noticed but completely misunderstood.

Depression is also associated with a 1.5- to 2-fold increased risk of cardiovascular disease, independent of other known risk factors, and is itself linked directly or indirectly to risk factors such as smoking and obesity. People with major depression are less likely to follow medical recommendations for treating cardiovascular disorders, which further increases their risk. In addition, cardiologists may not recognize underlying depression that complicates a cardiovascular problem under their care.

**History**

The Ancient Greek physician Hippocrates described a syndrome of melancholia as a distinct disease with particular mental and physical symptoms; he characterized all "fears and despondencies, if they last a long time" as being symptomatic of the ailment. It was a similar but far broader concept than today’s depression; prominence was given to a clustering of the symptoms of sadness, dejection, and despondency, and often fear, anger, delusions and obsessions were included.

The term depression itself was derived from the Latin verb deprimere, "to press down". From the 14th century, "to depress" meant to subjugate or to bring down in spirits. It was used in 1665 in English author Richard Baker's Chronicle to refer to someone having "a great depression of spirit", and by English author Samuel Johnson in a similar sense in 1753. The term also came in to use in physiology and economics. An early usage referring to a psychiatric symptom was by French psychiatrist Louis Delasauve in 1856, and by the 1860s it was appearing in medical dictionaries to refer to a physiological and metaphorical lowering of emotional function. Since Aristotle, melancholia had been associated with men of learning and intellectual brilliance, a hazard of contemplation and creativity. The newer concept abandoned these associations and through the 19th century, became more associated with women.

Although melancholia remained the dominant diagnostic term, depression gained increasing currency in medical treatises and was a synonym by the end of the century; German psychiatrist Emil Kraepelin may have been the first to use it as the overarching term, referring to different kinds of melancholia as depressive states.

Sigmund Freud likened the state of melancholia to mourning in his 1917 paper Mourning and Melancholia. He theorized that objective loss, such as the loss of a valued relationship through death or a romantic break-up, results in subjective loss as well; the depressed individual has identified with the object of affection through an unconscious, narcissistic process called the libidinal cathexis of the ego. Such loss results in severe melancholic symptoms more profound than mourning; not only is the outside world viewed negatively, but the ego itself is compromised. The patient’s decline of self-perception is revealed in his belief of his own blame, inferiority, and unworthiness. He also emphasized early life experiences as a predisposing factor. Meyer put forward a mixed social and biological framework emphasizing reactions in the context of an individual’s life, and argued that the
term depression should be used instead of melancholia. The first version of the DSM (DSM-I, 1952) contained depressive reaction and the DSM-II (1968) depressive neurosis, defined as an excessive reaction to internal conflict or an identifiable event, and also included a depressive type of manic-depressive psychosis within Major affective disorders.

In the mid-20th century, researchers theorized that depression was caused by a chemical imbalance in neurotransmitters in the brain, a theory based on observations made in the 1950s of the effects of reserpine and isoniazid in altering monoamine neurotransmitter levels and affecting depressive symptoms.

The term Major depressive disorder was introduced by a group of US clinicians in the mid-1970s as part of proposals for diagnostic criteria based on patterns of symptoms (called the "Research Diagnostic Criteria", building on earlier Feighner Criteria), and was incorporated into the DSM-III in 1980. To maintain consistency the ICD-10 used the same criteria, with only minor alterations, but using the DSM diagnostic threshold to mark a mild depressive episode, adding higher threshold categories for moderate and severe episodes. The ancient idea of melancholia still survives in the notion of a melancholic subtype.

The new definitions of depression were widely accepted, albeit with some conflicting findings and views. There have been some continued empirically based arguments for a return to the diagnosis of melancholia. There has been some criticism of the expansion of coverage of the diagnosis, related to the development and promotion of antidepressants and the biological model since the late 1950s.

Society and culture
Former American president Abraham Lincoln suffered from "melancholy", a condition which now may be referred to as clinical depression.

People's conceptualizations of depression vary widely, both within and among cultures. "Because of the lack of scientific certainty," one commentator has observed, "the debate over depression turns on questions of language. What we call it—'disease,' 'disorder,' 'state of mind'—affects how we view, diagnose, and treat it." There are cultural differences in the extent to which serious depression is considered an illness requiring personal professional treatment, or is an indicator of something else such as the need to address social or moral problems, the result of biological imbalances, or a reflection of individual differences in the understanding of distress that may reinforce feelings of powerlessness, and emotional struggle.

The diagnosis is less common in some countries, such as China. It has been argued that the Chinese traditionally deny or somatize emotional depression (although since the early 1980s the Chinese denial of depression may have modified drastically). Alternatively, it may be that Western cultures reframe and elevate some expressions of human distress to disorder status. Australian professor Gordon Parker and others have argued that the Western concept of depression "medicalizes" sadness or misery. Similarly, Hungarian-American psychiatrist Thomas Szasz and others argue that depression is a metaphorical illness that is inappropriately regarded as an actual disease. There has also been concern that the DSM, as well as the field of descriptive psychiatry that employs it, tends to reify abstract phenomena such as depression, which may in fact be social constructs. American archetypal psychologist James Hillman writes that depression can be healthy for the soul, insofar as "it brings refuge, limitation, focus, gravity, weight, and humble powerlessness." Hillman argues that therapeutic attempts to eliminate depression echo the Christian theme of resurrection, but have the unfortunate effect of demonizing a soulful state of being.

Historical figures were often reluctant to discuss or seek treatment for depression due to social stigma about the condition, or due to ignorance of diagnosis or treatments. Nevertheless, analysis or interpretation of letters, journals, artwork, writings or statements of family and friends of some historical personalities has led to the presumption that they may have had some form of depression. People who may have had depression include English author Mary Shelley, American-British writer Henry James, and American president Abraham Lincoln. Some well-known contemporary people with possible depression include Canadian songwriter Leonard Cohen and American playwright and novelist Tennessee Williams. Some pioneering psychologists, such as Americans William James and John B. Watson, dealt with their own depression.

There has been a continuing discussion of whether neurological disorders and mood disorders may be linked to creativity, a discussion that goes back to Aristotelian times. British literature gives many examples of reflections on depression. English philosopher John Stuart Mill experienced a several-months-long period of what he called "a dull state of nerves", when one is "unsusceptible to enjoyment or pleasurable excitement; one of those moods when what is pleasure at other times, becomes insipid or indifferent". He quoted English poet Samuel Taylor Coleridge's "Dejection" as a perfect description of his case: "A
grief without a pang, void, dark and drear, / A drowsy, stifled, unimpassioned grief, / Which finds no natural outlet or relief / In word, or sigh, or tear." English writer Samuel Johnson used the term "the black dog" in the 1780s to describe his own depression, and it was subsequently popularized by depression sufferer former British Prime Minister Sir Winston Churchill.

Social stigma of major depression is widespread, and contact with mental health services reduces this only slightly. Public opinions on treatment differ markedly to those of health professionals; alternative treatments are held to be more helpful than pharmacological ones, which are viewed poorly. In the UK, the Royal College of Psychiatrists and the Royal College of General Practitioners conducted a joint Five-year Defeat Depression campaign to educate and reduce stigma from 1992 to 1996; a MORI study conducted afterwards showed a small positive change in public attitudes to depression and treatment.

**Depression (differential diagnoses)**

![Image](image.png)

Neuroimaging can be a valuable tool in the diagnostic work-up of various psychiatric disorders including depression.

Depression, one of the most commonly diagnosed psychiatric disorders, is being diagnosed in increasing numbers in various segments of the population worldwide. Depression in the United States alone affects 17.6 million Americans each year or 1 in 6 people. Depressed patients are at increased risk of type 2 diabetes, cardiovascular disease and suicide. Within the next twenty years depression is expected to become the second leading cause of disability worldwide and the leading cause in high-income nations, including the United States. In approximately 75% of completed suicides the individuals had seen a physician within the prior year before their death, 45%-66% within the prior month. Approximately 33% - 41% of those who completed suicide had contact with mental health services in the prior year, 20% within the prior month.
There are many psychiatric and medical conditions that may mimic some or all of the symptoms of depression, or may occur comorbid to it. A disorder either psychiatric or medical that shares symptoms and characteristics of another disorder, and may be the true cause of the presenting symptoms is known as a differential diagnosis.

Many psychiatric disorders such as depression are diagnosed by allied health professionals with little or no medical training, and are made on the basis of presenting symptoms without proper consideration of the underlying cause; adequate screening of differential diagnoses is often not conducted. According to one study "non-medical mental health care providers may be at increased risk of not recognizing masked medical illnesses in their patients."

Misdiagnosis or missed diagnoses may lead to lack of treatment or ineffective and potentially harmful treatment which may worsen the underlying causative disorder. A conservative estimate is that 10% of all psychological symptoms may be due to medical reasons, with the results of one study suggesting that about 50% of individuals with a serious mental illness "have general medical conditions that are largely undiagnosed and untreated and may cause or exacerbate psychiatric symptoms."

In a case of misdiagnosed depression recounted in Newsweek, a writer received treatment for depression for years; during the last 10 years of her depression the symptoms worsened, resulting in multiple suicide attempts and psychiatric hospitalizations. When an MRI finally was performed it showed the presence of a tumor. She was however told by a neurologist that it was benign. After a worsening of symptoms, upon the second opinion of another neurologist, the tumor was removed. After the surgery she no longer suffered from "depression."

**Autoimmune disorders**

Celiac disease; is an autoimmune disorder in which the body is unable to digest gluten, found in various grains such as rye and barley. Current research has shown its neuropsychiatric symptoms may manifest without the gastrointestinal symptoms.

"However, more recent studies have emphasized that a wider spectrum of neurologic syndromes may be the presenting extraintestinal manifestation of gluten sensitivity with or without intestinal pathology."

Lupus: Systemic lupus erythematosus (SLE), is a chronic autoimmune connective tissue disease that can affect any part of the body. Lupus can cause or worsen depression.

**Bacterial-viral-parasitic infection**
Lyme disease; is a bacterial infection caused by Borrelia burgdorferi, a spirochete bacterium transmitted by the Deer tick (Ixodes scapularis). Lyme disease is one of a group of diseases which have earned the name the "great imitator" for their propensity to mimic the symptoms of a wide variety of medical and neuropsychiatric disorders. Lyme disease is an underdiagnosed illness, partially as a result of the complexity and unreliability of serologic testing.

"Because of the rapid rise of Lyme borreliosis nationwide and the need for antibiotic treatment to prevent severe neurologic damage, mental health professionals need to be aware of its possible psychiatric presentations.

- **Syphilis**: the prevalence of which is on the rise, is another of the "great imitators", which if left untreated can progress to neurosyphilis and affect the brain, can present with solely neuropsychiatric symptoms. "This case emphasises that neurosyphilis still has to be considered in the differential diagnosis within the context of psychiatric conditions and diseases. Owing to current epidemiological data and difficulties in diagnosing syphilis, routine screening tests in the psychiatric field are necessary."

- **Neurocysticercosis (NCC)**: is an infection of the brain or spinal cord caused by the larval stage of the pork tapeworm, Taenia solium. NCC is the most common helminthic (parasitic worm) infestation of the central nervous system worldwide. Humans develop cysticercosis when they ingest eggs of the pork tapeworm via contact with contaminated fecal matter or eating infected vegetables or undercooked pork. "While cysticercosis is endemic in Latin America, it is an emerging disease with increased prevalence in the United States." "The rate of depression in those with neurocysticercosis is higher than in the general population."

- **Toxoplasmosis**: is an infection caused by Toxoplasma gondii an intracellular protozoan parasite. Humans can be infected in 3 different ways: ingestion of tissue cysts, ingestion of oocysts, or in utero infection with tachyzoites. One of the prime methods for transmission to humans is contact with the feces of the host species, the
domesticated cat. Toxoplasma gondii infects approximately 30% of the world's human population, but causes overt clinical symptoms in only a small segment of those infected. Exposure to Toxoplasma gondii (seropositivity) without developing Toxoplasmosis has been proven to alter various characteristics of human behavior as well as being a causative factor in some cases of depression, in addition, studies have linked seropositivity with an increased rate of suicide.

- **West Nile virus (WNV)**; which can cause encephalitis has been reported to be a causal factor in developing depression in 31% of those infected in a study conducted in Houston, Texas and reported to the Center for Disease Control (CDC). The primary vectors for disease transmission to humans are various species of mosquito. WNV which is endemic to Southern Europe, Africa the Middle East and Asia was first identified in the United States in 1999. Between 1999 and 2006, 20,000 cases of confirmed symptomatic WNV were reported in the United States, with estimates of up to 1 million being infected. "WNV is now the most common cause of epidemic viral encephalitis in the United States, and it will likely remain an important cause of neurological disease for the foreseeable future."

**Blood disorders**

Anemia: is a decrease in normal number of red blood cells (RBCs) or less than the normal quantity of hemoglobin in the blood. Depressive symptoms are associated with anemia in a general population of older persons living in the community.

**Chronic fatigue syndrome**

Between 1 and 4 million Americans are believed to have Chronic Fatigue Syndrome (CFS), yet only 50% have consulted a physician for symptoms of CFS. In addition individuals with CFS symptoms often have an undiagnosed medical or psychiatric disorder such as diabetes, thyroid disease or substance abuse. CFS, at one time considered to be psychosomatic in nature, is now considered to be a valid medical condition in which early diagnosis and treatment can aid in alleviating or completely resolving symptoms. While frequently misdiagnosed as depression, differences have been noted in rate of cerebral blood flow.

CFS is underdiagnosed in more than 80% of the people who have it; at the same time, it is often misdiagnosed as depression.

**Dietary disorders**

Fructose malabsorption and lactose intolerance; deficient fructose transport by the duodenum, or by the deficiency of the enzyme, lactase in the mucosal lining, respectively. As a result of this malabsorption, the saccharides reach the colon and are digested by bacteria which convert them to short chain fatty acids, CO2, and H2. Approximately 50% of those afflicted exhibit the physical signs of irritable bowel syndrome.
"Fructose malabsorption may play a role in the development of depressed mood. Fructose malabsorption should be considered in patients with symptoms of major depression."

"Fructose and sorbitol reduced diet in subjects with fructose malabsorption does not only reduce gastrointestinal symptoms but also improves mood and early signs of depression."

**Endocrine system disorders**

Dysregulation of the endocrine system may present with various neuropsychiatric symptoms; irregularities in the hypothalamic-pituitary-adrenal (HPA) axis and the hypothalamic-pituitary-thyroid (HPT) axis have been shown in patients with primary depression.

**HPT and HPA axes abnormalities observed in patients with depression**
(Musselman DL, Nemeroff CB. 1996)

HPT axes irregularities:

- alterations in thyroid-stimulating hormone (TSH) response to thyrotropin-releasing hormone (TRH)
- an abnormally high rate of antithyroid antibodies
- elevated cerebrospinal fluid (CSF) TRH concentrations.

HPA axes irregularities:

- adrenocorticoid hypersecretion
- enlarged pituitary and adrenal gland size (organomegaly)
- elevated corticotropin-releasing factor (CSF) concentrations

Adrenal gland

Addison's disease: also known as chronic adrenal insufficiency, hypocortisolism, and hypocorticism) is a rare endocrine disorder wherein the adrenal glands, located above the kidneys, produce insufficient steroid hormones (glucocorticoids and often mineralocorticoids). "Addison's disease presenting with psychiatric features in the early stage has the tendency to be overlooked and misdiagnosed."

**Thyroid and parathyroid glands**
Thyroid and Parathyroid Glands

Location of the thyroid and parathyroid glands in front of the larynx.

- Graves' disease: an autoimmune disease where the thyroid is overactive, resulting in hyperthyroidism and thyrotoxicosis.
- Hashimoto's thyroiditis: also known chronic lymphocytic thyroiditis is an autoimmune disease in which the thyroid gland is gradually destroyed by a variety of cell and antibody mediated immune processes. Hashimoto's thyroiditis is associated with thyroid peroxidase and thyroglobulin autoantibodies.
- Hypothyroidism
- Hyperthyroidism
- Hypoparathyroidism; can affect calcium homeostasis, supplementation of which has completely resolved cases of depression in which hypoparathyroidism is the sole causative factor.

Pituitary tumors

Tumors of the pituitary gland are fairly common in the general population with estimates ranging as high as 25%. Most tumors are considered to be benign and are often an incidental finding discovered during autopsy or as of neuroimaging in which case they are dubbed "incidentalomas". Even in benign cases, pituitary tumors can affect cognitive, behavioral and emotional changes. Pituitary microadenomas are smaller than 10 mm in diameter and are generally considered benign, yet the presence of a microadenoma has been positively identified as a risk factor for suicide.

"... patients with pituitary disease were diagnosed and treated for depression and showed little response to the treatment for depression."

Pancreas

Hypoglycemia: an overproduction of insulin causes reduced blood levels of glucose. In one study of patients recovering from acute lung injury in intensive care, those patients who developed hypoglycemia while hospitalized showed an increased rate of depression.
Neurological

In addition to pituitary tumors, tumors in various locations in the central nervous system may cause depressive symptoms and be misdiagnosed as depression.

Post concussion syndrome

Post-concussion syndrome (PCS), is a set of symptoms that a person may experience for weeks, months, or occasionally years after a concussion with a prevalence rate of 38–80% in mild traumatic brain injuries, it may also occur in moderate and severe cases of traumatic brain injury. A diagnosis may be made when symptoms resulting from concussion, depending on criteria, last for more than three to six months after the injury, in which case it is termed persistent postconcussive syndrome (PPCS). In a study of the prevalence of post concussion syndrome symptoms in patients with depression utilizing the British Columbia Postconcussion Symptom Inventory: "Approximately 9 out of 10 patients with depression met liberal self-report criteria for a postconcussion syndrome and more than 5 out of 10 met conservative criteria for the diagnosis." These self reported rates were significantly higher than those obtained in a scheduled clinical interview. Normal controls have exhibited symptoms of PCS as well as those seeking psychological services. There is considerable debate over the diagnosis of PCS in part because of the medico-legal and thus monetary ramifications of receiving the diagnosis.

Pseudobulbar affect

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>PBA</th>
<th>Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration</td>
<td>Seconds to minutes</td>
<td>Weeks to months</td>
</tr>
<tr>
<td>Voluntary control</td>
<td>None to minimal</td>
<td>Can be modulated by the situation</td>
</tr>
<tr>
<td>Affect</td>
<td>Unrelated to or independent of mood</td>
<td>Sad, worried, guilty, depressed; most often congruent with mood</td>
</tr>
<tr>
<td>Behavior</td>
<td>Does not change</td>
<td>Fatigued, apathetic, occasionally agitated</td>
</tr>
<tr>
<td>Perception</td>
<td>No misperceptions</td>
<td>Distorted and negative view of self, others, and future</td>
</tr>
<tr>
<td>Insight</td>
<td>Usually not impaired</td>
<td>May be impaired</td>
</tr>
<tr>
<td>Stimulus</td>
<td>Nonspecific, minimal or inappropriate to situation</td>
<td>Specific mood-related situations</td>
</tr>
</tbody>
</table>

Diagnostic differences between PBA and depression

Pseudobulbar affect (PBA) is an affective disinhibition syndrome that is largely unrecognized in clinical settings and thus often untreated due to ignorance of the clinical
manifestations of the disorder; it may be misdiagnosed as depression. It often occurs secondary to various neurodegenerative diseases such as amyotrophic lateral sclerosis, and also can result from head trauma. PBA is characterized by involuntary and inappropriate outbursts of laughter and/or crying. PBA has a high prevalence rate with estimates of 1.5 - 2 million cases in the United States alone.

**Neurotoxicity**

Various compounds have been shown to have neurotoxic effects many of which have been implicated as having a causal relationship in the development of depression.

**Cigarette smoking**

There has been research which suggests a correlation between cigarette smoking and depression. The results of one recent study suggest that smoking cigarettes may have a direct causal effect on the development of depression. There have been various studies done showing a positive link between smoking, suicidal ideation and suicide attempts.

In a study conducted among nurses, those smoking between 1-24 cigarettes per day had twice the suicide risk; 25 cigarettes or more, 4 times the suicide risk, than those who had never smoked. In a study of 300,000 male U.S. Army soldiers, a definitive link between suicide and smoking was observed with those smoking over a pack a day having twice the suicide rate of non-smokers.

**Link Between Smoking Depression and Suicide**

"Current daily smoking, but not past smoking, predicted the subsequent occurrence of suicidal thoughts or attempt."

"It would seem unwise, nevertheless, to rule out the possibility that smoking might be among the antecedent factors associated with the development of depression."

"Abstinence from cigarettes for prolonged periods may be associated with a decrease in depressive symptomatology."

"The stress induction model of smoking suggests, however, that smoking causes stress and concomitant negative affect."

**Medication**

Various medications have been suspected of having a causal relation in the development of depression; this has been classified as "organic mood syndrome". Some classes of medication such as those used to treat hypertension, have been recognized for decades as having a definitive relationship with the development of depression.
Monitoring of those taking medications which have shown a relationship with depression is often indicated, as well as the necessity of factoring in the use of such medications in the diagnostic process.

Topical Tretinoin (Retin-A); derived from Vitamin A and used for various medical conditions such as in topical solutions used to treat acne vulgaris. Although applied externally to the skin, it may enter the bloodstream and cross the blood brain barrier where it may have neurotoxic effects.

Interferons; proteins produced by the human body, three types have been identified alpha, beta and gamma. Synthetic versions are utilized in various medications used to treat different medical conditions such as the use of interferon-alpha in cancer treatment and hepatitis C treatment. All three classes of interferons may cause depression and suicidal ideation.

**Chronic Exposure to Organophosphates**

The neuropsychiatric effects of chronic organophosphate exposure include mood disorders, suicidal thinking and behaviour, cognitive impairment and chronic fatigue.

**Neuropsychiatric**

**Bipolar disorder**

Bipolar disorder is frequently misdiagnosed as major depression, and is thus treated with antidepressants alone which is not only not efficacious it is often contraindicated as it may exacerbate hypomania, mania, or cycling between moods. J Clin Psychiatry. 2005 Nov;66(11):1432-40.

"Misdiagnosed bipolar patients received inappropriate and costly treatment regimens involving overuse of antidepressants and underuse of potentially effective medications.... It is recommended that steps be taken to minimize misdiagnosis in clinical settings."

**Nutritional deficiencies**

Nutrition plays a key role in every facet of maintaining proper physical and psychological well being. Insufficient or inadequate nutrition can have a profound effect on mental health. The emerging field of Nutritional Neuroscience explores the various connections between diet, neurological functioning and mental health.

- Vitamin B6:pyridoxal phosphate (PLP) the active form of B6 is a cofactor in the dopamine serotonin pathway, a deficiency in Vitamin B6 may cause depressive symptoms.
- Folate (vitamin B9) - Vitamin B12 cobalamin : Low blood plasma and particularly red cell folate and diminished levels of Vitamin B12 have been found in patients with depressive disorders. "[W]e suggest that oral doses of both folic acid (800
μg/(mcg) daily) and vitamin B12 (1 mg daily) should be tried to improve treatment outcome in depression."

Long chain fatty acids: higher levels of omega-6 and lower levels of omega-3 fatty acids has been associated with depression and behavioral change.

**Sleep disorders**

- **Insomnia:** While the inability to fall asleep is often a symptom of depression, it can also in some instances serve as the trigger for developing a depressive disorder. It can be transient, acute or chronic. It can be a primary disorder or a co-morbid one.

- **Restless legs syndrome:** (RLS), also known as Wittmaack-Ekbom's syndrome, is characterized by an irresistible urge to move one’s body to stop uncomfortable or odd sensations. It most commonly affects the legs, but can also affect the arms or torso, and even phantom limbs. Restless Leg syndrome has been associated with Major depressive disorder. "Adjusted odds ratio for diagnosis of major depressive disorder... suggested a strong association between restless legs syndrome and major depressive disorder and/or panic disorder."

- **Sleep apnea:** is a sleep disorder characterized by pauses in breathing during sleep. Each episode, called an apnea, lasts long enough for one or more breaths to be missed; such episodes occur repeatedly throughout the sleep cycle. Undiagnosed sleep apnea may cause or contribute to the severity of depression.

- **Circadian rhythm sleep disorders:** of which too few clinicians are aware, often go untreated or are treated inappropriately, as when misdiagnosed as either primary insomnia or as a psychiatric condition.

**Management of depression**

Depression, for the purposes of this article, refers to the mental disorder known as major depressive disorder. This kind of depression is a recognized clinical condition and is becoming a common condition in developed countries, where up to 20% of the population is affected by this disorder at some stage of their lives. Patients are usually assessed and managed as outpatients, and only admitted to an inpatient mental health unit if they are considered to pose a risk to themselves or others.

The three most commonly indicated treatments for depression are psychotherapy, psychiatric medication, and (in severe cases) electroconvulsive therapy. Psychiatric medication are the primary therapy for major depression. Psychotherapy is the treatment of choice in those under the age of 18, with medication offered only in conjunction with the former and generally not as a first line agent. Furthermore, pathology in the parents may need to be looked for and addressed in parallel.

**Psychotherapy**
There are a number of different psychotherapies for depression, which may be provided to individuals or groups. Psychotherapy can be delivered by a variety of mental health professionals, including psychotherapists, psychiatrists, psychologists, clinical social workers, counselors, and psychiatric nurses. With more complex and chronic forms of depression the most effective treatment is often considered to be a combination of medication and psychotherapy. As mentioned earlier, psychotherapy is the treatment of choice in people under 18.

The most studied form of psychotherapy for depression is cognitive behavioral therapy (CBT), thought to work by teaching clients to learn a set of cognitive and behavioral skills, which they can employ on their own. Earlier research suggested that cognitive-behavioral therapy was not as effective as antidepressant medication in the treatment of depression; however, more recent research suggests that it can perform as well as antidepressants in treating patients with moderate to severe depression.

Behavior therapy for depression is sometimes referred to as behavioral activation Studies exist showing behavioral activation to be superior to CBT. In addition, behavioral activation appears to take less time and lead to longer lasting change.

For the treatment of adolescent depression, CBT performed no better than placebo, and significantly worse than the antidepressant fluoxetine. Combining fluoxetine with CBT appeared to bring no additional benefit or, at the most, only marginal benefit.

A review of four studies on the effectiveness of mindfulness-based cognitive therapy (MBCT), a recently developed class-based program designed to prevent relapse, suggests that MBCT may have an additive effect when provided with the usual care in patients who have had three or more depressive episodes, although the usual care did not include antidepressant treatment or any psychotherapy, and the improvement observed may have reflected non-specific or placebo effects.

Interpersonal psychotherapy focuses on the social and interpersonal triggers that may cause depression. There is evidence that it is an effective treatment for depression. Here, the therapy takes a structured course with a set number of weekly sessions (often 12) as in the case of CBT, however the focus is on relationships with others. Therapy can be used to help a person develop or improve interpersonal skills in order to allow him or her to communicate more effectively and reduce stress.

Psychoanalysis, a school of thought founded by Sigmund Freud that emphasizes the resolution of unconscious mental conflicts, is used by its practitioners to treat clients presenting with major depression. A more widely practiced technique, called psychodynamic psychotherapy, is loosely based on psychoanalysis and has an additional social and interpersonal focus. In a meta-analysis of three controlled trials, psychodynamic psychotherapy was found to be as effective as medication for mild to moderate depression.

**Medication**
To find the most effective pharmaceutical treatment, the dosages of medications must often be adjusted, different combinations of antidepressants tried, or antidepressants changed. Response rates to the first agent administered may be as low as 50%. It may take anywhere from three to eight weeks after the start of medication before its therapeutic effects can be fully discovered. Patients are generally advised not to stop taking an antidepressant suddenly and to continue its use for at least four months to prevent the chance of recurrence. People with chronic depression need to take the medication for the rest of their lives.

Selective serotonin reuptake inhibitors (SSRIs), such as sertraline (Zoloft, Lustral), escitalopram (Lexapro, Cipralex), fluoxetine (Prozac), paroxetine (Seroxat), and citalopram (Cipralex), are the primary medications considered, due to their relatively mild side effects and broad effect on the symptoms of depression and anxiety, as well as reduced risk in overdose, compared to their older tricyclic alternatives. Those who do not respond to the first SSRI tried can be switched to another; such a switch results in improvement in almost 50% of cases. Another popular option is to switch to the atypical antidepressant bupropion (Wellbutrin) or to add bupropion to the existing therapy; this strategy is possibly more effective. It is not uncommon for SSRIs to cause or worsen insomnia; the sedating antidepressant mirtazapine (Zispin, Remeron) can be used in such cases. Cognitive Behavioral Therapy for Insomnia can also help to alleviate the insomnia without additional medication. Venlafaxine (Effexor) may be moderately more effective than SSRIs; however, it is not recommended as a first-line treatment because of the higher rate of side effects, and its use is specifically discouraged in children and adolescents. Fluoxetine is the only antidepressant recommended for people under the age of 18. Evidence of effectiveness of SSRIs in those with depression complicated by dementia is lacking.

Tricyclic antidepressants have more side effects than SSRIs and are usually reserved for the treatment of inpatients, for whom the tricyclic antidepressant amitriptyline, in particular, appears to be more effective. A different class of antidepressants, the monoamine oxidase inhibitors, have historically been plagued by questionable efficacy and life-threatening
adverse effects. They are still used only rarely, although newer agents of this class (RIMA), with a better side effect profile, have been developed.

**Augmentation**

Physicians often add a medication with a different mode of action to bolster the effect of an antidepressant in cases of treatment resistance; a 2002 large community study of 244,859 depressed Veterans Administration patients found that 22% had received a second agent, most commonly a second antidepressant. Lithium has been used to augment antidepressant therapy in those who have failed to respond to antidepressants alone. Furthermore, lithium dramatically decreases the suicide risk in recurrent depression. Addition of atypical antipsychotics when the patient has not responded to an antidepressant is also known to increase the effectiveness of antidepressant drugs, albeit at the cost of more frequent side effects. There is some evidence for the addition of a thyroid hormone, triiodothyronine, in patients with normal thyroid function.

**Efficacy of medication and psychotherapy**

Antidepressants are statistically superior to placebo but their overall effect is low-to-moderate. In that respect they often did not exceed the National Institute for Health and Clinical Excellence criteria for a "clinically significant" effect. In particular, the effect size was very small for moderate depression but increased with severity reaching "clinical significance" for very severe depression. These results were consistent with the earlier clinical studies in which only patients with severe depression benefited from either psychotherapy or treatment with an antidepressant, imipramine, more than from the placebo treatment. Despite obtaining similar results, the authors argued about their interpretation. One author concluded that there "seems little evidence to support the prescription of antidepressant medication to any but the most severely depressed patients, unless alternative treatments have failed to provide benefit." The other author agreed that "antidepressant 'glass' is far from full" but disagreed "that it is completely empty". He pointed out that the first-line alternative to medication is psychotherapy, which does not have superior efficacy.

Antidepressants in general are as effective as psychotherapy for major depression, and this conclusion holds true for both severe and mild forms of MDD. In contrast, medication gives better results for dysthymia. The subgroup of SSRIs may be slightly more efficacious than psychotherapy. On the other hand, significantly more patients drop off from the antidepressant treatment than from psychotherapy, likely because of the side effects of antidepressants. Successful psychotherapy appears to prevent the recurrence of depression even after it has been terminated or replaced by occasional "booster" sessions. The same degree of prevention can be achieved by continuing antidepressant treatment.

Two studies suggest that the combination of psychotherapy and medication is the most effective way to treat depression in adolescents. Both TADS (Treatment of Adolescents with Depression Study) and TORDIA (Treatment of Resistant Depression in Adolescents) showed very similar results. TADS resulted in 71% of their teen subjects having a "much"
or "very much" improvement in mood over the 60.6% with medication alone and the 43.2% with CBT alone. Similarly, TORDIA showed a 54.8% improvement with CBT and drugs verses a 40.5% with drug therapy alone.

**Other medications**

Numerous alternative treatments have been used to treat depression, whether medications or other kinds of intervention.

**Opiates**

Various opiates were commonly used as antidepressants until the mid-1950s, when they fell out of favor with medical orthodoxy due to their addictive nature, tolerance buildup issues and their side-effect profile. Today the use of opioids in treating depression is a large taboo in the medical field due to associations with drug abuse; hence, research has proceeded at a very slow rate. A small clinical trial conducted at Harvard Medical School in 1995, demonstrated that a majority of treatment-refractory, unipolar, non-psychotic, major depression patients could be successfully treated with an opioid medication called Buprenorphine, which is a partial μ-agonist and potent κ antagonist. The exact mechanism of its action in depression is not known, as κ (kappa) antagonists are antidepressants in their own right.

In 2006, The Journal of European Neuropsychopharmacology published a follow-up study to the 1995 Harvard experiment, with results consistent with the original Harvard findings. Eleven severely depressed patients, refractory to all the conventional depression treatments, were given small doses of buprenorphine. Most of these patients found the buprenorphine to be of significant benefit. The researchers theorized that "Possibly, the response to opiates describes a special subtype of depressive disorders e.g. corresponding to a dysregulation of the endogenous opioid system and not of the monaminergic system."

Another scientific paper was published in the American Journal of Psychiatry in 1999, detailing how researchers found Oxycodeone/Oxymorphone to help 5 out of 6 "incurable" refractory severe depression patients.

Buprenorphine was found to be effective as a treatment for depression in patients that had responded to neither antidepressants nor electroconvulsive therapy.

**Other treatments**

- Gamma-Hydroxybutyric acid (GHB) has been used by some as an antidepressant. Claude Rifat, a French biologist, conducted some early research into GHB’s antidepressant potential. Rifat noted that GHB did not cause the emotional blunting effects caused by conventional antidepressants, but instead intensified pleasurable and rewarding feelings in the user while powerfully suppressing depression. However, GHB has now been outlawed, except for use as a prescription treatment for narcolepsy.
NMDA antagonists such as ketamine and dextromethorphan have recently gained some interest in this field as their apparent ability to reverse opioid tolerance, and can give fast-acting dramatic effects. However, their acute psychoactive effects have been a problem.

Memantine, a moderate affinity NMDA antagonist, has been used to avoid tolerance buildup, and has seen use in opioid tolerance reversal. Proglumide is used to induce acute reversal of tolerance prior to this maintenance strategy; it does not work by itself in the long term, due to tolerance to its effects.

Marijuana – The use of marijuana, in moderation, has shown to be of benefit in severely depressed patients. Many people that do not respond well to the use of traditional antidepressants, or who do not like the many unpleasant side effects, prove to do rather well using this plant in moderation.

Treatment using medical devices or equipment

A variety of medical devices are in use or under consideration for treatment of depression including devices which offer electroconvulsive therapy, vagus nerve stimulation, repetitive transcranial magnetic stimulation, and cranial electrotherapy stimulation. Use of such devices in the United States requires approval by the U.S. Food and Drug Administration (FDA) after field trials. In 2010 a FDA advisory panel considered the question of how such field trials should be managed. Factors considered were whether drugs had been effective, how many different drugs had been tried, and what tolerance for suicides should be in field trials.

Electroconvulsive therapy

Electroconvulsive therapy (ECT) is a treatment where seizures are electrically induced in anesthetized patients for therapeutic effect. ECT is most often used as a "last resort" (from the perspective of hospital psychiatrists) for severe major depression which has not responded to trials of antidepressant or, less often, psychotherapy or supportive interventions. It has a quicker effect than antidepressant therapy, and thus may be the treatment of choice in emergencies such as catatonic depression where the patient has ceased oral intake of fluid or nutrients, or where there is severe suicidality. Some evidence suggests it is the most effective treatment for depression in the short-term and one study, without a comparison group or assessment of additional treatments given, suggested that in the minority who remit it may be related to improved self-rated quality of life in both the short-term (which was correlated with the degree of amnesia) and after six months. However, the first systematic documentation of the effectiveness of ECT in community practice in the 65 years of its use found much lower remission rates than in prior research, and most of those relapsed. ECT on its own does not usually have a sustained benefit, as virtually all those who remit end up relapsing within 6 months following a course, even when given a placebo. The relapse rate in the first six months may be reduced by the use of psychiatric medications or further ECT (though the latter is not recommended by some authorities, such as NICE), but remains high. Short-term memory loss, disorientation, headache and other adverse effects are common, as are long-term memory and other neurocognitive deficits, which may persist. The American Psychiatric Association and the
National Institute for Health and Clinical Excellence have concluded that the evidence they had suggested that the procedure, when administered according to their standards and without complications, does not cause brain damage in adults.

**Deep brain stimulation**

Deep brain stimulation (DBS) is a neurosurgical treatment that has been used especially to treat movement disorders such as Parkinson's disease. It requires a neurosurgeon to drill a hole in the skull and insert an electrode into the patient's tissue. Then, a device located in the chest transmits a signal to the implanted electrode through wires located underneath the scalp.

Clinical trials are focused on the use of DBS for epilepsy and depression but the FDA has not approved this use. It requires brain surgery and it is therefore the most invasive form of brain stimulation in the treatment of depression.

**Other conventional methods of treatment**

![St John's wort](image)

**St. John's Wort (Hypericum perforatum)**

St John's wort extract is used extensively in Europe to treat mild and moderate depression. It is a prescription antidepressant in several European countries but is classified as an herbal supplement and sold over the counter in the US. Opinions on its efficacy for major depression differ. A systematic meta-analysis of 37 trials conducted by Cochrane Collaboration indicated statistically significant weak-to-moderate effect as compared to
placebo. The same meta-analysis found that St John’s wort efficacy for major depression is not different from prescription antidepressants. NCCAM and other NIH-affiliated organizations hold that St John’s wort has minimal or no effects beyond placebo in the treatment of major depression, based primarily on one study with negative outcome conducted by NCCAM.

**SAMe**

S-Adenosyl methionine (SAMe) is available as a prescription antidepressant in Europe and an over-the-counter dietary supplement in the US. Fairly strong evidence from 16 clinical trials suggests it to be more effective than placebo and as effective as standard antidepressant medication for the treatment of major depression.

**Repetitive transcranial magnetic stimulation**

Repetitive transcranial magnetic stimulation (rTMS) use in treatment-resistant depression is supported by multiple controlled studies, and it has been approved for this indication in Europe, Canada and Australia, and now in the US. A 2008 meta-analysis based on 32 trials found a robust effect of this method on depression, and it appeared similarly effective for both uncomplicated depression and depression that is resistant to medication. However, it was inferior to ECT in a side-by-side randomized trial.

**Vagus nerve stimulation**

Vagus nerve stimulation (VNS) uses an implanted electrode and generator to deliver electrical pulses to the vagus nerve, one of the primary nerves emanating from the brain. It is an approved therapy for treatment-resistant depression and is sometimes used as an adjunct to existing antidepressant treatment. The support for this method comes mainly from open-label trials, which indicate that several months may be required to see a benefit. The only large double-blind trial conducted lasted only 10 weeks and yielded inconclusive results; VNS failed to show superiority over a sham treatment on the primary efficacy outcome, but the results were more favorable for the secondary outcome.

**Alternative treatments**

**Bright light therapy**
Bright light therapy is sometimes used to treat depression, especially in its seasonal form.

A meta-analysis of bright light therapy commissioned by the American Psychiatric Association found it to be more effective than placebo—usually dim light—for both seasonal affective disorder and for nonseasonal depression, with effect sizes similar to those for conventional antidepressants. For non-seasonal depression, adding light therapy to the standard antidepressant treatment was not effective. A meta-analysis of light therapy for non-seasonal depression conducted by Cochrane Collaboration, studied a different set of trials, where light was used mostly as an addition to medication or sleep deprivation. A moderate statistically significant effect of light therapy was found; however, it disappeared if a different statistical technique was used. Both analyses noted poor quality of most studies and their small size, and urged caution in the interpretation of their results. The short 1–2 weeks duration of most trials makes it unclear whether the effect of light therapy could be sustained in the longer term.

**Acupuncture**

A 2004 Cochrane Review concluded that based on the low quality of the evidence base there is "insufficient evidence to determine whether acupuncture is effective in the management of depression." Clinical trials have shown the effect of acupuncture to be comparable with amitriptyline; in addition, specifically electroacupuncture has been found to be more effective in depressive patients with decreased excretion of 3-methyl-4-hydroxy-phenylglycol (the principal metabolite of the central neurotransmitter norepinephrine), while amitriptyline is more effective for those with inhibition in the dexamethasone suppression test. Acupuncture has also been proven to prompt the body to produce greater levels of endorphins.

**Exercise**

"A 2001 study by the Duke University in North Carolina found that exercise is a more effective treatment for depression than antidepressants, with fewer relapses and a higher recovery rate." An earlier Duke study likewise found patients who completed 30 minutes of
brisk exercise at least three times a week had a significantly lower incidence of relapse; "Only 8 percent of patients in the exercise group had their depression return, while 38 percent of the drug-only group and 31 percent of the exercise-plus-drug group relapsed."

Vigorous exercise has significant physiological effects which help to reduce stress and counter depression. Also, by improving fitness and self-esteem, exercise may enable the sufferer to cope better with demanding events and situations and so reduce the likelihood of depressing failure.

Exercise in natural surroundings such as the countryside or parks is especially recommended because contact with nature and green spaces has a positive effect upon mental health. Gardening is an ideal activity of this sort, providing mental, practical and social benefits. The benefits of such exercise in improving mood and self-esteem are experienced primarily in the first five minutes and are strongest in young people.

**Deep brain stimulation**

The support for the use of deep brain stimulation in treatment-resistant depression comes from a handful of case studies, and this treatment is still in a very early investigational stage. A March 2010 systematic review found that "about half the patients did show dramatic improvement" and that adverse events were "generally trivial" given the younger psychiatric patient population than with movements disorders.

**Cold Water (Shower, Bath) Therapy**

Taking cold showers according to a study led by Nikolai Shevchuk may be an effective way to help treat depression. Shevchuk believes the biological explanation as to why cold showers help with depression involves the stimulation of locus ceruleus or blue spot which is the brain’s primary source of norepinephrine. Also affected are beta-endorphin levels.

**Tryptophan**

Although tryptophan and 5-hydroxytryptophan may be more effective than placebo in alleviating depression according to the Cochrane Collaboration meta-analysis, only 2 out of 108 trials were of sufficient quality to be included in this analysis. The reviewers concluded that they were unable to recommend the drugs for use in major depression.

Tryptophan is the precursor of the neurotransmitter serotonin. It has shown some promise as an antidepressant alone and as an augmenter of antidepressant drugs. Foods rich in tryptophan include chickpeas, milk products, eggs, pork, beef, chicken, fish, oats, dates, mangoes, seeds, nuts and spirulina.

**Low fructose diet**

Fructose malabsorption is poor absorption of fructose and fructans in the intestines. Subjects with this condition show a significantly higher score in the Beck Depression
Inventory than normal fructose absorbers. Some minerals and amino acids (among others, tryptophan) are also poorly absorbed. Because of the inadequate supply of precursor molecules, some hormones and neurotransmitters (among others, serotonin) may not be synthesized in sufficient quantities. Treatment is a diet that is low in fructose, fructans and sorbitol. Depression scores were reduced by 65.2% after four weeks on this diet.

**Omega-3 fatty acids**

Omega-3 fatty acids have been studied in clinical trials for major depression primarily as an adjunctive to antidepressant therapy. A systematic review of 18 such trials found little evidence of a beneficial effect.

**DHEA**

Dehydroepiandrosterone (DHEA), a metabolic precursor for several hormones including estrogen and testosterone, has been promoted as a remedy for many ailments. Sold in the 1970s and 1980s as a weight-loss aid, it was subsequently banned for over-the-counter sale, but then unbanned, and is currently available as a supplement in the US. It has been shown to be more effective than placebo in two small double-blind trials: in one as an adjunct to antidepressant treatment, and as monotherapy in another. However, a larger placebo-controlled randomized clinical trial reported in the New England Journal of Medicine in 2006 found that DHEA supplementation in elderly men and women had no beneficial effects on quality of life.

**Chromium picolinate**

Chromium picolinate was found to be equivalent to placebo for atypical depression overall but possibly efficacious in the sub-group of patients with severe carbohydrate craving.

**Zinc**

Zinc supplementation was found in a small study to augment the effect of antidepressants.

Serum levels of zinc are found to be low in depressed patients and supplementation with zinc has been demonstrated to be of benefit. Most of the zinc found in the human body is located in the brain, mainly in the hippocampus and cerebral cortex area. Lack of zinc influences zinc homeostasis and leads to a change in learning, behavior, mood swings, mental function and epilepsy. Zinc is found in beans, meat, nuts, oysters, whole grains and seeds.

**Lithium**

In the late 1800s there was a vogue for consumption of lithia water which contained a significant quantity of lithium. Some claimed that this cured depression, but its effectiveness is not clear.
In May 2009, the BBC reported that a Japanese study of lithium in drinking water in the Japan prefecture of Oita, which has a population of more than one million, revealed that the suicide rate was significantly lower in those areas with the highest levels of lithium.

Lithium is also used as the standard drug to treat different mood disorders including depression. See Lithium pharmacology.

**Magnesium**

Magnesium deficiency is common and may cause depression. Supplementation or changes in diet may therefore be helpful. Foodstuffs rich in magnesium include whole grains, beans and seeds, halibut and spinach.

**Cranial electrotherapy stimulation**

Cranial electrotherapy stimulation (CES, electrosleep) devices currently on the market have been granted marketing authorization by the FDA based on the legacy waiver, that is because a sufficiently similar device had been marketed before 1976, when the new regulations requiring controlled testing were introduced. The FDA considers them to be the class III devices—"devices for which insufficient information exists to ... provide reasonable assurance of safety and effectiveness" The effects of CES on depression were inconclusive or negative in multiple double-blind studies of psychiatric patients. In one of them, four out of six clinically depressed patients dropped out of the study because of the massive worsening of depressive symptoms, with two of them becoming actively suicidal. One of the authors of the latter study cautioned that CES "should not be used as a treatment of choice" for the patients with the primary diagnosis of depression, "and should be used with caution if this diagnosis is suspected." Nevertheless, the CES practitioners continue to employ it as a treatment of choice for depression.

**Eleuthero**

Eleuthero or Siberian Ginseng. The plant is an adaptogen or tonic and has been shown to have significant antidepressant effects in rats.

**Saffron**

Saffron, the flowers of Crocus sativus have been shown to have antidepressant properties. Two of the active ingredients are crocin and safranal.

**Inositol (Vitamin B8)**

Inositol or Vitamin B8 has been tested as a treatment for depression in four RCTs, but there is insufficient evidence of therapeutic benefit.
Kanna

Kanna (Sceletium tortuosum) is a succulent herb commonly found in South Africa. In doses as low as 50 mg, users have reported improvements in mood, decreased anxiety, relaxation and a sense of well-being. It contains about 1-1.5% alkaloids and those which are believed to be psychoactive include mesembrine, mesembrenone, mesembrenol and tortuosamine.

There is about 0.3% mesembrine in the leaves and 0.86% in the stems of the plant. This has been shown to be a potent serotonin reuptake inhibitor.

**Flower remedies**

Bach flower remedies and Australian bush flower essences are prepared from various flowers. Current clinical evidence does not support any hypothesized action or efficacy beyond placebo effects.

**Meditation**

Mindfulness meditation has been shown to be of medical benefit in a number of ways, including lowering blood pressure and stress levels. The most helpful and gentle form of meditation for a clinically depressed person may be the repetition—silently or aloud—of a mantra.

**Neurofeedback**

Neurofeedback is a form of biofeedback therapy in which brain activity is monitored using an EEG. The output is presented to the patient who is then able to see any variation in the brain waves associated with depression and may then develop some ability to reduce them, so improving their mood. The resulting direct control of mental state is thought to be similar to that achieved by the mental exercises of yoga.
Reiki

Reiki is a form of energy medicine which originated in 1922 by Mikao Usui. In the UK, it has been recommended as a complementary medicine for pain management, anxiety and depression by NHS Trusts and Princess of Wales's Foundation of Integrative Medicine, but there is no evidence of its efficacy in the treatment of any disorder.

Religion

Numerous studies and clinical trials have looked at the relationship between religion and depression. These have looked at the matter from Buddhist, Christian and Muslim perspectives. These indicate that religious faith helps to prevent the onset of depression and assists recovery if depression should still occur.

Sleep

Depression is commonly associated with poor sleep (difficulty going to sleep, early waking, and general lassitude during the day). The interaction of the two results in each condition worsening. Good sleep hygiene is therefore important to help break this vicious circle. This would include measures such as regular bed times, avoidance of stimulants such as caffeine and management of disturbances such as sleep apnea. Ironically, sleep deprivation (such as wake therapy) is a temporary treatment for depression.

Chi Kung

The traditional Chinese exercise of Chi Kung and related martial arts such as Tai Chi can help to prevent and relieve depression.

Music Therapy

Studies have demonstrated that music can bring about different moods, conditioned by different emotional states. Music has the property of facilitating self-expression and in this way giving vent to disturbing emotional upheavals and dissipating them. Music has been proven that it can reach the sub-cortical centers of the brain and thereby helps to integrate the personality that is being disrupted by unhealthy emotions. Researchers have shown that music therapy is effective in patients. It has been shown that clinically depressed patients who were made to listen to soft, dissonant-free, melodic music gradually became more emotional and rhythmical.

Green/White Tea

A frequent consumption of green tea was associated with a lower prevalence of depressive symptoms in a Japanese study. Researchers conducted a cross-sectional study in 1,058 community-dwelling elderly Japanese individuals 70 years of age. The prevalence of mild and severe depressive symptoms was 34.1 percent and 20.2 percent, respectively. After
adjustment for confounding factors, the odds ratios for mild and severe depressive symptoms when higher green tea consumption was compared with green tea consumption of one cup/day were: two to three cups green tea/day and four cups green tea/day. Similar relations were also observed in the case of severe depressive symptoms.

Biology of depression

Scientific studies have found that numerous brain areas show altered activity in depressed patients. It has not been possible to determine a single cause of depression.

Monoamine hypothesis

Illustration of the major elements in a prototypical synapse. Synapses are gaps between nerve cells. These cells convert their electrical impulses into bursts of chemical relayers, called neurotransmitters, which travel across the synapses to receptors on adjacent cells, triggering electrical impulses to travel down the latter cells.

Most antidepressants increase synaptic levels of the monoamine neurotransmitter serotonin. They may also enhance the levels of two other neurotransmitters, norepinephrine and dopamine. This observation gave rise to the monoamine hypothesis of depression. In its contemporary formulation, the monoamine hypothesis postulates that the deficit of certain neurotransmitters is responsible for the corresponding features of depression: "Norepinephrine may be related to alertness and energy as well as anxiety, attention, and interest in life; [lack of] serotonin to anxiety, obsessions, and compulsions; and dopamine to attention, motivation, pleasure, and reward, as well as interest in life." The proponents of this hypothesis recommend choosing the antidepressant with the mechanism of action impacting the most prominent symptoms. The anxious and irritable
patients should be treated with SSRIs or norepinephrine reuptake inhibitors, and the ones with the loss of energy and enjoyment of life—with norepinephrine and dopamine enhancing drugs.

Monoamine receptors affect phospholipase C and adenylyl cyclase inside of the cell. Green arrows mean stimulation and red arrows inhibition. Serotonin receptors are blue, norepinephrine orange, and dopamine yellow. Phospholipase C and adenylyl cyclase start a signaling cascade which turn on or off genes in the cell. The 5HT-3 receptor is associated with gastrointestinal adverse effects and has no relationship to the other monoamine receptors.

Consistent with the monoamine hypothesis, a longitudinal study uncovered a moderating effect of the serotonin transporter (5-HTT) gene on stressful life events in predicting depression. Specifically, depression seems especially likely to follow stressful life events, but even more so for people with one or two short alleles of the 5-HTT gene. Serotonin may help to regulate other neurotransmitter systems, and decreased serotonin activity may "permit" these systems to act in unusual and erratic ways. Facets of depression may be emergent properties of this dysregulation.

In the past two decades, research has uncovered multiple limitations of the monoamine hypothesis, and its inadequacy has been criticized within the psychiatric community. Intensive investigation has failed to find convincing evidence of a primary dysfunction of a specific monoamine system in patients with major depressive disorders. The antidepressants that do not act through the monoamine system, such as tianeptine and opipramol, have been known for a long time. Experiments with pharmacological agents that cause depletion of monoamines have shown that this depletion does not cause depression in healthy people nor does it worsen the symptoms in depressed patients.
Already limited, the monoamine hypothesis has been further oversimplified when presented to the general public.

An offshoot of the monoamine hypothesis suggests that monoamine oxidase A (MAO-A), an enzyme which metabolizes monoamines, may be overly active in depressed people. This would, in turn, cause the lowered levels of monoamines. This hypothesis received support from a PET study, which found significantly elevated activity of MAO-A in the brain of some depressed people. In genetic studies, the alterations of MAO-A-related genes have not been consistently associated with depression. Contrary to the assumptions of the monoamine hypothesis, lowered but not heightened activity of MAO-A was associated with the depressive symptoms in youth. This association was observed only in maltreated youth, indicating that both biological (MAO genes) and psychological (maltreatment) factors are important in the development of depressive disorders. In addition, some evidence indicates that problems in information processing within neural networks, rather than changes in chemical balance, might underlie depression.

**Circadian rhythm**

Depression may be related to the same brain mechanisms that control the cycles of sleep and wakefulness. Depression may be related to abnormalities in the circadian rhythm, or biological clock. For example, rapid eye movement (REM) sleep—the stage in which dreaming occurs—may be quick to arrive and intense in depressed people. REM sleep depends on decreased serotonin levels in the brain stem, and is impaired by compounds, such as antidepressants, that increase serotonergic tone in brain stem structures. Overall, the serotonergic system is least active during sleep and most active during wakefulness. Prolonged wakefulness due to sleep deprivation activates serotonergic neurons, leading to processes similar to the therapeutic effect of antidepressants, such as the selective serotonin reuptake inhibitors (SSRIs). Depressed individuals can exhibit a significant lift in mood after a night of sleep deprivation. SSRIs may directly depend on the increase of central serotonergic
neurotransmission for their therapeutic effect, the same system that impacts cycles of sleep and wakefulness.

Research on the effects of light therapy on seasonal affective disorder suggests that light deprivation is related to decreased activity in the serotonergic system and to abnormalities in the sleep cycle, particularly insomnia. Exposure to light also targets the serotonergic system, providing more support for the important role this system may play in depression. Sleep deprivation and light therapy both target the same brain neurotransmitter system and brain areas as antidepressant drugs, and are now used clinically to treat depression. Light therapy, sleep deprivation and sleep time displacement (sleep phase advance therapy) are being used in combination quickly to interrupt a deep depression in hospitalized patients.

**Abnormalities by brain region**

Research on the brains of depressed patients usually shows disturbed pattern of interaction between multiple parts of the brain. Here are the areas that are most strongly affected:

**Raphe nuclei**

The raphe nuclei are a group of small nuclei in the upper brain stem, located directly at the midline of the brain. They are the sole source of serotonin in the brain. Despite their small size, they project very widely, and are involved in a very diverse set of functions. Most antidepressants are serotonergic. Serotonin system dysfunction cannot be the sole cause of depression, though: antidepressants usually bring serotonin levels up to normal very quickly, but it often takes at least two to four weeks before mood improves significantly. The 5-HTT gene regulates a chemical called serotonin. Serotonin works as the neurotransmitter and helps with the modulation of things such as anxiety, anger, appetite, sexuality, sleep, mood, and several other things. People with depression often have impaired 5-HTT genes. There are two forms of the 5-HTT gene and everyone has two 5-HTT genes. (Levinson) There is a long form of 5-HTT and a short form of 5-HTT. Research shows that people with both 5-HTT genes being the long form are less likely to become depressed while people with one short and one long or two short forms are more likely to develop depression. Research is still being conducted to find more information. The functions of serotonin are difficult to describe in a simple way. In some circumstances serotonin seems to act as a signal of "repletion" or "satisfaction". Thus, satiation after eating, and orgasm following sex, both produce release of serotonin. In animals that have hierarchical social structures, dominant individuals show higher levels of serotonin metabolites than lower-status individuals. In the brain, serotonin exerts a suppressive effect on both the reward system and punishment system, and therefore is likely to reduce the intensity of motivation whether aversive or appetitive. (One of the most common but least-discussed side effects of antidepressants is to reduce sex drive).

**Suprachiasmatic nucleus (SCN)**
The suprachiasmatic nucleus (SCN) is the control center for the body's "biological clock". It contains neurons whose activity waxes and wanes throughout the day. The output from the SCN controls the sleep/wake cycle as well as a number of other biological rhythms, such as fluctuations in body temperature. Disturbances of these cycles are a consistent symptom of depression, especially of the melancholic type. The "classic" pattern is for depressed people to have great difficulty falling asleep at night, and then to wake bolt upright at around 3 AM. The waking is usually preceded by a rise in body temperature, which in non-depressed people does not usually occur until several hours later. It is a common observation that antidepressants produce a return to normal sleep patterns before they produce an improvement in mood: if good sleep does not return, it is a strong sign that the treatment is not going to be effective. Conversely, disruptions to sleep are often the first indication of impending relapse.

There is a powerful interaction between the Raphe nuclei and the SCN. On one hand, the Raphe nuclei send a strong serotonergic projection to the SCN. In animal studies, this input has been shown to modulate the ability of light to reset the timing of the biological clock: the more serotonin, the stronger the effects of light. On the other hand, the biological clock exerts a strong influence on the Raphe nuclei: serotonin levels drop during sleep, and fall almost to nothing during REM (dreaming) sleep. It is worth noting that one of the characteristics of sleep in depressed people is that REM tends to appear very soon after sleep onset, whereas in non-depressed people it does not usually dominate sleep until the last hours, in the early morning. Antidepressants are powerful suppressors of REM.

**Ventral tegmental area (VTA)**

The ventral tegmentum (or ventral tegmental area) is a small area in the basal midbrain which is a critical part of the brain's reward system. It sends projections to the nucleus accumbens that use the neurotransmitter dopamine. Addictive drugs universally increase the effects of dopamine in this system, whereas drugs that oppose dopamine produce anhedonia of the sort seen in depressed people. Dopamine-enhancers such as cocaine often relieve the lack-of-pleasure in depression, but the effects only last as long as a drug is present in the body: that is, they temporarily alleviate one of the main symptoms, but do not help to cure the disease.

**Nucleus accumbens (NAc)**

Long-term exposure to various unavoidable stress factors decreases dopamine release in the NAc shell, as it was shown in the forced swimming test, an animal model of depression.

**Anterior cingulate cortex (ACC)**

The anterior cingulate cortex is activated by negative experiences of many types, and consistently shows higher levels of activity in depressed people than in non-depressed people. The functions of the ACC are controversial, but one proposal is that it mediates the conscious experience of suffering. Several decades ago, trials were made of ablating parts of the ACC in an attempt to relieve intolerable pain in patients who were terminally ill.
These patients reported that after the surgery, they could still perceive the physical sensations of pain, but they no longer found them distressing. (The effects of heroin and morphine are sometimes described in the same way.) Very recently, clinical experiments were made in using deep brain stimulation to temporarily inactivate the ACC in severely depressed patients. This was not effective in all cases, but in some patients very striking results were achieved, with a perceptible lifting of mood immediately apparent to the patient as soon as the stimulus was applied.

**Subgenual cingulate**

Recent studies have shown that Brodmann area 25, also known as Subgenual cingulate is metabolically overactive in treatment-resistant depression. This region is extremely rich in serotonin transporters and is considered as a governor for a vast network involving areas like hypothalamus and brain stem, which influences changes in appetite and sleep; the amygdala and insula, which affect the mood and anxiety; the hippocampus, which plays an important role in memory formation; and some parts of the frontal cortex responsible for self-esteem. Thus disturbances in this area or a smaller than normal size of this area contributes to depression. Deep Brain Stimulations of this area have been successful in reducing its elevated activity and thus curing depression in patients that could not be cured by anti-depressants.

**Altered neuroplasticity**

Recent studies have called attention to the role of altered neuroplasticity in depression. Pittenger and Duman have reviewed extensive research, demonstrating a convergence of three phenomena:

- Chronic stress reduces synaptic and dendritic plasticity
- Depressed subjects show evidence of impaired neuroplasticity (eg. shortening and reduced complexity of dendritic trees)
- Anti-depressant medications enhance neuroplasticity at both a molecular and dendritic level.

They conclude that disrupted neuroplasticity is an underlying feature of depression, and is reversed by antidepressants.

**Hypothalamic-Pituitary-Adrenal (HPA) axis**

The Hypothalamic-pituitary-adrenal axis is a chain of endocrine structures that are activated during the body's response to stressors of various sorts. It often shows increased activation in depressed people, and drugs that reduce its activity are sometimes effective in reducing symptoms. The HPA influences many parts of the brain, including the Raphe nuclei.

**Genetic factors**
In 2003 Science published an influential study of Avshalom Caspi et al. who found that a gene-environment interaction (GxE) may explain why life stress is a predictor for depressive episodes in some individuals, but not in others, depending on an allelic variation of the serotonin-transporter-linked promoter region (5-HTTLPR). Soon after, the results were replicated by Kenneth Kendler’s group, raising hopes in the psychiatric genetics community. By 2007 there were 11 replications, 3 partial replication and 3 non-replications of this proposed GxE. However, two of the largest studies were negative. Two 2009 meta-analyses were also negative; one included 14 studies, the other just five, owing to different study selection criteria. A 2010 review of studies in this area found 17 replications, 8 partial replications (interaction only in females or only with one of several types of adversity), and 9 non-replications (no interaction or an interaction in the opposite direction). It also found a systematic relationship between the method used to assess environmental adversity and the results of the studies; all studies using objective indicators or structured interviews to assess stress replicated the gene-environment interaction fully or partially, whereas all non-replications relied on brief self-report measures of adversity. This review also found that both 2009 meta-analyses were significantly biased toward negative studies.

Other hypothesized genomic influences are BDNF polymorphisms, but the replications studies have been mixed and insufficient as of 2005 for a meta-analysis. Studies also indicate an association of BDNF to suicidal behavior. However, findings from the gene-environment interactions studies suggest that the current BDNF models of depression are too simplistic. A 2008 study found interactions (biological epistasis) in the signaling pathways of the BDNF and the serotonin transporter; the BDNF Val66Met allele, which was predicted to have reduced responsivity to serotonin, was found to exercise protective effects in individuals with the short 5-HTTLPR allele that is otherwise believed to predispose individuals to depressive episodes after stressful events. Thus, the BDNF-mediated signalling involved in neuroplastic responses to stress and antidepressants is influenced by other genetic and environmental modifiers.

**Fructose malabsorption**

A series of 50 subjects examined in an Australian study showed increased depressive scores on Beck Depression Inventory associated with reduced plasma tryptophan, due to Fructose malabsorption

**Evolutionary approaches to depression**

Major depression is the leading cause of disability worldwide, and in 2000 was the fourth leading contributor to the global burden of disease (measured in DALYs); it is also an important risk factor for suicide. It is understandable then, that clinical depression is thought to be a pathology — a major dysfunction of the brain. In most cases, rates of organ dysfunction increase with age, with low rates in adolescents and young adults, and the highest rates in the elderly. These patterns are consistent with evolutionary theories of
aging which posit that selection against dysfunctional traits decreases with age (because there is a decreasing probability of surviving to later ages).

In contrast to these patterns, prevalence of clinical depression is high in all age categories, including otherwise healthy adolescents and young adults. In one study of the US population, for example, the 12 month prevalence for a major depression episode was highest in the youngest age category (15–24 year olds). The high prevalence of depression is also an outlier when compared to the prevalence of major mental retardation, autism, and schizophrenia, all with prevalence rates about one tenth that of depression, or less.

The common occurrence and persistence of a trait like clinical depression with such negative effects early in life is difficult to explain. (Rates of infectious disease are high in young people, of course, but clinical depression is not thought to be caused by an infection.) Evolutionary psychology and its application in evolutionary medicine suggest how behaviour and mental states, including seemingly harmful states such as depression, may be past adaptations to recurring reproductive problems faced by our ancestors, actually having improved (however disadvantageous in the modern world), the fitness of either the individual or their relatives. It has been argued, for example, that Abraham Lincoln's lifelong depression was a source of insight and strength. Some even suggest that "we aren't designed to have happiness as our natural default" and so a state of depression is the evolutionary norm.

The following hypotheses attempt to identify a benefit of depression that outweighs its obvious costs. All take as their starting point the fact that one of the most potent, well-established causes of major depression is a severe negative life event.

**Psychic pain hypothesis**

One reason depression is thought to be a pathology is that it causes so much psychic pain and distress. However, physical pain is also very distressful, yet it has an evolved function: to inform the organism that it is suffering damage, to motivate it to withdraw from the source of damage, and to learn to avoid such damage-causing circumstances in the future.

According to the psychic pain hypothesis, depression is analogous to physical pain in that it informs the sufferer that current circumstances, such as the loss of a friend, are imposing a threat to biological fitness, it motivates the sufferer to cease activities that led to the costly situation, if possible, and it causes him or her to learn to avoid similar circumstances in the future. Proponents of this view tend to focus on low mood, and regard clinical depression as a dysfunctional extreme of low mood.

**Rank theory**

Rank theory is the hypothesis that, if an individual is involved in a lengthy fight for dominance in a social group and is clearly losing, then depression causes the individual to back down and accept the submissive role. In doing so, the individual is protected from unnecessary harm. In this way, depression helps maintain a social hierarchy. This theory is
a special case of a more general theory derived from the psychic pain hypothesis: that the
cognitive response that produces modern-day depression evolved as a mechanism that
allows people to assess whether they are in pursuit of an unreachable goal, and if they are,
to motivate them to desist.

**Honest signaling theory**

Another reason depression is thought to be a pathology is that key symptoms, such as loss
of interest in virtually all activities, are extremely costly to the sufferer. Biologists and
economists have proposed, however, that signals with inherent costs can credibly signal
information when there are conflicts of interest. In the wake of a serious negative life event,
such as those that have been implicated in depression (e.g., death, divorce), "cheap" signals
of need, such as crying, might not be believed when social partners have conflicts of
interest. The symptoms of major depression, such as loss of interest in virtually all
activities and suicidality, are inherently costly, but, as costly signaling theory requires, the
costs differ for individuals in different states. For individuals who are not genuinely in
need, the fitness cost of major depression is very high because it threatens the flow of
fitness benefits. For individuals who are in genuine need, however, the fitness cost of major
depression is low, because the individual is not generating many fitness benefits. Thus, only
an individual in genuine need can afford to suffer major depression. Major depression
therefore serves as an honest, or credible, signal of need.

For example, individuals suffering a severe loss such as the death of a spouse are often in
need of help and assistance from others. Such individuals who have few conflicts with their
social partners are predicted to experience grief—a means, in part, to signal need to others.
Such individuals who have many conflicts with their social partners, in contrast, are
predicted to experience depression—a means, in part, to credibly signal need to others
who might be skeptical that the need is genuine. (A theologian might say that depression is
like the difference between attrition and contrition. If it doesn't hurt it doesn't work). Put in
simple language, depression may function to enforce sincerity.

**Social navigation or niche change theory**

The social navigation, bargaining, or niche change hypothesis suggests that depression,
operationally defined as a combination of prolonged anhedonia and psychomotor
retardation or agitation, provides a focused sober perspective on socially imposed
constraints hindering a person's pursuit of major fitness enhancing projects. Simultaneously, publicly displayed symptoms, which reduce the depressive's ability to
conduct basic life activities, serve as a social signal of need; the signal's costliness for the
depressive certifies its honesty. Finally, for social partners who find it uneconomical to
respond helpfully to an honest signal of need, the same depressive symptoms also have the
potential to extort relevant concessions and compromises. Depression's extortionary
power comes from the fact that it retards the flow of just those goods and services such
partners have come to expect from the depressive under status quo socioeconomic
arrangements.
Thus depression may be a social adaptation especially useful in motivating a variety of social partners, all at once, to help the depressive initiate major fitness-enhancing changes in their socioeconomic life. There are diverse circumstances under which this may become necessary in human social life, ranging from loss of rank or a key social ally which makes the current social niche uneconomic to having a set of creative new ideas about how to make a livelihood which begs for a new niche. The social navigation hypothesis emphasizes that an individual can become tightly ensnared in an overly restrictive matrix of social exchange contracts, and that this situation sometimes necessitates a radical contractual upheaval that is beyond conventional methods of negotiation. Regarding the treatment of depression, this hypothesis calls into question any assumptions by the clinician that the typical cause of depression is related to maladaptive perverted thinking processes or other purely endogenous sources. The social navigation hypothesis calls instead for analysis of the depressive’s talents and dreams, identification of relevant social constraints (especially those with a relatively diffuse non-point source within the social network of the depressive), and practical social problem-solving therapy designed to relax those constraints enough to allow the depressive to move forward with their life under an improved set of social contracts. This theory has been the subject of criticism.

**Bargaining theory**

Depression is not only costly to the sufferer, it also imposes a significant burden on family, friends, and society at large—yet another reason it is thought to be pathological. Yet if sufferers of depression have real but unmet needs, they might have to provide an incentive to others to address those needs.

The bargaining theory of depression is similar to the honest signaling, niche change, and social navigation theories of depression described above. It draws on theories of labor strikes developed by economists to basically add one additional element to honest signaling theory: The fitness of social partners is generally correlated. When a wife suffers depression and reduces her investment in offspring, for example, the husband’s fitness is also put at risk. Thus, not only do the symptoms of major depression serve as costly and therefore honest signals of need, they also compel reluctant social partners to respond to that need in order to prevent their own fitness from being reduced.

**Prevention of infection**

It has been hypothesized that depression is an evolutionary adaptation because it helps prevent infection in both the affected individual and his/her kin.

First, the associated symptoms of depression, such as inactivity and lethargy, encourage the affected individual to rest. Energy conserved through such methods is highly crucial, as immune activation against infections is relatively costly; there must be, for instance, a 10% increase in metabolic activity for even a 1°C change in body temperature. Therefore, depression allows one to conserve and allocate energy to the immune system more efficiently.
Depression further prevents infection by discouraging social interactions and activities that may result in exchange of infections. For example, the loss of interest discourages one from engaging in sexual activity, which, in turn, prevents the exchange of sexually transmitted diseases. Similarly, depressed mothers may interact less with their children, reducing the probability of the mother infecting her kin. Lastly, the lack of appetite associated with depression may also reduce exposure to food-borne parasites.

**Analytical rumination hypothesis**

This hypothesis suggests that depression is an adaptation that causes the affected individual to concentrate his or her attention and focus on a complex problem in order to analyze and solve it.

One way depression increases the individual’s focus on a problem is by inducing rumination. Depression activates the left ventrolateral prefrontal cortex, which increases attention control and maintains problem-related information in an “active, accessible state” referred to as “working memory,” or WM. As a result, depressed individuals have been shown to ruminate, reflecting on the reasons for their current problems. Feelings of regret associated with depression also cause individuals to reflect and analyze past events in order to determine why they happened and how they could have been prevented.

Another way depression increases an individual’s ability to concentrate on a problem is by reducing distraction from the problem. For example, anhedonia, which is often associated with depression, decreases an individual’s desire to participate in activities that provide short-term rewards, and instead, allows the individual to concentrate on long-term goals. In addition, “psychomotor changes,” such as solitariness, decreased appetite, and insomnia also reduce distractions. For instance, insomnia enables conscious analysis of the problem to be maintained by preventing sleep from disrupting such processes. Likewise, solitariness, lack of physical activity, and lack of appetite all eliminate sources of distraction, such as social interactions, navigation through the environment, and “oral activity,” which disrupt stimuli from being processed.

**Social risk hypothesis**

This hypothesis explains the evolutionary origin of depression in the ancestral context in which depression was an adaptation that enabled an individual to maintain social ties critical for survival and reproduction. During the Pleistocene period, for instance, such social ties were vital in food foraging and protection from predators.

Depression, resulting from one’s loss of “Social Attention Holding Power,” or SAHP, may have signalled the individual’s submissiveness to the more dominant males of the social group through associated symptoms, such as low confidence and anxiety. This, in turn, reduced tension among group members, and enabled bonds to be maintained. Secondly, depression may also have served to signal other members of an individual’s need for aid and desire to reform lost bonds.
Behavioral shutdown model

If an organism faces more risk or expenditure than reward from activities, the best evolutionary strategy may be to withdraw from them. The behavioral shutdown model proposes that emotional pain, like physical pain, serves a useful adaptive purpose. Negative emotions like disappointment, sadness, grief, fear, anxiety, anger, and guilt are described as "evolved strategies that allow for the identification and avoidance of specific problems, especially in the social domain." Depression is characteristically associated with anhedonia and lack of energy, and those experiencing it are risk-aversive and perceive more negative and pessimistic outcomes because they are focused on preventing further loss. Although the model views depression as an adaptive response, it does not suggest that it is beneficial by the standards of current society; but it does suggest that many approaches to depression treat symptoms rather than causes, and underlying social problems need to be addressed.

Possibilities of depression as a dysregulated adaptation

Depression, especially in the modern context, may not necessarily be adaptive. The ability to feel pain, have diarrhea, and experience depression, are adaptive defense mechanisms, but when they are “too easily triggered, too intense, or long lasting,” they can become “dysregulated.” In such a case, defense mechanisms, too, can become diseases, such as “chronic pain or dehydration from diarrhea.” Depression, which may be a similar kind of defense mechanism, may have become dysregulated as well.

Major depressive episode

A major depressive episode is the cluster of symptoms of major depressive disorder. The description has been formalised in psychiatric diagnostic criteria such as the DSM-IV and ICD-10, and is characterized by severe, highly persistent depression, and a loss of interest or pleasure in everyday activities, which is often manifested by lack of appetite, chronic fatigue, and sleep disturbances (somnipathy). The individual may think about suicide, and indeed an increased risk of actual suicide is present.

In addition to the emotional pain endured by those suffering from depression, significant economic costs are associated with depression. In fact, American and Canadian studies have indicated that the costs associated with depression are greater than those associated with hypertension, and equal to those of heart disease, diabetes, and back problems.

Criteria

The criteria below are based on the formal DSM-IV criteria for a Major Depressive Episode. A diagnosis of major depressive episode requires that the patient has—over a two-week period—experienced five or more of the symptoms below, and these must be outside the patient’s normal behaviour. Either depressed mood or decreased interest or pleasure must be one of the five (although both are frequently concomitant).
Mood

- For the better part of nearly every day, the patient reports a depressed mood or appears depressed to others.
- The patient may state that he or she has been feeling sad, depressed, blue, empty, "down in the dumps," hopeless, etc. If the patient is in denial about these feelings, yet appears to be on the verge of tearfulness, manifests a depressed facial expression and disposition, or appears to be overly irritable, these may also indicate the presence of depressed mood. Some people may report physical complaints (i.e., aches, pains, headaches) rather than depressed mood, and physical symptoms without physical cause are sometimes indicators of depression. (See Myalgia and Neuralgia.)

Anhedonia and loss of interest

- For most of nearly every day, interest or pleasure is markedly decreased in nearly all activities (noted by the patient or by others). (See Anhedonia)
- People suffering with depression tend to lose interest in things they once found enjoyable. Activities are no longer enjoyable and there is often a loss of interest in or desire for sex. People who are depressed may say, "I just don't care anymore," or "nothing matters anymore." Friends and family of the depressed person may notice that he/she has withdrawn from friends, or has neglected or quit doing activities that were once a source of enjoyment.

Change in eating, appetite, or weight

- Although not dieting, there is a marked loss or gain of weight (such as 5% in one month) or appetite is markedly decreased or increased nearly every day.
- Changes in appetite take on two manifestations: under- or over-eating.
- In the first instance, some people never feel hungry, can go long periods without wanting to eat, may forget to eat, or if they do eat a small amount of food may be sufficient. A reduction in weight is often associated with a melancholic type of depression.
- In the second instance, some people tend toward an increase in appetite and may gain significant amounts of weight. They may tend to crave certain types of food such as sweets or carbohydrates. People with seasonal affective disorder (SAD) often crave foods high in carbohydrates. Weight gain is often associated with atypical depression.

Sleep

- Nearly every day the patient sleeps excessively, known as hypersomnia, or not enough, known as insomnia.
- Insomnia is the most common type of sleep disturbance for people who are clinically depressed. Waking in the middle of the night and being unable to go back to sleep is known as "middle insomnia"; waking too early as "terminal insomnia",...
and; having difficulty falling asleep at night is "initial" insomnia. Insomnia is often associated with a melancholic type of depression.

- A less frequent sleeping problem is oversleeping (called "hypersomnia"). This may occur in the form of sleeping for prolonged periods at night or increased sleeping during the daytime. Even with excess sleep, a person may still feel tired and sluggish during the day. People with seasonal affective disorder (SAD) may sleep longer during the winter months. Hypersomnia is often associated with an atypical depression.

Motor activity

- Nearly every day others can see that the patient's activity is agitated or slow.
- People suffering from depression may be either quite agitated (psychomotor agitation), or very lethargic (psychomotor retardation) in their mannerisms and behavior. If a person is agitated, he or she may find it difficult to sit still, may pace the room, wring his/her hands, or fidget with clothes or objects. Someone with psychomotor retardation tend to move sluggishly, may move across a room very slowly, avert his/her eyes, sit slumped in a chair and speak slowly, saying little.
- In terms of diagnosis, the agitation or slowing down of one's demeanor must be to the degree that it can be observed by others.

Fatigue

- Nearly every day the person experiences extreme fatigue.
- A decrease in energy and feeling fatigued are very common symptoms for those who are clinically depressed. A person may feel tired without having engaged in any physical activity, and day-to-day tasks become difficult, including getting washed and dressed in the morning. Job tasks or housework become very tiring, and the person finds that his/her work at home, school, or on the job suffers.

Self-worth

- Nearly every day the patient feels worthless or inappropriately guilty. These feelings are not just about being depressed, they may be delusional.
- Depressed people may think of themselves in very negative, unrealistic ways such as manifesting a preoccupation with past "failures", personalisation of trivial events, or believing that minor mistakes prove their inadequacy. They also may have an unrealistic sense of personal responsibility and see things beyond their control as being their fault. Additionally, self-loathing is common in clinical depression, and can lead to a downward spiral when combined with other symptoms.

Concentration

- Noted by the patient or by others, nearly every day the patient is indecisive or has trouble thinking or concentrating.
A person with depression frequently experiences negative and pessimistic thoughts, and reports that his/her ability to think, concentrate, or make decisions becomes impaired. Memory and distraction problems are common. This problem can be notably pronounced, causing significant difficulty in functioning for those involved in intellectually demanding activities.

**Thoughts of death**

- The patient has had repeated thoughts about death (other than the fear of dying), suicide (with or without a plan) or has made a suicide attempt.
- The frequency and intensity of thoughts about suicide can range from believing that friends and family would be better off if one were dead, to frequent thoughts about committing suicide (generally related to wishing to stop the emotional pain), to detailed plans about how the suicide would be carried out. Less severely suicidal people may have regular thoughts of suicide, while those who are more severely suicidal may have made specific plans and decided upon a day and location for the suicide attempt.
- Thoughts of suicide occur mostly when triggered. Thoughts of suicide happen more frequently than normal.

**Diagnostic caveats**

In diagnosing the symptoms a trained therapist must take the following into account:

- These symptoms must cause clinically important distress, or impair work, social or personal functioning, and they should not fulfil the criteria for Mixed Episode.
- The symptoms are not due to the direct physiological effects of a substance (e.g., abuse of a drug or medication) or a general medical condition (e.g., hypothyroidism).
- Other than in the case of severe symptoms (severely impaired functioning, severe preoccupation with worthlessness, ideas of suicide, delusions or hallucinations or psychomotor retardation), the episode should not have begun within two months of the loss of a loved one. (See Bereavement.)

**Treatment**

If left untreated, a typical major depressive episode may last for about six months, while about 20% of these episodes can last two years or more, with 50% of depressive episodes ending spontaneously. However, even after the major depressive episode is over, 20% to 30% of patients have residual symptoms, which can be distressing and associated with disability.

Regarding the treatment of major depressive episodes of severe intensity (multiple symptoms, minimal mood reactivity, severe functional impairment), combined psychotherapy plus antidepressant medications are more effective than psychotherapy alone.
Demographics

Estimates of the numbers of people suffering from major depressive episodes and Major Depressive Disorder (MDD) vary significantly. Between 10% and 25% of women, and between 5% and 12% of men will suffer a major depressive episode.

Fewer people, between 5% and 9% of women and between 2% and 3% of men, will have MDD, or full-blown depression. Depression occurs nearly twice as often in adolescent and adult females as in males, and the peak period of development is between the ages of 25 and 44 years.

Onset of major depressive episodes or MDD often occurs to people in their mid-20s, and less often to those over 65. Prepubescent girls and boys are affected equally. Additionally, socio-economic or environmental factors do not appear to have any bearing on the incidence of a major depressive episode or MDD.

Dysthymia

Dysthymia (pronounced dis-thigh-mee-uh, from Ancient Greek δυσθυμία, "melancholy") is a mood disorder consisting of chronic depression, but with less severity than major depressive disorder. The concept was coined by Dr Robert Spitzer (an editor of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III) as a replacement for the term "depressive personality" in the late 1970s.

According to the DSM's new definition, dysthymia is a type of mild depression. Harvard Health Publications states that "the Greek word dysthymia means 'bad state of mind' or 'ill humor'. As one of the two chief forms of clinical depression, it usually has fewer or less serious symptoms than major depression but lasts longer." Harvard Health Publications also says, "at least three-quarters of patients with dysthymia also have a chronic physical illness or another psychiatric disorder such as one of the anxiety disorders, drug addiction, or alcoholism". The prevalence estimate for dysthymia of "clinical significance" among the adult US population is 1.7 percent (CI: 1.5–1.9) based on the Epidemiologic Catchment Area Program and 1.8 percent (CI: 1.4–2.2) based on the National Comorbidity Survey. Harvard Health Publications says: "The rate of depression in the families of people with dysthymia is as high as fifty percent for the early-onset form of the disorder. [...] Most people with dysthymia can't tell for sure when they first became depressed".

People with dysthymia have a higher-than-average chance of developing major depression. When an intense episode of depression occurs on top of dysthymia the state is called "double depression."

As dysthymia is a chronic disorder, sufferers may experience symptoms for many years before it is diagnosed, if diagnosis occurs at all. As a result, they may believe that
depression is a part of their character, so they may not even discuss their symptoms with doctors, family members, or friends.

Dysthymia, like major depression, tends to run in families. Some sufferers describe being under chronic stress. When treating diagnosed individuals, it is often difficult to tell whether they are under unusually high environmental stress or the dysthymia is causing them to be more psychologically stressed in a standard environment.

**Signs and symptoms**

Dysthymia is a chronic long-lasting form of depression sharing many characteristic symptoms of major depressive disorder (in the form of the melancholic depression subtype). These symptoms tend to be less severe but do fluctuate in intensity. Signs and symptoms can include:

- Feelings of hopelessness
- Insomnia or hypersomnia
- Poor concentration or difficulty making decisions
- Poor appetite or overeating
- Low energy or fatigue
- Low self-esteem
- Low sex drive
- Irritability

Symptoms exclude "manic, hypomanic or mixed episodes commonly associated with bipolar disorder". (If a person experiences these episodes, they may suffer from cyclothymia.)

**Diagnostic criteria**

The Diagnostic and Statistical Manual of Mental Disorders (DSM), published by the American Psychiatric Association, characterizes dysthymic disorder. The essential symptom involves the individual feeling depressed for the majority of days and parts of the day for at least two years. Low energy, disturbances in sleep or in appetite, and low self-esteem typically contribute to the clinical picture as well. Sufferers have often experienced dysthymia for many years before it is diagnosed. People around them come to believe that the sufferer is 'just a moody person'. Note the following diagnostic criteria:

- During a majority of days for two years or more, the adult patient reports depressed mood or appears depressed to others for most of the day.
- When depressed, the patient has two or more of:
  - decreased or increased appetite
  - decreased or increased sleep (insomnia or hypersomnia)
  - Fatigue or low energy
  - Reduced self-esteem
  - Decreased concentration or problems making decisions
o Feels hopeless or pessimistic
  - During this two-year period, the above symptoms are never absent longer than two consecutive months.
  - During the first two years of this syndrome, the patient has not had a major depressive episode.
  - The patient has not had any manic, hypomanic, or mixed episodes.
  - The patient has never fulfilled criteria for cyclothymic disorder.
  - The depression does not exist only as part of a chronic psychosis (such as schizophrenia or delusional disorder).
  - The symptoms are often not directly caused by a medical illness or by substances, including drug abuse, or other medications.
  - The symptoms may cause significant problems or distress in social, work, academic, or other major areas of life functioning.

People suffering from dysthymia aren't always capable of coping well with their everyday lives. Dysthymics who cope well with daily life tend to follow particular routines that provide certainty.

In children and adolescents, mood can be irritable, and duration must be at least one year, in contrast to two years needed for diagnosis in adults.

**Treatments**

**Medications**

If medication is deemed necessary, the most commonly prescribed anti-depressants for this disorder are the selective serotonin reuptake inhibitors (SSRIs), which include fluoxetine (Prozac), sertraline (Zoloft), escitalopram (Lexapro), paroxetine (Paxil), and citalopram (Celexa). Other anti-depressants which may be used include newer dual-acting agents such as bupropion (Wellbutrin), venlafaxine (Effexor), mirtazapine (Remeron, Avanza), and duloxetine (Cymbalta).

Sometimes two different anti-depressant medications are prescribed together, or a physician may prescribe a mood stabilizer or anti-anxiety medication in combination with an anti-depressant.

**Side-effects of medications**

Some side-effects for SSRIs are "sexual dysfunction, nausea...diarrhea, sleepiness or insomnia, short-term memory loss and tremors". Antidepressant medications can also cause suicidality and aggression in some cases, in particular in children and teens. Some antidepressants are ineffective in some patients. Older antidepressants such as a tricyclic antidepressant or an MAOI can be tried in such cases. Tricyclic antidepressants are more effective but have worse side-effects. Side-effects for tricyclic antidepressants are "weight gain, dry mouth, blurry vision, sexual dysfunction, and low blood pressure".
Depressive Disorder Not Otherwise Specified

Depressive Disorder Not Otherwise Specified (DD-NOS) is designated by the code 311 in the DSM-IV for depressive disorders that are impairing but do not fit any the officially specified diagnoses. According to the DSM-IV, DD-NOS encompasses "any depressive disorder that does not meet the criteria for a specific disorder."

Examples of disorders in this category include those sometimes described as Minor Depressive Disorder and Recurrent Brief Depressive Disorder.

Concerns

Accurately assessing for a specific Depressive Disorder diagnosis requires an expenditure of time that is deemed unreasonable for most primary care physicians. For this reason, physicians often use this code as a proxy for a more thorough diagnosis. There is concern that this may lead to a "waste basket" mindset for certain disorders. In addition reimbursement through Medicare may be lower for certain non specific diagnosis.

According to DSM IV-TR classification

311- Depressive Disorder Not Otherwise Specified (NOS)

The Depressive disorder NOS category includes disorders with depressive features that do not meet the criteria for Major Depressive Disorder, Dysthymic disorder, Adjustment Disorder with Depressed Mood or Adjustment Disorder with Mixed Anxiety and Depressed Mood. Sometimes depressive symptoms can present as part of an Anxiety Disorder Not otherwise Specified. Examples of Depressive Disorder Not Otherwise Specified include.

- Premenstrual Dysphoric Disorder: in most menstrual cycles during the past years, (e.g., markedly depressed mood, marked anxiety, marked affective liability, decreased interest in activities) regularly occurred during the onset of menses. These symptoms must be severe enough to markedly interfere with work, school, or usual activities and be entirely absent for at least 1 week post menses.
- Minor depressive disorder :episodes of at least 2 weeks of depressive symptoms but with fewer than the five items required for Major Depressive Disorder.
- Recurrent brief depressive disorder: depressive episodes lasting from 2 days up to 2 weeks, occurring at least once a month for 12 months(not associated with the menstrual cycle)
- Post psychotic depressive Disorder of schizophrenia :a Major Depressive Episode that occurs during the residual phase of schizophrenia.
- A Major Depressive Episode superimposed on Delusional Disorder, Psychotic Disorder Not Otherwise Specified, or the active phase of Schizophrenia.
- Situations in which the clinician has concluded that a depressive disorder is present but is unable to determine whether it is primary, due to a general medical condition, or substance induced.
Bipolar disorder

Bipolar disorder or manic-depressive disorder, also referred to as bipolar affective disorder or manic depression, is a psychiatric diagnosis that describes a category of mood disorders defined by the presence of one or more episodes of abnormally elevated energy levels, cognition, and mood with or without one or more depressive episodes. The elevated moods are clinically referred to as mania or, if milder, hypomania. Individuals who experience manic episodes also commonly experience depressive episodes, or symptoms, or a mixed state in which features of both mania and depression are present at the same time. These events are usually separated by periods of "normal" mood; but, in some individuals, depression and mania may rapidly alternate, which is known as rapid cycling. Extreme manic episodes can sometimes lead to such psychotic symptoms as delusions and hallucinations. The disorder has been subdivided into bipolar I, bipolar II, cyclothymia, and other types, based on the nature and severity of mood episodes experienced; the range is often described as the bipolar spectrum.

Estimates of the lifetime prevalence of bipolar disorder vary, with studies typically giving values of the order of 1%, with higher figures given in studies with looser definitions of the condition. The onset of full symptoms generally occurs in late adolescence or young adulthood. Diagnosis is based on the person’s self-reported experiences, as well as observed behavior. Episodes of abnormality are associated with distress and disruption and an elevated risk of suicide, especially during depressive episodes. In some cases, it can
be a devastating long-lasting disorder. In others, it has also been associated with creativity, goal striving, and positive achievements. There is significant evidence to suggest that many people with creative talents have also suffered from some form of bipolar disorder. It is often suggested that creativity and bipolar disorder are linked.

Genetic factors contribute substantially to the likelihood of developing bipolar disorder, and environmental factors are also implicated. Bipolar disorder is often treated with mood stabilizing medications and, sometimes, other psychiatric drugs. Psychotherapy also has a role, often when there has been some recovery of the subject’s stability. In serious cases, in which there is a risk of harm to oneself or others, involuntary commitment may be used. These cases generally involve severe manic episodes with dangerous behavior or depressive episodes with suicidal ideation. There are widespread problems with social stigma, stereotypes, and prejudice against individuals with a diagnosis of bipolar disorder. People with bipolar disorder exhibiting psychotic symptoms can sometimes be misdiagnosed as having schizophrenia, another serious mental illness.

The current term bipolar disorder is of fairly recent origin and refers to the cycling between high and low episodes (poles). A relationship between mania and melancholia had long been observed, although the basis of the current conceptualisation can be traced back to French psychiatrists in the 1850s. The term "manic-depressive illness" or psychosis was coined by German psychiatrist Emil Kraepelin in the late nineteenth century, originally referring to all kinds of mood disorder. German psychiatrist Karl Leonhard split the classification again in 1957, employing the terms unipolar disorder (major depressive disorder) and bipolar disorder.

Signs and symptoms

Bipolar disorder is a condition in which people experience abnormally elevated (manic or hypomanic) and, in many cases, abnormally depressed states for periods of time in a way that interferes with functioning. Not everyone’s symptoms are the same, and there is no simple physiological test to confirm the disorder. Bipolar disorder can appear to be unipolar depression. Diagnosing bipolar disorder is often difficult, even for mental health professionals. What distinguishes bipolar disorder from unipolar depression is that the affected person experiences states of mania and depression. Often bipolar is inconsistent among patients because some people feel depressed more often than not and experience little mania whereas others experience predominantly manic symptoms. Additionally, the younger the age of onset—bipolar disorder starts in childhood or early adulthood in most patients—the more likely the first few episodes are to be depression. Because a bipolar diagnosis requires a manic or hypomanic episode, many patients are initially diagnosed and treated as having major depression.

Depressive episode

Signs and symptoms of the depressive phase of bipolar disorder include persistent feelings of sadness, anxiety, guilt, anger, isolation, or hopelessness; disturbances in sleep and appetite; fatigue and loss of interest in usually enjoyable activities; problems
concentrating; loneliness, self-loathing, apathy or indifference; depersonalization; loss of interest in sexual activity; shyness or social anxiety; irritability, chronic pain (with or without a known cause); lack of motivation; and morbid suicidal ideation. In severe cases, the individual may become psychotic, a condition also known as severe bipolar depression with psychotic features. These symptoms include delusions or, less commonly, hallucinations, usually unpleasant. A major depressive episode persists for at least two weeks, and may continue for over six months if left untreated.

**Manic episode**

Mania is the signature characteristic of bipolar disorder and, depending on its severity, is how the disorder is classified. Mania is generally characterized by a distinct period of an elevated mood, which can take the form of euphoria. People commonly experience an increase in energy and a decreased need for sleep, with many often getting as little as 3 or 4 hours of sleep per night, while others can go days without sleeping. A person may exhibit pressured speech, with thoughts experienced as racing. Attention span is low, and a person in a manic state may be easily distracted. Judgment may become impaired, and sufferers may go on spending sprees or engage in behavior that is quite abnormal for them. They may indulge in substance abuse, particularly alcohol or other depressants, cocaine or other stimulants, or sleeping pills. Their behavior may become aggressive, intolerant, or intrusive. People may feel out of control or unstoppable, or as if they have been "chosen" and are "on a special mission" or have other grandiose or delusional ideas. Sexual drive may increase. At more extreme phases of bipolar I, a person in a manic state can begin to experience psychosis, or a break with reality, where thinking is affected along with mood. Some people in a manic state experience severe anxiety and are very irritable (to the point of rage), while others are euphoric and grandiose.

To be diagnosed with mania according to the Diagnostic and Statistical Manual of Mental Disorders (DSM), a person must experience this state of elevated or irritable mood, as well as other symptoms, for at least one week, less if hospitalization is required.

Severity of manic symptoms can be measured by rating scales such as self-reported Altman Self-Rating Mania Scale and clinician-based Young Mania Rating Scale.

**Hypomanic episode**

Hypomania is generally a mild to moderate level of mania, characterized by optimism, pressure of speech and activity, and decreased need for sleep. Generally, hypomania does not inhibit functioning like mania. Many people with hypomania are actually in fact more productive than usual, while manic individuals have difficulty completing tasks due to a shortened attention span. Some people have increased creativity, while others demonstrate poor judgment and irritability. Many people experience signature hypersexuality. These persons generally have increased energy and tend to become more active than usual. They do not, however, have delusions or hallucinations. Hypomania can be difficult to diagnose because it may masquerade as mere happiness, though it carries the same risks as mania.
Hypomania may feel good to the person who experiences it. Thus, even when family and friends learn to recognize the mood swings, the individual often will deny that anything is wrong. Also, the individual may not be able to recall the events that took place while they were experiencing hypomania. What might be called a "hypomanic event", if not accompanied by complementary depressive episodes ("downs", etc.), is not typically deemed as problematic: The "problem" arises when mood changes are uncontrollable and, more importantly, volatile or "mercurial". If unaccompanied by depressive counterpart episodes or otherwise general irritability, this behavior is typically called hyperthymia, or happiness, which is, of course, perfectly normal. Indeed, the most elementary definition of bipolar disorder is an often "violent" or "jarring" state of essentially uncontrollable oscillation between hyperthymia and dysthymia. If left untreated, an episode of hypomania can last anywhere from a few days to several years. Most commonly, symptoms continue for a few weeks to a few months.

Mixed affective episode

In the context of bipolar disorder, a mixed state is a condition during which symptoms of mania and clinical depression occur simultaneously. Typical examples include tearfulness during a manic episode or racing thoughts during a depressive episode. Individuals may also feel incredibly frustrated in this state, since one may feel like a failure and at the same time have a flight of ideas. Mixed states are often the most dangerous period of mood disorders, during which substance abuse, panic disorder, suicide attempts, and other complications increase greatly.

Associated features

Associated features are clinical phenomena that often accompany the disorder but are not part of the diagnostic criteria for the disorder. There are several childhood precursors in children who later receive a diagnosis of bipolar disorder. They may show subtle early traits such as mood abnormalities, full major depressive episodes, and ADHD. BD is also accompanied by changes in cognitive processes and abilities. This include reduced attentional and executive capabilities and impaired memory. How the individual processes the world also depends on the phase of the disorder, with differential characteristics between the manic, hypomanic and depressive states. Some studies have found a significant association between bipolar disorder and creativity.

Causes

The causes of bipolar disorder likely vary between individuals. Twin studies have been limited by relatively small sample sizes but have indicated a substantial genetic contribution, as well as environmental influence. For bipolar I, the (probandwise) concordance rates in modern studies have been consistently put at around 40% in monozygotic twins (same genes), compared to 0 to 10% in dizygotic twins. A combination of bipolar I, II and cyclothymia produced concordance rates of 42% vs 11%, with a relatively lower ratio for bipolar II that likely reflects heterogeneity. The overall heritability of the bipolar spectrum has been put at 0.71. There is overlap with unipolar depression and
if this is also counted in the co-twin the concordance with bipolar disorder rises to 67% in monozygotic twins and 19% in dizygotic. The relatively low concordance between dizygotic twins brought up together suggests that shared family environmental effects are limited, although the ability to detect them has been limited by small sample sizes.

**Genetic**

Genetic studies have suggested many chromosomal regions and candidate genes appearing to relate to the development of bipolar disorder, but the results are not consistent and often not replicated.

Although the first genetic linkage finding for mania was in 1969, the linkage studies have been inconsistent. Meta-analyses of linkage studies detected either no significant genome-wide findings or, using a different methodology, only two genome-wide significant peaks, on chromosome 6q and on 8q21. Genome-wide association studies neither brought a consistent focus — each has identified new loci.

Findings point strongly to heterogeneity, with different genes being implicated in different families. A review seeking to identify the more consistent findings suggested several genes related to serotonin (SLC6A4 and TPH2), dopamine (DRD4 and SLC6A3), glutamate (DAOA and DTNBP1), and cell growth and/or maintenance pathways (NRG1, DISC1 and BDNF), although noting a high risk of false positives in the published literature. It was also suggested that individual genes are likely to have only a small effect and to be involved in some aspect related to the disorder (and a broad range of "normal" human behavior) rather than the disorder per se.

Advanced paternal age has been linked to a somewhat increased chance of bipolar disorder in offspring, consistent with a hypothesis of increased new genetic mutations.

**Physiological**

Abnormalities in the structure and/or function of certain brain circuits could underlie bipolar. Two meta-analyses of MRI studies in bipolar disorder report a increase in the volume of the lateral ventricles, globus pallidus and increase in the rates of deep white matter hyperintensities.

The "kindling" theory asserts that people who are genetically predisposed toward bipolar disorder can experience a series of stressful events, each of which lowers the threshold at which mood changes occur. Eventually, a mood episode can start (and become recurrent) by itself. There is evidence of hypothalamic-pituitary-adrenal axis (HPA axis) abnormalities in bipolar disorder due to stress.

Other brain components which have been proposed to play a role are the mitochondria, and a sodium ATPase pump, causing cyclical periods of poor neuron firing (depression) and hypersensitive neuron firing (mania). This may only apply for type one, but type two
apparently results from a large confluence of factors. Circadian rhythms and melatonin activity also seem to be altered.

**Environmental**

Evidence suggests that environmental factors play a significant role in the development and course of bipolar disorder, and that individual psychosocial variables may interact with genetic dispositions. There is fairly consistent evidence from prospective studies that recent life events and interpersonal relationships contribute to the likelihood of onsets and recurrences of bipolar mood episodes, as they do for onsets and recurrences of unipolar depression. There have been repeated findings that between a third and a half of adults diagnosed with bipolar disorder report traumatic/abusive experiences in childhood, which is associated on average with earlier onset, a worse course, and more co-occurring disorders such as PTSD. The total number of reported stressful events in childhood is higher in those with an adult diagnosis of bipolar spectrum disorder compared to those without, particularly events stemming from a harsh environment rather than from the child’s own behavior. Early experiences of adversity and conflict are likely to make subsequent developmental challenges in adolescence more difficult, and are likely a potentiating factor in those at risk of developing bipolar disorder.

**Diagnosis**

Diagnosis is based on the self-reported experiences of an individual as well as abnormalities in behavior reported by family members, friends or co-workers, followed by secondary signs observed by a psychiatrist, nurse, social worker, clinical psychologist or other clinician in a clinical assessment. There are lists of criteria for someone to be so diagnosed. These depend on both the presence and duration of certain signs and symptoms. Assessment is usually done on an outpatient basis; admission to an inpatient facility is considered if there is a risk to oneself or others. The most widely used criteria for diagnosing bipolar disorder are from the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders, the current version being DSM-IV-TR, and the World Health Organization’s International Statistical Classification of Diseases and Related Health Problems, currently the ICD-10. The latter criteria are typically used in Europe and other regions while the DSM criteria are used in the USA and other regions, as well as prevailing in research studies.

An initial assessment may include a physical exam by a physician. Although there are no biological tests which confirm bipolar disorder, tests may be carried out to exclude medical illnesses such as hypo- or hyperthyroidism, metabolic disturbance, a systemic infection or chronic disease, and syphilis or HIV infection. An EEG may be used to exclude epilepsy, and a CT scan of the head to exclude brain lesions. Investigations are not generally repeated for relapse unless there is a specific medical indication.

Several rating scales for the screening and evaluation of BD exist, such as the Bipolar spectrum diagnostic scale. The use of evaluation scales can not substitute a full clinical
interview but they serve to systematize the recollection of symptoms. On the other hand, instruments for the screening of BD have low sensitivity and limited diagnostic validity.

**Criteria and subtypes**

There is no clear consensus as to how many types of bipolar disorder exist. In DSM-IV-TR and ICD-10, bipolar disorder is conceptualized as a spectrum of disorders occurring on a continuum. The DSM-IV-TR lists three specific subtypes and one for non-specified:

**Bipolar I disorder**
- One or more manic episodes. Subcategories specify whether there has been more than one episode, and the type of the most recent episode. A depressive or hypomanic episode is not required for diagnosis, but it frequently occurs.
- Bipolar II disorder
- No manic episodes, but one or more hypomanic episodes and one or more major depressive episode. However, a bipolar II diagnosis is not a guarantee that they will not eventually suffer from such an episode in the future. Hypomanic episodes do not go to the full extremes of mania (i.e., do not usually cause severe social or occupational impairment, and are without psychosis), and this can make bipolar II more difficult to diagnose, since the hypomanic episodes may simply appear as a period of successful high productivity and is reported less frequently than a distressing, crippling depression.
- Cyclothymia
- A history of hypomanic episodes with periods of depression that do not meet criteria for major depressive episodes. There is a low-grade cycling of mood which appears to the observer as a personality trait, and interferes with functioning.
- Bipolar Disorder NOS (Not Otherwise Specified)
- This is a catchall category, diagnosed when the disorder does not fall within a specific subtype. Bipolar NOS can still significantly impair and adversely affect the quality of life of the patient.

The bipolar I and II categories have specifiers that indicate the presentation and course of the disorder. For example, the "with full interepisode recovery" specifier applies if there was full remission between the two most recent episodes.

**Rapid cycling**

Most people who meet criteria for bipolar disorder experience a number of episodes, on average 0.4 to 0.7 per year, lasting three to six months. Rapid cycling, however, is a course specifier that may be applied to any of the above subtypes. It is defined as having four or more episodes per year and is found in a significant fraction of individuals with bipolar disorder. The definition of rapid cycling most frequently cited in the literature (including the DSM) is that of Dunner and Fieve: at least four major depressive, manic, hypomanic or mixed episodes are required to have occurred during a 12-month period. Ultra-rapid (days) or ultra-ultra rapid or ultradian (within a day) cycling have also been described.
Differential diagnosis

There are several other mental disorders which may involve similar symptoms to bipolar disorder. These include schizophrenia, schizoaffective disorder, drug intoxication, brief drug-induced psychosis, schizophreniform disorder and borderline personality disorder. Both borderline personality and bipolar disorder can involve what are referred to as "mood swings". In bipolar disorder, the term refers to the cyclic episodes of elevated and depressed mood which generally last weeks or months. The term in borderline personality refers to the marked lability and reactivity of mood, known as emotional dysregulation, due to response to external psychosocial and intrapsychic stressors; these may arise or subside suddenly and dramatically and last for seconds, minutes, hours or days. A bipolar depression is generally more pervasive with sleep, appetite disturbance and nonreactive mood, whereas the mood in dysthymia of borderline personality remains markedly reactive and sleep disturbance not acute. Some hold that borderline personality disorder represents a subthreshold form of mood disorder while others maintain the distinctness, though noting they often coexist.

Challenges

The experiences and behaviors involved in bipolar disorder are often not understood by individuals or recognized by mental health professionals, so diagnosis may sometimes be delayed for over 10 years. The treatment lag is apparently not decreasing, even though there is increased public awareness of the condition.

Individuals are commonly misdiagnosed. An individual may appear simply depressed when they are seen by a health professional. This can result in misdiagnosis of Major Depressive Disorder. However, there is also a long-standing issue in the research literature as to whether a categorical classificatory divide between unipolar and bipolar depression is actually valid, or whether it is more accurate to talk of a continuum involving dimensions of depression and mania.

It has been noted that the bipolar disorder diagnosis is officially characterised in historical terms such that, technically, anyone with a history of (hypo)mania and depression has bipolar disorder whatever their current or future functioning and vulnerability. This has been described as "an ethical and methodological issue", as it means no one can be considered as being recovered (only "in remission") from bipolar disorder according to the official criteria. This is considered especially problematic given that brief hypomanic episodes are widespread among people generally and not necessarily associated with dysfunction.

Flux is the fundamental nature of bipolar disorder. Individuals with the illness have continual changes in energy, mood, thought, sleep, and activity. The diagnostic subtypes of bipolar disorder are thus static descriptions—snapshots, perhaps—of an illness in continual flux, with a great diversity of symptoms and varying degrees of severity. Individuals may stay in one subtype, or change into another, over the course of their illness.
The DSM-V, to be published in 2013, will likely include further and more accurate sub-typing (Akiskal and Ghaemi, 2006).

The diagnosis of bipolar disorder can be complicated by coexisting psychiatric conditions such as obsessive-compulsive disorder, social phobia, panic disorder, or attention-deficit/hyperactivity disorder. Substance abuse may predate the appearance of bipolar symptoms, further complicating the diagnosis. A careful longitudinal analysis of symptoms and episodes, enriched if possible by discussions with friends and family members, is crucial to establishing a treatment plan where these comorbidities exist.

Management

There are a number of pharmacological and psychotherapeutic techniques used to treat bipolar disorder. Individuals may use self-help and pursue recovery.

Hospitalization may be required especially with the manic episodes present in bipolar I. This can be voluntary or (if mental health legislation allows and varying state-to-state regulations in the USA) involuntary (called civil or involuntary commitment). Long-term inpatient stays are now less common due to deinstitutionalization, although these can still occur. Following (or in lieu of) a hospital admission, support services available can include drop-in centers, visits from members of a community mental health team or Assertive Community Treatment team, supported employment and patient-led support groups, intensive outpatient programs. These are sometimes referred to partial-inpatient programs.

Psychosocial

Psychotherapy is aimed at alleviating core symptoms, recognizing episode triggers, reducing negative expressed emotion in relationships, recognizing prodromal symptoms before full-blown recurrence, and, practicing the factors that lead to maintenance of remission Cognitive behavioural therapy, family-focused therapy, and psychoeducation have the most evidence for efficacy in regard to relapse prevention, while interpersonal and social rhythm therapy and cognitive-behavioural therapy appear the most effective in regard to residual depressive symptoms. Most studies have been based only on bipolar I, however, and treatment during the acute phase can be a particular challenge. Some clinicians emphasize the need to talk with individuals experiencing mania, to develop a therapeutic alliance in support of recovery.

Medication
Sodium valproate is a common mood stabilizer

The mainstay of treatment is a mood stabilizers such as lithium carbonate or lamotrigine. Lamotrigine has been found to be best for preventing depressions, while lithium is the only drug proven to reduce suicide in people with bipolar disorder. These two drugs comprise several unrelated compounds which have been shown to be effective in preventing relapses of manic, or in the one case, depressive episodes. The first known and "gold standard" mood stabilizer is lithium, while almost as widely used is sodium valproate, also used as an anticonvulsant. Other anticonvulsants used in bipolar disorder include carbamazepine, reportedly more effective in rapid cycling bipolar disorder, and lamotrigine, which is the first anticonvulsant shown to be of benefit in bipolar depression. Depending on the severity of the case, anti-convulsants may be used in combination with lithium-based products or on their own.

Atypical antipsychotics have been found to be effective in managing mania associated with bipolar disorder. Antidepressants have not been found to be of any benefit over that found with mood stabilizers.

Omega 3 fatty acids, in addition to normal pharmacological treatment, may have beneficial effects on depressive symptoms, although studies have been scarce and of variable quality. The effectiveness of topiramate is unknown.

**Prognosis**

For many individuals with bipolar disorder a good prognosis results from good treatment, which, in turn, results from an accurate diagnosis. Because bipolar disorder can have a high rate of both under-diagnosis and misdiagnosis, it is often difficult for individuals with the condition to receive timely and competent treatment.
Bipolar disorder can be a severely disabling medical condition. However, many individuals with bipolar disorder can live full and satisfying lives. Quite often, medication is needed to enable this. Persons with bipolar disorder may have periods of normal or near normal functioning between episodes.

Prognosis depends on many factors such as the right medicines and dosage, comprehensive knowledge of the disease and its effects; a positive relationship with a competent medical doctor and therapist; and good physical health, which includes exercise, nutrition, and a regulated stress level. There are other factors that lead to a good prognosis, such as being very aware of small changes in a person’s energy, mood, sleep and eating behaviors.

**Functioning**

A recent 20-year prospective study on bipolar I and II found that functioning varied over time along a spectrum from good to fair to poor. During periods of major depression or mania (in BPI), functioning was on average poor, with depression being more persistently associated with disability than mania. Functioning between episodes was on average good — more or less normal. Subthreshold symptoms were generally still substantially impairing, however, except for hypomania (below or above threshold) which was associated with improved functioning.

Another study confirmed the seriousness of the disorder as "the standardized all-cause mortality ratio among patients with BD is increased approximately two-fold." Bipolar disorder is currently regarded "as possibly the most costly category of mental disorders in the United States." Episodes of abnormality are associated with distress and disruption, and an elevated risk of suicide, especially during depressive episodes.

**Recovery and recurrence**

A naturalistic study from first admission for mania or mixed episode (representing the hospitalized and therefore most severe cases) found that 50% achieved syndromal recovery (no longer meeting criteria for the diagnosis) within six weeks and 98% within two years. 72% achieved symptomatic recovery (no symptoms at all) and 43% achieved functional recovery (regaining of prior occupational and residential status). However, 40% went on to experience a new episode of mania or depression within 2 years of syndromal recovery, and 19% switched phases without recovery.

Symptoms preceeding a relapse (prodromal), specially those related to mania, can be reliably identified by people with BD. There have been intents to teach patients coping strategies when noticing such symptoms with encouraging results.

**Mortality**

Bipolar disorder can cause suicidal ideation that leads to suicidal attempts. One out of 3 people with bipolar disorder report past attempts of suicide or complete it, and the annual
average suicide rate is 0.4%, which is 10 to 20 times that of the general population. The standardized mortality ratio from suicide in BD is between 18 and 25.

**Epidemiology**

When broadly defined 4% of people experience bipolar at some point in their life. The lifetime prevalence of bipolar disorder type I, which includes at least a lifetime manic episode, has generally been estimated at 2%. It is equally prevalent in men and women and is found across all cultures and ethnic groups.

A reanalysis of data from the National Epidemiological Catchment Area survey in the United States, however, suggested that 0.8 percent experience a manic episode at least once (the diagnostic threshold for bipolar I) and 0.5 a hypomanic episode (the diagnostic threshold for bipolar II or cyclothymia). Including sub-threshold diagnostic criteria, such as one or two symptoms over a short time-period, an additional 5.1 percent of the population, adding up to a total of 6.4 percent, were classed as having a bipolar spectrum disorder. A more recent analysis of data from a second US National Comorbidity Survey found that 1% met lifetime prevalence criteria for bipolar 1, 1.1% for bipolar II, and 2.4% for subthreshold symptoms. There are conceptual and methodological limitations and variations in the findings. Prevalence studies of bipolar disorder are typically carried out by lay interviewers who follow fully structured/fixed interview schemes; responses to single items from such interviews may suffer limited validity. In addition, diagnosis and prevalence rates are dependent on whether a categorical or spectrum approach is used. Concerns have arisen about the potential for both underdiagnosis and overdiagnosis.

Late adolescence and early adulthood are peak years for the onset of bipolar disorder. One study also found that in 10% of bi-polar cases, the onset of mania had happened after the patient had turned 50.

**History**

Variations in moods and energy levels have been observed as part of the human experience since time immemorial. The words "melancholia" (an old word for depression) and "mania" have their etymologies in Ancient Greek. The word melancholia is derived from melas/μελας, meaning "black", and chole/χολη, meaning "bile" or "gall", indicative of the term's origins in pre-Hippocratic humoral theories. Within the humoral theories, mania was viewed as arising from an excess of yellow bile, or a mixture of black and yellow bile. The linguistic origins of mania, however, are not so clear-cut. Several etymologies are proposed by the Roman physician Caelius Aurelianus, including the Greek word 'ania', meaning to produce great mental anguish, and 'manos', meaning relaxed or loose, which would contextually approximate to an excessive relaxing of the mind or soul (Angst and Marneros 2001). There are at least five other candidates, and part of the confusion surrounding the exact etymology of the word mania is its varied usage in the pre-Hippocratic poetry and mythologies (Angst and Marneros 2001).
The basis of the current conceptualisation of manic-depressive illness can be traced back to the 1850s; on January 31, 1854, Jules Baillarger described to the French Imperial Academy of Medicine a biphasic mental illness causing recurrent oscillations between mania and depression, which he termed folie à double forme (‘dual-form insanity’). Two weeks later, on February 14, 1854, Jean-Pierre Falret presented a description to the Academy on what was essentially the same disorder, and designated folie circulaire (‘circular insanity’) by him. (Sedler 1983) The two bitterly disputed as to who had been the first to conceptualise the condition.

These concepts were developed by the German psychiatrist Emil Kraepelin (1856–1926), who, using Kahlbaum’s concept of cyclothymia, categorized and studied the natural course of untreated bipolar patients. He coined the term manic depressive psychosis, after noting that periods of acute illness, manic or depressive, were generally punctuated by relatively symptom-free intervals where the patient was able to function normally.

The term "manic-depressive reaction" appeared in the first American Psychiatric Association Diagnostic Manual in 1952, influenced by the legacy of Adolf Meyer who had introduced the paradigm illness as a reaction of biogenetic factors to psychological and social influences. Subclassification of bipolar disorder was first proposed by German psychiatrist Karl Leonhard in 1957; he was also the first to introduce the terms bipolar (for those with mania) and unipolar (for those with depressive episodes only).

**Society and culture**

**Stigma**

There are widespread problems with social stigma, stereotypes, and prejudice against individuals with a diagnosis of bipolar disorder.

**Cultural references**

Kay Redfield Jamison, a clinical psychologist and Professor of Psychiatry at the Johns Hopkins University School of Medicine, profiled her own bipolar disorder in her memoir An Unquiet Mind (1995). In her book, Touched with Fire (1993), she argued for a connection between bipolar disorder and artistic creativity.

Several films have portrayed characters with traits suggestive of the diagnosis that has been the subject of discussion by psychiatrists and film experts alike. A notable example is Mr. Jones (1993), in which Mr. Jones (Richard Gere) swings from a manic episode into a depressive phase and back again, spending time in a psychiatric hospital and displaying many of the features of the syndrome. In The Mosquito Coast (1986), Allie Fox (Harrison Ford) displays some features including recklessness, grandiosity, increased goal-directed activity and mood lability, as well as some paranoia.

In the Australian TV drama Stingers, Detective Luke Harris (Gary Sweet) is portrayed as having bipolar disorder and shows how his paranoia interfered with his work. As research
for the role, Sweet visited a psychiatrist to learn about manic-depressive illness. He said that he left the sessions convinced he had the condition. TV specials, for example the BBC’s The Secret Life of the Manic Depressive, MTV’s True Life: I’m Bipolar, talk shows, and public radio shows, and the greater willingness of public figures to discuss their own bipolar disorder, have focused on psychiatric conditions, thereby, raising public awareness.

On April 7, 2009, the nighttime drama 90210 on the CW network, aired a special episode where the character Silver was diagnosed with bipolar disorder. A public service announcement (PSA) aired after the episode, directing teens and young adults to the Child and Adolescent Bipolar Foundation website for information and to chat with other teens.

Stacey Slater, a character from the popular BBC soap EastEnders, has been diagnosed with the disorder. After losing her friend Danielle Jones, Stacey began acting strangely; and the character had to come to terms with the prospect that, like her mother, Jean Slater, she suffers from bipolar disorder. The high-profile storyline was developed as part of the BBC’s Headroom campaign. The Channel 4 soap Brookside had earlier featured a story about bipolar disorder when the character Jimmy Corkhill was diagnosed with the condition. Dean Sullivan, the actor who played Jimmy, was presented with a Special Achievement Award at the 2003 British Soap Awards for the role.

Specific populations

In children

Emil Kraepelin in the 1920s noted that mania episodes were rare before puberty. In general BD in children was not recognized in the first half of the XX century this issue

Lithium carbonate is the only drug approved for children by the FDA
diminishing with an increased following of the DSM criteria in the last part of the XXth
century.

While in adults the course of BD is characterized by discrete episodes of depression and
mania with no clear symptomatology between them, in children and adolescents very fast
mood changes or even chronic symptoms are the norm. On the other hand pediatric BD
instead of euphoric mania commonly develops with outbursts of anger, irritability and
psychosis, less common in adults.

The diagnosis of childhood BD is controversial, although it is not under discussion that BD
typical symptoms have negative consequences for minors suffering them. Main discussion
is centered on whether what is called BD in children refers to the same disorder than when
diagnosing adults, and the related question on whether adults criteria for diagnosis are
useful and accurate when applied to children. Regarding diagnosis of children some experts
recommend to follow the DSM criteria. Others believe that these criteria do not separate
correctly children with BD from other problems such as ADHD, and emphasize fast mood
cycles. Still others argue that what accurately differentiates children with BD is irritability.
The practice parameters of the AACAP encourage the first strategy. American children and
adolescents diagnosed of BD in community hospitals increased 4-fold reaching rates of up
to 40% in 10 years around the beginning of the current century, while in outpatient clinics
it doubled reaching the 6%. The data suggest that doctors had been more aggressively
applying the diagnosis to children. The reasons for this increase are unclear. Consensus
regarding the diagnosis in the pediatric age seems to apply only to the USA. Studies using
DSM criteria show that up to 1% of youth may have BD.

Treatment involves medication and psychotherapy. Drug prescription usually consists in
mood stabilizers and atypical antipsychotics. Among the formers lithium is the only
compound approved by the FDA for children. Psychological treatment combines normally
education on the disease, group therapy and cognitive behavioral therapy. Chronic
medication is often needed.

Current research directions for BD in children include optimizing treatments, increasing
the knowledge of the genetic and neurobiological basis of the pediatric disorder and
improving diagnostic criteria. The DSM-V has proposed a new diagnosis which is
considered to cover some presentations currently thought of as childhood-onset bipolar.

In the elderly

There is a relative lack of knowledge about bipolar disorder in late life. There is evidence
that it becomes less prevalent with age but nevertheless accounts for a similar percentage
of psychiatric admissions; that older bipolar patients had first experienced symptoms at a
later age; that later onset of mania is associated with more neurologic impairment; that
substance abuse is considerably less common in older groups; and that there is probably a
greater degree of variation in presentation and course, for instance individuals may
develop new-onset mania associated with vascular changes, or become manic only after
recurrent depressive episodes, or may have been diagnosed with bipolar disorder at an
early age and still meet criteria. There is also some weak evidence that mania is less intense and there is a higher prevalence of mixed episodes, although there may be a reduced response to treatment. Overall there are likely more similarities than differences from younger adults. In the elderly, recognition and treatment of bipolar disorder may be complicated by the presence of dementia or the side effects of medications being taken for other conditions.

**Mania**

Mania, the presence of which is a criterion for certain psychiatric diagnoses, is a state of abnormally elevated or irritable mood, arousal, and/or energy levels. In a sense, it is the opposite of depression. The word derives from the Greek "μανία" (mania), "madness, frenzy" and that from the verb "μαίνομαι" (mainomai), "to be mad, to rage, to be furious".

In addition to mood disorders, individuals may exhibit manic behavior as a result of drug intoxication (notably stimulants such as cocaine or methamphetamine), medication side effects (notably steroids), or malignancy. However, mania is most often associated with bipolar disorder, where episodes of mania may alternate with episodes of major depression. Gelder, Mayou and Geddes (2005) suggests that it is vital that mania is predicted in the early stages because the patient becomes reluctant to comply to the treatment. The criteria for bipolar do not include depressive episodes and the presence of mania in the absence of depressive episodes is sufficient for a diagnosis. Regardless, even those who never experience depression experience cyclical changes in mood. These cycles are often affected by changes in sleep cycle (too much or too little), diurnal rhythms and environmental stressors.

Mania varies in intensity, from mild mania known as hypomania to full-blown mania with psychotic features including hallucinations, delusion of grandeur, suspiciousness, catatonic behavior, aggression, and a preoccupation with thoughts and schemes that may lead to self neglect. Since mania and hypomania have also been associated with creativity and artistic talent, it is not always the case that the clearly manic bipolar person will need or want medical assistance; such people will often either retain sufficient amount of control to function normally or be unaware that they have "gone manic" severely enough to be committed or to commit themselves ('commitment' means admission to a psychiatric facility). Manic individuals can often be mistaken for being on drugs or other mind-altering substances.

**Classification**

**Mixed states**

Mania can be experienced at the same time as depression, in a mixed episode. Dysphoric mania is primarily manic and agitated depression is primarily depressed. This has caused speculation amongst doctors that mania and depression are two independent axes in a bipolar spectrum, rather than opposites.
There is an increased probability of suicide in the mixed state, as depressed individuals who are also manic have the energy needed to commit the act and the thoughts of depression that would lead them initially to suicide.

Mania can be the result of using drugs. Quitting drugs can create situations in one's mind similar to the symptoms of mania, such as constant racing of the mind. A diagnosis of mania in these situations is often temporary.

**Hypomania**

Hypomania is a lowered state of mania that does little to impair function or decrease quality of life. In hypomania there is less need for sleep, and both goal-motivated behavior and metabolism increase. Though the elevated mood and energy level typical of hypomania could be seen as a benefit, mania itself generally has many undesirable consequences including suicidal tendencies.

**Associated disorders**

A single manic episode is sufficient to diagnose Bipolar I Disorder. Hypomania may be indicative of Bipolar II Disorder or Cyclothymia. However, if prominent psychotic symptoms are present for a duration significantly longer than the mood episode, a diagnosis of Schizoaffective Disorder is more appropriate. Several types of Mania such as kleptomania and pyromania are related more closely to OCD than to Bipolar Disorder, depending on the seriousness of these disorders. For instance, someone with kleptomania who suffers from impulses to steal things such as pencils, pens, and paperclips is better diagnosed with a form of OCD or Hypomania, but someone with pyromania who receives impulses to commit serious acts of arson (setting fire to large areas of private and/or public property) would be diagnosed with a very serious case of Mania or Bipolar Disorder.

B12 deficiency can also cause characteristics of mania and psychosis.

**Signs and symptoms**

A manic episode is defined in the American Psychiatric Association's diagnostic manual as a period of seven or more days (or any period if admission to hospital is required) of unusually and continuously effusive and open elated or irritable mood, where the mood is not caused by drugs or a medical illness (e.g., hyperthyroidism), and (a) is causing obvious difficulties at work or in social relationships and activities, or (b) requires admission to hospital to protect the person or others, or (c) the person is suffering psychosis.

To be classed as a manic episode, while the disturbed mood is present at least three (or four if only irritability is present) of the following must have been consistently prominent: grand or extravagant style, or expanded self-esteem; reduced need of sleep (e.g. three hours may be sufficient); talks more often and feels the urge to talk longer; ideas flit through the mind in quick succession, or thoughts race and preoccupy the person; over
indulgence in enjoyable behaviors with high risk of a negative outcome (e.g., extravagant shopping, sexual adventures or improbable commercial schemes).

If the person is concurrently depressed, they are said to be having a mixed episode.

The World health organization's classification system defines a manic episode as one where mood is higher than the person's situation warrants and may vary from relaxed high spirits to barely controllable exuberance, accompanied by hyperactivity, a compulsion to speak, a reduced sleep requirement, difficulty sustaining attention and, often, increased distractability. Frequently, confidence and self-esteem are excessively enlarged, and grand, extravagant ideas are expressed. Behavior that is out of character and risky, foolish or inappropriate may result from a loss of normal social restraint.

Some people also have physical symptoms, such as sweating, pacing, and weight loss. In full-blown mania, often the manic person will feel as though his or her goal(s) trump all else, that there are no consequences or that negative consequences would be minimal, and that they need not exercise restraint in the pursuit of what they are after. Hypomania is different, as it may cause little or no impairment in function. The hypomanic person's connection with the external world, and its standards of interaction, remain intact, although intensity of moods is heightened. But those who suffer from prolonged unresolved hypomania do run the risk of developing full mania, and indeed may cross that "line" without even realizing they have done so.

One of the most signature symptoms of mania (and to a lesser extent, hypomania) is what many have described as racing thoughts. These are usually instances in which the manic person is excessively distracted by objectively unimportant stimuli. This experience creates an absentmindedness where the manic individual's thoughts totally preoccupy him or her, making him or her unable to keep track of time, or be aware of anything besides the flow of thoughts. Racing thoughts also interfere with the ability to fall asleep.

Mania is always relative to the normal rate of intensity of the person being diagnosed with it; therefore, an easily-angered person may exhibit mania by getting even angrier even more quickly, and an intelligent person may adopt seemingly "genius" characteristics and an ability to perform and to articulate thought beyond what they can do in a normal mood. But perhaps the easiest indicator of mania would be if a noticeably clinically depressed person becomes suddenly cheerful, optimistic, happy, and full of energy. Other elements of mania may include delusions (of grandeur, potential, or otherwise), hypersensitivity, hypersexuality, hyper-religiosity, hyperactivity, impulsiveness, talkativeness, an internal pressure to keep talking (over-explanation) or rapid speech, grandiose ideas and plans, and decreased need for sleep (e.g. feeling rested after 3 or 4 hours of sleep). In manic and hypomanic cases, the afflicted person may engage in out-of-character behavior, such as questionable business transactions, wasteful expenditures of money, risky sexual activity, recreational drug abuse, abnormal social interaction, or highly vocal arguments uncharacteristic of previous behaviors. These behaviors may increase stress in personal relationships, lead to problems at work and increase the risk of altercations with law
enforcement. There is a high risk of impulsively taking part in activities potentially harmful to self and others.

Although "severely elevated mood" sounds somewhat desirable and enjoyable, the experience of mania is ultimately often quite unpleasant and sometimes disturbing, if not frightening, for the person involved and for those close to them, and it may lead to impulsive behavior that may later be regretted. It can also often be complicated by the sufferer's lack of judgment and insight regarding periods of exacerbation of characteristic states. Manic patients are frequently grandiose, obsessive, impulsive, irritable, belligerent, and frequently deny anything is wrong with them. Because mania frequently encourages high energy and decreased perception of need or ability to sleep, within a few days of a manic cycle, sleep-deprived psychosis may appear, further complicating the ability to think clearly. Racing thoughts and misperceptions lead to frustration and decreased ability to communicate with others.

There are different "stages" or "states" of mania. A minor state is essentially hypomania and, like hypomania's characteristics, may involve increased creativity, wit, gregariousness, and ambition. Full-blown mania will make a person feel elated, but perhaps also irritable, frustrated, and even disconnected from reality.

**Cause**

Mania is a complex neurophysiological phenomenon. Predisposing factors to develop mania are primarily genetic and are no longer considered to be psychological, although stress triggers to a particular manic episode may include significant psychological and social conflicts. The primary trigger for (and the primary symptom of) acute mania is sleep deprivation. Social problems, medications, or illness may initiate manic hyperarousal but genetic predisposition or brain illnesses are most likely to be the main causations for classic and persistent manic symptoms. Some medications, including all stimulants, may mimic manic symptoms but differ substantially in duration and intensity compared with true manic episodes. The primary mediator of all mood disease is the brain's limbic system. A full description of the cause of mania is complex and should be referenced elsewhere.

Some medications may cause symptoms that mimic mania. Some medications may trigger a manic episode through hyperarousal of the limbic system and subsequent sleep deprivation. These may include: amphetamines and other stimulants (Provigil, Nuvigil, Adipex), caffeine (caffeine/taurine energy drinks), cocaine and various illegal drugs, serotonin reuptake inhibitors (SSRI, SNRI), tricyclic compounds (TCA, excluding carbamazepine), steroid medications (Prednisone, oral cortisone), serotonin agonists, dopamine agonists (Mirapex, Sinemet), and several other groups of medicines. One common over the counter medication group that can be stimulating in large doses is cough and cold medications that contain agents meant to stimulate blood vessels which shrink nasal mucosa thereby enlarging space for nasal air flow (decongestants).
For example, Phenylpropanolamine (PPA) is a sympathomimetic drug similar in structure to amphetamine which was formerly present in over 130 medications, primarily decongestants, cough/cold remedies, and anorectic agents.

A report on PPA, from the Dept. of Psychiatry, F. Edward Hebert School of Medicine, Uniformed Services University of the Health Sciences, Bethesda, Maryland. Pharmacopsychiatry 1988 stated:

We have reviewed 37 cases (published in North America and Europe since 1960) that received diagnoses of acute mania, paranoid schizophrenia, and organic psychosis and that were attributed to PPA product ingestion. Of the 27 North American case reports, more reactions followed the ingestion of combination products than preparations containing PPA alone; more occurred after ingestion of over-the-counter products than those obtained by prescription or on-the-street; and more of the cases followed ingestion of recommended doses rather than overdoses.

Failure to recognize PPA as an etiological agent in the onset of symptoms usually led to a diagnosis of schizophrenia or mania, lengthy hospitalization, and treatment with substantial doses of neuroleptics or lithium.

PPA is no longer available in any medication in the United States as of the year 2000.

**Treatment**

Before beginning treatment for mania, careful differential diagnosis must be performed to rule out non-psychiatric causes.

Acute mania in bipolar disorder is typically treated with mood stabilizers and/or antipsychotic medication. Note that these treatments need to be prescribed and monitored carefully to avoid harmful side-effects such as neuroleptic malignant syndrome with the antipsychotic medications. It may be necessary to temporarily admit the patient involuntarily until the patient is stabilized. Antipsychotics and mood stabilizers help stabilize mood of those with mania or depression. They work by blocking the receptor for the neurotransmitter dopamine and allowing serotonin to still work, but in diminished capacity.

When the manic behaviours have gone, long-term treatment then focuses on prophylactic treatment to try to stabilize the patient’s mood, typically through a combination of pharmacotherapy and psychotherapy.

Lithium is the classic mood stabilizer to prevent further manic and depressive episodes. Anticonvulsants such as valproic acid and carbamazepine are also used for prophylaxis. More recent drug solutions include lamotrigine. Clonazepam (Rivotril, Ravotril or Rivatril) is also used.
Verapamil, a calcium-channel blocker, is useful in the treatment of hypomania and in those cases where lithium and mood stabilizers are contraindicated or ineffective. Verapamil is effective for both short-term and long-term treatment.

**Medications**

The biological mechanism by which mania occurs is not yet known. One hypothesised cause of mania (among others) is that the amount of the neurotransmitter serotonin in the temporal lobe may be excessively high. Dopamine, norepinephrine, glutamate and gamma-aminobutyric acid also appear to play important roles. Imaging studies have shown that the left amygdala is more active in women who are manic and the orbitofrontal cortex is less active.

Antidepressant monotherapy is not recommended for the treatment of depression in patients with bipolar disorders I or II, and no benefit has been demonstrated by combining antidepressants with mood stabilizers in these patients.

**Society and culture**

In Electroboy: A Memoir of Mania by Andy Behrman, he describes his experience of mania as "the most perfect prescription glasses with which to see the world...life appears in front of you like an oversized movie screen". Behrman indicates early in his memoir that he sees himself not as a person suffering from an uncontrollable disabling illness, but as a director of the movie that is his vivid and emotionally alive life. "When I'm manic, I'm so awake and alert, that my eyelashes fluttering on the pillow sound like thunder".

**Hypomania**

Hypomania (literally, "below mania") is a mood state characterized by persistent and pervasive elevated (euphoric) or irritable mood, as well as thoughts and behaviors that are consistent with such a mood state.

**Presentation**

Individuals in a hypomanic state have a decreased need for sleep, are extremely outgoing and competitive, and have a great deal of energy. However, unlike with full mania, those with hypomanic systems are fully functioning, and are often actually more productive than usual.

**Distinctive markers**

Specifically, hypomania is distinguished from mania by the absence of psychotic symptoms and grandiosity, and by its lesser degree of impact on functioning. Hypomania is a feature of bipolar II disorder and cyclothymia, but can also occur in schizoaffective disorder. Hypomania is also a feature of bipolar I disorder as it arises in sequential procession as the
mood disorder fluctuates between normal mood and mania. Hypomania can occur when moods progress downwards from a manic mood state to a normal mood. Hypomania is sometimes credited with increasing creativity and productive energy. A significant number of people with creative talents have reportedly experienced hypomania or other symptoms of bipolar disorder and attribute their success to it. Classic symptoms of hypomania include mild euphoria, a flood of ideas, endless energy, and a desire and drive for success. A lesser form of hypomania is called hyperthymia.

Definitions

Hypomania is also a side effect of numerous medications, often—though not always—those used in psychopharmacotherapy. Patients suffering from severe depression who experience hypomania as a side effect of (for example) antidepressants, may prove to have a form of bipolar disorder that has previously gone unrecognized. However, drug-induced hypomania is not invariably indicative of bipolar affective disorders. The difference between uni- and bi-polar disorders is essential for analysis of switches (mood changes). Consequently, it is important for researchers and mental health professionals to distinguish drug-induced hypomania in bipolar patients from drug-induced hypomania in unipolar (non-bipolar) depressives. Nevertheless if antidepressants trigger the first episode of hypomania, it is strongly suggestive of an underlying diagnosis of Bipolar Disorder, particularly if the manic symptoms (mild, moderate or severe) last for a lengthy period of time after they start. In cases of true drug-induced hypomania, cessation of the antidepressant or whichever drug has triggered this mood state - for example steroid therapy or stimulants such as amphetamine - usually causes a fairly swift return to normal mood. It is far less likely to be a side effect in those with pure Clinical Unipolar Depression, unless for example tricyclic antidepressants are given in very high doses. SSRIs are less likely to trigger manic symptoms except in those individuals where there is an underlying Bipolar Disorder, particularly if administered without a mood stabilizer.

Occurrence

Often in those who have experienced their first episode of hypomania (which is a level of mild to moderate mania) - generally without psychotic features - there will have been a long or recent history of depression prior to the emergence of manic symptoms, and commonly this surfaces in the mid to late teens. Due to this being an emotionally charged time, it is not unusual for mood swings to be passed off as hormonal or teenage ups and downs and for a diagnosis of Bipolar Disorder to be missed until there is evidence of an obvious manic/hypomaniac phase.

Hypomania may also occur as a side effect of pharmaceuticals prescribed for conditions/diseases other than psychological states or mood disorders. In those instances, as in cases of drug-induced hypomanic episodes in unipolar depressives, the hypomania can almost invariably be eliminated by lowering medication dosage, withdrawing the drug entirely, or changing to a different medication if discontinuation of treatment is not possible.
Some, such as Johns Hopkins psychologist John Gartner, argue that hypomania is better understood as a stable non-pathological temperament rather than an episode of mental illness. The DSM however clearly defines hypomania as an aberrant state, not a stable trait.

**Symptomatic recognition**

The DSM-IV-TR defines a hypomanic episode as including, over the course of at least four days, elevated mood plus three of the following symptoms OR irritable mood plus four of the following symptoms:

- pressured speech
- inflated self-esteem or grandiosity
- decreased need for sleep
- flight of ideas or the subjective experience that thoughts are racing
- easy distractibility and attention-deficit similar to attention deficit hyperactivity disorder
- increase in psychomotor agitation
- involvement in pleasurable activities that may have a high potential for negative psycho-social or physical consequences (e.g., the person engages in unrestrained buying sprees, sexual indiscretions, reckless driving, or foolish business investments).

**Possible benefits**

Some commentators believe that hypomania actually has an evolutionary advantage. People with hypomania are generally perceived as being energetic, euphoric, visionary, overflowing with new ideas, and sometimes over-confident and very charismatic, yet—unlike those with full-blown mania—are sufficiently capable of coherent thought and action to participate in everyday activities. Like mania, there seems to be a significant correlation between hypomania and creativity. A person in the state of hypomania might be immune to fear and doubt and have little social and sexual inhibition. People experiencing hypomania are often the "life of the party." They may talk to strangers easily, offer solutions to problems, and find pleasure in small activities. Such advantages may render them unwilling to submit to treatment, especially when disadvantages are minimal.

**Relationship with disorders**

Cyclothymia is a condition of continued mood fluctuations between hypomania and depressive symptoms that do not meet the criteria for a major depressive episode. These are often interspersed with periods of normal moods.

When a patient presents with a history of one or more hypomanic episodes and one or more depressive episodes that meet the criteria for a major depressive episode, bipolar II disorder is diagnosed.
If left untreated, hypomania can transition into mania and sometimes psychosis, in which case, bipolar I disorder is often diagnosed. (See also, Kindling model)

**Treatment**

Medications typically prescribed for hypomania include mood stabilizers such as valproic acid and lithium carbonate as well as atypical antipsychotics such as olanzapine and quetiapine.

**Notable individuals with hypomaniac symptoms**

John Gartner's book The Hypomaniac Edge claims notable people including Christopher Columbus, Alexander Hamilton, Andrew Carnegie, Howard Zinn and Louis B. Mayer owe their innovation and drive, as well as their eccentricities, to hypomaniac temperaments. Gartner suggests that the constructive behaviors associated with hypomania may contribute to bipolar disorder's evolutionary survival. Critics charge that Gartner vastly overstates his case, however.

**Mixed state (psychiatry)**

In the context of mental disorder, a mixed state (also known as dysphoric mania, agitated depression, or a mixed episode) is a condition during which symptoms of mania and depression occur simultaneously (e.g., agitation, anxiety, fatigue, guilt, impulsiveness, irritability, morbid or suicidal ideation, panic, paranoia, pressured speech and rage). Typical examples include tearfulness during a manic episode or racing thoughts during a depressive episode. One may also feel incredibly frustrated or be prone to fits of rage in this state, since one may feel like a failure and at the same time have a flight of ideas. Mixed states are often the most dangerous period of mood disorders, during which susceptibility to substance abuse, panic disorder, commission of violence, suicide attempts, and other complications increase greatly.

**Diagnostic criteria**

As affirmed by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), a mixed state must meet the criteria for a major depressive episode and a manic episode nearly every day for at least one week. However, mixed episodes rarely conform to these qualifications; they may be described more practically as any combination of depressive and manic symptoms. The Merck Manual of Diagnosis and Therapy (MMDT) splits the DSM-IV diagnosis into dysphoric mania and an agitated depression state.

A dysphoric mania consists of a manic episode with depressive symptoms. Increased energy and some form of anger, from irritability to full blown rage, are the most common symptoms (MMDT). Symptoms may also include auditory hallucinations, confusion, insomnia, persecutory delusions, racing thoughts, restlessness, and suicidal ideation.
Alcohol, drug abuse, and some antidepressant drugs may trigger dysphoric mania in susceptible individuals.

An agitated depression is a "major depressive [episode] with superimposed hypomanic symptoms". Mixed episodes in which major depression is the primary state, concurrent with atypical manic features have been described. A study by Goodwin and Ghaemi (2003) reported manic symptoms in two-thirds of patients with agitated depression, which they suggest calling "mixed-state agitated depression".

**Treatment**

Successful treatment of mixed states require administration of mood stabilizing medication, which may include anticonvulsants such as lamotrigine and valproic acid; atypical antipsychotics such as olanzapine, aripiprazole, and ziprasidone; or first-generation antipsychotics such as haloperidol. There is question of lithium's efficacy for treatment of mixed states due to conflicting conclusions drawn from various trials and research. Mood stabilizers work to reduce the manic symptoms associated with the mixed state, but they are not considered particularly effective for improving concurrent depressive symptoms.

**Associated features of bipolar disorder**

The associated features of bipolar disorder are clinical phenomena that often accompany bipolar disorder (BD) but are not part of the diagnostic criteria for the disorder. There are several childhood precursors in children who later receive a diagnosis of bipolar disorder. They may show subtle early traits such as mood abnormalities, full major depressive episodes, and ADHD. BD is also accompanied by changes in cognition processes and abilities. This include reduced attentional and executive capabilities and impaired memory. How the individual processes the world also depends on the phase of the disorder, with differential characteristics between the manic, hypomanic and depressive states. Some studies have found a significant association between bipolar disorder and creativity.

**Childhood precursors**

Some limited long-term studies indicate that children who later receive a diagnosis of bipolar disorder may show subtle early traits such as subthreshold cyclical mood abnormalities, full major depressive episodes, and possibly ADHD with mood fluctuation. There may be hypersensitivity and irritability. There is some disagreement whether the experiences are necessarily fluctuating or may be chronic. Having parents with bipolar disorder is associated with increased risk of psychiatric disorders. A history of stimulant use in childhood is found in high numbers of bipolar patients and has been found to cause an earlier onset of bipolar disorder and a worse clinical course, independent of attention deficit hyperactivity disorder.
Cognitive functioning

Reviews have indicated that most individuals diagnosed with bipolar disorder, but who are euthymic (not experiencing major depression or mania), do not show neuropsychological deficits on most tests. Meta-analyses have indicated, by averaging the variable findings of many studies, cognitive deficits on some measures of sustained attention, executive function and verbal memory, in terms of group averages. On some tests, functioning is superior; however, and sub-threshold mood states and psychiatric medications may account for some deficits. A 2010 study found that "excellent performance" at school at age 15–16 was associated in males with a higher rate of developing bipolar disorder, but so was the poorest performance. A 2005 study of young adult males found that poor performance on visuospatial tasks was associated with a higher rate of developing bipolar disorder, but so was high performance in arithmetic reasoning.

Psychological studies of bipolar disorder have examined the development of a wide range of both the core symptoms of psychomotor activation and related clusterings of depression/anxiety, increased hedonic tone, irritability/aggression and sometimes psychosis. The existing evidence has been described as patchy in terms of quality but converging in a consistent manner. The findings suggest that the period leading up to mania is often characterized by depression and anxiety at first, with isolated sub-clinical symptoms of mania such as increased energy and racing thoughts. The latter increase and lead to increased activity levels, the more so if there is disruption in circadian rhythms or goal attainment events. There is some indication that once mania has begun to develop, social stressors, including criticism from significant others, can further contribute. There are also indications that individuals may hold certain beliefs about themselves, their internal states, and their social world (including striving to meet high standards despite it causing distress) that may make them vulnerable during changing mood states in the face of relevant life events. In addition, subtle frontal-temporal and subcortical difficulties in some individuals, related to planning, emotional regulation and attentional control, may play a role. Symptoms are often subthreshold and likely continuous with normal experience. Once (hypo)mania has developed, there is an overall increase in activation levels and impulsivity. Negative social reactions or advice may be taken less notice of, and a person may be more caught up in their own thoughts and interpretations, often along a theme of feeling criticised. There is some suggestion that the mood variation in bipolar disorder may not be cyclical as often assumed, nor completely random, but results from a complex interaction between internal and external variables unfolding over time; there is mixed evidence as to whether relevant life events are found more often in early than later episodes. Many sufferers report inexplicably varied cyclical patterns, however.

A series of authors have described mania or hypomania as being related to a high motivation to achieve, ambitious goal-setting, and sometimes high achievement. One study indicated that the pursuit of goals, encouraged by sometimes achieving them, can become emotionally dysregulated and involve the development of mania. Individuals may have low self-esteem and difficulties in social adjustment, however, and by definition there are periods of depression with difficulty in motivation and functioning.
Bipolar disorder has been associated with people involved in the arts but it is an ongoing question as to whether many creative geniuses had bipolar disorder. Some studies have found a significant association between bipolar disorder and creativity, although it is unclear in which direction the cause lies or whether both conditions are caused by a third unknown factor; temperament has been hypothesized to be one such factor.

**Self-medication**

Often bipolar individuals are subject to self-medication with non-prescribed drugs such as alcohol, tobacco and other recreational drugs.

There is some evidence that, although bipolar patients in general do not appear to smoke significantly more than other people, the subset of bipolar patients with a history of psychosis may smoke more heavily than the general population.

**Creativity and mental illness**

There is anecdotal evidence for a relationship between creativity and psychosis, particularly schizophrenia. James Joyce had a daughter with schizophrenia and had many schizotypal traits. Albert Einstein had a son with schizophrenia and was also somewhat schizotypal and eccentric. Bertrand Russell had many family members with schizophrenia or psychosis: his aunt, uncle, son and grand-daughter. Psychotic individuals are said to display a capacity to see the world in a novel and original way, literally, to see things that others cannot.

**History**

The association between bipolar disorder and creativity first appeared in literature in the 1970s, but the idea of a link between "madness" and "genius" is much older, dating back at least to the time of Aristotle. The Ancient Greeks believed that creativity came from the gods, and in particular the Muses: the nine daughters of Zeus, the god of arts and sciences. The idea of a complete work of art emerging without conscious thought or effort was reinforced by the views of the Romantic era. It has been proposed that there's a particular link in the case of bipolar disorder, whereas major depressive disorder appears to be significantly more common among playwrights, novelists, biographers, and artists.

**Positive mood, mental illness and creativity**

Mood-creativity research reveals that people are most creative when they're in a positive mood and that mental illnesses such as depression or schizophrenia actually decrease creativity. People who have worked in the field of arts throughout the history have had problems with poverty, persecution, social alienation, psychological trauma, substance abuse, high stress and other such environmental factors which are associated with developing and perhaps causing mental illnesses. It is thus likely that when creativity itself is associated with positive moods, happiness, and mental health, pursuing a career in the
arts may bring problems with stressful environment and income. Other factors such as the centuries-old stereotype of the suffering of a mad artist help to fuel the link by putting expectations on how an artist should act. It also helps the field to be more attractive to those with mental disorders.

Creativity and bipolar disorder

Bipolar disorder

There is a range of types of bipolar disorder. Individuals with Bipolar I Disorder experience severe episodes of mania and depression with periods of wellness between episodes. The severity of the manic episodes can mean that the person is seriously disabled and unable to express the heightened perceptions and flight of thoughts and ideas in a practical way. Individuals with Bipolar II Disorder experience milder periods of hypomania during which the flight of ideas, faster thought processes and ability to take in more information can be converted to art, poetry or design.

Creativity and psychopathology

Many famous historical figures gifted with creative talents may have been affected by bipolar disorder. Ludwig van Beethoven, Virginia Woolf, Isaac Newton, and Robert Schumann are some people whose lives have been researched to discover signs of mood disorder. In many instances, creativity and psychopathology share some common traits, such as a tendency for "thinking outside the box," flights of ideas, speeding up of thoughts and heightened perception of visual, auditory and somatic stimuli.

Creativity and the emotions of bipolar disorder

Many people with bipolar disorder may feel powerful emotions during both depressive and manic phases, potentially aiding in creativity. Because (hypo)mania decreases social inhibition, performers are often daring and bold. As a consequence, creators commonly exhibit characteristics often associated with mental illness. The frequency and intensity of these symptoms appear to vary according to the magnitude and domain of creative achievement. At the same time, these symptoms are not equivalent to the full-blown psychopathology of a clinical manic episode which, by definition, entails significant impairment.

Posthumous diagnosis

Some creative people have been posthumously diagnosed as suffering from bipolar or unipolar disorder based on biographies, letters, correspondence, contemporaneous accounts, or other anecdotal material, most notably in Kay Redfield Jamison’s book Touched with Fire: Manic-Depressive Illness and the Artistic Temperament. Touched With Fire presents the argument that bipolar disorder, and affective disorders more generally, may be found in a disproportionate number of people in creative professions such as actors, artists, comedians, musicians, authors, performers and poets.
Positive correlation

Several recent clinical studies have also suggested that there is a positive correlation between creativity and bipolar disorder, although the relationship between the two is unclear. Temperament may be an intervening variable.

The 2005 Stanford study

A 2005 study at the Stanford University School of Medicine measured creativity by showing children figures of varying complexity and symmetry and asking whether they like or dislike them. The study showed for the first time that a sample of children who either have or are at high risk for bipolar disorder tend to dislike simple or symmetric symbols more. Children with bipolar parents who were not bipolar themselves also scored higher dislike scores.

The 'Sylvia Plath' effect

Questions swirl around a supposed link between creativity and mental illness.

By DEBORAH SMITH BAILEY

Monitor Staff

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Popular culture has long stereotyped poets as depressed and creative scientists as mad. In fact, the idea of a link between creativity and mental illness goes back to the time of Aristotle, when he wrote that eminent philosophers, politicians, poets and artists all have tendencies toward "melancholia."

Indeed, there are numerous examples of famous creators--writers like Virginia Woolf, painters like Vincent Van Gogh, composers like Robert Schumann--who have been highly successful but had or are suspected to have had a mental illness.

Some studies have backed up this notion, suggesting that writers, artists and others are more likely to have a mental illness and that people with certain mental illnesses, such as depression and mood disorders, appear somewhat more likely to be creative. While some researchers have found that creative people are slightly more at-risk, others have found more grave connections, such as that they are 30 percent more likely to have bipolar disorder.

However, such research is often fraught with methodological problems, including selection bias, controls that are not blinded, reliance on biographies that might play up mental illness, retrospective designs and unclear definitions of creativity. And considering that not
all studies have found a link between creativity and mental illness, the jury is still out on the specific nature of the relationship, says psychologist and creativity researcher James Kaufman, PhD, of California State University, San Bernardino.

Still, the findings raise interesting questions about the relationship between mental illness and creativity, including:

Does creativity cause mental illness? There isn’t a link between mental illness and the actual process of creating, says psychiatrist Albert Rothenberg, MD, of Harvard Medical School, who has studied Nobel laureates, Pulitzer Prize winners and other highly creative individuals. Rather, he argues that mental illnesses such as anxiety, thought disorder and depression disrupt the cognitive and emotional processes necessary for successful creativity.

In fact, in his book, "Creativity and Madness: New Findings and Old Stereotypes" (Johns Hopkins University Press, 1990), Rothenberg proposes that highly creative people do better when they are treated for their mental illnesses.

"That doesn’t mean people who create haven’t often had mental illnesses," he adds, but that their subject matter and the field they are in perhaps have more bearing on their mental health than creativity itself.

Does the type of creativity matter? Creative people in the artistic professions are more likely to have a mental illness than those in less artistic professions, such as science and business, according to research by Arnold M. Ludwig, MD, in his book, "The Price of Greatness" (Guilford, 1995).

Moreover, in a more recent retrospective study of 1,629 writers, Kaufman found that poets--and in particular female poets --were more likely than fiction writers, nonfiction writers and playwrights to have signs of mental illness, such as suicide attempts or psychiatric hospitalizations.

In a second analysis of 520 eminent American women, he again found that poets were more likely to have mental illnesses and to experience personal tragedy than eminent journalists, visual artists, politicians and actresses--a finding Kaufman has dubbed "the Sylvia Plath effect" after the noted poet who had depression and eventually committed suicide. The findings appear in The Journal of Creative Behavior (Vol. 35, No. 1).

Are creative people’s motivations a factor? Kaufman and psychologist John Baer, PhD, of Rider University, theorize in the Review of General Psychology (Vol. 6, No. 3) that creative people--specifically, eminent female poets--may be more prone to mental illness if they are more vulnerable to extrinsic motivational constraints, such as interpersonal relationships.

Valuing such external factors may harm poets’ mental health, they speculate, because high levels of creativity require people to "defy the crowd" and ignore what other people think.
That means eminent writing could produce more stress--leading to a higher incidence of mental illness.

Could the stigma of mental illness be a factor? "In the fields of art and literature and music, there is much more toleration of mental illness than there is in the rest of society," explains Rothenberg. That might allow people with mental illnesses to climb the ranks of poetry in a way they couldn't have in business.

How does creative writing interact with mental illness? In several studies, University of Texas at Austin psychologist James Pennebaker, PhD, has found positive health and mental health benefits from writing--but only when the writer crafts a narrative or makes connections between thought and feelings. Kaufman theorizes that poets may not garner the same benefits from writing that other writers do because poems seldom form a narrative.

However, Pennebaker cautions that there is no data yet that proves that poetry writing isn't beneficial. "It's very possible that writing poetry may have kept Sylvia Plath alive longer than she would have," he says. "One of the counterarguments is that being in poetry is a real tough way to make a living. There are very few jobs that have a higher rejection rate."

**Are Genius and Madness Related?**

*Contemporary Answers to an Ancient Question*

By Dean Keith Simonton, Ph.D. | May 31, 2005

The idea that creativity and psychopathology are somehow linked goes way back to antiquity--to the time of Aristotle. Centuries later, this belief was developed and expanded by various psychiatrists, psychoanalysts and psychologists. For instance, Cesare Lombroso, M.D., argued toward the end of the 19th century that genius and madness were closely connected manifestations of an underlying degenerative neurological disorder. To be sure, this idea has not gone without challenge. On the contrary, humanistic psychologists were inclined to associate creativity with mental health. Nevertheless, the prevailing view appears to be that psychopathology and creativity are positively associated.

But what is the scientific evidence supporting this hypothesized association? And what does this evidence suggest is the basis for the relationship?

**Empirical Evidence**

Scientific data addressing this issue come from three main sources: historiometric, psychiatric and psychometric. Although each source has distinct methodological problems, the findings all converge on the same general conclusions.
Historiometric research. In this approach, historical data are subjected to objective and quantitative analyses. In particular, the biographies of eminent creators are systematically analyzed to discern the presence of symptoms associated with various psychopathological syndromes. Such historiometric inquiries lead to four conclusions.

First, the rate and intensity of psychopathological symptoms appear to be higher among eminent creators than in the general population (Ellis, 1926; Raskin, 1936). Although the differential depends on the specific definition used, a reasonable estimate is that highly creative individuals are about twice as likely to experience some mental disorder as otherwise comparable noncreative individuals (Ludwig, 1995). Depression seems to be the most common symptom, along with the correlates of alcohol/drug information on alcoholism and suicide (Goertzel et al., 1978; Ludwig, 1990; Post, 1996).

Second, on average, the more eminent the creator, the higher is the expected rate and intensity of the psychopathological symptoms (Ludwig, 1995).

Third, the rate and intensity of symptoms varies according to the specific domain of creativity (Ludwig, 1992; Post, 1994). For example, psychopathology is higher among artistic creators than among scientific creators (Post, 1994; Raskin, 1936). Thus, according to one study, 87% of famous poets experienced psychopathology whereas only 28% of the eminent scientists did so, a figure close to the population baseline (Ludwig, 1995).

Fourth, those family lines that produce the most eminent creators also tend to be characterized by a higher rate and intensity of psychopathological symptoms (Jamison, 1993; Juda, 1949; Karlsson, 1970).

Hence, even though there is some evidence that the lifestyle of creative activity can have adverse consequences for mental health (Schaller, 1997), it remains the case that there may be a common genetic component to both creativity and psychopathology (Ludwig, 1995).

Psychiatric research. This type of evidence depends on the incidence of clinical diagnosis and therapeutic treatment in samples of contemporary creators. Hence, the research does not require retrospective analysis as in historiometric research, and the assessment of psychopathology reflects modern standards. In any case, psychiatric studies also seem to find higher rate and intensity of symptoms among distinguished creators, especially those engaged in artistic creativity (Andreasen and Canter, 1974; Jamison, 1989). Once more, depression, alcoholism and suicide appear to be the most common indicators. Furthermore, the evidence suggests that creativity and mental illness run in the same family lines (Andreasen, 1987; McNeil, 1971; Myerson and Boyle, 1941).

Psychometric research. Here, standard assessment instruments are applied to contemporary creators. The sampled creators either vary substantially in creative achievement or else they are compared to a control group of noncreative participants who are otherwise comparable. The psychometric measures include the Minnesota Multiphasic Personality Inventory (MMPI) and the Eysenck Personality Questionnaire (EPQ) (Gough,
1953). In general, highly creative individuals score above normal level on several dimensions associated with psychopathology (Barron, 1963). For instance, creativity is positively correlated with psychoticism scores on the EPQ (Eysenck, 1995, 1994). In addition, the higher the level of creativity displayed, the higher the scores tend to be on the clinical scales. Nonetheless, artistic creators still have more elevated scores than do scientific creators (Simonton, 2004).

The days of getting eminent creators to take the MMPI or EPQ are long gone, with the classic studies done in the 1950s and 60s. Eysenck’s work is a research integration of work published much earlier. More recent work tends to focus on specific components, such as the research on latent inhibition discussed later. Also, the psychometric literature provides some unique empirical results that can shed some light on the specific nature of the relationship between creativity and psychopathology. The following two sets of findings stand out.

First, although highly creative individuals tend to exhibit elevated scores on certain psychopathological symptoms, their scores are seldom so high as to represent bona fide psychopathology. Instead, the scores lie somewhere between the normal and abnormal ranges (Barron, 1963; Eysenck, 1995). For example, although successful writers score higher than normals on most clinical scales of the MMPI, and highly creative writers score higher still, scores for both groups remain below those received by individuals who are psychotic (Figure). At these moderate levels, the individual will possess traits that can actually be considered adaptive from the standpoint of creative behavior. For instance, higher than average scores on psychoticism are associated with independence and nonconformity, features that lend support to innovative activities (Eysenck, 1995). In addition, elevated scores on psychoticism are associated with the capacity for defocused attention (e.g., reduced negative priming and latent inhibition), thereby enabling ideas to enter the mind that would normally be filtered out during information processing (Eysenck, 1993). This less restrictive mode of information processing is also associated with openness to experience, a cognitive inclination that is positively associated with creativity (Peterson and Carson, 2000; Peterson et al., 2002).

Second, creative individuals score high on other characteristics that would seem to dampen the effects of any psychopathological symptoms. In particular, creators display high levels of ego strength and self-sufficiency (Barron, 1963; Cattell and Butcher, 1968). Accordingly, they can exert meta-cognitive control over their symptoms, taking advantage of bizarre thoughts, rather than having the bizarre thoughts take advantage of them. Furthermore, the capacity to exploit unusual ideas is supported by general intelligence. Although intelligence is not correlated with creativity in the upper levels of the intelligence distribution, a certain minimal level of intelligence is required for exceptional creativity (Simonton, 2000). That threshold level is in the gifted range, roughly equivalent to an IQ 120. Creators do not necessarily have genius-grade IQs, but they do have sufficient information processing power to select, develop, elaborate and refine original ideas into creative contributions.
Theoretical Interpretation

Do these results imply that creativity and psychopathology are intimately connected? Are genius and madness tantamount to the same thing? The answer to the first question is affirmative, but the response to the second is negative. The affirmation comes from the fact that various indicators of mental health appear to be negatively associated with creative achievement. This fact is demonstrated by historiometric, psychiatric and psychometric sources. The negation emerges from the equally crucial reality that few creative individuals can be considered truly mentally ill. Indeed, outright psychopathology usually inhibits rather than helps creative expression.

Even more significant is the fact that a very large proportion of creators exhibit no pathological symptoms, at least not to any measurable degree. Hence, psychopathology is by no means a sine qua non of creativity. Instead, it is probably more accurate to say that creativity shares certain cognitive and dispositional traits with specific symptoms, and that the degree of that commonality is contingent on the level and type of creativity that an individual displays. To be more specific, the relationship can be expressed as follows.

In general, creativity requires the cognitive ability and the dispositional willingness to "think outside the box"; to explore novel, unconventional and even odd possibilities; to be open to serendipitous events and fortuitous results; and to imagine the implausible or consider the unlikely. From this requirement arises the need for creators to have such traits as defocused attention, divergent thinking, openness to experience, independence and nonconformity. Let us call this complex configuration of traits the "creativity cluster."

The higher the level of creativity displayed, the higher the likelihood that the individual manifests this cluster. In addition, some domains require this cluster more than others do. For instance, scientific creativity tends to be more constrained by logic and fact than artistic creativity. Accordingly, this creativity cluster of attributes will be more apparent in artists than in scientists (Simonton, 2004). However, there will be some differences even with each of these general domains. For example, artists operating in formal, classical or academic styles will operate under more constraints than artists working in more expressive, subjective or romantic styles (Ludwig, 1998). The extent to which they exhibit the creativity cluster will reflect this stylistic contrast.

Because some psychopathological symptoms correlate with several of the characteristics making up the creativity cluster, moderate amounts of these symptoms will be positively associated with creative behavior. Moreover, more creative individuals will display these traits to a higher degree. Creators operating in less-constrained domains will also exhibit these symptoms to a greater extent.

To the extent that these symptoms have a genetic foundation, creativity can be said to be partly biologically determined. Nevertheless, psychopathological symptoms are not the only possible source for the cognitive and dispositional attributes underlying creativity. Many environmental experiences and conditions can also nurture the development of the same cluster. Although some of these developmental influences are also associated with
psychopathology, others are not. Thus, on the one hand, creative development is frequently associated with traumatic experiences in childhood or adolescence, experiences that may also contribute to depression and suicidal tendencies (Eisenstadt, 1978; Goertzel and Goertzel, 1962). On the other hand, development is also linked to an enriched and diverse intellectual and cultural environment, an environment that is neutral with respect to psychopathology (Simonton, 1999). Growing up under such conditions fosters the emergence of many cognitive and dispositional traits that define the creativity cluster.

**Implications**

The theoretical interpretation just provided holds that creativity and psychopathology share a common set of traits. As a consequence, creators will commonly exhibit symptoms often associated with mental illness. The frequency and intensity of these symptoms will vary according to the magnitude and domain of creative achievement. At the same time, these symptoms are not equivalent to out-and-out psychopathology. Besides the fact that characteristics are normally at subclinical levels, their effects are tempered by positive attributes, such as high ego strength and exceptional intellect. Moreover, many of the relevant components can be nurtured by environmental factors that lessen their dependence on any psycho-pathological inclinations. Taken altogether, this means that creativity is not incompatible with mental and emotional health. This affirmation is reinforced by the existence of numerous creative individuals who display little or no symptoms beyond normal baselines.

As a result, creators should have no fear that therapeutic treatment for disabling mental or emotional disorders would undermine their creative potential. Because the relationships between certain symptoms and creativity are described by curvilinear inverted-U curves, one goal of psychiatric intervention should be to identify the optimum level of functioning and then maintain the creative individual at that level.

Furthermore, treatment can also concentrate on those aspects of the creative personality that have a positive linear association with both creativity and mental health. Examples include ego strength and openness to experience. Although such an intervention clearly requires a delicate balancing act, the task is not by any means impossible. Executed carefully, it should be possible to help clients become more creative and more healthy at the same time.

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Children Of Bipolar Parents Score Higher On Creativity Test, Stanford Study Finds

ScienceDaily (Nov. 9, 2005) — Researchers at the Stanford University School of Medicine have shown for the first time that a sample of children who either have or are at high risk for bipolar disorder score higher on a creativity index than healthy children. The findings add to existing evidence that a link exists between mood disorders and creativity.

The small study, published in the November issue of the Journal of Psychiatric Research, compared creativity test scores of children of healthy parents with the scores of children of bipolar parents. Children with the bipolar parents - even those who were not bipolar themselves - scored higher than the healthy children.

"I think it’s fascinating," said Kiki Chang, MD, assistant professor of psychiatry and behavioral sciences and co-author of the paper. "There is a reason that many people who have bipolar disorder become very successful, and these findings address the positive aspects of having this illness."

Many scientists believe that a relationship exists between creativity and bipolar disorder, which was formerly called manic-depressive illness and is marked by dramatic shifts in a person’s mood, energy and ability to function. Numerous studies have examined this link; several have shown that artists and writers may have two to three times more incidences of psychosis, mood disorders or suicide when compared with people in less creative professions.

Terence Ketter, MD, professor of psychiatry and behavioral sciences and a study co-author, said he became interested in the link between mental illness and creativity after noticing that patients who came through the bipolar clinic, despite having problems, were extraordinarily bright, motivated people who "tended to lead interesting lives." He began a scholarly pursuit of this link and in 2002 published a study that showed healthy artists were more similar in personality to individuals with bipolar disorder (the majority of whom were on medication) than to healthy people in the general population.

Some researchers believe that bipolar disorder or mania, a defining symptom of the disease, causes creative activity. Ketter said he believes that bipolar patients’ creativity stems from their mobilizing energy that results from negative emotion to initiate some sort of solution to their problems. "In this case, discontent is the mother of invention," he said.

The researchers point out that creativity and bipolar may have important genetic components that are transmitted together inter-generationally. There have only been limited studies investigating this; the Stanford study is the first to specifically examine creativity in the offspring of bipolar parents.

During the study, the researchers looked at creative characteristics in 40 bipolar patients and 40 offspring, comparing them with 18 healthy adults and 18 healthy offspring. The
children in the study ranged in age from 10 to 18. Half of the children of bipolar patients also had bipolar disorder; the other half had attention deficit hyperactivity disorder or ADHD, which appears to be an early sign of bipolar disorder in offspring of parents with the condition. The majority of participants with bipolar or ADHD were on medication.

The researchers included children with ADHD so they could study creativity before the onset of full bipolar disorder. "We wanted to see whether having a manic episode is necessary for this sort of creativity," said Chang, who also directs the Pediatric Bipolar Disorders Program at Lucile Packard Children’s Hospital.

Study participants were given psychiatric evaluations and then completed the Barron-Welsh Art Scale, or BWAS, a test that seeks to provide an objective measure of creativity. The scoring is based on "like" and "dislike" responses to figures of varying complexity and symmetry; past studies suggest that creative people tend to dislike the simple and symmetric symbols.

The researchers found that the bipolar parents had 120 percent higher BWAS "dislike" scores than the healthy parents. The children with bipolar and the children with ADHD had, respectively, 107 and 91 percent higher BWAS dislike scores than the healthy children.

"The results of this study support an association between bipolar disease and creativity and contribute to a better understanding of possible mechanisms of transmission of creativity in families with genetic susceptibility for bipolar disease," the researchers wrote in their paper.

The researchers had hypothesized that the scores of children with ADHD would differ significantly from the scores of bipolar children so they were surprised when the scores did not. Chang said this indicates that mania is not what is fueling the creativity. "The kids with ADHD who hadn’t been manic yet still had very high levels of creativity," he said.

The researchers also found a link between the length of a bipolar child’s illness and creativity: the longer a child was sick or manic, the lower the BWAS dislike score. It makes sense, Chang said, that this illness could, over time, erode one’s creativity. "After awhile you aren’t able to function and you can’t access your creativity," he explained.

BWAS dislike scores tend to decrease with age even in healthy individuals, so more research is needed, Ketter said. Further studies are also needed to assess the role of genetic and environmental factors in creativity and bipolar, he added. The team plans to next examine whether the degree of creativity in parents correlates with the degree of creativity in their children.

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Treatment of bipolar disorder

The emphasis of the treatment of bipolar disorder is on effective management of the long-term course of the illness, which can involve treatment of emergent symptoms. Treatment methods include pharmacological and psychological techniques.

A variety of medications are used to treat bipolar disorder; most people with bipolar disorder require combinations of medications.

Principles

The primary treatment for bipolar disorder consists of medications called mood stabilizers, which are used to prevent or control episodes of mania or depression. Proven mood stabilizers include lithium, and anticonvulsants such as Valproic acid (Depakote), carbamazepine (Tegretol), and lamotrigine (Lamictal). The atypical antipsychotics are all FDA approved for acute treatment of mania; these include quetiapine (Seroquel), olanzapine (Zyprexa), and risperidone (Risperdal). Generally speaking, mood stabilizers are more effective at treating or preventing manic episodes; however, some medications (i.e. lamotrigine, quetiapine) have been shown to be effective for the treatment of bipolar depression. In particular, lamotrigine is often the first-line treatment for bipolar II, where depression, rather than mania, tends to be the dominant symptom. Antidepressants are often prescribed to treat depressive symptoms, however the role of antidepressants remains controversial because antidepressants have been known to induce mania, and some researchers believe that they can worsen the course of the disease (see below). Medications are not the only available treatment for bipolar disorder; other treatments include omega 3 fatty acids, therapy such as psychotherapy and cognitive behavioral therapy, and exercise. Stress, alcohol abuse, and drug abuse can all cause bipolar disorder to worsen, and so effective treatment may require management of stress and moderation or elimination of alcohol and drug use. The goal of treatment is not to cure the disorder but rather to control the symptoms and the course of the disorder. Generally speaking, maintenance treatment of bipolar disorder continues long after symptoms have been brought under control.

Drugs used for bipolar disorder sometimes have significant side effects, which vary from drug to drug. Lithium may be associated with gastrointestinal upset (e.g. nausea, diarrhea), memory problems, weight gain and other side effects. Higher doses equal more side effects, but lower doses (within the therapeutic window) have little to no side effects. Anticonvulsant medications commonly cause sedation, weight gain, electrolyte disturbances, or other side effects. If one cannot tolerate one of the anticonvulsants, it's advisable to try another anticonvulsant. Two or more anticonvulsants in combination can often result in a lower effective dose of each and lower side effects. The side effect profile of the atypical antipsychotics vary widely between agents. Generally speaking, the most common side effects of the atypicals are sedation and metabolic disturbances (i.e. weight gain, dyslipidemia, hyperglycemia). Atypical antipsychotics may also cause extrapyramidal side effects and restlessness. Atypical antipsychotics also carry a risk of causing tardive
dyskinesia; however, the risk with the newer atypical agents is much less than the risk associated with older antipsychotics (e.g. haloperidol). The risk of TD is thought to be proportionate to the duration of neuroleptic/antipsychotic use (roughly 5% per year in non-elderly patients treated with older antipsychotics). Patients and physicians need to be careful to watch for symptoms of this side effect carefully so that an antipsychotic can be reduced in dosage, or changed to another medication, before the condition progresses. The physician should, of course, be consulted about any change in dosage.

A recent large-scale study found that severe depression in patients with bipolar disorder responds no better to a combination of antidepressant medications and mood stabilizers than it does to mood stabilizers alone. Furthermore, this federally funded study found that antidepressant use does not hasten the emergence of manic symptoms in patients with bipolar disorder.

Medications work differently in each person, and it takes considerable time to determine in any particular case whether a given drug is effective at all, since bipolar disorder is by nature episodic, and patients may experience remissions whether or not they receive treatment. For this reason, neither patients nor their doctors should expect immediate relief, although psychosis with mania can respond quickly to anti psychotics, and bipolar depression can be alleviated quickly with electroconvulsive therapy (ECT). Many doctors emphasize that patients should not expect full stabilization for at least 3–4 weeks (some antidepressants, for example, take 4–6 weeks to take effect), and should not give up on a medication prematurely, nor should they discontinue medication with the disappearance of symptoms as the depression may return.

Compliance with medications can be a major problem. Some people, as they become manic, may lose the awareness of having an illness, and they therefore discontinue medications. Patients also often quit taking medication when symptoms disappear, erroneously thinking themselves "cured", and some people enjoy the effects of unmedicated hypomania. Other reasons cited by individuals for discontinuing medication are side effects, expense, and the stigma of having a psychiatric disorder. In a relatively small number of cases stipulated by law (varying by locality but typically, according to the law, only when a patient poses a threat to himself or others), patients who do not agree with their psychiatric diagnosis and treatment can legally be required to have treatment without their consent. Throughout North America and the United Kingdom, involuntary treatment or detention laws exist for severe cases of bipolar disorder and other mental illnesses where there is a potential for harm to oneself or others.

**Mood Stabilizers**

**Lithium salts**

The use of lithium salts as a treatment of bipolar disorder was first discovered by Dr. John Cade, an Australian psychiatrist who published a paper on the use of lithium in 1949.
Lithium salts had long been used as a first-line treatment for bipolar disorder. In ancient times, doctors would send their mentally ill patients to drink from "alkali springs" as a treatment. They did not know it, but they were really prescribing lithium, which was present in high concentration in the waters. The therapeutic effect of lithium salts appears to be entirely due to the lithium ion, Li⁺.

The two lithium salts used for bipolar therapy are lithium carbonate (mostly) and lithium citrate (sometimes). Approved for the treatment of acute mania in 1970 by the Food and Drug Administration (FDA), lithium has been an effective mood-stabilizing medication for many people with bipolar disorder. Lithium is also noted for reducing the risk of suicide. Although lithium is among the most effective mood stabilizers, persons taking it may experience side effects similar to the effects of ingesting too much table salt, such as high blood pressure, water retention, and constipation. Regular blood testing is required when taking lithium to determine the correct lithium levels since the therapeutic dose is close to the toxic dose.

The mechanism of lithium salt treatment is believed to work as follows: some symptoms of bipolar disorder appear to be caused by the enzyme inositol monophosphatase (IMPase), an enzyme that splits inositol monophosphate into free inositol and phosphate. It is involved in signal transduction and is believed to create an imbalance in neurotransmitters in bipolar patients. The lithium ion is believed to produce a mood stabilizing effect by inhibiting IMPase by substituting for one of two magnesium ions in IMPase's active site, slowing down this enzyme.

Lithium orotate is used as an alternative treatment to lithium carbonate by some individuals with bipolar disorder, mainly because it is available without a doctor's prescription. It is sometimes sold as "organic lithium" by nutritionists, as well as under a wide variety of brand names. There seems to be little evidence for its use in clinical treatment in preference to lithium carbonate. Individuals with bipolar disorder have complained that it is much weaker than lithium carbonate and, therefore, less effective.

Lithium has problems with its side effects, including hand trembling and intolerance of hot weather. Benztrapine is sometimes used to control the trembling, but itself causes sedation. Lithium has a very narrow window of effectiveness. Below that level it has no effect, and above it is toxic. For that reason blood must be sampled frequently to determine if the proper blood level is currently present.

**Anticonvulsants**

Anticonvulsant medications, particularly valproate and carbamazepine, are often used instead of, or along with, lithium. Valproate (Depakote, Epival) was FDA approved for the treatment of acute mania in 1995, and is now considered by some doctors to be the first line of therapy for bipolar disorder. A similar medication, valproic acid (Depakene) is also used. For some, it is preferable to lithium because its side effect profile seems to be less severe, compliance with the medication is better, and fewer breakthrough manic episodes occur. However, valproate is not as effective as lithium in preventing or managing
depressive episodes, so patients taking valproate may also need an antidepressant as an adjunct medicinal therapy.

New research suggests that different combinations of lithium and anticonvulsants may be helpful. Anticonvulsants are also used in combination with antipsychotics. Newer anticonvulsant medications, including lamotrigine and oxcarbazepine, are also effective as mood stabilizers in bipolar disorder. Lamotrigine is particularly promising, as it alleviates bipolar depression and prevents recurrence at higher rates. Lamotrigine is also well tolerated by many patients.

Zonisamide (trade name Zonegran), another anticonvulsant, also may show promise in treating bipolar depression according to Frederick K. Goodwin M.D. on a recent Medscape webcast titled "The Accurate Diagnosis and Long-Term Treatment of Bipolar Depression" to view the webcast click here. (free reg required).

Topiramate has not done well in clinical trials; it seems to help a few patients very much but most not at all. It appears to be useful in some treatment resistant cases and for anxiety issues when clonazepam cannot be prescribed. Gabapentin has failed to distinguish itself from placebo as a mood stabilizer.

According to studies conducted in Finland in patients with epilepsy, valproate may increase testosterone levels in teenage girls and produce polycystic ovary syndrome in women who began taking the medication before age 20. Increased testosterone can lead to polycystic ovary syndrome with irregular or absent menses, obesity, and abnormal growth of hair. Therefore, young female patients taking valproate should be monitored carefully by a physician. However, the therapeutic dose for a patient taking valproate for epilepsy is much higher than the therapeutic dose of valproate for an individual with bipolar disorder.

Other anticonvulsants effective in some cases and being studied closer include phenytoin, levetiracetam, pregabalin and valnoctamide.

**Atypical antipsychotic drugs**

The newer atypical antipsychotic drugs such as risperidone, quetiapine, and olanzapine are often used in acutely manic patients, because these medications have a rapid onset of psychomotor inhibition, which may be lifesaving in the case of a violent or psychotic patient. Parenteral and orally disintegrating (in particular, Zydus wafers) forms are favoured in emergency room settings. These drugs can also be used as adjunctives to lithium or anticonvulsants in refractory bipolar disorder and in prevention of mania recurrence. They also have fewer side effects, and are often used in place of lithium, in combination with an antidepressant, an anticonvulsant, or both.

In light of recent evidence, olanzapine (Zyprexa) has been FDA approved as an effective monotherapy for the maintenance of bipolar disorder. A head-to-head randomized control trial in 2005 has also shown olanzapine monotherapy to be just as effective and safe as
lithium in prophylaxis. Eli Lilly and Company also offers Symbyax, a combination of olanzapine and fluoxetine.

In addition, quetiapine (Seroquel and Seroquel XR) has been approved for the treatment of bipolar mania, bipolar depression, and for long-term maintenance treatment of bipolar disorder when used in conjunction with lithium or divalproex.

Ziprasidone (Geodon) and aripiprazole (Abilify) also show promise according to Gary Sachs M.D. of Harvard’s Massachusetts General Hospital Bipolar Clinic and Research Program. (View the webcast above at the Bipolar Clinic and Research Program link).

The atypical antipsychotics have some potential for causing weight gain and diabetes, and in larger doses over long periods may sometimes create tardive dyskinesia, though with much lesser probability than with the typical antipsychotics, such as Thorazine, Stelazine, or Haloperidol (Haldol).

New treatments

Modafinil (Provigil) and Pramipexole (Mirapex) show promise in treating the cognitive deterioration related to bipolar depression. In addition Riluzole, an ALS treatment, has been shown to be effective treatment. The breast cancer medicine tamoxifien has shown quick response to manic phases.

Antidepressants

Depression is one of the major symptoms of bipolar disorder (indeed, in bipolar II it is frequently the dominant symptom) and so antidepressants are often used to treat the disorder. Antidepressants are typically administered along with a mood stabilizer rather than as the primary treatment for bipolar disorder. Antidepressants include serotonin reuptake inhibitors (SSRIs) such as Prozac and Paxil, the serotonin-norepinephrine reuptake inhibitor Effexor, and the dopamine reuptake inhibitor Wellbutrin. Older antidepressants include the monoamine oxidase inhibitors (MAOIs). The herbal supplement Saint John’s Wort has also been shown to be an effective antidepressant in a number of clinical studies, although the precise mechanism of action remains unclear.

However, some doctors and researchers argue that antidepressants should be used with caution, if at all, in treating bipolar disorder. Fredrick K. Goodwin M.D.(1), coauthor of Manic Depressive Illness with Kay Redfield Jamison PhD and the NIMH’s Robert M. Post gleaned evidence by comparing the life charts of individuals with BP I and BP II who were medicated with certain mood stabilizers only versus any combination of those mood stabilizers plus certain antidepressants. The life chart trends indicate that use of certain antidepressants (over months to years) caused a long-term worsening of the illness over the life course compared to certain mood stabilizers alone in both bipolar I and bipolar II disorders. Specifically, they observed increased cycle frequency, increased mood episode severity, the emergence of mixed states and more treatment-resistant (difficult to treat) bipolar disorder. Some studies have also found that antidepressants pose a risk of inducing
hypomania or mania, sometimes in individuals with no prior history of mania. Saint John's Wort, although a naturally occurring compound, is thought to function in a fashion similar to man-made antidepressants, and so unsurprisingly, there are reports that suggest that it can also induce mania. For these reasons, some psychiatrists are hesitant to prescribe antidepressants for the treatment bipolar disorder unless mood stabilizers have failed to have an effect, however, others feel that antidepressants still have an important role to play in treatment of bipolar disorder.

**Psychotherapy and Cognitive Behavioral Therapy**

Certain types of psychotherapy, used in combination with medication, may provide some benefit in the treatment of bipolar disorders. Psychoeducation has been shown to be effective in improving patients' compliance with their lithium treatment. Several studies of family therapy report it can improve family communication, social functioning and lithium compliance, though it appears to be effective mainly on females. Evidence for the efficacy of other psychotherapies is absent or weak, often not being performed under randomized and controlled conditions. Well-designed studies have found interpersonal and social rhythm therapy to be ineffective.

Although medication and psychotherapy cannot cure the illness, therapy can often be valuable in helping to address the effects of disruptive manic or depressive episodes that have hurt a patient's career, relationships or self-esteem. Therapy is available not only from psychiatrists but from social workers, psychologists and other licensed counselors.

**Lifestyle Changes**

**Understanding One's Symptoms**

Understanding the symptoms, when they occur and ways to control them using appropriate medications and psychotherapy has given many people diagnosed with bipolar disorder a chance at a better life. Technically this is called prodrome detection and this is partly what is meant by becoming an expert on one's illness.

**Stress reduction**

Forms of stress may include having too much to do, too much complexity and conflicting demands among others. There are also stresses that come from the absence of elements such as human contact, a sense of achievement, constructive creative outlets, and occasions or circumstances that will naturally elicit positive emotions. Stress reduction will involve reducing things that cause anxiety and increasing those that generate happiness. It is not enough to just reduce the anxiety.

**Co-morbid substance use disorder**

Co-occurring substance misuse disorders, which are extremely common in bipolar patients can cause a significant worsening of bipolar symptomatology and can cause the emergence
of affective symptoms. The treatment options and recommendations for substance use disorders is wide but may include certain pharmacological and nonpharmacological treatment options.

Other Treatments

Omega-3 fatty acids

Omega-3 fatty acids may also be used as a treatment for bipolar disorder, particularly as a supplement to medication. An initial clinical trial by Stoll et al. produced positive results. However, since 1999 attempts to confirm this finding of beneficial effects of omega-3 fatty acids in several larger double-blind clinical trials have produced inconclusive results. It was hypothesized that the therapeutic ingredient in omega-3 fatty acid preparations is eicosapentaenoic acid (EPA) and that supplements should be high in this compound to be beneficial. Omega-3 fatty acids may be found in fish, fish oils, algae, and to a lesser degree in other foods such as flaxseed, flaxseed oil and walnuts. Although the benefits of Omega-3 fatty acids remain debated, they are readily available at drugstores and supermarkets, relatively inexpensive, and have no known side effects.

Exercise

Exercise has also been shown to have antidepressant effects. Its major advantages are that it appears to provide lasting improvement in mood, has no side effects, and is free.

Electroconvulsive therapy

Electroconvulsive therapy (ECT) is sometimes used to treat severe bipolar depression in cases where other treatments have failed and is 60 to 70 percent effective. Although it has proved to be a highly effective treatment, doctors are reluctant to use it except as a treatment of last resort because of the side-effects and possible temporary memory loss complications of ECT, particularly when repeated treatments ("maintenance ECT") are needed.

Ketogenic diet

A ketogenic diet similar to the diet used for pediatric epilepsy was thought to have mood stabilizing and antidepressant effects. Stanford University Medical School attempted a study using a ketogenic diet protocol on bipolar patients. However due to the lack of ability to attract subjects the trial was never started. Studies have shown it to have anti-depressant properties in rats.

Cannabinoids

While some reports indicate that cannabis can lessen the severity of mania and depression symptoms, others indicate cannabis can trigger mania and has been noted to have "a detrimental and potentially causative role in the development of psychosis." However, a
recent study noted neurocognitive functioning improved in bipolar patients who used cannabis. The study added that further research was needed.

THC can relieve depressive phases through its euphoriant action, while the tranquilizing effects of CBD can alleviate manic phases. CBD, another active constituent of cannabis, has proven anti-psychotic effects.

One recent online survey questioned the notion that marijuana smoking increases a user’s risk for depression. The authors of the study show that marijuana users reported fewer somatic symptoms and daily users reported less depressed mood and more positive effect than non-users. These self-report data suggest that adults apparently do not increase their risk for depression by using marijuana. Many find that the calming sedation associated with the use of cannabis helps to alleviate depression.

Current medical marijuana pharmaceuticals (such as dronabinol, marketed as Marinol) exist in the U.S. while Sativex, a whole-plant cannabis extract, is currently being marketed in Canada, the UK and Spain. Sativex is used for various illnesses, such as MS, cancer, and depression. Individuals who want to access pharmaceutical cannabis, however, may not be able to receive it due to drug laws against marijuana. Although illegal in many places around the world, cannabis remains easy to grow and purchase. See legal issues of cannabis for more information.

A study listed on clinicaltrials.gov states that the University of British Columbia is conducting a study for bipolar using Cannabinoids.

**Schizophrenia and Delusional Disorders**

**Schizophrenia**

Cloth embroidered by a patient diagnosed with schizophrenia
Schizophrenia is a mental disorder characterized by a disintegration of thought processes and of emotional responsiveness. It most commonly manifests as auditory hallucinations, paranoid or bizarre delusions, or disorganized speech and thinking, and it is accompanied by significant social or occupational dysfunction. The onset of symptoms typically occurs in young adulthood, with a global lifetime prevalence of about 0.3–0.7%. Diagnosis is based on observed behavior and the patient’s reported experiences.

Genetics, early environment, neurobiology, and psychological and social processes appear to be important contributory factors; some recreational and prescription drugs appear to cause or worsen symptoms. Current research is focused on the role of neurobiology, although no single isolated organic cause has been found. The many possible combinations of symptoms have triggered debate about whether the diagnosis represents a single disorder or a number of discrete syndromes. Despite the etymology of the term from the Greek roots skhizein (σχίζειν, "to split") and phren, phren- (φρήν, φρέν; "mind"), schizophrenia does not imply a "split mind" and it is not the same as dissociative identity disorder—also known as "multiple personality disorder" or "split personality"—a condition with which it is often confused in public perception.

The mainstay of treatment is antipsychotic medication, which primarily suppresses dopamine, and sometimes serotonin, receptor activity. Psychotherapy and vocational and social rehabilitation are also important in treatment. In more serious cases—where there is risk to self and others—involuntary hospitalization may be necessary, although hospital stays are now shorter and less frequent than they were.

The disorder is thought mainly to affect cognition, but it also usually contributes to chronic problems with behavior and emotion. People with schizophrenia are likely to have additional (comorbid) conditions, including major depression and anxiety disorders; the lifetime occurrence of substance abuse is almost 50%. Social problems, such as long-term unemployment, poverty and homelessness, are common. The average life expectancy of people with the disorder is 12 to 15 years less than those without, the result of increased physical health problems and a higher suicide rate (about 5%).

Signs and symptoms

A person diagnosed with schizophrenia may experience hallucinations (most reported are hearing voices), delusions (often bizarre or persecutory in nature), and disorganized thinking and speech. The latter may range from loss of train of thought, to sentences only loosely connected in meaning, to incoherence known as word salad in severe cases. Social withdrawal, sloppiness of dress and hygiene, and loss of motivation and judgement are all common in schizophrenia. There is often an observable pattern of emotional difficulty, for example lack of responsiveness. Impairment in social cognition is associated with schizophrenia, as are symptoms of paranoia; social isolation commonly occurs. In one uncommon subtype, the person may be largely mute, remain motionless in bizarre postures, or exhibit purposeless agitation, all signs of catatonia.
Late adolescence and early adulthood are peak periods for the onset of schizophrenia, critical years in a young adult's social and vocational development. In 40% of men and 23% of women diagnosed with schizophrenia the condition manifested itself before the age of 19. To minimize the developmental disruption associated with schizophrenia, much work has recently been done to identify and treat the prodromal (pre-onset) phase of the illness, which has been detected up to 30 months before the onset of symptoms. Those who go on to develop schizophrenia may experience transient or self-limiting psychotic symptoms and the non-specific symptoms of social withdrawal, irritability, dysphoria, and clumsiness during the prodromal phase.

**Schneiderian classification**

The term schizophrenia was coined by Eugen Bleuler.

The psychiatrist Kurt Schneider (1887–1967) listed the forms of psychotic symptoms that he thought distinguished schizophrenia from other psychotic disorders. These are called first-rank symptoms or Schneider’s first-rank symptoms, and they include delusions of being controlled by an external force; the belief that thoughts are being inserted into or withdrawn from one's conscious mind; the belief that one’s thoughts are being broadcast to other people; and hearing hallucinatory voices that comment on one’s thoughts or actions or that have a conversation with other hallucinated voices. Although they have significantly contributed to the current diagnostic criteria, the specificity of first-rank symptoms has been questioned. A review of the diagnostic studies conducted between 1970 and 2005 found that they allow neither a reconfirmation nor a rejection of Schneider’s claims, and suggested that first-rank symptoms be de-emphasized in future revisions of diagnostic systems.

**Positive and negative symptoms**

Schizophrenia is often described in terms of positive and negative (or deficit) symptoms. Positive symptoms are those that most individuals do not normally experience but are present in people with schizophrenia. They can include delusions, disordered thoughts and speech, and tactile, auditory, visual, olfactory and gustatory hallucinations, typically regarded as manifestations of psychosis. Hallucinations are also typically related to the content of the delusional theme. Positive symptoms generally respond well to medication. Negative symptoms are deficits of normal emotional responses or of other thought
processes, and respond less well to medication. They commonly include flat or blunted affect and emotion, poverty of speech (alogia), inability to experience pleasure (anhedonia), lack of desire to form relationships (asociality), and lack of motivation (avolation). Research suggests that negative symptoms contribute more to poor quality of life, functional disability, and the burden on others than do positive symptoms. People with prominent negative symptoms often have a history of poor adjustment before the onset of illness, and response to medication is often limited.

Causes of schizophrenia

The causes of schizophrenia have been the subject of much debate, with various factors proposed and discounted or modified. The language of schizophrenia research under the medical model is scientific. Such studies suggest that genetics, prenatal development, early environment, neurobiology and psychological and social processes are important contributory factors.

Current psychiatric research into the development of the disorder is often based on a neurodevelopmental model (proponents of which see schizophrenia as a syndrome.) However, schizophrenia is diagnosed on the basis of symptom profiles. Neural correlates do not provide sufficiently useful criteria. "Current research into schizophrenia has remained highly fragmented, much like the clinical presentation of the disease itself".

Although no common cause of schizophrenia has been identified in all individuals diagnosed with the condition, currently most researchers and clinicians believe it results from a combination of both brain vulnerabilities (either inherited or acquired) and life events. This widely adopted approach is known as the 'stress-vulnerability' model, and much scientific debate now focuses on how much each of these factors contributes to the development and maintenance of schizophrenia.

Schizophrenia is most commonly first diagnosed during late adolescence or early adulthood, suggesting it is often the end process of childhood and adolescent development. There is on average a somewhat earlier onset for men than women, with the possible influence of the female hormone estrogen being one hypothesis and sociocultural influences another.

Genetics

Evidence suggests that genetic vulnerability and environmental factors can act in combination to result in diagnosis of schizophrenia. Research suggests that genetic vulnerability to schizophrenia is multifactorial, caused by interactions of several genes.

Both individual twin studies and meta-analyses of twin studies estimate the heritability of risk for schizophrenia to be approximately 80% (this refers to the proportion of variation between individuals in a population that is influenced by genetic factors, not the degree of genetic determination of individual risk). Concordance rates between monozygotic twins was close to 50%; whereas dizygotic twins was 17%. Adoption studies have also indicated
a somewhat increased risk in those with a parent with schizophrenia even when raised apart. Studies suggest that the phenotype is genetically influenced but not genetically determined; that the variants in genes are generally within the range of normal human variation and have low risk associated with them each individually; and that some interact with each other and with environmental risk factors; and that they may not be specific to schizophrenia.

Some twin studies have found rates as low as 11.0%–13.8% among monozygotic twins, and 1.8%–4.1% among dizygotic twins, however. Tyrone Cannon reviewed the situation, stating: "Previous twin studies have reported estimates of broad heritability ranging from 0.41 to 0.87" Yet, in the "Pairs of Veteran Twins" study, for example, 338 pairs were schizophrenic with only 26 pairs concordant, and it was concluded in one report: "the role of the suggested genetic factor appears to be a limited one; 85 percent of the affected monozygotic pairs in the sample were discordant for schizophrenia". In addition, some scientists criticize the methodology of the twin studies, and have argued that the genetic basis of schizophrenia is still largely unknown or open to different interpretations.

For example, although the concordance of schizophrenia occurrence in monozygotic twins has traditionally been used to estimate a genetic component to the illness, the results could be skewed because of environmental factors like a shared placenta

In fact, researchers, have used the phenomenon of 'fetal programming' to account for familial patterns in epidemiological studies. "Intra-uterine growth is a complex outcome that is influenced by a wide range of factors including fetal genotype, maternal physiology and behaviour as well as the function of that crucial interface—the placenta," said one journal.

After reviewing techniques like: Genome Wide Association Studies; Single Nucleotide Polymorphisms and Copy Number Variations; the Nature journal reports: the basic observation is that, "You have this clear tangible phenomenon in which children resemble their parents"...."Despite what children get told in elementary school science we just don't know how that works," as Professor of ecology and evolutionary biology at Princeton, Leonid Kruglyak says (in reviewing hereditibility in general). It cites schizophrenia as a trait in which the genes have gone missing.

A great deal of effort has been put into molecular genetic studies of schizophrenia, which attempt to identify specific genes which may increase risk. A 2003 review of linkage studies listed seven genes as likely to increase risk for a later diagnosis of the disorder. Two recent reviews suggested that the evidence was strongest for two genes known as dysbindin (DTNBP1) and neuregulin (NRG1), and that a number of other genes (such as COMT, RGS4, PPP3CC, ZDHHC8, DISC1, and AKTI1) showed some early promising results. Variations near the gene FXYD6 have also been associated with schizophrenia in the UK but not in Japan. In 2008, rs7341475 SNP of the reelin gene was associated with an increased risk of schizophrenia in women, but not in men. This female-specific association was replicated in several populations. Still another review found evidence that the protein phosphatase 2B (calcineurin) might be involved in susceptibility to schizophrenia.
The largest most comprehensive genetic study of its kind, involving tests of several hundred single nucleotide polymorphisms (SNPs) in nearly 1,900 individuals with schizophrenia or schizoaffective disorder and 2,000 comparison subjects, reported in 2008 that there was no evidence of any significant association between the disorders and any of 14 previously identified candidate genes (RGS4, DISC1, DTNBP1, STX7, TAAR6, PPP3CC, NRG1, DRD2, HTR2A, DAOA, AKT1, CHRNA7, COMT, and ARVCF). The statistical distributions suggested nothing more than chance variation. The authors concluded that the findings make it unlikely that common SNPs in these genes account for a substantial proportion of the genetic risk for schizophrenia, although small effects could not be ruled out.

The perhaps largest analysis of genetic associations in schizophrenia is with the SzGene database at the Schizophrenia Research Forum. One 2008 meta-analysis examined genetic variants in 16 genes and found nominally significant effects.

Other research has suggested that a greater than average number of rare deletions or duplications of tiny DNA sequences within genes (known as copy number variants) are linked to increased risk for schizophrenia, especially in those "sporadic" cases not linked to family history of schizophrenia, and that the genetic factors and developmental pathways can thus be different in different individuals. A genome wide survey of 3,391 individuals with schizophrenia found CNVs in less than 1% of cases. Within them, deletions in regions related to psychosis were observed, as well as deletions on chromosome 15q13.3 and 1q21.1.

CNVs occur due to non-allelic homologous recombination mediated by low copy repeats (sequentially similar regions). This results in deletions and duplications of dosage sensitive genes. It has been speculated that CNVs underlie a significant proportion of normal human variation, including differences in cognitive, behavioral, and psychological features, and that CNVs in at least three loci can result in increased risk for schizophrenia in a few individuals. Epigenetics may also play a role in schizophrenia, with the expression of Protocadherin 11 X/Protocadherin Y playing a possible role in schizophrenia.

A 2009 study was able to create mice matching schizophrenic symptoms by the deletion of only one gene set, those of the neuregulin post-synaptic receptor. The result showed that although the mice mostly developed normally, on further brain development, glutamate receptors broke down. This theory supports the glutamate hypothesis of schizophrenia.

Another study in 2009 by Simon Fraser University researchers identifies a link between Autism and Schizophrenia:

"The SFU group found that variations in four sets of genes are related to both autism and schizophrenia. People normally have two copies of each gene, but in autistics some genome locations have only single copies and in schizophrenics extra copies are present at the same locations." "Source"

Prenatal
It is well established that obstetric complications or events are associated with an increased chance of the child later developing schizophrenia, although overall they constitute a non-specific risk factor with a relatively small effect. Obstetric complications occur in approximately 25 to 30% of the general population and the vast majority do not develop schizophrenia, and likewise the majority of individuals with schizophrenia have not had a detectable obstetric event. Nevertheless, the increased average risk is well-replicated, and such events may moderate the effects of genetic or other environmental risk factors. The specific complications or events most linked to schizophrenia, and the mechanisms of their effects, are still under examination.

One epidemiological finding is that people diagnosed with schizophrenia are more likely to have been born in winter or spring (at least in the northern hemisphere). However, the effect is not large. Explanations have included a greater prevalence of viral infections at that time, or a greater likelihood of vitamin D deficiency. A similar effect (increased likelihood of being born in winter and spring) has also been found with other, healthy populations, such as chess players.

Women who were pregnant during the Dutch famine of 1944, where many people were close to starvation (experiencing malnutrition) had a higher chance of having a child who would later develop schizophrenia. Studies of Finnish mothers who were pregnant when they found out that their husbands had been killed during the Winter War of 1939–1940 have shown that their children were significantly more likely to develop schizophrenia when compared with mothers who found out about their husbands’ death after pregnancy, suggesting that maternal stress may have an effect.

Fetal growth

Lower than average birth weight has been one of the most consistent findings, indicating slowed fetal growth possibly mediated by genetic effects. Almost any factor adversely affecting the fetus will affect growth rate, however, so the association has been described as not particularly informative regarding causation. In addition, the majority of birth cohort studies have failed to find a link between schizophrenia and low birth weight or other signs of growth retardation.

Animal models have suggested links between intrauterine growth restriction and specific neurological abnormalities similar to those that may be involved in the development of schizophrenia, including ventricular enlargement and reduced hippocampal volume in guinea pigs.

Hypoxia

It has been hypothesized since the 1970s that brain hypoxia (low oxygen levels) before, at or immediately after birth may be a risk factor for the development of schizophrenia.
Hypoxia is now being demonstrated as relevant to schizophrenia in animal models, molecular biology and epidemiology studies. One study in Molecular Psychiatry was able to differentiate 90% of schizophrenics from controls based on hypoxia and metabolism. Hypoxia has been recently described as one of the most important of the external factors that influence susceptibility, although studies have been mainly epidemiological. Such studies place a high degree of importance on hypoxic influence, but because of familial pattern of the illness in some families, propose a genetic factor also; stopping short of concluding hypoxia to be the sole cause. Fetal hypoxia, in the presence of certain unidentified genes, has been correlated with reduced volume of the hippocampus, which is in turn correlated with schizophrenia.

Although most studies have interpreted hypoxia as causing some form of neuronal dysfunction or even subtle damage, it has been suggested that the physiological hypoxia that prevails in normal embryonic and fetal development, or pathological hypoxia or ischemia, may exert an effect by regulating or dysregulating genes involved in neurodevelopment. A literature review judged that over 50% of the candidate genes for susceptibility to schizophrenia met criteria for "ischemia-hypoxia regulation and/or vascular expression" even though only 3.5% of all genes were estimated to be involved in hypoxia/ischemia or the vasculature.

A longitudinal study found that obstetric complications involving hypoxia were one factor associated with neurodevelopmental impairments in childhood and with the later development of schizophreniform disorders. Fetal hypoxia has been found to predict unusual movements at age 4 (but not age 7) among children who go on to develop schizophrenia, suggesting that its effects are specific to the stage of neurodevelopment. A Japanese case study of monozygotic twins discordant for schizophrenia (one has the diagnosis while the other does not) draws attention to their different weights at birth and concludes hypoxia may be the differentiating factor.

The unusual functional laterality in speech production (e.g. right hemisphere auditory processing) found in some individuals with schizophrenia could be due to aberrant neural networks established as a compensation for left temporal lobe damage induced by pre- or perinatal hypoxia. Prenatal and perinatal hypoxia appears to be important as one factor in the neurodevelopmental model, with the important implication that some forms of schizophrenia may thus be preventable.

Research on rodents seeking to understand the possible role of prenatal hypoxia in disorders such as schizophrenia has indicated that it can lead to a range of sensorimotor and learning/memory abnormalities. Impairments in motor function and coordination, evident on challenging tasks when the hypoxia was severe enough to cause brain damage, were long-lasting and described as a "hallmark of prenatal hypoxia".

Several animal studies have indicated that fetal hypoxia can affect many of the same neural substrates implicated in schizophrenia, depending on the severity and duration of the hypoxic event as well as the period of gestation, and in humans moderate or severe (but not mild) fetal hypoxia has been linked to a series of motor, language and cognitive deficits.
in children, regardless of genetic liability to schizophrenia. One paper restated that cerebellum neurological disorders were frequently found in schizophrenics and speculated hypoxia may cause the subsequent cognitive dysmetria.

Whereas most studies find only a modest effect of hypoxia in schizophrenia, a longitudinal study using a combination of indicators to detect possible fetal hypoxia, such as early equivalents of Neurological Soft Signs or obstetric complications, reported that the risk of schizophrenia and other nonaffective psychoses was "strikingly elevated" (5.75% versus 0.39%). Although objective estimates of hypoxia did not account for all schizophrenic cases; the study revealed increasing odds of schizophrenia according to graded increase in severity of hypoxia.

Other factors

There is an emerging literature on a wide range of prenatal risk factors, such as prenatal stress, intrauterine (in the womb) malnutrition, and prenatal infection. Increased paternal age has been linked to schizophrenia, possibly due to "chromosomal aberrations and mutations of the aging germline." Maternal-fetal rhesus or genotype incompatibility has also been linked, via increasing the risk of an adverse prenatal environment. Also, in mothers with schizophrenia, an increased risk has been identified via a complex interaction between maternal genotype, maternal behavior, prenatal environment and possibly medication and socioeconomic factors. References for many of these environmental risk factors have been collected in an online database.

There may be an association between celiac disease (gluten intolerance) and schizophrenia in a small proportion of patients, though large randomized controlled trials and epidemiological studies will be needed before such an association can be confirmed. Withdrawal of gluten from the diet is an inexpensive measure which may improve the symptoms in a small (≤3%) number of schizophrenic patients.

In addition, there is some evidence that exposure to toxins such as lead can also increase the risk of later development of schizophrenia spectrum disorders.

Infections

Numerous viral infections, in utero or in childhood, have been associated with an increased risk of later developing schizophrenia. Schizophrenia is somewhat more common in those born in winter to early spring, when infections are more common.

Influenza has long been studied as a possible factor. A 1988 study found that individuals who were exposed to the Asian flu as second trimester fetuses were at increased risk of eventually developing schizophrenia. This result was corroborated by a later British study of the same pandemic, but not by a 1994 study of the pandemic in Croatia. A Japanese study also found no support for a link between schizophrenia and birth after an influenza epidemic.
Polio, measles, varicella-zoster, rubella, herpes simplex virus type 2, maternal genital infections, Borna disease virus, and more recently Toxoplasma gondii, have been correlated with the later development of schizophrenia. Psychiatrists E. Fuller Torrey and R.H. Yolken have hypothesized that the latter, a common parasite in humans, contributes to some, if not many, cases of schizophrenia.

In a meta-analysis of several studies, they found moderately higher levels of Toxoplasma antibodies in those with schizophrenia and possibly higher rates of prenatal or early postnatal exposure to Toxoplasma gondii, but not acute infection. However, in another study of postmortem brain tissue, the authors have reported equivocal or negative results, including no evidence of herpes virus or T. gondii involvement in schizophrenia.

There is some evidence for the role of autoimmunity in the development of some cases of schizophrenia. A statistical correlation has been reported with various autoimmune diseases and direct studies have linked dysfunctional immune status to some of the clinical features of schizophrenia.

This is known as the pathogenic theory of schizophrenia or germ theory of schizophrenia. It is a pathogenic theory of disease in which it is thought that a proximal cause of certain cases of schizophrenia is the interaction of the developing fetus with pathogens such as viruses, or with antibodies from the mother created in response to these pathogens (in particular, Interleukin 8). Substantial research suggests that exposure to certain illnesses (e.g., influenza) in the mother of the neonate (especially at the end of the second trimester) causes defects in neural development which may emerge as a predisposition to schizophrenia around the time of puberty, as the brain grows and develops.

**Childhood antecedents**

In general, the antecedents of schizophrenia are subtle and those who will go on to develop schizophrenia do not form a readily identifiable subgroup - which would lead to identification of a specific cause. Average group differences from the norm may be in the direction of superior as well as inferior performance. Overall, birth cohort studies have indicated subtle nonspecific behavioral features, some evidence for psychotic-like experiences (particularly hallucinations), and various cognitive antecedents. There have been some inconsistencies in the particular domains of functioning identified and whether they continue through childhood and whether they are specific to schizophrenia.

A prospective study found average differences across a range of developmental domains, including reaching milestones of motor development at a later age, having more speech problems, lower educational test results, solitary play preferences at ages four and six, and being more socially anxious at age 13. Lower ratings of the mother's skills and understanding of the child at age 4 were also related.

Some of the early developmental differences were identified in the first year of life in a study in Finland, although generally related to psychotic disorders rather than schizophrenia in particular. The early subtle motor signs persisted to some extent, showing
a small link to later school performance in adolescence. An earlier Finnish study found that childhood performance of 400 individuals diagnosed with schizophrenia was significantly worse than controls on subjects involving motor co-ordination (sports and handcrafts) between ages 7 and 9, but there were no differences on academic subjects (contrary to some other IQ findings). (Patients in this age group with these symptoms were significantly less likely to progress to high school, despite academic ability)

However, reanalysis of the data from the later Finnish study, on older children (14 to 16) in a changed school system, using narrower diagnostic criteria and with less cases but more controls, did not support a significant difference on sports and handicraft performance. However, another study found that unusual motor coordination scores at 7 years of age were associated in adulthood with both those with schizophrenia and their unaffected siblings, while unusual movements at ages 4 and 7 predicted adult schizophrenia but not unaffected sibling status.

A birth cohort study in New Zealand found that children who went on to develop schizophreniform disorder had, as well as emotional problems and interpersonal difficulties linked to all adult psychiatric outcomes measured, significant impairments in neuromotor, receptive language, and cognitive development. A retrospective study found that adults with schizophrenia had performed better than average in artistic subjects at ages 12 and 15, and in linguistic and religious subjects at age 12, but worse than average in gymnastics at age 15.

Some small studies on offspring of individuals with schizophrenia have identified various neurobehavioral deficits, a poorer family environment and disruptive school behaviour, poor peer engagement, immaturity or unpopularity or poorer social competence and increasing schizophrenic symptomatology emerging during adolescence.

A minority "deficit syndrome" subtype of schizophrenia is proposed to be more marked by early poor adjustment and behavioral problems, as compared to non-deficit subtypes.

**Substance use**

The relationship between schizophrenia and drug use is complex, meaning that a clear causal connection between drug use and schizophrenia has been difficult to tease apart. There is strong evidence that using certain drugs can trigger either the onset or relapse of schizophrenia in some people. It may also be the case, however, that people with schizophrenia use drugs to overcome negative feelings associated with both the commonly prescribed antipsychotic medication and the condition itself, where negative emotion, paranoia and anhedonia are all considered to be core features.

The rate of substance use is known to be particularly high in this group. In a recent study, 60% of people with schizophrenia were found to use substances and 37% would be diagnosable with a substance use disorder.

**Cannabis**
There is some evidence that cannabis use can contribute to schizophrenia. Some studies suggest that cannabis is neither a sufficient nor necessary factor in developing schizophrenia, but that cannabis may significantly increase the risk of developing schizophrenia and may be, among other things, a significant causal factor. Nevertheless, some previous research in this area has been criticised as it has often not been clear whether cannabis use is a cause or effect of schizophrenia. To address this issue, a recent review of studies from which a causal contribution to schizophrenia can be assessed has suggested that cannabis statistically doubles the risk of developing schizophrenia on the individual level, and may, assuming a causal relationship, be responsible for up to 8% of cases in the population.

An older longitudinal study, published in 1987, suggested a sixfold increase of schizophrenia risks for high consumers of cannabis (use on more than fifty occasions) in Sweden.

Despite increases in cannabis consumption in the 1960s and 1970s in western society, rates of psychotic disorders such as schizophrenia remained relatively stable over time. Also, Sweden and Japan, where self-reported marijuana use is very low, do not have lower rates of psychosis than the U.S. and Canada do. Thus, there remains controversy over whether or not the apparent association between cannabis and schizophrenia is a causal relationship.

**Amphetamines and other stimulants**

As amphetamines trigger the release of dopamine and excessive dopamine function is believed to be responsible for many symptoms of schizophrenia (known as the dopamine hypothesis of schizophrenia), amphetamines may worsen schizophrenia symptoms. In addition, amphetamines are known to cause a stimulant psychosis in otherwise healthy individuals that superficially resembles schizophrenia, and may be misdiagnosed as such by some healthcare professionals.

**Hallucinogens**

Drugs such as ketamine, PCP, and LSD have been used to mimic schizophrenia for research purposes. Using LSD and other psychedelics as a model has now fallen out of favor with the scientific research community, as the differences between the drug induced states and the typical presentation of schizophrenia have become clear. The dissociatives ketamine and PCP, however, are still considered to produce states that are remarkably similar however, and are considered to be even better models than stimulants since they produce both positive and negative symptoms.

**Tobacco use**

People with schizophrenia tend to smoke significantly more tobacco than the general population. The rates are exceptionally high amongst institutionalized patients and
homeless people. In a UK census from 1993, 74% of people with schizophrenia living in institutions were found to be smokers. A 1999 study that covered all people with schizophrenia in Nithsdale, Scotland found a 58% prevalence rate of cigarette smoking, to compare with 28% in the general population. An older study found that as much as 88% of outpatients with schizophrenia were smokers.

Despite the higher prevalence of tobacco smoking, people diagnosed with schizophrenia have a much lower than average chance of developing and dying from lung cancer. While the reason for this is unknown, it may be because of a genetic resistance to the cancer, a side effect of drugs being taken, or a statistical effect of increased likelihood of dying from causes other than lung cancer.

A 2003 study of over 50,000 Swedish conscripts found that there was a small but significant protective effect of smoking cigarettes on the risk of developing schizophrenia later in life. While the authors of the study stressed that the risks of smoking far outweigh these minor benefits, this study provides further evidence for the 'self-medication' theory of smoking in schizophrenia and may give clues as to how schizophrenia might develop at the molecular level. Furthermore, many people with schizophrenia have smoked tobacco products long before they are diagnosed with the illness, and a cohort study of Israeli conscripts found that healthy adolescent smokers were more likely to develop schizophrenia in the future than their non-smoking peers.

It is of interest that cigarette smoking affects liver function such that the antipsychotic drugs used to treat schizophrenia are broken down in the blood stream more quickly. This means that smokers with schizophrenia need slightly higher doses of antipsychotic drugs in order for them to be effective than do their non-smoking counterparts.

The increased rate of smoking in schizophrenia may be due to a desire to self-medicate with nicotine. One possible reason is that smoking produces a short term effect to improve alertness and cognitive functioning in persons who suffer this illness. It has been postulated that the mechanism of this effect is that people with schizophrenia have a disturbance of nicotinic receptor functioning which is temporarily abated by tobacco use. However, some researchers have questioned whether self-medication is really the best explanation for the association.

A study from 1989 and a 2004 case study show that when haloperidol is administered, nicotine limits the extent to which the antipsychotic increases the sensitivity of the dopamine 2 receptor. Dependent on the dopamine system, symptoms of Tardive Dyskinesia are not found in the nicotine administered patients despite a roughly 70% increase in dopamine receptor activity, but the controls have more than 90% and do develop symptoms. A 1997 study showed that akathisia was significantly reduced upon administration of nicotine when the akathisia was induced by antipsychotics. This gives credence to the idea tobacco could be used to self medicate by limiting effects of the illness, the medication, or both.
Life experiences

Social adversity

The chance of developing schizophrenia has been found to increase with the number of adverse social factors (e.g. indicators of socioeconomic disadvantage or social exclusion) present in childhood. Stressful life events generally precede the onset of schizophrenia. A personal or recent family history of migration is a considerable risk factor for schizophrenia, which has been linked to psychosocial adversity, social defeat from being an outsider, racial discrimination, family dysfunction, unemployment and poor housing conditions.

Childhood experiences of abuse or trauma are risk factors for a diagnosis of schizophrenia later in life. Recent large-scale general population studies indicate the relationship is a causal one, with an increasing risk with additional experiences of maltreatment, although a critical review suggests conceptual and methodological issues require further research. There is some evidence that adversities may lead to cognitive biases and/or altered dopamine neurotransmission, a process that has been termed "sensitization".

Specific social experiences have been linked to specific psychological mechanisms and psychotic experiences in schizophrenia. In addition, structural neuroimaging studies of victims of sexual abuse and other traumas have sometimes reported findings similar to those sometimes found in psychotic patients, such as thinning of the corpus callosum, loss of volume in the anterior cingulate cortex, and reduced hippocampal volume.

Urbanicity

A particularly stable and replicable finding has been the association between living in an urban environment and the development of schizophrenia, even after factors such as drug use, ethnic group and size of social group have been controlled for. A recent study of 4.4 million men and women in Sweden found a 68%–77% increased risk of diagnosed psychosis for people living in the most urbanized environments, a significant proportion of which is likely to be described as schizophrenia.

The effect does not appear to be due to a higher incidence of obstetric complications in urban environments. The risk increases with the number of years and degree of urban living in childhood and adolescence, suggesting that constant, cumulative, or repeated exposures during upbringing occurring more frequently in urbanized areas are responsible for the association.

Various possible explanations for the effect have been judged unlikely based on the nature of the findings, including infectious causes or a generic stress effect. It is thought to interact with genetic dispositions and, since there appears to be nonrandom variation even across different neighborhoods, and an independent association with social isolation, it has been proposed that the degree of "social capital" (e.g. degree of mutual trust, bonding and safety
in neighborhoods) can exert a developmental impact on children growing up in these environments.

**Close relationships**

Evidence is consistent that negative attitudes from others increase the risk of schizophrenia relapse, in particular critical comments, hostility, authoritarian, and intrusive or controlling attitudes (termed 'high expressed emotion' by researchers). Although family members and significant others are not held responsible for schizophrenia - the attitudes, behaviors and interactions of all parties are addressed - unsupportive dysfunctional relationships may also contribute to an increased risk of developing schizophrenia.

**Other proposed etiologies**

Psychiatrists R. D. Laing, Silvano Arieti, Theodore Lidz and others have argued that the symptoms of what is called mental illness are comprehensible reactions to impossible demands that society and particularly family life places on some sensitive individuals. Laing, Arieti and Lidz were notable in valuing the content of psychotic experience as worthy of interpretation, rather than considering it simply as a secondary and essentially meaningless marker of underlying psychological or neurological distress. Laing described eleven case studies of people diagnosed with schizophrenia and argued that the content of their actions and statements was meaningful and logical in the context of their family and life situations.

In 1956, Gregory Bateson and his colleagues Paul Watzlawick, Donald Jackson, and Jay Haley articulated a theory of schizophrenia, related to Laing's work, as stemming from double bind situations where a person receives different or contradictory messages. Madness was therefore an expression of this distress and should be valued as a cathartic and transformative experience. In the books Schizophrenia and the Family and The Origin and Treatment of Schizophrenic Disorders Lidz and his colleagues explain their belief that parental behaviour can result in mental illness in children. Arieti's Interpretation of Schizophrenia won the 1975 scientific National Book Award in the United States.

The concept of schizophrenia as a result of civilization has been developed further by psychologist Julian Jaynes in his 1976 book The Origin of Consciousness in the Breakdown of the Bicameral Mind; he proposed that until the beginning of historic times, schizophrenia or a similar condition was the normal state of human consciousness. This would take the form of a "bicameral mind" where a normal state of low affect, suitable for routine activities, would be interrupted in moments of crisis by "mysterious voices" giving instructions, which early people characterized as interventions from the gods. Researchers into shamanism have speculated that in some cultures schizophrenia or related conditions may predispose an individual to becoming a shaman; the experience of having access to multiple realities is not uncommon in schizophrenia, and is a core experience in many shamanic traditions. Equally, the shaman may have the skill to bring on and direct some of the altered states of consciousness psychiatrists label as illness. Psychohistorians, on the
other hand, accept the psychiatric diagnoses. However, unlike the current medical model of mental disorders they may argue that poor parenting in tribal societies causes the shaman’s schizoid personalities. Commentators such as Paul Kurtz and others have endorsed the idea that major religious figures experienced psychosis, heard voices and displayed delusions of grandeur.

Modern clinical psychological research has indicated a number of processes which may cause or bring on episodes of schizophrenia.

A number of cognitive biases and deficits have been identified. These include attribution biases in social situations, difficulty distinguishing inner speech from speech from an external source (source monitoring), difficulty in adjusting speech to the needs of the hearer, difficulties in the very earliest stages of processing visual information (including reduced latent inhibition), and an attentional bias towards threats.

Some of these tendencies have been shown to worsen or appear when under emotional stress or in confusing situations. As with related neurological findings, they are not shown by all individuals with a diagnosis of schizophrenia, and it is not clear how specific they are to schizophrenia. However, the findings regarding cognitive difficulties in schizophrenia are reliable and consistent enough for some researchers to argue that they are diagnostic.

Impaired capacity to appreciate one’s own and others’ mental states has been reported to be the single-best predictor of poor social competence in schizophrenia, and similar cognitive features have been identified in close relatives of people diagnosed with schizophrenia.

A number of emotional factors have been implicated in schizophrenia, with some models putting them at the core of the disorder. It was thought that the appearance of blunted affect meant that sufferers did not experience strong emotions, but more recent studies indicate there is often a normal or even heightened level of emotionality, particularly in response to negative events or stressful social situations. Some theories suggest positive symptoms of schizophrenia can result from or be worsened by negative emotions, including depressed feelings and low self-esteem and feelings of vulnerability, inferiority or loneliness. Chronic negative feelings and maladaptive coping skills may explain some of the association between psychosocial stressors and symptomology. Critical and controlling behaviour by significant others (high expressed emotion) causes increased emotional arousal and lowered self-esteem and a subsequent increase in positive symptoms such as unusual thoughts. Countries or cultures where schizotypal personalities or schizophrenia symptoms are more accepted or valued appear to be associated with reduced onset of, or increased recovery from, schizophrenia.

Related studies suggest that the content of delusional and psychotic beliefs in schizophrenia can be meaningful and play a causal or mediating role in reflecting the life history, or social circumstances of the individual. Holding minority socio-cultural beliefs, for example due to ethnic background, has been linked to increased diagnosis of schizophrenia. The way an individual interprets his or her delusions and hallucinations
(e.g. as threatening or as potentially positive) has also been found to influence functioning and recovery.

Some experts think autonomy vs intimacy is a motivation for schizophrenic symptoms.

Other lines of work relating to the self in schizophrenia have linked it to psychological dissociation or abnormal states of awareness and identity as understood from phenomenological and other perspectives.

Psychiatrist Tim Crow has argued that schizophrenia may be the evolutionary price we pay for a left brain hemisphere specialization for language. Since psychosis is associated with greater levels of right brain hemisphere activation and a reduction in the usual left brain hemisphere dominance, our language abilities may have evolved at the cost of causing schizophrenia when this system breaks down.

In alternative medicine, some practitioners believe that there are a vast number of physical causes of what ends up being diagnosed as schizophrenia. While some of these explanations may stretch credulity, others (such as heavy metal poisoning and nutritional imbalances) have been supported at least somewhat by research. However, it is not entirely clear how many patients initially diagnosed with schizophrenia these alternative explanations may account for.

**Mechanisms**

A number of attempts have been made to explain the link between altered brain function and schizophrenia. One of the most common is the dopamine hypothesis, which attributes psychosis to the mind’s faulty interpretation of the misfiring of dopaminergic neurons.

**Psychological**

Many psychological mechanisms have been implicated in the development and maintenance of schizophrenia. Cognitive biases have been identified in those with the diagnosis or those at risk, especially when under stress or in confusing situations. Some cognitive features may reflect global neurocognitive deficits such as memory loss, while others may be related to particular issues and experiences.

Despite a demonstrated appearance of "blunted affect", recent findings indicate that many individuals diagnosed with schizophrenia are emotionally responsive, particularly to stressful or negative stimuli, and that such sensitivity may cause vulnerability to symptoms or to the disorder. Some evidence suggests that the content of delusional beliefs and psychotic experiences can reflect emotional causes of the disorder, and that how a person interprets such experiences can influence symptomatology. The use of “safety behaviors” to avoid imagined threats may contribute to the chronicity of delusions. Further evidence for the role of psychological mechanisms comes from the effects of psychotherapies on symptoms of schizophrenia.
Neurological

Functional magnetic resonance imaging (fMRI), and other brain imaging technologies, allow for the study of differences in brain activity in people diagnosed with schizophrenia. The image shows two levels of the brain, with areas that were more active in healthy controls than in schizophrenia patients shown in red, during an fMRI study of working memory.

Those with a diagnosis of schizophrenia have changes in both brain structure and chemistry. Studies using neuropsychological tests and brain imaging technologies such as fMRI and PET to examine functional differences in brain activity have shown that differences seem to most commonly occur in the frontal lobes, hippocampus and temporal lobes. Reductions in brain volume, smaller than those found in Alzheimer's disease, have been reported in areas of the frontal cortex and temporal lobes. It is uncertain whether these volumetric changes are progressive or preexist prior to the onset of the disease. These differences have been linked to the neurocognitive deficits often associated with schizophrenia. Because neural circuits are altered, it has alternatively been suggested that schizophrenia should be thought of as a collection of neurodevelopmental disorders.

Particular attention has been paid to the function of dopamine in the mesolimbic pathway of the brain. This focus largely resulted from the accidental finding that phenothiazine drugs, which block dopamine function, could reduce psychotic symptoms. It is also supported by the fact that amphetamines, which trigger the release of dopamine, may exacerbate the psychotic symptoms in schizophrenia. The influential dopamine hypothesis of schizophrenia proposed that excessive activation of D2 receptors was the cause of (the positive symptoms of) schizophrenia. Although postulated for about 20 years based on the D2 blockade effect common to all antipsychotics, it was not until the mid-1990s that PET and SPET imaging studies provided supporting evidence. The dopamine hypothesis is now thought to be simplistic, partly because newer antipsychotic medication (atypical antipsychotic medication) can be just as effective as older medication (typical antipsychotic medication), but also affects serotonin function and may have slightly less of a dopamine blocking effect.

Interest has also focused on the neurotransmitter glutamate and the reduced function of the NMDA glutamate receptor in schizophrenia, largely because of the abnormally low levels of glutamate receptors found in the postmortem brains of those diagnosed with schizophrenia, and the discovery that glutamate-blocking drugs such as phencyclidine and ketamine can mimic the symptoms and cognitive problems associated with the condition.
Reduced glutamate function is linked to poor performance on tests requiring frontal lobe and hippocampal function, and glutamate can affect dopamine function, both of which have been implicated in schizophrenia, have suggested an important mediating (and possibly causal) role of glutamate pathways in the condition. But positive symptoms fail to respond to glutamatergic medication.

**Diagnosis**

Schizophrenia is diagnosed based on criteria in either the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders, version DSM-IV-TR, or the World Health Organization’s International Statistical Classification of Diseases and Related Health Problems, the ICD-10. These criteria use the self-reported experiences of the person and reported abnormalities in behavior, followed by a clinical assessment by a mental health professional. Symptoms associated with schizophrenia occur along a continuum in the population and must reach a certain severity before a diagnosis is made. As of 2009 there is no objective test.

John Nash, a U.S. mathematician and joint winner of the 1994 Nobel Prize for Economics, suffered from schizophrenia. His life has been the subject of the 2001 Academy Award-winning film A Beautiful Mind.

**Criteria**
The ICD-10 criteria are typically used in European countries, while the DSM-IV-TR criteria are used in the United States and the rest of the world, and are prevailing in research studies. The ICD-10 criteria put more emphasis on Schneiderian first-rank symptoms. In practice, agreement between the two systems is high.

According to the revised fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), to be diagnosed with schizophrenia, three diagnostic criteria must be met:

Characteristic symptoms: Two or more of the following, each present for much of the time during a one-month period (or less, if symptoms remitted with treatment).

- Delusions
- Hallucinations
- Disorganized speech, which is a manifestation of formal thought disorder
- Grossly disorganized behavior (e.g. dressing inappropriately, crying frequently) or catatonic behavior
- Negative symptoms: Blunted affect (lack or decline in emotional response), alogia (lack or decline in speech), or avolition (lack or decline in motivation)

If the delusions are judged to be bizarre, or hallucinations consist of hearing one voice participating in a running commentary of the patient's actions or of hearing two or more voices conversing with each other, only that symptom is required above. The speech disorganization criterion is only met if it is severe enough to substantially impair communication.

- Social or occupational dysfunction: For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, or self-care, are markedly below the level achieved prior to the onset.
- Significant duration: Continuous signs of the disturbance persist for at least six months. This six-month period must include at least one month of symptoms (or less, if symptoms remitted with treatment).

If signs of disturbance are present for more than a month but less than six months, the diagnosis of schizophreniform disorder is applied. Psychotic symptoms lasting less than a month may be diagnosed as brief psychotic disorder, and various conditions may be classed as psychotic disorder not otherwise specified. Schizophrenia cannot be diagnosed if symptoms of mood disorder are substantially present (although schizoaffective disorder could be diagnosed), or if symptoms of pervasive developmental disorder are present unless prominent delusions or hallucinations are also present or if the symptoms are the direct physiological result of a general medical condition or a substance, such as abuse of a drug or medication.

Subtypes
The **DSM-IV-TR** contains five sub-classifications of schizophrenia, although the developers of DSM-5 are recommending they be dropped from the new classification:

- **Paranoid type**: Where delusions and hallucinations are present but thought disorder, disorganized behavior, and affective flattening are absent. (DSM code 295.3/ICD code F20.0)
- **Disorganized type**: Named hebephrenic schizophrenia in the ICD. Where thought disorder and flat affect are present together. (DSM code 295.1/ICD code F20.1)
- **Catatonic type**: The subject may be almost immobile or exhibit agitated, purposeless movement. Symptoms can include catatonic stupor and waxy flexibility. (DSM code 295.2/ICD code F20.2)
- **Undifferentiated type**: Psychotic symptoms are present but the criteria for paranoid, disorganized, or catatonic types have not been met. (DSM code 295.9/ICD code F20.3)
- **Residual type**: Where positive symptoms are present at a low intensity only. (DSM code 295.6/ICD code F20.5)

The ICD-10 defines two additional subtypes:

- Post-schizophrenic depression: A depressive episode arising in the aftermath of a schizophrenic illness where some low-level schizophrenic symptoms may still be present. (ICD code F20.4)
- Simple schizophrenia: Insidious and progressive development of prominent negative symptoms with no history of psychotic episodes. (ICD code F20.6)

**Differential**

Psychotic symptoms may be present in several other mental disorders, including bipolar disorder, borderline personality disorder, drug intoxication and drug-induced psychosis. Delusions ("non-bizarre") are also present in delusional disorder, and social withdrawal in social anxiety disorder, avoidant personality disorder and schizotypal personality disorder. Schizophrenia is complicated with obsessive-compulsive disorder (OCD) considerably more often than could be explained by pure chance, although it can be difficult to distinguish obsessions that occur in OCD from the delusions of schizophrenia.

A more general medical and neurological examination may be needed to rule out medical illnesses which may rarely produce psychotic schizophrenia-like symptoms, such as metabolic disturbance, systemic infection, syphilis, HIV infection, epilepsy, and brain lesions. It may be necessary to rule out a delirium, which can be distinguished by visual hallucinations, acute onset and fluctuating level of consciousness, and indicates an underlying medical illness. Investigations are not generally repeated for relapse unless there is a specific medical indication or possible adverse effects from antipsychotic medication.

**Prevention**
Evidence for the effectiveness of early intervention is inconclusive. While there is some evidence that early intervention in those with a psychotic episode may improve short term outcomes, there is little benefit from these measures after five years. Attempting to prevent schizophrenia in the prodrome phase is of uncertain benefit and therefore as of 2009 is not recommended. Prevention is difficult as there are no reliable markers for the later development of the disease.

**Management**

The primary treatment of schizophrenia is antipsychotic medications, often in combination with psychological and social supports. Hospitalization may occur for severe episodes either voluntarily or (if mental health legislation allows it) involuntarily. Long-term hospitalization is uncommon since deinstitutionalization beginning in the 1950s, although still occurs. Community support services including drop-in centers, visits by members of a community mental health team, supported employment and support groups are common. Some evidence indicates that regular exercise has a positive effect on the physical and mental health of those with schizophrenia.

**Medication**

Risperidone (trade name Risperdal) is a common atypical antipsychotic medication.

The first-line psychiatric treatment for schizophrenia is antipsychotic medication, which can reduce the positive symptoms of psychosis in about 7–14 days. Antipsychotics however fail to significantly ameliorate the negative symptoms and cognitive dysfunction.
The choice of which antipsychotic to use is based on benefits, risks, and costs. It is debatable whether, as a class, typical or atypical antipsychotics are better. Both have equal drop-out and symptom relapse rates when typicals are used at low to moderate dosages. There is a good response in 40–50%, a partial response in 30–40%, and treatment resistance (failure of symptoms to respond satisfactorily after six weeks to two of three different antipsychotics) in 20% of people. Clozapine is an effective treatment for those who respond poorly to other drugs, but it has the potentially serious side effect of agranulocytosis (lowered white blood cell count) in 1–4%.

With respect to side effects typical antipsychotics are associated with a higher rate of extrapyramidal side effects while atypicals are associated with considerable weight gain, diabetes and risk of metabolic syndrome. While atypicals have fewer extrapyramidal side effects these differences are modest. Some atypicals such as quetiapine and risperidone are associated with a higher risk of death compared to the atypical perphenazine, while clozapine is associated with the lowest risk of death. It remains unclear whether the newer antipsychotics reduce the chances of developing neuroleptic malignant syndrome, a rare but serious neurological disorder.

For people who are unwilling or unable to take medication regularly, long-acting depot preparations of antipsychotics may be used to achieve control. When used in combination with psychosocial interventions they may improve long-term adherence to treatment.

Psychosocial

A number of psychosocial interventions may be useful in the treatment of schizophrenia including family therapy, assertive community treatment, supported employment, cognitive remediation, skills training, cognitive behavioral therapy (CBT), token economic interventions, and psychosocial interventions for substance use and weight management. Family therapy or education, which addresses the whole family system of an individual, may reduce relapses and hospitalizations. The evidence for CBT’s effectiveness in either reducing symptoms or preventing relapse is minimal. The benefits of art or drama therapy are currently unknown.

Prognosis

Schizophrenia has great human and economic costs. It results in a decreased life expectancy of 12–15 years, primarily because of its association with obesity, sedentary lifestyles, and smoking, with an increased rate of suicide playing a lesser role. These differences in life expectancy increased between the 1970s and 1990s, and between the 1990s and first decade of the 21st century did not change substantially in a health system with open access to care (Finland).

Schizophrenia is a major cause of disability, with active psychosis ranked as the third-most-disabling condition after quadriplegia and dementia and ahead of paraplegia and blindness. Approximately three-fourths of people with schizophrenia have ongoing disability with relapses. Some people do recover completely and others function well in society. Most
people with schizophrenia live independently with community support. In people with a first episode of psychosis a good long-term outcome occurs in 42%, an intermediate outcome in 35% and a poor outcome in 27%. Outcomes for schizophrenia appear better in the developing than the developed world. These conclusions however have been questioned.

There is a higher than average suicide rate associated with schizophrenia. This has been cited at 10%, but a more recent analysis of studies and statistics revises the estimate to 4.9%, most often occurring in the period following onset or first hospital admission. Several times more (20 to 40%) attempt suicide at least once. There are a variety of risk factors, including male gender, depression, and a high intelligence quotient.

Schizophrenia and smoking have shown a strong association in studies world-wide. Use of cigarettes is especially high in individuals diagnosed with schizophrenia, with estimates ranging from 80% to 90% being regular smokers, as compared to 20% of the general population. Those who smoke tend to smoke heavily, and additionally smoke cigarettes with high nicotine content.

**Epidemiology**

Schizophrenia affects around 0.3–0.7% of people at some point in their life, or 24 million people worldwide as of 2011. It occurs 1.4 times more frequently in males than females and typically appears earlier in men—the peak ages of onset are 20–28 years for males and 26–32 years for females. Onset in childhood is much rarer, as is onset in middle- or old age. Despite the received wisdom that schizophrenia occurs at similar rates worldwide, its prevalence varies across the world, within countries, and at the local and neighborhood level. It causes approximately 1% of worldwide disability adjusted life years. The rate of schizophrenia varies up to threefold depending on how it is defined.

**History**

Accounts of a schizophrenia-like syndrome are thought to be rare in the historical record before the 19th century, although reports of irrational, unintelligible, or uncontrolled behavior were common. A detailed case report in 1797 concerning James Tilly Matthews, and accounts by Phillipe Pinel published in 1809, are often regarded as the earliest cases of the illness in the medical and psychiatric literature. Schizophrenia was first described as a distinct syndrome affecting teenagers and young adults by Bénédict Morel in 1853, termed démence précoce (literally ‘early dementia’). The term dementia praecox was used in 1891 by Arnold Pick in a case report of a psychotic disorder. In 1893 Emil Kraepelin introduced a broad new distinction in the classification of mental disorders between dementia praecox and mood disorder (termed manic depression and including both unipolar and bipolar depression). Kraepelin believed that dementia praecox was primarily a disease of the brain, and particularly a form of dementia, distinguished from other forms of dementia such as Alzheimer’s disease which typically occur later in life.
The word schizophrenia—which translates roughly as "splitting of the mind" and comes from the Greek roots schizein (σχίζειν, "to split") and phrēn, phren- (φρήν, φρεν-, "mind")—was coined by Eugen Bleuler in 1908 and was intended to describe the separation of function between personality, thinking, memory, and perception. Bleuler described the main symptoms as 4 A's: flattened Affect, Autism, impaired Association of ideas and Ambivalence. Bleuler realized that the illness was not a dementia, as some of his patients improved rather than deteriorated, and thus proposed the term schizophrenia instead. Treatment was revolutionized in the mid-1950s with the development and introduction of chlorpromazine.

Molecule of chlorpromazine (trade name Thorazine), which revolutionized treatment of schizophrenia in the 1950s

In the early 1970s, the diagnostic criteria for schizophrenia was the subject of a number of controversies which eventually led to the operational criteria used today. It became clear after the 1971 US-UK Diagnostic Study that schizophrenia was diagnosed to a far greater extent in America than in Europe. This was partly due to looser diagnostic criteria in the US, which used the DSM-II manual, contrasting with Europe and its ICD-9. David Rosenhan’s 1972 study, published in the journal Science under the title "On being sane in insane places", concluded that the diagnosis of schizophrenia in the US was often subjective and unreliable. These were some of the factors leading to the revision not only of the diagnosis of schizophrenia, but the revision of the whole DSM manual, resulting in the publication of the DSM-III in 1980.

The term schizophrenia is commonly misunderstood to mean that affected persons have a "split personality". Although some people diagnosed with schizophrenia may hear voices and may experience the voices as distinct personalities, schizophrenia does not involve a person changing among distinct multiple personalities. The confusion arises in part due to
the literal interpretation of Bleuler's term schizophrenia. The first known misuse of the term to mean "split personality" was in an article by the poet T. S. Eliot in 1933.

**Society and culture**

Social stigma has been identified as a major obstacle in the recovery of patients with schizophrenia. In a large, representative sample from a 1999 study, 12.8% of Americans believed that individuals with schizophrenia were "very likely" to do something violent against others, and 48.1% said that they were "somewhat likely" to. Over 74% said that people with schizophrenia were either "not very able" or "not able at all" to make decisions concerning their treatment, and 70.2% said the same of money management decisions. The perception of individuals with psychosis as violent has more than doubled in prevalence since the 1950s, according to one meta-analysis.

In 2002 the term for schizophrenia in Japan was changed from Seishin-Bunretsu-Byō (mind-split-disease) to Tōgō-shitchō-shō (integration disorder) to reduce stigma. The new name was inspired by the biopsychosocial model; it increased the percentage of patients who were informed of the diagnosis from 37% to 70% over three years.

In the United States, the cost of schizophrenia—including direct costs (outpatient, inpatient, drugs, and long-term care) and non-health care costs (law enforcement, reduced workplace productivity, and unemployment)—was estimated to be $62.7 billion in 2002.

The book and film A Beautiful Mind chronicle the life of John Forbes Nash, a Nobel Prize-winning mathematician who was diagnosed with schizophrenia.

**Psychosis**

Psychosis (from the Greek ψυχή "psyche", for mind/soul, and -ωσις "-osis", for abnormal condition) means abnormal condition of the mind, and is a generic psychiatric term for a mental state often described as involving a "loss of contact with reality". People suffering from psychosis are described as psychotic. Psychosis is given to the more severe forms of psychiatric disorder, during which hallucinations and delusions and impaired insight may occur. Some professionals say that the term psychosis is not sufficient as some illnesses grouped under the term "psychosis" have nothing in common (Gelder, Mayou & Geddes 2005).

People experiencing psychosis may report hallucinations or delusional beliefs, and may exhibit personality changes and thought disorder. Depending on its severity, this may be accompanied by unusual or bizarre behavior, as well as difficulty with social interaction and impairment in carrying out the daily life activities.

A wide variety of central nervous system diseases, from both external poisons and internal physiologic illness, can produce symptoms of psychosis.
Signs and symptoms

People with psychosis may have one or more of the following: hallucinations, delusions, or a thought disorder, as described below.

Hallucinations

A hallucination is defined as sensory perception in the absence of external stimuli. Hallucinations are different from illusions, or perceptual distortions, which are the misperception of external stimuli. Hallucinations may occur in any of the five senses and take on almost any form, which may include simple sensations (such as lights, colors, tastes, and smells) to more meaningful experiences such as seeing and interacting with fully formed animals and people, hearing voices, and having complex tactile sensations.

Auditory hallucinations, particularly experiences of hearing voices, are a common and often prominent feature of psychosis. Hallucinated voices may talk about, or to, the person, and may involve several speakers with distinct personas. Auditory hallucinations tend to be particularly distressing when they are derogatory, commanding or preoccupying. However, the experience of hearing voices need not always be a negative one. One research study has shown that the majority of people who hear voices are not in need of psychiatric help. The Hearing Voices Movement has subsequently been created to support voice hearers, regardless of whether they are considered to have a mental illness or not.

Delusions

Psychosis may involve delusional beliefs, some of which are paranoid in nature. Karl Jaspers has classified psychotic delusions into primary and secondary types. Primary delusions are defined as arising suddenly and not being comprehensible in terms of normal mental processes, whereas secondary delusions may be understood as being influenced by the person’s background or current situation (e.g., ethnic or sexual orientation, religious beliefs, superstitious belief).

Thought disorder

Thought disorder describes an underlying disturbance to conscious thought and is classified largely by its effects on speech and writing. Affected persons show loosening of associations, that is, a disconnection and disorganization of the semantic content of speech and writing. In the severe form speech becomes incomprehensible and it is known as "word-salad".

Causes

Causes of symptoms of mental illness were customarily classified as "organic" or "functional". Organic disorders were those held to be caused by physical illness affecting the brain (that is, psychiatric disorders secondary to other conditions), while functional disorders were considered to be disorders of the functioning of the mind in the absence of
physical disorders (that is, primary psychological or psychiatric disorders). The materialistic view of the mind-body problem holds that mental disorders arise from physical processes; in this view, the distinction between brain and mind, and therefore between organic and functional disease, is an artificial one. Subtle physical abnormalities have been found in illnesses traditionally considered functional, such as schizophrenia. The DSM-IV-TR avoids the functional/organic distinction, and instead lists traditional psychotic illnesses, psychosis due to general medical conditions, and substance-induced psychosis.

**Psychiatric disorders**

Primary psychiatric causes of psychosis include the following:

- schizophrenia and schizophreniform disorder
- affective (mood) disorders, including severe depression, and severe depression or mania in bipolar disorder (manic depression). People experiencing a psychotic episode in the context of depression may experience persecutory or self-blaming delusions or hallucinations, while people experiencing a psychotic episode in the context of mania may form grandiose delusions.
- schizoaffective disorder, involving symptoms of both schizophrenia and mood disorders
- brief psychotic disorder, or acute/transient psychotic disorder
- delusional disorder (persistent delusional disorder)
- chronic hallucinatory psychosis

Psychotic symptoms may also be seen in

- schizotypal disorder
- certain personality disorders at times of stress (including paranoid personality disorder, schizoid personality disorder, and borderline personality disorder)
- induced delusional disorder
- and occasionally in obsessive-compulsive disorder

Stress is known to contribute to and trigger psychotic states. A history of psychologically traumatic events, and the recent experience of a stressful event, can both contribute to the development of psychosis. Short-lived psychosis triggered by stress is known as brief reactive psychosis, and patients may spontaneously recover normal functioning within two weeks. In some rare cases, individuals may remain in a state of full-blown psychosis for many years, or perhaps have attenuated psychotic symptoms (such as low intensity hallucinations) present at most times.

**Normal states**

Brief hallucinations are not uncommon in those without any psychiatric disease. Causes or triggers include...
- falling asleep and waking: hypnagogic and hypnopompic hallucinations, which are entirely normal
- bereavement, in which hallucinations of a deceased loved one are common
- severe sleep deprivation
- sensory deprivation and sensory impairment

**Medical conditions**

A very large number of medical conditions can cause psychosis, sometimes called secondary psychosis. Examples include:

- disorders causing delirium (toxic psychosis), in which consciousness is disturbed
- neurodevelopmental disorders and chromosomal abnormalities, including velocardiofacial syndrome
- neurodegenerative disorders, such as Alzheimer’s disease, dementia with Lewy bodies, and Parkinson’s disease
- focal neurological disease, such as stroke, brain tumors, multiple sclerosis, and some forms of epilepsy
- malignancy (typically via masses in the brain, paraneoplastic syndromes, or drugs used to treat cancer)
- infectious and postinfectious syndromes, including infections causing delirium, viral encephalitis, HIV, malaria, Lyme disease, syphilis
- endocrine disease, such as hypothyroidism, hyperthyroidism, adrenal failure, Cushing’s syndrome, hypoparathyroidism and hyperparathyroidism; sex hormones also affect psychotic symptoms and sometimes childbirth can provoke psychosis, termed puerperal psychosis
- inborn errors of metabolism, such as porphyria and metachromatic leukodystrophy
- nutritional deficiency, such as vitamin B12 deficiency
- other acquired metabolic disorders, including electrolyte disturbances such as hypocalcemia, hypernatremia, hyponatremia, hypokalemia, hypomagnesemia, hypermagnesemia, hypercalcemia, and hypophosphatemia, but also hypoglycemia, hypoxia, and failure of the liver or kidneys
- autoimmune and related disorders, such as systemic lupus erythematosus (lupus, SLE), sarcoidosis, Hashimoto’s encephalopathy, and anti-NMDA-receptor encephalitis
- poisoning, by therapeutic drugs (see below), recreational drugs (see below), and a range of plants, fungi, metals, organic compounds, and a few animal toxins
- some sleep disorders, including hallucinations in narcolepsy (in which REM sleep intrudes into wakefulness)

Psychosis can even be caused by familiar ailments such as flu or mumps.

**Recreational drugs**

Various psychoactive substances (both legal and illegal) have been implicated in causing, exacerbating, and/or precipitating psychotic states and/or disorders in users. This may be
upon intoxication, for a more prolonged period after use, or upon withdrawal. Drugs that can induce psychotic symptoms include amphetamine, caffeine (which can worsen psychotic symptoms in schizophrenia and produce olfactory hallucinations at very high doses in normal volunteers), cannabis, cocaine, desoxypipradrol, dimethyltryptamine, alcohol (ethanol), inhalants, gammahydroxybutyric acid (and its precursors gammabutyrolactone and 1,4-butanediol), ketamine, LSD, mephedrone and methcathinone, mescaline and other phenethylamine hallucinogens, methamphetamine, MDMA (very rarely), opiates such as heroin, phencyclidine, piperazine-based drugs, psilocybin, and anabolic steroids at high doses.

Frequent users of cannabis have twice the likelihood of developing both psychosis and schizophrenia. Older studies indicate that certain strains containing large proportions of tetrahydrocannabinol and low proportions of cannabidiol merely lowers the threshold for psychosis, and thus helps to trigger full-blown psychosis in some people. On the other hand, cannabis use has increased dramatically over the past few decades but declined in the last decade, whereas the rate of psychosis has not increased. This suggests that a direct causal link is unlikely for all users.

**Medication**

Administration, or sometimes withdrawal, of a large number of medications may provoke psychotic symptoms. Drugs that can induce psychosis experimentally and/or in a significant proportion of patients include amphetamine and other sympathomimetics, dopamine agonists, ketamine, corticosteroids (often with mood changes in addition), and some anticonvulsants such as vigabatrin.

**Pathophysiology**

The first brain image of an individual with psychosis was completed as far back as 1935 using a technique called pneumoencephalography (a painful and now obsolete procedure where cerebrospinal fluid is drained from around the brain and replaced with air to allow the structure of the brain to show up more clearly on an X-ray picture).

The purpose of the brain is to collect information from the body (pain, hunger, etc.), and from the outside world, interpret it to a coherent world view, and produce a meaningful response. The information from the senses enter the brain in the primary sensory areas. They process the information and send it to the secondary areas where the information is interpreted. Spontaneous activity in the primary sensory areas may produce hallucinations which are misinterpreted by the secondary areas as information from the real world.

For example, a PET or fMRI scan of a person who claims to be hearing voices may show activation in the primary auditory cortex, or parts of the brain involved in the perception and understanding of speech.

Tertiary brain cortex collects the interpretations from the secondary cortices and creates a coherent world view of it. A study investigating structural changes in the brains of people
with psychosis showed there was significant grey matter reduction in the right medial temporal, lateral temporal, and inferior frontal gyrus, and in the cingulate cortex bilaterally of people before and after they became psychotic. Findings such as these have led to debate about whether psychosis itself causes excitotoxic brain damage and whether potentially damaging changes to the brain are related to the length of psychotic episode. Recent research has suggested that this is not the case although further investigation is still ongoing.

Studies with sensory deprivation have shown that the brain is dependent on signals from the outer world to function properly. If the spontaneous activity in the brain is not counterbalanced with information from the senses, loss from reality and psychosis may occur after some hours. A similar phenomenon is paranoia in the elderly when poor eyesight, hearing and memory causes the person to be abnormally suspicious of the environment.

On the other hand, loss from reality may also occur if the spontaneous cortical activity is increased so that it is no longer counterbalanced with information from the senses. The 5-HT2A receptor seems to be important for this, since drugs which activate them produce hallucinations.

However, the main feature of psychosis is not hallucinations, but the inability to distinguish between internal and external stimuli. Close relatives to psychotic patients may hear voices, but since they are aware that they are unreal they can ignore them, so that the hallucinations do not affect their reality perception. Hence they are not considered to be psychotic.

Psychosis has been traditionally linked to the neurotransmitter dopamine. In particular, the dopamine hypothesis of psychosis has been influential and states that psychosis results from an overactivity of dopamine function in the brain, particularly in the mesolimbic pathway. The two major sources of evidence given to support this theory are that dopamine receptor D2 blocking drugs (i.e., antipsychotics) tend to reduce the intensity of psychotic symptoms, and that drugs which boost dopamine activity (such as amphetamines and cocaine) can trigger psychosis in some people (see amphetamine psychosis). However, increasing evidence in recent times has pointed to a possible dysfunction of the excitatory neurotransmitter glutamate, in particular, with the activity of the NMDA receptor. This theory is reinforced by the fact that dissociative NMDA receptor antagonists such as ketamine, PCP and dextromethorphan/dextrophan (at large overdoses) induce a psychotic state more readily than dopaminergic stimulants, even at "normal" recreational doses. The symptoms of dissociative intoxication are also considered to mirror the symptoms of schizophrenia, including negative psychotic symptoms, more closely than amphetamine psychosis. Dissociative induced psychosis happens on a more reliable and predictable basis than amphetamine psychosis, which usually only occurs in cases of overdose, prolonged use or with sleep deprivation, which can independently produce psychosis. New antipsychotic drugs which act on glutamate and its receptors are currently undergoing clinical trials.
The connection between dopamine and psychosis is generally believed to be complex. While dopamine receptor D2 suppresses adenylate cyclase activity, the D1 receptor increases it. If D2-blocking drugs are administered the blocked dopamine spills over to the D1 receptors. The increased adenylate cyclase activity affects genetic expression in the nerve cell, a process which takes time. Hence antipsychotic drugs take a week or two to reduce the symptoms of psychosis. Moreover, newer and equally effective antipsychotic drugs actually block slightly less dopamine in the brain than older drugs whilst also blocking 5-HT2A receptors, suggesting the 'dopamine hypothesis' may be oversimplified. Soyka and colleagues found no evidence of dopaminergic dysfunction in people with alcohol-induced psychosis and Zoldan et al. reported moderately successful use of ondansetron, a 5-HT3 receptor antagonist, in the treatment of levodopa psychosis in Parkinson's disease patients.

Psychiatrist David Healy has criticised pharmaceutical companies for promoting simplified biological theories of mental illness that seem to imply the primacy of pharmaceutical treatments while ignoring social and developmental factors which are known to be important influences in the aetiology of psychosis.

Some theories regard many psychotic symptoms to be a problem with the perception of ownership of internally generated thoughts and experiences. For example, the experience of hearing voices may arise from internally generated speech that is mislabeled by the psychotic person as coming from an external source.

It has been suggested that persons with bipolar disorder may have increased activity of the left hemisphere compared to the right hemisphere of the brain, while persons with schizophrenia have increased activity in the right hemisphere.

Increased level of right hemisphere activation has also been found in people who have high levels of paranormal beliefs and in people who report mystical experiences. It also seems to be the case that people who are more creative are also more likely to show a similar pattern of brain activation. Some researchers have been quick to point out that this in no way suggests that paranormal, mystical or creative experiences are in any way by themselves a symptom of mental illness, as it is still not clear what makes some such experiences beneficial and others distressing.

**Diagnosis**

Diagnosing the presence and/or extent of psychosis may be distinguished from diagnosing the cause of psychosis.

The presence of psychosis is typically diagnosed by clinical interview, incorporating mental state examination. Its extent may be established by formal rating scales. The Brief Psychiatric Rating Scale (BPRS) assesses the level of 18 symptom constructs of psychosis such as hostility, suspicion, hallucination, and grandiosity. It is based on the clinician's interview with the patient and observations of the patient’s behavior over the previous 2–3 days. The patient’s family can also provide the behavior report. During the initial
assessment and the follow-up, both positive and negative symptoms of psychosis can be assessed using the 30 item Positive and Negative Symptom Scale (PANSS).

Establishing the cause of psychosis requires clinical examination, and sometimes special investigations, to diagnose or exclude secondary causes of psychosis; if these are excluded, a primary psychiatric diagnosis can be established.

**Treatment**

The treatment of psychosis depends on the cause or diagnosis or diagnoses (such as schizophrenia, bipolar disorder and/or substance intoxication). The first line treatment for many psychotic disorders is antipsychotic medication (oral or intramuscular injection), and sometimes hospitalization is needed. There is growing evidence that cognitive behavior therapy and family therapy can be effective in managing psychotic symptoms. When other treatments for psychosis are ineffective, electroconvulsive therapy or ECT (also known as shock treatment) is sometimes applied to relieve the underlying symptoms of psychosis due to depression. There is also increasing research suggesting that animal-assisted therapy can contribute to the improvement in general well-being of people with schizophrenia.

**Early intervention**

Early intervention in psychosis is a relatively new concept based on the observation that identifying and treating someone in the early stages of a psychosis can significantly improve their longer term outcome. This approach advocates the use of an intensive multi-disciplinary approach during what is known as the critical period, where intervention is the most effective, and prevents the long term morbidity associated with chronic psychotic illness.

Newer research into the effectiveness of cognitive behavioural therapy during the early pre-cursory stages of psychosis (also known as the "prodrome" or "at risk mental state") suggests that such input can prevent or delay the onset of psychosis.

**History**

The word psychosis was first used by Ernst von Feuchtersleben in 1845 as an alternative to insanity and mania and stems from the Greek ψυχωσίς (psychosis), "a giving soul or life to, animating, quickening" and that from ψυχή (psyche), "soul" and the suffix -ωσίς (-osis), in this case "abnormal condition". The word was used to distinguish disorders which were thought to be disorders of the mind, as opposed to "neurosis", which was thought to stem from a disorder of the nervous system. The psychoses thus became the modern equivalent of the old notion of madness, and hence there was much debate on whether there was only one (unitary) or many forms of the new disease.

The division of the major psychoses into manic depressive illness (now called bipolar disorder) and dementia praecox (now called schizophrenia) was made by Emil Kraepelin,
who attempted to create a synthesis of the various mental disorders identified by 19th century psychiatrists, by grouping diseases together based on classification of common symptoms. Kraepelin used the term 'manic depressive insanity' to describe the whole spectrum of mood disorders, in a far wider sense than it is usually used today. In Kraepelin’s classification this would include 'unipolar' clinical depression, as well as bipolar disorder and other mood disorders such as cyclothymia. These are characterised by problems with mood control and the psychotic episodes appear associated with disturbances in mood, and patients will often have periods of normal functioning between psychotic episodes even without medication. Schizophrenia is characterized by psychotic episodes which appear to be unrelated to disturbances in mood, and most non-medicated patients will show signs of disturbance between psychotic episodes.

During the 1960s and 1970s, psychosis was of particular interest to counterculture critics of mainstream psychiatric practice, who argued that it may simply be another way of constructing reality and is not necessarily a sign of illness. For example, R. D. Laing argued that psychosis is a symbolic way of expressing concerns in situations where such views may be unwelcome or uncomfortable to the recipients. He went on to say that psychosis could be also seen as a transcendental experience with healing and spiritual aspects. Arthur J. Deikman suggested use of the term "mystical psychosis" to characterize first-person accounts of psychotic experiences that are similar to reports of mystical experiences. Thomas Szasz focused on the social implications of labeling people as psychotic, a label he argues unjustly medicalises different views of reality so such unorthodox people can be controlled by society. Psychoanalysis has a detailed account of psychosis which differs markedly from that of psychiatry. Freud and Lacan outlined their perspective on the structure of psychosis in a number of works.

Since the 1970s, the introduction of a recovery approach to mental health, which has been driven mainly by people who have experienced psychosis (or whatever name is used to describe their experiences), has led to a greater awareness that mental illness is not a lifelong disability, and that there is an expectation that recovery is possible, and probable with effective support.

**Schizophreniform disorder**

Schizophreniform disorder is a mental disorder diagnosed when symptoms of schizophrenia are present for a significant portion of the time within a one-month period, but signs of disruption are not present for the full six months required for the diagnosis of schizophrenia.

The symptoms of both disorders can include delusions, hallucinations, disorganized speech, disorganized or catatonic behavior and social withdrawal. While impairment in social, occupational, or academic functioning is required for the diagnosis of schizophrenia, in schizophreniform disorder an individual’s level of functioning may or may not be affected. While the onset of schizophrenia is often gradual over a number of months or years, the onset of schizophreniform disorder can be relatively rapid.
Like schizophrenia, schizophreniform disorder is often treated with antipsychotic medications, especially the atypicals, along with a variety of social supports (such as individual psychotherapy, family therapy, occupational therapy, etc.) designed to reduce the social and emotional impact of the illness. The prognosis varies depending upon the nature, severity, and duration of the symptoms, but about two-thirds of individuals diagnosed with schizophreniform disorder go on to develop schizophrenia.

**Symptoms and Diagnosis**

Schizophreniform disorder is a type of mental illness that is characterized by psychosis and closely related to schizophrenia. Both schizophrenia and schizophreniform disorder, as defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), have the same symptoms and essential features except for two differences: the level of functional impairment and the duration of symptoms. Impairment in social, occupational, or academic functioning is always present in schizophrenia, but such impairment may or may not be present in schizophreniform disorder. In schizophreniform disorder, the symptoms (including prodromal, active, and residual phases) must last at least 1 month but not more than 6 months, while in schizophrenia the symptoms must be present for a minimum of 6 months.

If the symptoms have persisted for at least one month, a provisional diagnosis of schizophreniform disorder can be made while waiting to see if recovery occurs. If the symptoms resolve within 6 months of onset, the provisional qualifier is removed from the diagnosis. However, if the symptoms persist for 6 months or more, the diagnosis of schizophreniform disorder must be revised. The diagnosis of brief psychotic disorder may be considered when the duration of symptoms is less than one month.

The main symptoms of both schizophreniform disorder and schizophrenia can include:

- delusions,
- hallucinations,
- disorganized speech resulting from formal thought disorder,
- disorganized or catatonic behavior, and negative symptoms, such as
  - an inability to show emotion (flat affect),
  - an inability to experience pleasure (anhedonia),
  - impaired or decreased speech (aphasia),
  - a lack of desire to form relationships (asociality), and
  - a lack of motivation (avolition).

**Prognosis**

The following specifiers for schizophreniform disorder may be used to indicate the presence or absence of features that may be associated with a better prognosis:

With Good Prognostic Features, used if at least two of the following features are present:
- onset of prominent psychotic symptoms within 4 weeks of the first noticeable change in usual behavior or functioning,
- confusion or perplexity at the height of the psychotic episode,
- good premorbid social and occupational functioning, and
- absence of blunted or flat affect.

Without Good Prognostic Features, used if two or more of the above features have not been present.

The presence of negative symptoms and poor eye contact both appear to be prognostic of a poor outcome. Many of the anatomic and functional changes seen in the brains of patients with schizophrenia also occur in patients with schizophreniform disorder. However, at present there is no consensus among scientists regarding whether or not ventricular enlargement, a poor prognostic factor in schizophrenia, has any prognostic value in patients with schizophreniform disorder. According to the American Psychiatric Association, approximately two-thirds of patients diagnosed with schizophreniform disorder are subsequently diagnosed with schizophrenia.

Etiology

The exact etiology of the disorder remains unknown, and relatively few studies have focused exclusively on the etiology of schizophreniform disorder. Like other psychotic disorders, a two-hit hypothesis has been proposed, suggesting that some individuals have an underlying multifactorial genetic vulnerability to the disorder that can be triggered by certain environmental factors. Schizophreniform disorder is more likely to occur in people with family members who have schizophrenia or bipolar disorder.

Prevalence

Schizophreniform disorder is equally prevalent among men and women. The most common ages of onset are 18–24 for men and 18–35 for women. While the symptoms of schizophrenia often develop gradually over a period of years, the diagnostic criteria for schizophreniform disorder require a much more rapid onset.

Available evidence suggests variations in incidence across sociocultural settings. In the United States and other developed countries, the incidence is low, possibly fivefold less than that of schizophrenia. In developing countries, the incidence is substantially higher, especially for the subtype "With Good Prognostic Features". In some of these settings schizophreniform disorder may be as common as schizophrenia.

Treatment

Various modalities of treatment, including pharmacotherapy, psychotherapy, and various other psychosocial and educational interventions, are used in the treatment of schizophreniform disorder. Pharmacotherapy is the most commonly used treatment
modality as psychiatric medications can act quickly to both reduce the severity of symptoms and shorten their duration. The medications used are largely the same as those used to treat schizophrenia, with an atypical antipsychotic as the usual drug of choice. Patients who do not respond to the initial atypical antipsychotic may benefit from being switched to another atypical antipsychotic, the addition of a mood stabilizer such as lithium or an anticonvulsant, or being switched to a typical antipsychotic.

Treatment of schizophreniform disorder can occur in inpatient, outpatient, partial hospitalization settings. In selecting the treatment setting, the primary aims are to minimize the psychosocial consequences for the patient and maintain the safety of the patient and others. While the need to quickly stabilize the patient’s symptoms almost always exists, consideration of the patient’s severity of symptoms, family support, and perceived likelihood of compliance with outpatient treatment can help determine if stabilization can occur in the outpatient setting. Patients who receive inpatient treatment may benefit from a structured intermediate environment, such as a sub-acute unit, step-down unit, partial hospital, or day hospital, during the initial phases of returning to the community.

As improvement progresses during treatment, help with coping skills, problem-solving techniques, psychoeducational approaches, and eventually occupational therapy and vocational assessments are often very helpful for patients and their families. Virtually all types of individual psychotherapy are used in the treatment of schizophreniform disorder, except for insight-oriented therapies as patients often have limited insight as a symptom of their illness.

Since schizophreniform disorder has such rapid onset of severe symptoms, patients are sometimes in denial about their illness, which also would limit the efficacy of insight-oriented therapies. Supportive forms of psychotherapy such as interpersonal psychotherapy, supportive psychotherapy, and cognitive behavior therapy are particularly well suited for the treatment of the disorder. Group psychotherapy is usually not indicated for patients with schizophreniform disorder because they may be distressed by the symptoms of patients with more advanced psychotic disorders.

**Brief psychotic disorder**

Brief psychotic disorder is a period of psychosis whose duration is generally shorter, non re-occurring, and not better accounted for by another condition.

The disorder is characterized by a sudden onset of psychotic symptoms, which may include delusions, hallucinations, disorganized speech or behavior, or catatonic behavior. The symptoms must not be better accounted for by schizophrenia, schizoaffective disorder, delusional disorder or mania in bipolar disorder. They must also not be caused by a drug (such as amphetamines) or medical condition (such as a brain tumor).
Symptoms generally last at least a day, but not more than a month, and there is an eventual return to full baseline functioning. It may occur in response to a significant stressor in a person’s life, or in other situations where a stressor is not apparent, including in the weeks following birth. In diagnosis, a careful distinction is considered for culturally appropriate behaviors, such as religious beliefs and activities. It is believed to be connected to or synonymous with a variety of culture-specific phenomena such as latah, koro, and amok.

**Frequency**

The condition is considered uncommon in the United States, and is 10 times more frequent in developing countries. Internationally, it occurs twice as often in women than men, and even more often in women in the United States. It typically occurs in the late 30s and early 40s.

**Chronic hallucinatory psychosis**

Chronic hallucinatory psychosis is a psychosis subtype, classified under "Other nonorganic psychosis" by the ICD-10 Chapter V: Mental and behavioural disorders. Other abnormal mental symptoms in the early stages are, as a rule, absent. The patient is most usually quiet and orderly, with a good memory.

It has often been a matter of the greatest difficulty to decide under which heading of the recognized classifications individual members of this group should be placed. As the hallucinations give rise to slight depression, some might possibly be included under melancholia. In others, paranoia may develop. Others, again, might be swept into the widespread net of dementia precox. This state of affairs cannot be regarded as satisfactory, for they are not truly cases of melancholia, paranoia, dementia precox or any other described affection.

What this disease is, as its name suggests, a hallucinatory case, for it is its main feature. These may be of all senses, but auditory hallucinations are the most prominent. At the beginning the patient may realize that the hallucination is a morbid phenomenon and unaccountable. They may admit that thought they hear a "voice" speaking, there is no one in the flesh actually doing so. Such a state of affairs may last for years and possibly, though rarely, for life, and the subject would not be deemed insane in the ordinary sense of the word.

It's probable, however, that this condition forms the first stage of the illness, which eventually develops on definite lines. What usually happens is the patient seeks an explanation for the hallucinations. As none is forthcoming he/she tries to account for their presence and the result is a delusion, and most frequently, a delusion of persecution. Also, it needs to be noted that the delusion is a comparatively late arrival and is the logical result of the hallucinations.
Folie à deux

Folie à deux (or shared psychosis) is a psychiatric syndrome in which symptoms of a delusional belief are transmitted from one individual to another. The same syndrome shared by more than two people may be called folie à trois, folie à quatre, folie en famille or even folie à plusieurs ("madness of many"). Recent psychiatric classifications refer to the syndrome as shared psychotic disorder (DSM-IV) (297.3) and induced delusional disorder (F.24) in the ICD-10, although the research literature largely uses the original name. The disorder was first conceptualized in 19th century French psychiatry.

Presentation

This case study is taken from Enoch and Ball’s 'Uncommon Psychiatric Syndromes' (2001, p181): Margaret and her husband Michael, both aged 34 years, were discovered to be suffering from folie à deux when they were both found to be sharing similar persecutory delusions. They believed that certain persons were entering their house, spreading dust and fluff and "wearing down their shoes". Both had, in addition, other symptoms supporting a diagnosis of emotional contagion, which could be made independently in either case.

This syndrome is most commonly diagnosed when the two or more individuals concerned live in proximity and may be socially or physically isolated and have little interaction with other people.

Various sub-classifications of folie à deux have been proposed to describe how the delusional belief comes to be held by more than one person.

- Folie imposée is where a dominant person (known as the 'primary', 'inducer' or 'principal') initially forms a delusional belief during a psychotic episode and imposes it on another person or persons (known as the 'secondary', 'acceptor' or 'associate') with the assumption that the secondary person might not have become deluded if left to his or her own devices. If the parties are admitted to hospital separately, then the delusions in the person with the induced beliefs usually resolve without the need of medication.

- Folie simultanée describes either the situation where two people considered to suffer independently from psychosis influence the content of each other's delusions so they become identical or strikingly similar, or one in which two people "morbidly predisposed" to delusional psychosis mutually trigger symptoms in each other.

Folie à deux and its more populous cousins are in many ways a psychiatric curiosity. The current Diagnostic and Statistical Manual of Mental Disorders states that a person cannot be diagnosed as being delusional if the belief in question is one "ordinarily accepted by other members of the person's culture or subculture" (see entry for delusion). It is not clear at what point a belief considered to be delusional escapes from the folie à... diagnostic category and becomes legitimate because of the number of people holding it. When a large
number of people may come to believe obviously false and potentially distressing things based purely on hearsay, these beliefs are not considered to be clinical delusions by the psychiatric profession and are labelled instead as mass hysteria.

In a well-publicised case in the United Kingdom, the condition was one of two possible diagnoses of a Swedish woman, Sabina Eriksson, who stabbed a man to death after he took her into his home, offering food and shelter. Eriksson had just been released from police custody following an incident on a motorway which grabbed news headlines. Caught on camera by a police documentary filmmaker, her twin sister ran into the path of an oncoming articulated lorry, sustaining severe injuries. Eriksson then immediately duplicated her twin’s actions by stepping into the path of an oncoming car; she survived the impact. The defence counsel in the ultimate murder trial claimed that Eriksson was a ‘secondary’ sufferer of folie à deux, influenced by the presence or perceived presence of her twin sister — the ‘primary’.

Related phenomena

Reports have stated that a similar phenomenon to folie à deux had been induced by the military incapacitating agent BZ in the late 60s, and most recently again by anthropologists in the South American rainforest consuming the hallucinogen ayahuasca (Metzner, 1999).

Similar experiences of folie à deux or even folie à plusieurs have been reported during Shamanic journeying in a group setting. A typical example is that of the interaction between power animals of two or more people who were guided in a Power-Animal retrieval journey during the same session and by the same Shaman.

Cultural References

The 19th episode of season 5 of the X-Files TV series and the fifth studio album of the American rock band Fall Out Boy are named after the syndrome.

Early intervention in psychosis

Early intervention in psychosis is a clinical approach to those experiencing symptoms of psychosis for the first time. It forms part of the new prevention paradigm for psychiatry and is leading to reform of mental health services, especially in the United Kingdom. There has been considerable academic interest over the past decade.

This approach centers on the early detection and treatment of early symptoms of psychosis during the formative years of the psychotic condition. The first three to five years are believed to be a critical period. The aim is to reduce the usual delays to treatment for those in their first episode of psychosis. The provision of optimal treatments in these early years is thought to prevent relapses and reduce the long term impact of the condition. It is considered a secondary prevention strategy.
The duration of untreated psychosis (DUP) has been shown as an indicator of prognosis, with a longer DUP associated with more long term disability.

**Components of the model**

There are a number of functional components of the early psychosis model, and they can be structured as different sub-teams within early psychosis services. The emerging pattern of sub-teams are currently:

**Early psychosis treatment teams**

Multiple discipline clinical teams providing an intensive case management approach for the first three to five years. The approach is similar to assertive community treatment, but with an increased focus on the engagement and treatment of this previously untreated population and the provision of evidence based, optimal interventions for clients in their first episode of psychosis. For example, the use of low-dose antipsychotic medication is promoted ("start low, go slow"), with a need for monitoring of side effects and an intensive and deliberate period of psycho-education for patients and families that are new to the mental health system. Interventions to prevent a further episodes of psychosis (a "relapse") and strategies that encourage a return to normal vocation and social activity are a priority. There is a concept of phase specific treatment for acute, early recovery and late recovery periods in the first episode of psychosis.

**Early detection function**

Interventions aimed at improving the detection and engagement of those early in the course of their psychotic conditions. Key tasks include being aware of early signs of psychosis and improving pathways into treatment. Teams provide information and education to the general public and assist GPs with recognition and response to those with suspected signs, for example: EPPIC’s Youth Access Team (YAT) (Melbourne); OPUS (Denmark); TIPS (Norway); REDIRECT (Birmingham); LEO CAT (London).

**Prodrome or "at risk mental state" clinics**

There are specialist services for those with subclinical symptoms of psychosis or other strong indicators of risk of transition to psychosis. The Pace Clinic in Melbourne, Australia, is considered one of the origins of this strategy, along with the Institute of Psychiatry based service OASIS in South London, Yale Medical School based clinic, PRIME, The Center of Prevention and Evaluation (COPE) at the Columbia University Medical Center in New York City, the Recognition and Prevention Program based at the Zucker Hillside Hospital in Glen Oaks, New York, and the NAPLS site based at the Emory University Department of Clinical Psychology in Atlanta, Georgia. These services are able to reliably identify those at high risk of developing psychosis and are beginning to publish encouraging outcomes from randomised controlled trials that reduce the chances of becoming psychotic, including evidence that psychological therapy and high doses of fish oil have a role in the prevention of psychosis.
History

Early intervention in psychosis is a preventative approach for psychosis that has evolved as contemporary recovery views of psychosis and schizophrenia have gained acceptance. It subscribes to a "post Kraepelin" concept of schizophrenia, challenging the current assumptions originally promoted by Emil Kraepelin in the 19th century, that schizophrenia (or dementia praecox) was a condition move toward with a progressing and deteriorating course. Psychosis is now formulated within a diathesis–stress model, allowing a more hopeful view of prognosis, and expects full recovery for those with early emerging psychotic symptoms. It is more aligned with psychosis as continuum (such as with the concept of schizotypy) with multiple contributing factors, rather than schizophrenia as simply a neurobiological disease.

Within this changing view of psychosis and schizophrenia, the model has developed from a divergence of several different ideas, and from a number of sites beginning with the closure of psychiatric institutions signaling move toward community based care. In 1986, the Northwick Park study discovered an association between delays to treatment and disability, questioning the service provision for those with their first episode of schizophrenia. In the 1990s, evidence began to emerge that cognitive behavioural therapy was an effective treatment for delusions and hallucinations. The next step came with the development of the EPPIC early detection service in Melbourne, Australia in 1996 and the prodrome clinic led by Alison Yung. This service was an inspiration to other services, such as the West Midlands IRIS group, including the consumer non-governmental organisation Rethink; the TIPS early detection randomised control trial in Norway; and the Danish OPUS trial. In 2001, the United Kingdom Department of Health called the development of early psychosis teams "a priority". The International Early Psychosis Association, founded in 1998, issued an international consensus declaration together with the World Health Organisation in 2004. Clinical practice guidelines have been written by consensus.

Clinical outcome evidence

An early psychosis approach has been shown in formal studies to reduce the severity of symptoms, improve relapse rates, and decrease the use of inpatient care, in comparison to standard care, at 18 months follow up. These studies also clearly show greater levels of user satisfaction with the service. Although the evidence for an ongoing positive impact has yet to be established, some have noted that the underlying assumptions and lack of evidence for the current late intervention standard service approaches make the rationale early intervention "overwhelming".

The earlier 2006 Cochrane review continues to report a lack of strong research evidence for specific early detection and early intervention programmes, although it does acknowledge the need to intervene earlier for those with psychosis. Since that time, the emerging evidence on treatment outcome for early psychosis is positive.
Current literature on cost

Evidence from the United Kingdom suggests that the costs of an early psychosis service are considerably less compared to standard care with one year costs for early psychosis teams (£9,422) two thirds the cost of standard teams (£14,394). This is maintained at Year 3 and is thought to be due to the reduced inpatient costs with the more intensive community follow up provided by early psychosis services.

An Australian historical comparison of direct health costs found a clear economic advantage for an early psychosis approach compared to standard care, at 12 month follow up. Another report, commissioned by Orygen Research Centre in Melbourne, concludes: EI not only costs nearly AU$2000 less per person annually than TAU (treatment as usual) in trial-related costs, it also saves nearly AU$1500 in health system and other financial costs...total saving to society of nearly AU$9000 per patient per year. This does not take into account the potential benefits of EI in reducing suicides and positive impact on vocational outcomes.

Reform of mental health services

United Kingdom

The United Kingdom has probably made the most significant service reform with their adoption of early psychosis teams, with early psychosis now considered as an integral part of comprehensive community mental health services. The Mental Health Policy Implementation Guide outlines service specifications and forms the basis of a newly developed fidelity tool. There is a requirement for services to reduce the duration of untreated psychosis, as this has been shown to be associated with better long term outcome. The implementation guideline recommends:

- 14 to 35 year age entry criteria
- First three years of psychotic illness
- Aim to reduce the duration of untreated psychosis to less than 3 months
- Maximum caseload ratio of 1 care coordinator to 10-15 clients
- For every 250,000 (depending on population characteristics), one team
- Total caseload 120 to 150
- 1.5 doctors per team
- Other specialist staff to provide specific evidence based interventions

Australia and New Zealand

Services have spread from the origin founding EPPI-C initiative in Melbourne (Victoria, Australia) since the 1990s.

New Zealand has operated significant early psychosis teams for more than ten years, following the inclusion of early psychosis in a mental health policy document in 1997.
There is a national early psychosis professional group, New Zealand Early Intervention in Psychosis Steering Group, organising training events and producing local resources. Scandinavia

Early psychosis programmes have continued to develop from the original TIPS services in Norway and the OPUS randomised trial in Denmark.

**North America**

Canada has extensive coverage across most provinces, including established clinical services and comprehensive academic research in British Columbia (Vancouver), Alberta (EPT in Calgary), and Ontario (PEPP, FEPP). In the United States, the Early Assessment and Support Alliance is implementing early psychosis intervention statewide.

**Asia**

The first meeting of the Asian Network of Early Psychosis (ANEPA) was held in 2004. There are now established services in Singapore and Hong Kong.

**Delusional disorder**

Delusional disorder is a psychiatric diagnosis denoting a psychotic mental disorder that is characterized by holding one or more non-bizarre delusions in the absence of any other significant psychopathology. Non-bizarre delusions are fixed beliefs that are certainly and definitely false, but that could possibly be plausible, for example, someone who thinks he or she is under police surveillance. For the diagnosis to be made, auditory and visual hallucinations cannot be prominent, though olfactory or tactile hallucinations related to the content of the delusion may be present.

To be diagnosed with delusional disorder, the delusion or delusions cannot be due to the effects of a drug, medication, or general medical condition, and delusional disorder cannot be diagnosed in an individual previously diagnosed with schizophrenia. A person with delusional disorder may be high functioning in daily life and may not exhibit odd or bizarre behavior aside from these delusions. The Diagnostic and Statistical Manual of Mental Disorders (DSM) defines six subtypes of the disorder characterized as erotomanic (believes that someone famous is in love with him/her), grandiose (believes that he/she is the greatest, strongest, fastest, most intelligent person ever), jealous (believes that the love partner is cheating on him/her), persecutory (believes that someone is following him/her to do some harm in some way), somatic (believes that he/she has a disease or medical condition), and mixed, i.e., having features of more than one subtype. Delusions also occur as symptoms of many other mental disorders, especially the other psychotic disorders.

The DSM-IV, and psychologists, generally agree that personal beliefs should be evaluated with great respect to complexity of cultural and religious differences since some cultures have widely accepted beliefs that may be considered delusional in other cultures. Specifically, to be a "delusion," a belief must be sustained despite what almost everyone
else believes, and not be one ordinarily accepted by other members of the person’s culture or subculture (e.g., it is not an article of religious faith).

**Indicators of a delusion**

The following can indicate a delusion:

- The patient expresses an idea or belief with unusual persistence or force.
- That idea appears to exert an undue influence on the patient’s life, and the way of life is often altered to an inexplicable extent.
- Despite his/her profound conviction, there is often a quality of secretiveness or suspicion when the patient is questioned about it.
- The individual tends to be humorless and oversensitive, especially about the belief.
- There is a quality of centrality: no matter how unlikely it is that these strange things are happening to him, the patient accepts them relatively unquestioningly.
- An attempt to contradict the belief is likely to arouse an inappropriately strong emotional reaction, often with irritability and hostility.
- The belief is, at the least, unlikely, and out of keeping with the patient’s social, cultural and religious background.
- The patient is emotionally over-invested in the idea and it overwhelms other elements of their psyche.
- The delusion, if acted out, often leads to behaviors which are abnormal and/or out of character, although perhaps understandable in the light of the delusional beliefs.
- Individuals who know the patient observe that the belief and behavior are uncharacteristic and alien.

**Features**

The following features are found:

- It is a primary disorder.
- It is a stable disorder characterized by the presence of delusions to which the patient clings with extraordinary tenacity.
- The illness is chronic and frequently lifelong.
- The delusions are logically constructed and internally consistent.
- The delusions do not interfere with general logical reasoning (although within the delusional system the logic is perverted) and there is usually no general disturbance of behavior. If disturbed behavior does occur, it is directly related to the delusional beliefs.
- The individual experiences a heightened sense of self-reference. Events which, to others, are nonsignificant are of enormous significance to him or her, and the atmosphere surrounding the delusions is highly charged.

**Types**
Diagnosis of a specific type of delusional disorder can sometimes be made based on the content of the delusions. The Diagnostic and Statistical Manual of Mental Disorders (DSM) enumerates six types:

- **Erotomanic Type (erotomania):** delusion that another person is in love with the individual, quite frequently a famous person. The individual may breach the law as he/she tries to obsessively make contact with the desired person.
- **Grandiose Type:** delusion of inflated worth, power, knowledge, identity or believes himself/herself to be a famous person, claiming the actual person is an impostor or an impersonator.
- **Jealous Type:** delusion that the individual's sexual partner is unfaithful when it is untrue. The patient may follow the partner, check text messages, emails, phone calls etc. in an attempt to find "evidence" of the infidelity.
- **Persecutory Type:** This delusion is the most common. It includes the belief that the person (or someone to whom the person is close) is being malevolently treated in some way. The patient may believe that he/she has been drugged, spied-on, harassed and so on and may seek "justice" by making police reports, taking court action or even acting violently.
- **Somatic Type:** delusions that the person has some physical defect or general medical condition (for example, see delusional parasitis).

(Lippincott, 2008).

Mixed Type: delusions with characteristics of more than one of the above types but with no one theme predominateing.

A diagnosis of 'unspecified type' may also be given if the delusions fall into several or none of these categories.

**Causes**

When delusional disorders occur late in life they suggest a hereditary predisposition. Researchers also suggest that these disorders are the result of early childhood experiences with an authoritarian family structure. According to other researchers, any person with a sensitive personality is particularly vulnerable to developing a delusional disorder.

Although its exact cause is unknown, it is believed that genetic, biochemical and environmental factors play a significant role in the development of delusional disorder.

**Diagnosis**

The symptoms expressed by a delusional disorder can also be part of a much more serious problem, such as bipolar disorder or schizophrenia, therefore diagnosing the delusional disorder is conducted partially by process of elimination. This occurs because delusions can be part of many other illnesses including dementia, schizophrenia and schizoaffective disorder. They may also be part of a response to physical, medical conditions, or reactions when drugs are ingested.
Interviews are useful tools to obtain information about the patient's life situation and past history to help identifying the delusional disorder. Clinicians may review earlier medical records, with the patient's permission. Clinicians also interview the patient’s immediate family. This is a very helpful measure in determining the presence of delusions. The mental status examination is used to assess the patient’s memory, concentration, and understanding the individual’s situation and logical thinking.

Another psychological test used in the diagnosis of the delusional disorder is the Peters Delusion Inventory (PDI) which focuses on identifying and understanding delusional thinking. However, this test is more likely used in research than in clinical practice.

Treatment

Treatment of delusional disorders includes a combination of drug therapy and psychotherapy although it is a challenging disorder to treat for many reasons such as the patient’s denial that they have a problem of a psychological nature.

Atypical antipsychotic medications (also known as novel or newer-generation) are used in the treatment of delusional disorder as well as in schizophrenic disorders. Some examples of such medications are risperidone (Risperdal), quetiapine (Seroquel), and olanzapine (Zyprexa). These medications work by blocking postsynaptic dopamine receptors and reduce the incidence of psychotic symptoms including hallucinations and delusions. They also relieve anxiety and agitation. When these drugs are tried but the symptoms do not improve, other types of antipsychotics may be prescribed. Some examples are: fluphenazine decanoate and fluphenazine enanthate. One very effective drug in delusional disorders is also pimozide.

In some cases agitation may occur as a response to severe or harsh confrontation when dealing with the existence of the delusions. If agitation occurs, different antipsychotics can be administered to conclude its outbreak. For instance, an injection of haloperidol (Haldol) can decrease anxiety and slow behavior, it is often combined with medications including lorazepam (Ativan).

In cases when severely ill patients do not respond to standard treatment, Clozapine may be prescribed although it may cause drowsiness, sedation, excessive salivation, tachycardia, dizziness, seizures and agranulocytosis.

To treat long term symptoms, an oral novel antipsychotic is often prescribed on a daily basis. Antidepressants and anxiolytics are also prescribed to control associated symptoms.

Psychotherapy for patients with delusional disorder include cognitive therapy which is conducted with the use of empathy. During the process, the therapist asks hypothetical questions in a form of therapeutic Socratic dialogue. This therapy has been mostly studied in patients with the persecutory type. The combination of pharmacotherapy with cognitive therapy integrates treating the possible underlying biological problems and decreasing the symptoms with psychotherapy as well.
Supportive therapy has also shown to be helpful. Its goal is to facilitate treatment adherence and provide education about the illness and its treatment.

Furthermore, providing social skills training has been applicable to a high number of persons. It should focus on promoting interpersonal competence as well as confidence and comfort when interacting with those individuals perceived as a threat.

Reports have shown successful use of insight-oriented therapy although it may also be contraindicated for delusional disorder. Its goals are to develop therapeutic alliance, containment of projected feelings of hatred, impotence, and badness; measured interpretation as well as the development of a sense of creative doubt in the internal perception of the world. The latter requires the empathy with the patient’s defensive position.

**Schizoaffective disorder**

Schizoaffective disorder is a psychiatric diagnosis that describes a mental disorder characterized by recurring episodes of elevated or depressed mood, or of simultaneously elevated and depressed mood, that alternate with, or occur together with, distortions in perception.

Schizoaffective disorder most commonly affects cognition and emotion. Auditory hallucinations, paranoia, bizarre delusions, or disorganized speech and thinking with significant social and occupational dysfunction are typical. The division into depressive and bipolar types is based on whether the individual has ever had a manic, hypomanic or mixed episode. Symptoms usually begin in early adulthood, which makes diagnosis prior to age 13 rare.

In addition to schizophrenia, schizoaffective disorder is part of a "schizophrenic spectrum" (comparable to the autistic spectrum) that includes schizotypal personality disorder. Each named disorder on this continuum shares symptoms with the others, and some professionals (including the working group for the DSM-5) contend that the boundaries are so unclear that separate labels are not necessarily warranted.

**Overview**

The lifetime prevalence of the disorder is probably less than 1 percent, in the range of 0.5 to 0.8 percent. Diagnosis is based on the patient’s self-reported experiences and observed behavior. No laboratory test for schizoaffective disorder currently exists, though extensive evidence exists for abnormalities in the metabolism of tetrahydrobiopterin (BH4), dopamine, and glutamate in people with schizophrenia and schizoaffective disorders. As a group, individuals with schizoaffective disorder have a more favorable prognosis than those with schizophrenia, but a worse prognosis than those with other mood disorders.
Genetics, early environment, neurobiology, psychological and social processes are important contributory factors. Some recreational and prescription drugs may cause or worsen symptoms. Current research is focused on the role of neurobiology, but no single organic cause has been found.

The mainstay of treatment is antipsychotic medication combined with mood stabilizer medication or antidepressant medication, or both. Psychotherapy, and vocational and social/psychiatric rehabilitation are also important for recovery. In cases where there is risk to self and others, brief involuntary hospitalization may be necessary.

People with schizoaffective disorder are likely to have comorbid conditions, including anxiety disorders and substance abuse. Social problems, such as long-term unemployment, poverty and homelessness, are common. The average life expectancy of people with the disorder is shorter than those without, due to increased physical health problems and a higher suicide rate.

The diagnosis was introduced in 1933 and may be removed in the next iteration of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5), to be published in May 2013.

**Signs and symptoms**

Late adolescence and early adulthood are the peak years for the onset of schizoaffective disorder, although it has been diagnosed (very rarely) in childhood.

Schizoaffective disorder is a mental illness characterized by recurring episodes of mood disorder and psychosis. Psychosis is defined by paranoia, delusions and hallucinations. Mood disorders are defined by discrete periods of clinical depression, mixed episodes and manic episodes. Individuals with the disorder may experience psychotic symptoms before, during or (commonly) after their depressive, mixed or manic episodes.

The illness tends to be difficult to diagnose since the symptoms are similar to other disorders with prominent mood and psychotic symptoms like bipolar disorder with psychotic features, recurrent depression with psychotic features and schizophrenia. By contrast, in schizoaffective disorder, as it is presently defined, psychosis must also occur during periods without mood symptoms. In schizophrenia, mood episodes have been thought to be absent or less prominent than in schizoaffective disorder. Since these differences can be difficult to detect, a firm diagnosis of schizoaffective disorder may thus require an extended period of observation and treatment.

Untreated, the individual with schizoaffective disorder may experience delusions. It should be noted that delusions in schizoaffective disorder are acute manifestations of an active psychosis and are not personality traits; that is, they go away when the psychosis subsides. Manifestations of delusions include the individual being convinced that he or she is Jesus or the Antichrist, has some special purpose or destiny (such as to save the world), or is being monitored, watched or persecuted by something (commonly government agencies), when
in reality they often are not. Individuals may also feel extremely paranoid. Other delusions may include the belief that an external force is controlling the individual’s thought processes. (See thought insertion.)

Hallucinations involving all five senses can also occur in untreated or undertreated schizoaffective disorder. That is, the individual may see, hear, smell, feel or taste things that aren’t there. For example, the individual may see overt visual hallucinations such as monsters, the devil or more subtle ones such as shadowy apparitions. Individuals may hear voices or, in some cases, music. Things may look or sound different. Individuals may also experience strange sensations. These hallucinations may worsen when the individual is intoxicated.

The untreated individual may quickly change their mind about their romantic partner, friends or family if they hear something negative being said about them; as a result they may attack or, conversely, isolate themself from the person or group until they regain normal thoughts.

Comorbid or co-occurring anxiety disorders may also play a role in the subjective experience of schizoaffective disorder and thus may shape the individual’s delusional thought content. For example, the individual may feel anxious, have trouble swallowing, and then believe that outside forces are controlling their throat functions. They may also suffer from various phobias which may also manifest as delusions.

There may be a decline in work or school functioning during episodes of illness. As stated above, individuals with schizoaffective disorder may withdraw socially and become isolated.

The untreated individual may sleep too much, or be unable to sleep.

Difficulties with executive function may also be a problem for individuals with schizoaffective disorder. This may include difficulties with concentration, attention, logical reasoning and impulse control.

Without treatment, the individual with schizoaffective disorder may further worsen in their delusional thought processes.

With comprehensive treatment, many individuals with schizoaffective disorder may recover much, most or even all of their functionality.

**Diagnosis**

Diagnosis is based on the self-reported experiences of the person as well as abnormalities in behavior reported by family members, friends or co-workers to a psychiatrist, psychiatric nurse, social worker or psychologist in a clinical assessment. There is a list of criteria that must be met for someone to be so diagnosed. These depend on both the presence and duration of certain signs and symptoms.
As discussed above, there are several psychiatric illnesses which may present with a similar range of psychotic symptoms; these include bipolar disorder with psychotic features, major depression with psychotic features, schizophrenia, drug intoxication, brief drug-induced psychosis, and schizoaffective disorder. These disorders need to be ruled out before a firm diagnosis of schizoaffective disorder can be made.

An initial assessment includes a comprehensive history and physical examination by a physician. Although there are no biological tests which confirm schizoaffective disorder, tests are carried out to exclude medical illnesses which rarely may be associated with psychotic symptoms. These include blood tests measuring TSH to exclude hypo- or hyperthyroidism, basic electrolytes and serum calcium to rule out a metabolic disturbance, full blood count including ESR to rule out a systemic infection or chronic disease, and serology to exclude syphilis or HIV infection; two commonly ordered investigations are EEG to exclude epilepsy, and a CT scan of the head to exclude brain lesions. It is important to rule out a delirium which can be distinguished by visual hallucinations, acute onset and fluctuating level of consciousness and indicates an underlying medical illness.

Investigations are not generally repeated for relapse unless there is a specific medical indication. These may include serum BSL if olanzapine has previously been prescribed, thyroid function if lithium has previously been taken to rule out hypothyroidism, liver function tests if chlorpromazine has been prescribed, and CPK levels to exclude neuroleptic malignant syndrome. Assessment and treatment are usually done on an outpatient basis; admission to an inpatient facility is considered if there is a risk to self or others.

The most widely-used criteria for diagnosing schizoaffective disorder are from the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders, the current version being DSM-IV-TR.

**DSM-IV-TR criteria**

The following are the revised criteria for a diagnosis of schizoaffective disorder from the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR):

Two (or more) of the following symptoms are present for the majority of a one-month period (or a shorter period of time if symptoms got better with treatment):

- delusions
- hallucinations
- disorganized speech (e.g., frequent derailment or incoherence) which is a manifestation of formal thought disorder
- grossly disorganized behavior (e.g. dressing inappropriately, crying frequently) or catatonic behavior
- negative symptoms—e.g., affective flattening (lack or decline in emotional response), alogia (lack or decline in speech), avolition (lack or decline in motivation), anhedonia (lack or decline in ability to experience pleasure), social
withdrawal (sometimes called social anhedonia). Negative symptoms refers to symptoms that are not present or that are diminished in the affected persons but are normally found in healthy persons.

If the delusions are judged to be bizarre, or hallucinations consist of hearing one voice participating in a running commentary of the individual’s actions or of hearing two or more voices conversing with each other, only that symptom is required to meet criterion A above. The speech disorganization criterion is only met if it is severe enough to substantially impair communication. and at some time during the illness there is either one, two or all three of the following:

- major depressive episode
- manic episode
- mixed episode
- During the illness, delusions or hallucinations were present for a minimum of two weeks, without major mood symptoms.
- For a substantial part of the overall duration of both the active and residual period of the illness, symptoms meeting criteria for a mood episode are present.
- Symptoms are not caused by drug abuse, medication or another medical condition.

**Subtypes**

Two subtypes of schizoaffective disorder exist and may be noted in a diagnosis based on the mood component of the disorder:

**Bipolar type**

if the disturbance includes

- a manic episode
- a mixed episode

Major depressive episodes usually, but not always, also occur in the bipolar subtype, however they are not required for DSM-IV diagnosis.

**Depressive type**

The depressive type is noted when the disturbance includes major depressive episodes exclusively.

This subtype applies if major depressive episodes only (and no manic or mixed episodes) are part of the presentation.

**Controversies and research directions**
Citing poor interrater reliability, some psychiatrists have totally contested the concept of schizoaffective disorder as a separate entity. The categorical distinction between mood disorders and schizophrenia, known as the Kraepelinian dichotomy, has also been challenged by data from genetic epidemiology. Consequently, some researchers have disputed that the term "schizoaffective disorder" refers to a well defined condition, and have recommended that the term be removed from or amended in future diagnostic manuals.

In April 2009, the DSM-5 Psychotic Disorders Work Group headed by psychiatrist William T. Carpenter of the University of Maryland, College Park School of Medicine, reported that they will be "developing new criteria for schizoaffective disorder to improve reliability and face validity," and that they will be "determining whether the dimensional assessment of mood will justify a recommendation to drop schizoaffective disorder as a diagnostic category." Speaking at the May 2009 annual conference of the American Psychiatric Association, Carpenter said,

"We had hoped to get rid of schizoaffective [disorder] as a diagnostic category because we don't think it's valid and we don't think it's reliable. On the other hand, we think it's absolutely indispensable to clinical practice."

**Cause**

Although the causes of schizoaffective disorder are unknown, it is suspected that this diagnosis represents a heterogeneous group of individuals, some with aberrant forms of schizophrenia and some with very serious forms of mood disorders. There is little evidence that schizoaffective disorder is a distinct variety of psychotic illness. Consequently, the disorder appears to be comorbid or (co-occurring) with schizophrenia and mood disorder. Schizoaffective disorder thus appears to exist on a continuum in-between schizophrenia and severe bipolar disorder and severe recurrent unipolar depression. It follows then that the etiology is probably more similar to that of schizophrenia in some cases and more similar to severe mood disorders in other cases.

Many different genes may be contributing to the genetic risk of acquiring this illness. In addition, many different biological and environmental factors are believed to interact with the person's genes in ways which can increase or decrease the person's risk for developing schizoaffective disorder. Schizophrenia spectrum disorders (of which schizoaffective disorder is a part) have been marginally linked to advanced paternal age at the time of conception, a common cause of mutations.

The physiology of patients diagnosed with schizoaffective disorder appears to be similar but not identical to that of those diagnosed with schizophrenia and severe bipolar disorder.

**Substance abuse**

A clear causal connection between drug use and psychotic spectrum disorders, including schizoaffective disorder, has been difficult to prove. The two most often used explanations
for this are "substance use causes schizoaffective disorder" and "substance use is a consequence of schizoaffective disorder", and they both may be correct. In the case of marijuana or cannabis, however, evidence is mounting that it can play a role in the development and morbidity of psychotic disorders, including schizoaffective disorder. For example, a 2007 meta-analysis showed that cannabis use is statistically associated with a dose-dependent increase in risk of development of psychotic disorders, including schizoaffective disorder. Moreover, a 2005 meta-analysis found that cannabis use is a significant independent risk factor for developing psychotic symptoms and psychosis. A 2009 Yale study stated that it is clear

"that in individuals with an established psychotic disorder, cannabinoids can exacerbate symptoms, trigger relapse, and have negative consequences on the course of the illness."

On the other hand, a meta-analysis published in 2008 concluded that "Confidence that most associations reported were specifically due to cannabis is low. Despite clinical opinion, it remains important to establish whether cannabis is harmful, what outcomes are particularly susceptible, and how such effects are mediated."

However, despite increases in cannabis consumption in the 1960s and 1970s in western society, rates of psychotic disorders generally remained relatively stable. Also, Sweden and Japan, where self-reported marijuana use is very low, do not have lower rates of psychosis than the U.S. and Canada do. For the theory of causality to be correct, other factors which are thought to contribute to psychosis would have to have converged almost flawlessly to mask the effect of increased cannabis usage. However, there may be other confounding factors, including social structure, family structure, typical diet, and ethnic genetic makeup that preclude a clear 1:1 comparison – either pro or con – between populations from different countries.

There is little evidence to suggest that other drugs including alcohol cause schizoaffective disorder, or that psychotic individuals choose specific drugs to self-medicate; there is some support for the theory that they use drugs to cope with unpleasant states such as depression, anxiety, boredom and loneliness. However, regarding psychosis itself, it is well understood that methamphetamine and cocaine use can result in methamphetamine- or cocaine-induced psychosis which presents very similar symptomatology and may persist even when users remain abstinent. The same can also be said for alcohol-induced psychosis, though to a somewhat lesser extent.

**Management**

Treatment for schizoaffective disorder consists of a combination of medicine, psychotherapy and psychosocial rehabilitation focused on recovery or symptom management.

A licensed psychiatrist will prescribe (usually combinations of) medicine for the individual. Each person responds differently to medication.
The only medicine that is FDA-approved for Schizoaffective Disorder is paliperidone (Invega). For psychotic symptoms neuroleptic medications such as risperidone may be used.

For manic symptoms, mood stabilizer medications may be prescribed along with a neuroleptic. Examples are:

- Lithium salt (Lithium)
- Valproate semisodium (Depakote ER)
- Carbamazepine (Tegretol)

For depression, antidepressant medications may be prescribed along with a neuroleptic. Examples are:

- SSRI antidepressants (includes Prozac and Zoloft among others)
- Lamictal (a mood stabilizer with antidepressant properties)

In schizoaffective individuals with manic symptoms, combining lithium, carbamazepine, or valproate with a neuroleptic has been shown to be superior to neuroleptics alone. Lithium-neuroleptic combinations, however, may produce severe extrapyramidal reactions or confusion in some patients.

When lithium is not effective or well tolerated in manic individuals with schizoaffective disorder, Tegretol or Depakote are frequently used. Granulocytopenia can occur during the first few weeks of carbamazepine treatment, and neuroleptic blood levels may be decreased substantially due to hepatic enzyme induction. Valproate can, in rare cases, cause liver toxicity and platelet dysfunction. Calcium channel blockers such as verapamil may also be an effective treatment for manic symptoms but are seldom prescribed for that purpose. The degree of benefit for an individual should be considered carefully, as each of these medications carries its own risks.

Benzodiazepines such as Ativan and Klonopin are effective adjunctive treatment agents for acute manic symptoms, but long-term use may result in dependency.

In schizoaffective individuals with depressive symptoms, an antidepressant (for example, Prozac or other SSRIs) may be prescribed with a neuroleptic. The SNRI antidepressants and Wellbutrin tend not to be prescribed in schizoaffective disorder because they may cause mixed episode symptoms and induce psychosis, respectively.

The anticonvulsant Lamictal is gaining prominence in treating depressed schizoaffective individuals because antidepressants appear to increase the risk of mood cycling in some individuals, which is a safety concern.

Often a sleeping pill will be prescribed initially to allow the individual rest from his or her anxiety, delusions or hallucinations. Long-term use of sleeping medications can, however,
cause dependence and can also cause delusions and hallucinations thereby exacerbating psychosis.

**Complications**

Complications are similar to those for schizophrenia and major mood disorders. These include:

- Problems following medical treatment and therapy
- Use of unsanctioned drugs in an attempt to self-medicate
- Short-term side effects and problems arising from long-term use of prescribed medications, including drug interactions.
- Problems resulting from manic behavior (for example, spending sprees, sexual indiscretion)
- Suicidal behavior due to depressive or psychotic symptoms

**Epidemiology**

Estimates of the prevalence of schizoaffective disorder vary widely, but schizoaffective manic patients appear to comprise 3-5% of psychiatric admissions to typical clinical centers. At one point it was widely believed that schizoaffective disorder was associated with increased risk of mood disorders in relatives. This may have been because of the number of patients with psychotic mood disorders who were included in schizoaffective study populations.

The current diagnostic criteria define a group of individuals with a mixed genetic picture. They are more likely to have schizophrenic relatives than individuals with mood disorders but more likely to have relatives with mood disorders than individuals with schizophrenia.

**History**

The term schizoaffective psychosis was introduced by the American psychiatrist Jacob Kasanin in 1933 to describe an episodic psychotic illness with predominant affective symptoms, that was thought at the time to be a good-prognosis schizophrenia. Kasanin's concept of the illness was influenced by the psychoanalytic teachings of Adolf Meyer and Kasanin postulated that schizoaffective psychosis was caused by "emotional conflicts" of a "mainly sexual nature" and that psychoanalysis "would help prevent the recurrence of such attacks." He based his description on a case study of nine individuals.

Other psychiatrists, before and after Kasanin, have made scientific observations of schizoaffective disorder based on assumptions of a biological and genetic etiology of the illness. In 1863, German psychiatrist Karl Kahlbaum (1828–1899) described schizoaffective disorders as a separate group in his vesania typica circularis. Kahlbaum distinguished between cross-sectional and longitudinal observations. (Cross-sectional refers to observation of a single, specific episode of the illness, for example, one episode of psychotic depression; while longitudinal refers to long-term observation of many distinct
episodes [similar or different] often occurring over the span of years.) In 1920, psychiatrist Emil Kraepelin (1856–1926), the founder of contemporary scientific psychiatry, observed a "great number" of cases that had characteristics of both groups of psychoses that he originally posited were two distinct and separate illnesses, dementia praecox (now called schizophrenia) and manic depressive insanity (now called bipolar disorder and recurrent depression).

Kraepelin acknowledged that "there are many overlaps in this area", that is, the area between schizophrenia and severe mood disorders. In 1939, psychiatrist Kurt Schneider (1887–1967) can be said to have been the first to begin to conceptualize the different forms that schizoaffective disorders can take since he observed "concurrent and sequential types". The concurrent type of illness he referred to is a longitudinal course of illness with episodes of mood disorder and psychosis occurring predominantly at the same time; while his sequential type refers to a longitudinal course predominantly marked by alternating mood and psychotic episodes.) Schneider described schizoaffective disorders as "cases in-between" the traditional Kraepelinian dichotomy of schizophrenia and mood disorders.

The historical phenomenological observation that schizoaffective disorder is an overlap of schizophrenia and severe mood disorders has more recently been assumed to be explained by genes for both illnesses being present in individuals with schizoaffective disorder. But recent research shows that schizophrenia and severe mood disorders appear to share common genes and polygenic variations also.

Schizoaffective disorder was included as a subtype of schizophrenia in DSM-I and DSM-II, though research showed a schizophrenic cluster of symptoms in individuals with a family history of mood disorders whose illness course, other symptoms and treatment outcome were otherwise more akin to bipolar disorder than to schizophrenia. DSM-III placed schizoaffective disorder in "Psychotic Disorders Not Otherwise Specified" before being formally recognized in DSM-III-R. DSM-III-R included its own diagnostic criteria as well as the subtypes, bipolar and depressive. In DSM-IV, published in 1994, schizoaffective disorders belonged to the category "Other Psychotic Disorders" and included almost the same criteria and the same subtypes of illness as DSM-III-R, with the addition of mixed bipolar symptomatology.

### Monothematic delusion

A monothematic delusion is a delusional state that only concerns one particular topic. This is contrasted by what is sometimes called multi-thematic or polythematic delusions where the person has a range of delusions (typically the case of schizophrenia). These disorders can occur within the context of schizophrenia or dementia or they can occur without any other signs of mental illness. When these disorders are found outside the context of mental illness, they are often caused by organic dysfunction as a result of traumatic brain injury, stroke, or neurological illness.

People who suffer from these delusions as a result of organic dysfunction often do not suffer from any obvious intellectual deficiency nor do they have any other symptoms.
Additionally, a few of these people even have some awareness that their beliefs are bizarre, yet they cannot be persuaded that their beliefs are false.

**Types**

The delusions that fall under this category are:

- Capgras delusion: the belief that (usually) a close relative or spouse has been replaced by an identical-looking impostor.
- Fregoli delusion: the belief that various people who the believer meets are actually the same person in disguise.
- Intermetamorphism: the belief that people in one’s environment swap identities with each other whilst maintaining the same appearance.
- Subjective doubles: a person believes there is a doppelgänger or double of him or herself carrying out independent actions.
- Cotard delusion: the belief that oneself is dead or does not exist; sometimes coupled with the belief that one is putrefying or missing internal organs.
- Mirrored self-misidentification: the belief that one’s reflection in a mirror is some other person.
- Reduplicative paramnesia: the belief that a familiar person, place, object or body part has been duplicated. For example, a person may believe that they are in fact not in the hospital to which they were admitted, but in an identical-looking hospital in a different part of the country.
- Somatoparaphrenia: the delusion where one denies ownership of a limb or an entire side of one’s body (often connected with stroke).

Note that some of these delusions are sometimes grouped under the umbrella term of delusional misidentification syndrome.

**Causes**

Current cognitive neuropsychology research points toward a two-factor approach to the cause of monothematic delusions. The first factor being the anomalous experience—often a neurological defect—which leads to the delusion and the second factor being an impairment of the belief formation cognitive process.

For example, one of these first factors, several studies point toward Capgras delusion being the result of a disorder of the affect component of face perception. As a result, while the person can recognize their spouse (or other close relation) they do not feel the typical emotional reaction and thus the spouse does not seem like the person they once knew.

As studies have shown, these neurological defects are not enough on their own to cause delusional thinking. An additional second factor, a bias or impairment of the belief formation cognitive process is required to solidify and maintain the delusion. Since we do not currently have a solid cognitive model of the belief formation process, this second factor is still somewhat of an unknown.
Some research has shown that delusional people are more prone to jumping to conclusions and thus they would be more likely to take their anomalous experience as veridical and make snap judgments based on these experiences. Additionally, studies have shown that they are more prone to making errors due to matching bias—indicative of a tendency to try and confirm the rule. These two judgment biases help explain how delusion prone people could grasp onto extreme delusions and be very resistant to change.

Researchers claim this is enough to explain the delusional thinking. However other researchers still argue that these biases are not enough to explain why they remain completely impervious to evidence over time. They believe that there must be some additional unknown neurological defect in the patient’s belief system (probably in the right hemisphere).

**Paranoia**

Paranoia is a thought process believed to be heavily influenced by anxiety or fear, often to the point of irrationality and delusion. Paranoid thinking typically includes persecutory beliefs concerning a perceived threat towards oneself. Historically, this characterization was used to describe any delusional state.

**History**

The word paranoia comes from the Greek "παράνοια" (paranoia), "madness" and that from "παρά" (para), "beside, by" + "νοος" (noos), "mind". The term was used to describe a mental illness in which a delusional belief is the sole or most prominent feature. In original attempt at classifying different forms of mental illness, Kraepelin used the term pure paranoia to describe a condition where a delusion was present, but without any apparent deterioration in intellectual abilities and without any of the other features of dementia praecox, the condition later renamed "schizophrenia". Notably, in his definition, the belief does not have to be persecutory to be classified as paranoid, so any number of delusional beliefs can be classified as paranoia. For example, a person who has the sole delusional belief that he is an important religious figure would be classified by Kraepelin as having 'pure paranoia'. According to Phelan, M. Padraig, W. Stern, J (2000) paranoia and paraphrenia are debated entities that were detached from dementia praecox by Kraepelin, who explained paranoia as a continuous systematized delusion arising much later in life with no presence of either hallucinations or a deteriorating course, paraphrenia as an identical syndrome to paranoia but with hallucinations. Even at the present time, a delusion need not be suspicious or fearful to be classified as paranoid. A person might be diagnosed as a paranoid schizophrenic without delusions of persecution, simply because their delusions refer mainly to themselves.

Use in modern psychiatry

In the DSM-IV-TR, paranoia is diagnosed in the form of:
- paranoid personality disorder
- paranoid schizophrenia (a subtype of schizophrenia)
- the persecutory type of delusional disorder, which is also called "querulous paranoia" when the focus is to remedy some injustice by legal action.

According to clinical psychologist P. J. McKenna, "As a noun, paranoia denotes a disorder which has been argued in and out of existence, and whose clinical features, course, boundaries, and virtually every other aspect of which is controversial. Employed as an adjective, paranoid has become attached to a diverse set of presentations, from paranoid schizophrenia, through paranoid depression, to paranoid personality—not to mention a motley collection of paranoid 'psychoses', 'reactions', and 'states'—and this is to restrict discussion to functional disorders. Even when abbreviated down to the prefix para-, the term crops up causing trouble as the contentious but stubbornly persistent concept of paraphrenia."

**Personality disorder**

Personality disorders, formerly referred to as character disorders, are a class of personality types and behaviors that the American Psychiatric Association (APA) defines in terms of supportive psychotherapy as "an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the culture of the individual who exhibits it". Personality disorders are noted on Axis II of the Diagnostic and Statistical Manual of Mental Disorders or DSM-IV-TR (fourth edition, text revision) of the American Psychiatric Association.

Personality disorders are also defined by the International Statistical Classification of Diseases and Related Health Problems (ICD-10), which is published by the World Health Organization. Personality disorders are categorized in ICD-10 Chapter V: Mental and behavioural disorders, specifically under Mental and behavioral disorders: 28F60-F69.29 Disorders of adult personality and behavior.

These behavioral patterns in personality disorders are typically associated with severe disturbances in the behavioral tendencies of an individual, usually involving several areas of the personality, and are nearly always associated with considerable personal and social disruption. Additionally, personality disorders are inflexible and pervasive across many situations, due in large part to the fact that such behavior is ego-syntonic (i.e. the patterns are consistent with the ego integrity of the individual) and are, therefore, perceived to be appropriate by that individual. This behavior can result in maladaptive coping skills, which may lead to personal problems that induce extreme anxiety, distress and depression.

The onset of these patterns of behavior can typically be traced back to early adolescence and the beginning of adulthood and, in rarer instances, childhood. General diagnostic guidelines applying to all personality disorders are presented below; supplementary descriptions are provided with each of the subtypes.
Diagnosis of personality disorders can be very subjective; however, inflexible and pervasive behavioral patterns often cause serious personal and social difficulties, as well as a general functional impairment. Rigid and ongoing patterns of feeling, thinking and behavior are said to be caused by underlying belief systems and these systems are referred to as fixed fantasies or "dysfunctional schemata" (cognitive modules).

**Classification**

**World Health Organization**

**ICD-10 groups for (F60.) Specific personality disorders:**

**Cluster A**
- (F60.0) Paranoid personality disorder
- (F60.1) Schizoid personality disorder

**Cluster B**
- (F60.2) Antisocial personality disorder
- (F60.3) Borderline personality disorder
- (F60.4) Histrionic personality disorder

**Cluster C**
- (F60.5) Obsessive–compulsive personality disorder
- (F60.6) Anxious (avoidant) personality disorder
- (F60.7) Dependent personality disorder

**(F60.8) Other specific personality disorders**
- Narcissistic personality disorder
- Passive-aggressive personality disorder

**(F60.9) Personality disorder, unspecified**

**(F61.) Mixed and other personality disorders**

The DSM-IV lists ten personality disorders, grouped into three clusters in Axis II. The DSM also contains a category for behavioral patterns that do not match these ten disorders, but nevertheless exhibit characteristics of a personality disorder. This category is labeled Personality disorder not otherwise specified.
Cluster A (odd or eccentric disorders)

- Paranoid personality disorder (DSM-IV code 301.0): characterized by irrational suspicions and mistrust of others.
- Schizoid personality disorder (DSM-IV code 301.20): lack of interest in social relationships, seeing no point in sharing time with others, anhedonia, introspection.
- Schizotypal personality disorder (DSM-IV code 301.22): characterized by odd behavior or thinking.

Cluster B (dramatic, emotional or erratic disorders)

- Antisocial personality disorder (DSM-IV code 301.7): a pervasive disregard for the law and the rights of others.
- Borderline personality disorder (DSM-IV code 301.83): extreme "black and white" thinking, instability in relationships, self-image, identity and behavior often leading to self-harm and impulsivity. Borderline personality disorder is diagnosed in 3 times as many females as males.
- Histrionic personality disorder (DSM-IV code 301.50): pervasive attention-seeking behavior including inappropriately seductive behavior and shallow or exaggerated emotions.
- Narcissistic personality disorder (DSM-IV code 301.81): a pervasive pattern of grandiosity, need for admiration, and a lack of empathy.

Cluster C (anxious or fearful disorders)

- Avoidant personality disorder (DSM-IV code 301.82): social inhibition, feelings of inadequacy, extreme sensitivity to negative evaluation and avoidance of social interaction.
- Dependent personality disorder (DSM-IV code 301.6): pervasive psychological dependence on other people.
- Obsessive-compulsive personality disorder (not the same as obsessive-compulsive disorder) (DSM-IV code 301.4): characterized by rigid conformity to rules, moral codes and excessive orderliness.

Appendix B: Criteria Sets and Axes Provided for Further Study

Appendix B contains the following disorders. They are still widely considered amongst psychiatrists as being valid disorders, for example by Theodore Millon.

- Depressive personality disorder - is a pervasive pattern of depressive cognitions and behaviors beginning by early adulthood.
- Passive-aggressive personality disorder (negativistic personality disorder) - is a pattern of negative attitudes and passive resistance in interpersonal situations.
Deleted

The following disorders are still considered to be valid disorders by Millon. They were in DSM-III-R but were deleted from DSM-IV. Both appeared in an appendix entitled "Proposed diagnostic categories needing further study", and so did not have any concrete diagnostic criteria.

- Sadistic personality disorder - is a pervasive pattern of cruel, demeaning and aggressive behavior.
- Self-defeating personality disorder (masochistic personality disorder) - is characterised by behaviour consequently undermining the person's pleasure and goals.

Cause

A study of almost 600 male college students, averaging almost 30 years of age and who were not drawn from a clinical sample, examined the relationship between childhood experiences of sexual and physical abuse and currently reported personality disorder symptoms. Childhood abuse histories were found to be definitively associated with greater levels of symptomatology. Severity of abuse was found to be statistically significant, but clinically negligible, in symptomatology variance spread over Cluster A, B and C scales.

Child abuse and neglect consistently evidence themselves as antecedent risks to the development of personality disorders in adulthood. In the following study, efforts were taken to match retrospective reports of abuse with a clinical population that had demonstrated psychopathology from childhood to adulthood who were later found to have experienced abuse and neglect. The sexually abused group demonstrated the most consistently elevated patterns of psychopathology. Officially verified physical abuse showed an extremely strong role in the development of antisocial and impulsive behavior. On the other hand, cases of abuse of the neglectful type that created childhood pathology were found to be subject to partial remission in adulthood.

Diagnosis

According to ICD-10, the diagnosis of a personality disorder must satisfy the following general criteria, in addition to the specific criteria listed under the specific personality disorder under consideration:

There is evidence that the individual's characteristic and enduring patterns of inner experience and behaviour as a whole deviate markedly from the culturally expected and accepted range (or "norm"). Such deviation must be manifest in two or more of the following areas:

- cognition (i.e., ways of perceiving and interpreting things, people, and events; forming attitudes and images of self and others);
affectivity (range, intensity, and appropriateness of emotional arousal and response);
- control over impulses and gratification of needs;
- manner of relating to others and of handling interpersonal situations.

The deviation must manifest itself pervasively as behaviour that is inflexible, maladaptive, or otherwise dysfunctional across a broad range of personal and social situations (i.e., not being limited to one specific "triggering" stimulus or situation).
- There is personal distress, or adverse impact on the social environment, or both, clearly attributable to the behaviour referred to in criterion 2.
- There must be evidence that the deviation is stable and of long duration, having its onset in late childhood or adolescence.
- The deviation cannot be explained as a manifestation or consequence of other adult mental disorders, although episodic or chronic conditions from sections F00-F59 or F70-F79 of this classification may coexist with, or be superimposed upon, the deviation.
- Organic brain disease, injury, or dysfunction must be excluded as the possible cause of the deviation. (If an organic causation is demonstrable, category F07.- should be used.)

Normal personality and personality disorders

The issue of the relationship between normal personality and personality disorders is one of the important issues in personality and clinical psychology. The personality disorders classification (DSM IV TR and ICD-10) follow categorical approach whereas the trait personality approach follows the dimensional approach. Thomas Widiger has contributed to this debate significantly. He discussed the constraints of the categorical approach and argued for the dimensional approach to the personality disorders. The Five Factor Model of personality has been proposed as a alternative to the classification of personality disorders. Many studies across cultures have explored the relationship between personality disorders and the Five Factor Model. This talks about Five-factor translations of DSM-III-R and DSM-IV personality disorders and expounds relevance of the FFM to a variety of patient populations, including patients with borderline personality disorder, narcissism, and bulimia nervosa as well as substance abusers, psychopaths, and sex offenders.

In children and adolescents

Early stages and preliminary forms of personality disorders need a multi-dimensional and early treatment approach. Personality development disorder is considered to be a childhood risk factor or early stage of a later personality disorder in adulthood.

In executives

In 2005, psychologists Belinda Board and Katarina Fritzon at the University of Surrey, UK, interviewed and gave personality tests to high-level British executives and compared their profiles with those of criminal psychiatric patients at Broadmoor Hospital in the UK. They
found that one out of eleven personality disorders were actually more pronounced in
executives than in the disturbed criminals:

Histrionic personality disorder: including superficial charm, insincerity, egocentricity
and manipulation

Furthermore, they found no significant difference in the average scores of executives and
the disturbed criminal offenders on two out of the eleven scales:

- Narcissistic personality disorder: including grandiosity, self-focused lack of empathy
  for others, exploitativeness and independence.
- Obsessive-compulsive personality disorder: including perfectionism, excessive
  devotion to work, rigidity, stubbornness and dictatorial tendencies.

According to leading leadership academic Manfred F.R. Kets de Vries, it seems almost
inevitable these days that there will be some personality disorders in a senior management

team.

History

Personality disorder is a term with a distinctly modern meaning owing in part to its clinical
usage and the institutional character of modern psychiatry. The currently accepted
meaning must be understood in the context of historical changing classification systems
such as DSM-IV and its predecessors. Although highly anachronistic, ignoring radical
differences in the character of subjectivity and social relations, some have suggested
similarities to other concepts going back to at least the ancient Greeks.

Personality development disorder

Personality development disorder is a concept which is currently discussed in Europe (e.g.
in Germany: Adam & Breithaupt-Peters, 2010). Personality development disorder is
considered to be a childhood risk factor or early stage of a later personality disorder in
adulthood. The term personality development disorder is used to emphasize the changes in
personality development which might still take place and the open outcome during
development. DSM-IV allows the diagnosis of personality disorders in children and
adolescents only as an exception.

Adults usually show personality patterns over a long duration of time. Children and
adolescents however still show marked changes in personality development. Some of these
children and adolescents have a hard time developing their personalities in an ordinary
way. DSM-IV states, for example, that children and adolescents are at higher risk to develop
an antisocial personality disorder if they showed signs of conduct disorder and attention
deficit disorder before the age of 10. This lead Adam & Breithaupt-Peters (2010) to the idea
that these children and adolescents need to be looked at more carefully. The therapy which
these children and adolescents need might be more intense and maybe even different from
looking at the disorders traditionally. The concept of personality development disorders also focuses on the severity of the disorder and the poor prognosis. An early diagnosis might help to get the right treatment at an early stage and thus might help to prevent a personality disorder outcome in adulthood.

Description

Similar to the adult diagnosis personality disorder these children display enduring patterns of inner experience and behavior deviating markedly from the expectations of the individual’s culture. These patterns are inflexible and pervasive across a broad range of personal and social situations, lead to clinically significant distress or impairment in social, occupational or other important areas of functioning and they are stable and of long duration (more than a year).

The term personality development disorder (Persönlichkeitsentwicklungsstörung) was first used in German by Spiel & Spiel (1987). Adam & Breithaupt-Peters (2010) adapted the term to a more modern concept and suggested the following definition.

Definition Personality Development Disorder

According to Adam und Breithaupt-Peters personality development disorders are defined as complex disorders

- which show similarity to a certain type of personality disorder in adulthood
- which persist over a long period of time (more than a year) and show a tendency towards being chronic
- which have a severe negative impact on more than one important area of functioning or social life
- which show resistance to traditional educational and therapeutic treatment methods
- which result in a reduced insight into or ignorance of the own problem behavior. The family usually suffers more than the child or adolescent and has a hard time dealing with the diminished introspection.
- which make positive interactions between the children/adolescents and other people merely impossible. Instead social collisions are part of every day life.
- which threaten the social integration of the young person into a social life and might result into an emotional disability.

Etiology and Diagnosis

Similar to adult personality disorders there are multiple causes and causal interactions for personality development disorders. In clinical practice it is important to view the disorder multi-perspectively and from an individual perspective. Biological and neurological causes need to be observed just as much as psychosocial factors. Looking at the disorder from only one perspective (e.g. (s)he had a bad childhood) often results in ignorance of important other factors or causal interactions. This might be one of the main reasons why traditional
treatment methods often fail with these disorders. Only a multi-perspective view can provide for a multi-dimensional treatment approach which seems to be the key for these disorders.

The diagnosis personality development disorder should only be given carefully and after a longer period of evaluation. Also a thorough diagnostic evaluation is necessary. Parents should be questioned separately and together with the child or adolescent to evaluate the severity and duration of the problems. In addition standardized personality tests might be helpful. It is also useful to ask the family what treatment approaches they have already tried so far without success.

**Treatment**

Personality development disorders usually need a complex and multi-dimensional treatment approach (Adam & Breithaupt-Peters, 2010). Since the problems are complex, treatment needs to affect the conditions in all impaired functional and social areas. Both educational and therapeutic methods are helpful and problem and strength based approaches work hand in hand. Parents need to be included as well as the school environment. Treatment methods need to be flexible and adjustable to the individual situation. Even elements of social work can be helpful when supporting the families and in some cases medication might be necessary. When suicidal behaviors or self-injuries are prominent treatment might best be done in a hospital.

For some personality development disorders (e.g. Borderline disorder) treatment methods from adults can be adapted (e.g. dialectical behavior therapy, Miller et al., 2006).

**Paranoid personality disorder**

Paranoid personality disorder is a psychiatric diagnosis characterized by paranoia and a pervasive, long-standing suspiciousness and generalized mistrust of others.

Those with the condition are hypersensitive, are easily slighted, and habitually relate to the world by vigilant scanning of the environment for clues or suggestions to validate their prejudicial ideas or biases. Paranoid individuals are eager observers. They think they are in danger and look for signs and threats of that danger, disregarding any facts. They tend to be guarded and suspicious and have quite constricted emotional lives. Their incapacity for meaningful emotional involvement and the general pattern of isolated withdrawal often lend a quality of schizoid isolation to their life experience.

**Causes**

A genetic contribution to paranoid traits and a possible genetic link between this personality disorder and schizophrenia exist. Psychosocial theories implicate projection of negative internal feelings and parental modeling.
Diagnosis

WHO

The World Health Organization’s ICD-10 lists paranoid personality disorder as (F60.0) Paranoid personality disorder.

It is characterized by at least 3 of the following:

- excessive sensitivity to setbacks and rebuffs;
- tendency to bear grudges persistently, i.e. refusal to forgive insults and injuries or slights;
- suspiciousness and a pervasive tendency to distort experience by misconstruing the neutral or friendly actions of others as hostile or contemptuous;
- a combative and tenacious sense of personal rights out of keeping with the actual situation;
- recurrent suspicions, without justification, regarding sexual fidelity of spouse or sexual partner;
- tendency to experience excessive self-importance, manifest in a persistent self-referential attitude;
- preoccupation with unsubstantiated "conspiratorial" explanations of events both immediate to the patient and in the world at large.

Includes:

expansive paranoid, fanatic, querulant and sensitive paranoid personality (disorder)

Excludes:

delusional disorder
schizophrenia

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

Cultural sensitivities

World Health Organization, in the ICD-10, points out for different cultures it may be necessary to develop specific sets of criteria with regard to social norms, rules and obligations.

Millon's subtypes

Theodore Millon identified five subtypes of paranoid. Any individual paranoid may exhibit none or one of the following:
fanatic paranoid - including narcissistic features
malignant paranoid - including sadistic features
obdurate paranoid - including compulsive features
querulous paranoid - including negativistic (passive-aggressive) features
insular paranoid - including avoidant features
litigious paranoia - including a form of paranoia in which the person seeks legal proof or justification for systematized delusions

Differential diagnosis

The following conditions commonly coexist (comorbid) with paranoid personality disorder:

- very brief psychotic episodes (lasting minutes to hours)
- delusional disorder
- schizophrenia
- major depressive disorder
- agoraphobia
- obsessive-compulsive disorder
- alcohol and substance-related disorders
- schizoid personality disorder
- schizotypal personality disorder
- narcissistic personality disorder
- avoidant personality disorder
- borderline personality disorder

Treatment

Because of reduced levels of trust, there can be challenges in treating paranoid personality disorder. However, psychotherapy, antidepressants, antipsychotics and anti-anxiety medications can play a role when an individual is receptive to intervention.

Epidemiology

Paranoid personality disorder occurs in about 0.5%-2.5% of the general population. It is seen in 2%-10% of psychiatric outpatients. It occurs more commonly in males.

A large long-term Norwegian twin study found paranoid personality disorder to be modestly heritable and to share a portion of its genetic and environmental risk factors with schizoid and schizotypal personality disorder.

Paranoid Personality Disorder: Summarized

People with paranoid personality disorder are generally characterized by having a long-standing pattern of pervasive distrust and suspiciousness of others. A person with
paranoid personality disorder will nearly always believe that other people's motives are suspect or even malevolent. Individuals with this disorder assume that other people will exploit, harm, or deceive them, even if no evidence exists to support this expectation. While it is fairly normal for everyone to have some degree of paranoia about certain situations in their lives (such as worry about an impending set of layoffs at work), people with paranoid personality disorder take this to an extreme -- it pervades virtually every professional and personal relationship they have.

Individuals with Paranoid Personality Disorder are generally difficult to get along with and often have problems with close relationships. Their excessive suspiciousness and hostility may be expressed in overt argumentativeness, in recurrent complaining, or by quiet, apparently hostile aloofness. Because they are hypervigilant for potential threats, they may act in a guarded, secretive, or devious manner and appear to be "cold" and lacking in tender feelings. Although they may appear to be objective, rational, and unemotional, they more often display a labile range of affect, with hostile, stubborn, and sarcastic expressions predominating. Their combative and suspicious nature may elicit a hostile response in others, which then serves to confirm their original expectations.

Because individuals with Paranoid Personality Disorder lack trust in others, they have an excessive need to be self-sufficient and a strong sense of autonomy. They also need to have a high degree of control over those around them. They are often rigid, critical of others, and unable to collaborate, and they have great difficulty accepting criticism.

**Symptoms of Paranoid Personality Disorder**

A pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

- Suspects, without sufficient basis, that others are exploiting, harming, or deceiving him or her
- Is preoccupied with unjustified doubts about the loyalty or trustworthiness of friends or associates
- Is reluctant to confide in others because of unwarranted fear that the information will be used maliciously against him or her
- Reads hidden demeaning or threatening meanings into benign remarks or events
- Persistently bears grudges, i.e., is unforgiving of insults, injuries, or slights
- Perceives attacks on his or her character or reputation that are not apparent to others and is quick to react angrily or to counterattack
- Has recurrent suspicions, without justification, regarding fidelity of spouse or sexual partner

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.
Paranoid personality disorder is more prevalent in males than females, and occurs somewhere between 0.5 and 2.5 percent in the general population.

Like most personality disorders, paranoid personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

**How is Paranoid Personality Disorder Diagnosed?**

Personality disorders such as paranoid personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose paranoid personality disorder.

Many people with paranoid personality disorder don't seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person's life. This most often happens when a person's coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for paranoid personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Paranoid Personality Disorder**

Researchers today don't know what causes paranoid personality disorder. There are many theories, however, about the possible causes of paranoid personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual’s personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

**Paranoid Personality Disorder Treatment**

**Psychotherapy**

As with most personality disorders, psychotherapy is the treatment of choice. Individuals with paranoid personality disorder, however, rarely present themselves for treatment. It
should not be surprising, then, that there has been little outcome research to suggest which types of treatment are most effective with this disorder.

It is likely that a therapy which emphasizes a simple supportive, client-centered approach will be most effective. Rapport-building with a person who has this disorder will be much more difficult than usual because of the paranoia associated with the disorder. Early termination, therefore, is common. As the therapy progresses, the patient will likely begin to trust the clinician more and more. The client then will likely begin disclosing some of his or her more bizarre paranoid ideation. The therapist must be careful to balance being objective in therapy and with regards to these thoughts, and of raising the suspicions of the client that he or she is not trusted. It is a difficult balance to maintain, even after a good working rapport has been established.

During times when the patient is acting upon his paranoid beliefs, the therapist’s loyalties and trust may be called into question. Care must be used not to challenge the client too firmly or risk the individual leaving therapy permanently. Control issues should be dealt with in much a similar manner, with great care. Since the paranoid beliefs are delusion and not based in reality, arguing them from a rational point of view is useless. Challenging the beliefs is also likely to result in more frustration on both the part of the therapist and client, too.

All clinicians and mental health personnel who come into contact with the individual who suffers from paranoid personality disorder should be more keenly aware of being straightforward with this individual. Subtle jokes are often lost on them and allusions to information about the client not received directly from the client's mouth will raise a great deal of suspicion. Therapists should typically avoid trying to have the patient sign a release of information for information not essential to the current therapy. Items in life which usually wouldn't give most people a second thought can easily become the focus of attention to this client, so care must be exercised in discussions with the client. An honest, concrete approach will likely gain the most results, focusing on current life difficulties which has brought the client into therapy at this time. Clinicians should generally not inquire too deeply into the client’s life or history, unless it’s directly relevant to clinical treatment.

Long-term prognosis for this disorder is not good. Individuals who suffer from this disorder often remain afflicted with prominent symptoms of it throughout their lifetime. It is not uncommon to see such people in day treatment programs or state hospitals. Other modalities, such as family or group therapy, are not recommended.

**Medications**

Medications are usually contraindicated for this disorder, since they can arouse unnecessary suspicion that will usually result in noncompliance and treatment dropout. Medications which are prescribed for specific conditions should be done so for the briefest time period possible to bring the condition under management.
An anti-anxiety agent, such as diazepam, is appropriate to prescribe if the client suffers from severe anxiety or agitation where it begins to interfere with normal, daily functioning. An anti-psychotic medication, such as thioridazine or haloperidol, may be appropriate if a patient decompensates into severe agitation or delusional thinking which may result in self-harm or harm to others.

**Self-Help**

There are not any self-help support groups or communities that we are aware of that would be conducive to someone suffering from this disorder. Such approaches would likely not be very effective because a person with this disorder is likely to be mistrustful and suspicious of others and their motivations, making group help and dynamics unlikely and possibly harmful.

**Schizoid personality disorder**

Schizoid personality disorder (SPD) is a personality disorder characterized by a lack of interest in social relationships, a tendency towards a solitary lifestyle, secretiveness, emotional coldness and sometimes (sexual) apathy, with a simultaneous rich, elaborate and exclusively internal fantasy world. SPD is not the same as schizophrenia, although they share some similar characteristics such as detachment or blunted affect; there is increased prevalence of the disorder in families with schizophrenia.

**Signs and symptoms**

People with SPD are seen as aloof, cold and indifferent, which causes some social problems. Most individuals diagnosed with SPD have difficulty establishing personal relationships or expressing their feelings in a meaningful way, and may remain passive in the face of unfavourable situations. Their communication with other people at times may be indifferent and concise. Because of their lack of meaningful communication with other people, those who are diagnosed with SPD are not able to develop accurate reflections of themselves with respect to how well they are getting along with others. Such reflections are important for a person’s self-awareness and their ability to assess the impact of their own actions in social situations. R. D. Laing suggests that without being enriched by injections of interpersonal reality there occurs an impoverishment in which one’s self-image becomes more and more empty and volatilized, leading the individual himself to feel unreal.

According to Gunderson, people with SPD “feel lost” without the people they are normally around because they need a sense of security and stability. However, when the patient’s personal space is violated, they feel suffocated and feel the need to free themselves and be independent. Those people who have SPD are happiest when they are in a relationship in which the partner places few emotional or intimate demands on them, as it is not people as such that they want to avoid, but both negative and positive emotions, emotional intimacy, and self-disclosure.
This means that it is possible for schizoid individuals to form relationships with others based on intellectual, physical, familial, occupational, or recreational activities as long as these modes of relating do not require or force the need for emotional intimacy, which the individual will reject.

Donald Winnicott sums up the schizoid need to modulate emotional interaction with others with his comment that schizoid individuals "prefer to make relationships on their own terms and not in terms of the impulses of other people," and that if they cannot do so, they prefer isolation.

'Secret schizoid'

According to Ralph Klein there are many fundamentally schizoid individuals who present with an engaging, interactive personality style which contradicts the timidity, reluctance, or avoidance of the external world and interpersonal relationships as emphasized by the DSM-IV and ICD-10 definitions of the schizoid personality. Klein classifies these individuals as secret schizoids who present themselves as socially available, interested, engaged, and involved in interacting in the eyes of the observer, while at the same time, he or she is apart, emotionally withdrawn, and sequestered in a safe place in his or her own internal world. So, while withdrawal or detachment from the outer world is a characteristic feature of schizoid pathology, it is sometimes overt and sometimes covert. While it is overt it matches the usual description of the schizoid personality offered in the DSM-IV. According to Klein, though, it is "just as often" a covert, hidden internal state of the patient in which what meets the objective eye may not be what is present in the subjective, internal world of the patient. Klein therefore cautions that one should not miss identifying the schizoid patient because one cannot see the patient’s withdrawal through the patient’s defensive, compensatory, engaging interaction with external reality. Klein suggests that one need only ask the patient what his or her subjective experience is in order to detect the presence of the schizoid refusal of emotional intimacy.

Descriptions of the schizoid personality as hidden behind an outward appearance of emotional engagement have long been recognized, beginning with Fairbain’s (1940) description of ‘schizoid exhibitionism’ in which he remarked that the schizoid individual is able to express quite a lot of feeling and to make what appear to be impressive social contacts but in reality giving nothing and losing nothing, because since he is only playing a part his own personality is not involved. According to Fairbain, the person "...disowns the part which he is playing and thus the schizoid individual seeks to preserve his own personality intact and immune from compromise." Further references to the secret schizoid come from Masud Khan, Jeffrey Seinfeld, and Philip Manfield, who gives a palpable description of an SPD individual who actually "enjoys" regular public speaking engagements, but experiences great difficulty in the breaks when audience members would attempt to engage him emotionally. These references expose the problems involved in relying singularly on outer observable behavior for assessing the presence of personality disorders in certain individuals.
Avoidant attachment style

The question of whether SPD qualifies as a full personality disorder or simply as an avoidant attachment style is a contentious one. If what has been known as schizoid personality disorder is no more than an attachment style requiring more distant emotional proximity, then many of the more problematic reactions these individuals show in interpersonal situations may be partly accounted for by the social judgments commonly imposed on those with this style. To date several sources have confirmed the synonymity of SPD and avoidant attachment style which leaves open the question of how researchers might approach this subject best in future diagnostic manuals, and in therapeutic practice. However, characteristically - and depending on the severity of the disorder - individuals do not seek social interactions merely due to lack of interest, as opposed to the avoidant personality type in which there is craving for interactions, but then fear of rejection.

Schizoid sexuality

People with SPD are sometimes sexually apathetic, though they do not normally suffer from anorgasmia. Many schizoids have a normal sex drive but some prefer to masturbate rather than deal with the social aspects of finding a sexual partner. Therefore, their need for sex may appear to be less than those who do not have SPD, as individuals with SPD prefer to remain alone and detached. When having sex, individuals with SPD often feel that their personal space is being violated, and they commonly feel that masturbation or sexual abstinence is preferable to the emotional closeness they must tolerate when having sex. Significantly broadening this picture are notable exceptions of SPD individuals who engage in occasional or even frequent sexual activities with others.

Harry Guntrip describes the "secret sexual affair" entered into by some married schizoid individuals as an attempt to reduce the quantity of emotional intimacy focused within a single relationship, a sentiment echoed by Karen Horney's resigned personality who may exclude sex as being "too intimate for a permanent relationship, and instead satisfy his sexual needs with a stranger. Conversely he may more or less restrict a relationship to merely sexual contacts and not share other experiences with the partner." More recently, Jeffrey Seinfeld, professor of social work at New York University, has published a volume on SPD in which he details examples of "schizoid hunger" which may manifest as sexual promiscuity. Seinfeld provides an example of a schizoid woman who would covertly attend various bars to meet men for the purposes of gaining impersonal sexual gratification, an act, says Seinfeld, which alleviated her feelings of hunger and emptiness.

Salman Akhtar describes this dynamic interplay of overt versus covert sexuality and motivations of some SPD individuals with greater accuracy. Rather than following the narrow proposition that schizoid individuals are either sexual or asexual, Akhtar suggests that these forces may both be present in an individual despite their rather contradictory aims. For Akhtar, therefore, a clinically accurate picture of schizoid sexuality must include both the overt signs: "asexual, sometimes celibate; free of romantic interests; averse to sexual gossip and innuendo," along with possible covert manifestations of "secret voyeuristic and pornographic interests; vulnerable to erotomania; tendency towards
compulsive masturbation and perversions,” although none of these necessarily apply to all people with SPD.

**Causes**

There is some evidence to suggest that there is an increased prevalence of schizoid personality disorder in relatives of people with schizophrenia or schizotypal personality disorder. Unloving or neglectful parenting is hypothesized to play a role.

**Diagnosis**

**DSM**

The Diagnostic and Statistical Manual of Mental Disorders fourth edition, a widely used manual for diagnosing mental disorders, defines schizoid personality disorder (in Axis II Cluster A) as:

A. A pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings, beginning by early adulthood (age eighteen or older) and present in a variety of contexts, as indicated by four (or more) of the following:

- neither desires nor enjoys close relationships, including being part of a family
- almost always chooses solitary activities
- has little, if any, interest in having sexual experiences with another person
- takes pleasure in few, if any, activities
- lacks close friends or confidants other than first-degree relatives
- appears indifferent to the praise or criticism of others
- shows emotional coldness, detachment, or flattened affect

B. Does not occur exclusively during the course of schizophrenia, a mood disorder with psychotic features, another psychotic disorder, or a pervasive developmental disorder and is not due to the direct physiological effects of a general medical condition.

It is a requirement of DSM-IV that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

In the draft of the DSM-V it is proposed that schizoid personality disorder should be represented and diagnosed by a combination of core impairment in personality functioning and specific pathological personality traits, rather than as a specific type.

**World Health Organization**

The World Health Organization’s ICD-10 lists schizoid personality disorder as (F60.1) Schizoid personality disorder.
It is characterized by at least four of the following criteria:

- Emotional coldness, detachment or reduced affection.
- Limited capacity to express either positive or negative emotions towards others.
- Consistent preference for solitary activities.
- Very few, if any, close friends or relationships, and a lack of desire for such.
- Indifference to either praise or criticism.
- Taking pleasure in few, if any, activities.
- Indifference to social norms and conventions.
- Preoccupation with fantasy and introspection.
- Lack of desire for sexual experiences with another person.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Millon's subtypes**

Theodore Millon identified four subtypes of schizoid. Any individual schizoid may exhibit none or one of the following:

- languid schizoid
- including depressive features
- remote schizoid
- including avoidant, schizotypal features
- depersonalised schizoid
- including schizotypal features
- affectless schizoid
- including compulsive features

**Dynamic diagnostic criteria**

Ralph Klein, 1995 brought new light into the commonly held beliefs about the schizoid which focus mainly on the schizoid's apparent disinterest in relationship. Clarifying the causes and conditions underlying the characteristics listed above, Klein describes a 'split' in the object relations of the schizoid. This split involves: on the one hand, the "slave/master" relationship, a relationship characterised by exploitation, appropriation, and dehumanisation, and on the other, the "self in exile". It is in aversion, or recoil from the exploitive relationship that the self goes into exile. It is this, the self in exile, that is the more commonly recognised aspect of the schizoid, as described in the DSM—the distanced or
unresponsive person. As Klein puts it: the..."seeming detachment from feelings should never be accepted as the real state of affairs" p. 135. Of particular significance is the correlation between the Narcissistic disorder and the schizoid. For example the "over entitlement" of the narcissist in a family can result in the "under-entitlement" of the schizoid. It is also the disavowed shame of the narcissist that is often absorbed by, or projected into the schizoid, thus giving rise to the experience of psychic invasion, and the vulnerability to intrusiveness. Paradoxically, a schizoid may also be attracted to exploitative relationships, where they long to experience significance and recognition by serving a need of the other. Yet this same person may be highly aware of any forms of corruption or exploitation outside of this relationship. In this approach diagnosis is based on the dynamic of this split, and all its consequences, as opposed to diagnosis on the basis of a list of external behaviours.

Guntrip criteria

Ralph Klein, Clinical Director of the Masterson Institute delineates the following nine characteristics of the schizoid personality as described by Harry Guntrip: introversion, withdrawnness, narcissism, self-sufficiency, a sense of superiority, loss of affect, loneliness, depersonalization, and regression.

Introversion

According to Guntrip, "By the very meaning of the term the schizoid is described as cut off from the world of outer reality in an emotional sense. All this libidinal desire and striving is directed inward toward internal objects and he lives an intense inner life often revealed in an astonishing wealth and richness of fantasy and imaginative life whenever that becomes accessible to observation. Though mostly his varied fantasy life is carried on in secret, hidden away." The schizoid person is cut off from outer reality to such a degree that he or she experiences outer reality as dangerous. It is a natural human response to turn away from sources of danger and toward sources of safety. The schizoid individual, therefore, is primarily concerned with avoiding danger and ensuring safety.

Withdrawnness

According to Guntrip, withdrawnness means detachment from the outer world, the other side of introversion. While there are many schizoid individuals who will present with obvious withdrawnness (a clear and obvious timidity, reluctance, or avoidance of the external world and interpersonal relationships), this defines only a portion of such individuals. Many fundamentally schizoid people present with an engaging, interactive personality style. Such a person can appear to be available, interested, engaged, and involved in interacting with others; however, in reality, he or she is emotionally withdrawn and sequestered in a safe place in an internal world. While withdrawnness or detachment from the outer world is a characteristic feature of schizoid pathology, it is sometimes overt and sometimes covert. When it is overt it matches the usual description of the schizoid personality. Just as often, it is a covert, hidden internal state of the patient.
Several points are important to review at this time. First, what meets the objective eye may not be what is present in the subjective, internal world of the patient. Second, one should not mistake introversion for indifference. Third, one should not miss identifying the schizoid patient because one cannot see the forest of the patient’s withdrawnness through the trees of the patient’s defensive, compensatory, engaging interaction with external reality.

**Narcissism**

Guntrip: "Narcissism is a characteristic that arises out of the predominately interior life the schizoid lives. His love objects are all inside him and moreover he is greatly identified with them so that his libidinal attachments appear to be in himself. The question, however, is whether the intense inner life of the schizoid is due to a desire for hungry incorporation of external objects or due to withdrawal from the outer to a presumed safer inner world." The need for attachment as a primary motivational force is as strong in the schizoid person as in any other human being. However, because the schizoid’s love objects are internal, he or she finds safety without connecting and attaching to objects in the real world.

**Self-sufficiency**

Guntrip writes, "This introverted narcissistic self-sufficiency, which does without real external relationships while all emotional relations are carried on in the internal world, is a safeguard against anxiety breaking out in dealing with actual people." The more that schizoids can rely on themselves, the less they have to rely on other people and expose themselves to the potential dangers and anxieties associated with that reliance or, even worse, dependence. The vast majority of schizoid individuals show an enormous capacity for self-sufficiency, for the ability to operate alone, independently and autonomously, in managing their worlds.

**Sense of superiority**

Guntrip states, "a sense of superiority naturally goes with self-sufficiency. One has no need of other people, they can be dispensed with... There often goes with it a feeling of being different from other people." The sense of superiority of the schizoid has nothing to do with the grandiose self of the narcissistic disorder. It does not find expression in the schizoid through the need to devalue or annihilate others who are perceived as offending, criticizing, shaming, or humiliating. This type of superiority was described by a young schizoid man:

"If I am superior to others, if I am above others, then I do not need others. When I say that I am above others, it does not mean that I feel better than them, it means that I am at a distance from them, a safe distance."

It is a feeling of being horizontally, rather than vertically distant.
Loss of affect

According to Guntrip, "Loss of affect in external situations is an inevitable part of the total picture." Because of the tremendous investment made in the self—the need to be self-contained, self-sufficient, and self-reliant—there is inevitable interference in the desire and ability to feel another person's experience, to be empathic and sensitive. Often these things seem secondary, a luxury that has to await securing one's own defensive, safe position. The subjective experience is one of loss of affect. For some patients, the loss of affect is present to such a degree that the insensitivity becomes manifest in the extreme as cynicism, callousness, or even cruelty. The patient appears to have no awareness of how his or her comments or actions affect and hurt other people. More frequently, the loss of affect is manifest within the patient as genuine confusion, a sense of something missing in his or her emotional life.

Loneliness

According to Guntrip, "Loneliness is an inescapable result of schizoid introversion and abolition of external relationships. It reveals itself in the intense longing for friendship and love which repeatedly break through. Loneliness in the midst of a crowd is the experience of the schizoid cut off from affective rapport." This is a central experience of the schizoid that is often lost to the observer. Contrary to the familiar caricature of the schizoid as uncaring and cold, the vast majority of schizoid persons who become patients express at some point in their treatment their longing for friendship and love. This is not the schizoid patient as described in the DSMs. Such longing, however, may not break through except in the schizoid's fantasy life, to which the therapist may not be allowed access for quite a long period in treatment. If longing is immediately present, however, it is more likely avoidant personality disorder.

There is a very narrow range of schizoid individuals—the classic DSM-defined schizoid—for whom the hope of relationship is so minimal as to be almost extinct; therefore, the longing for closeness and attachment is almost unidentifiable to the schizoid themselves. These individuals will not become patients. The schizoid individual who becomes a patient does so often because of the twin motivations of loneliness and longing. This schizoid patient still believes that some kind of connection and attachment is possible and is well suited to psychotherapy. Yet the irony of the DSMs is that they may lead the psychotherapist to approach the schizoid patient with a sense of therapeutic pessimism, if not nihilism, misreading the patient by believing that the patient's wariness is indifference and that caution is coldness.

Depersonalization

Guntrip describes depersonalization as a loss of a sense of identity and individuality. Depersonalization is a dissociative defense. Depersonalization is often described by the schizoid patient as a tuning out or a turning off, or as the experience of a separation between the observing and the participating ego. It is experienced by those with schizoid personality disorder when anxieties seem overwhelming. It is a more extreme form of loss
of affect than that described earlier. Whereas the loss of affect is a more chronic state in schizoid personality disorder, depersonalization is an acute defense against more immediate experiences of overwhelming anxiety or danger.

Regression

Guntip defined regression as "Representing the fact that the schizoid person at bottom feels overwhelmed by their external world and is in flight from it both inwards and as it were backwards to the safety of the metaphorical womb." Such a process of regression encompasses two different mechanisms: inward and backwards. Regression inward speaks to the magnitude of the reliance on primitive forms of fantasy and self-containment, often of an autoerotic or even objectless nature. Regression backwards to the safety of the womb is a unique schizoid phenomenon and represents the most intense form of schizoid defensive withdrawal in an effort to find safety and to avoid destruction by external reality. The fantasy of regression to the womb is the fantasy of regression to a place of ultimate safety.

The description of the nine characteristics first articulated by Guntip should bring more clearly into focus some of the major differences that exist between the traditional descriptive (track 1, DSM) portrait of the schizoid disorder and the traditional psychoanalytically informed (track 2, object relations) view. All nine characteristics are internally consistent. Most, if not all, should be present in order to diagnose a schizoid disorder.

Akhtar's phenomenological profile

In an article in the American Journal of Psychotherapy, Salman Akhtar, M.D., provides a comprehensive phenomenological profile of Schizoid Personality Disorder in which classic and contemporary descriptive views are synthesized with psychoanalytic observations. This profile is summarized in a table (reproduced below) listing clinical features, involving six areas of psychosocial functioning and designated by "overt" and "covert" manifestations. Dr. Akhtar states that "these designations do not imply conscious or unconscious but denote seemingly contradictory aspects that are phenomenologically more or less easily discernible," and that "this manner of organizing symptomology emphasizes the centrality of splitting and identity confusion in schizoid personality."

<table>
<thead>
<tr>
<th>Area</th>
<th>Overt Features</th>
<th>Covert Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-Concept</td>
<td>• compliant</td>
<td>• cynical</td>
</tr>
<tr>
<td></td>
<td>• stoic</td>
<td>• inauthentic</td>
</tr>
<tr>
<td></td>
<td>• noncompetitive</td>
<td>• depersonalized</td>
</tr>
<tr>
<td></td>
<td>• self-sufficient</td>
<td>• alternately feeling empty, robot-like, and full of omnipotent, vengeful fantasies</td>
</tr>
<tr>
<td></td>
<td>• lacking assertiveness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• feeling inferior and an outsider in life</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interpersonal Relations</td>
<td>Social Adaptation</td>
</tr>
<tr>
<td>------------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>• withdrawn</td>
<td>• prefer solitary occupational and recreational activities</td>
<td>• lack clarity of goals</td>
</tr>
<tr>
<td>• aloof</td>
<td>• marginal or eclectically sociable in groups</td>
<td>• weak ethnic affiliation</td>
</tr>
<tr>
<td>• have few close friends</td>
<td>• vulnerable to esoteric movements owing to a strong need to belong</td>
<td>• usually capable of steady work</td>
</tr>
<tr>
<td>• impervious to others’ emotions</td>
<td>• tend to be lazy and indolent</td>
<td>• quite creative and may make unique and original contributions</td>
</tr>
<tr>
<td>• afraid of intimacy</td>
<td>•</td>
<td>• capable of passionate endurance in certain spheres of interest</td>
</tr>
</tbody>
</table>

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<thead>
<tr>
<th></th>
<th>Love and Sexuality</th>
<th>Ethics, Standards, and Ideals</th>
</tr>
</thead>
<tbody>
<tr>
<td>• asexual, sometimes celibate</td>
<td>• idiosyncratic moral and political beliefs</td>
<td>• moral unevenness</td>
</tr>
<tr>
<td>• free of romantic interests</td>
<td>• tendency towards spiritual, mystical and para-psychological interests</td>
<td>• occasionally strikingly amoral and vulnerable to odd crimes, at other times altruistically self sacrificing</td>
</tr>
<tr>
<td>• averse to sexual gossip and innuendo</td>
<td>•</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Cognitive Style</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• absent-minded</td>
<td>• autistic thinking</td>
<td>• fluctuations between sharp contact with external reality and hyperreflectiveness about the self</td>
</tr>
<tr>
<td>• engrossed in fantasy</td>
<td>• vague and stilted speech</td>
<td>• autocentric use of language.</td>
</tr>
<tr>
<td>• alternations between eloquence and inarticulateness</td>
<td>•</td>
<td></td>
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</tbody>
</table>

One patient with SPD commented that he could not fully enjoy the life he has because he feels that he is living in a shell. Furthermore, he noted that his inability distressed his wife.
According to Beck and Freeman, "Patients with schizoid personality disorders consider themselves to be observers, rather than participants, in the world around them."

**Differential diagnosis**

Although SPD shares several aspects with other psychological conditions, there are some important differentiating features:

<table>
<thead>
<tr>
<th>Psychological condition</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>While people who have SPD can also suffer from clinical depression, this is certainly not always the case. Unlike depressed people, persons with SPD generally do not consider themselves inferior to others, although they will probably recognize that they are different.</td>
</tr>
<tr>
<td>Avoidant personality disorder</td>
<td>Unlike avoidant personality disorder, those affected with SPD do not avoid social interactions due to anxiety or feelings of incompetence, but because they are genuinely indifferent to social relationships; however, in a 1989 study, &quot;schizoid and avoidant personalities were found to display equivalent levels of anxiety, depression, and psychotic tendencies as compared to psychiatric control patients.&quot; One SPD patient remarked that previous knowledge, expectations, or assumptions may result in such elevated levels. Patients can mentally simulate damaging scenarios in order to flatten negative effects, should one occur.</td>
</tr>
<tr>
<td>Asperger syndrome</td>
<td>Asperger syndrome is an autism-spectrum disorder. Unlike AS, SPD does not involve an impairment in nonverbal communication (e.g., lack of eye-contact or unusual prosody) or a pattern of restricted interests or repetitive behaviors (e.g., a strict adherence to routines or rituals, or an unusually intense interest in a single topic). Compared to AS, SPD is characterized by prominent conduct disorder, better adult adjustment, and a slightly increased risk of schizophrenia.</td>
</tr>
</tbody>
</table>

Under stress, some people with schizoid personality features may occasionally experience instances of brief reactive psychosis. Schizoid individuals are also prone to developing pathological reliance on fantasizing activity as concomitant with their withdrawal from the world. Viewed in this fashion, fantasy constitutes a core component of the self-in-exile, though on closer examination fantasizing in schizoid individuals reveals as far more complicated than a means of facilitating withdrawal. Fantasy is also relationship with the world and with others by proxy. It is a substitute relationship, but a relationship nonetheless, characterized by idealized, defensive, and compensatory mechanisms. It is an expression of the self-in-exile because it is self-contained and free from the dangers and anxieties associated with emotional connection to real persons and situations. According to Klein it is "an expression of the self struggling to connect to objects, albeit internal objects. Fantasy permits schizoid patients to feel connected, and yet still free from the
imprisonment in relationships. In short, in fantasy one can be attached (to internal objects) and still be free." This aspect of schizoid pathology has been generously elaborated in works by Laing (1960); Winnicott; (1971); and Klein (1995).

According to Seinfeld, schizoid individuals frequently act out with substance and alcohol abuse and other addictions which serve as substitutes for human relationships. The substitute of a nonhuman for a human object serves as a schizoid defense. Providing examples of how the schizoid individual creates a personal relation with the drug, Seinfeld tells how "one addict called heroin his 'soothing white pet.' Another referred to crack as his 'bad mama.' I knew a female addict who termed crack her 'boyfriend.' Not all addicts name their drug, but there often is the trace of a personal feeling about the relationship." The object relations view emphasizes that the drug use and alcoholism reinforce the fantasy of union with an internal object, while enabling the addict to be indifferent to the external object world. Addiction is therefore viewed as a schizoid and symbiotic defense.

S. C. Ekleberry suggests that marijuana "may be the single most egosyntonic drug for individuals with SPD because it allows a detached state of fantasy and distance from others, provides a richer internal experience than these individuals can normally create, and reduces an internal sense of emptiness and failure to participate in life. Also, alcohol, readily available and safe to obtain, is another obvious drug of choice for these individuals. Some will use both marijuana and alcohol and see little point in giving up either. They are likely to use in isolation for the effect on internal processes."

According to Ralph Klein, suicide may also be a running theme for schizoid individuals, though they are not likely to actually attempt one. They might be down and depressed when all possible connections have been cut off, but as long as there is some relationship or even hope for one the risk will be low. The idea of suicide is a driving force against the person's schizoid defenses. As Klein says: "For some schizoid patients, its presence is like a faint, barely discernible background noise, and rarely reaches a level that breaks into consciousness. For others, it is an ominous presence, an emotional sword of Damocles. In any case, it is an underlying dread that they all experience."

**Treatment**

Since schizoid traits are very similar to negative schizophrenic symptoms, atypical antipsychotics may have efficacy in alleviating them. Those who do seek treatment have the option of medication or therapy. For medication, the schizoid personality disorder seems to have similar negative symptoms of schizophrenia such as anhedonia, blunted affect, and low energy. The medication that is most recently used to treat the negative symptoms is risperidone. Before this, there was no psychotropic medication that made an impact on the negative symptoms. According to Joseph, low doses of risperidone or olanzapine also work for the social deficits and blunted affect; Wellbutrin (bupropion) for anhedonia. Furthermore, the use of SSRIs, TCAs, MAOIs, low dose benzodiazepines, and beta-blockers may help social anxiety in the SPD. However, social anxiety may not be a main concern for the people who have SPD. Supportive psychotherapy is also used in an inpatient or outpatient setting by a trained personnel that focuses on areas such as: coping skills,
improving social skills and social interactions, communication, and self esteem issues. Mark Zimmerman suggested the following questions for evaluation of patients with SPD:

- Do you have close relationships with friends or family? If yes, with whom? If no, does this bother you?
- Do you wish you had close relationships with others?
- Some people prefer to spend time alone, others prefer to be with people. How would you describe yourself?
- Do you frequently choose to do things by yourself?
- Would it bother you to go a long time without a sexual relationship? Does your sex life seem important or could you get along as well without it?
- What kind of activities do you enjoy?
- Do you confide in anyone who is not in your immediate family?
- How do you react when someone criticizes you?
- How do you react when someone compliments you?

In the assessment process, note if these individuals make eye contact, smile or express affect nonverbally.

According to Beck and Freeman, people with SPD have "defective perceptual scanning which results in missing environmental cues. The defective perceptual scanning is characterized by a tendency to miss differences and to diffuse the varied elements of experience." The perception of varied events only increases their fear for intimacy and limits them in their interpersonal relationships. Also because of their aloofness, this barrier does not allow them to use their social skills and behavior to help them pursue relationships. Therefore, socialization groups may help these people with SPD. As said by Will, educational strategies also work with people who have SPD by having them identify their positive and negative emotions. They use the identification to learn about their own emotions; the emotions they draw out from others; and feeling the common emotions with other people whom they relate with. This can help people with SPD create empathy with the outside world.

**Shorter-term treatment**

According to Ralph Klein, Clinical Director of the Masterson Institute, the concept of closer compromise means that the schizoid patient may be encouraged to experience intermediate positions between the extremes of emotional closeness, and permanent exile.

As mentioned by Laing without being enriched by injections of interpersonal reality there occurs an impoverishment in which the schizoid individual's self-image becomes more and more empty and volatilized, leading the individual himself to feel unreal. Therefore to create a more adaptive and self-enriching interaction with others in which one "feels real" the patient is encouraged to take risks by creating less interpersonal distance through greater connection, communication, and the sharing of ideas, feelings, and actions. Closer compromise means that while the schizoid patient's vulnerability to the anxieties is not overcome, it is modified and managed more adaptively. Here the therapist repeatedly
conveys to the patient that anxiety is inevitable, yet manageable, without any illusion that the schizoid vulnerability to such anxiety can be permanently dispensed with. The limiting factor is the point at which the dangers of intimacy become overwhelming and the patient must again retreat.

Klein suggests that closer compromise must be directly stated as the patient’s responsibility; "It seems to me that in order to accomplish your goals, it is necessary to put yourself at risk," or "It seems to me that your willingness to come here (to treatment) and struggle with your anxieties must be mirrored by your willingness to challenge yourself outside of here," or "It seems to me that your efforts to connect with me are only half the battle; the other half must take place in the more dangerous arena of your life outside this office," i.e. therapist is always conveying that these are the therapists impressions. He or she is not reading the patient’s mind or imposing an agenda, but is simply stating a position. Also, the therapist’s position is an extension of the patient’s therapeutic wish ("your goals," "your willingness," and "your efforts"). Finally, the therapist specifically directs attention to the need for employing these actions outside the therapeutic setting.

**Longer-term therapy**

Klein suggests that working through is the second longer-term tier of psychotherapeutic work with schizoid patients. Its goals are to change fundamentally the old ways of feeling and thinking, and to rid oneself of the vulnerability to experiencing those emotions associated with old feelings and thoughts. A new therapeutic operation of ‘remembering with feeling’ is called for.

One must remember with feeling the coming into being of one’s false self through childhood. (The concept of false self and true self comes from D. W. Winnicott, and is viewed as representative of schizoid phenomenology.) This means that one must remember the conditions and proscriptions that were imposed on the individual’s freedom to experience the self in company with others. Ultimately, remembering with feeling leads the patient to the understanding that he or she had no choice in the process of developing a schizoid stance toward others. The patient did not have the opportunity to choose from a selection of possible ways of experiencing the self and of relating with others, rather, the patient had few if any options. The false self was simply the best way in which the patient could experience repetitive predictable acknowledgment, affirmation, and approval (the emotional supplies necessary for emotional survival), while warding off the effects associated with the abandonment depression.

If the goal of shorter-term therapy is for patients to understand that they are not the way they appear to be and can act differently, then the longer-term goal of working through is for patients to understand who and what they are as human beings, what they truly are like and what they truly contain. The goal of working through is not achieved by the patient’s sudden discovering of a hidden, fully formed talented and creative self living inside but is a process of slowly freeing oneself from the confinement of abandonment depression in order to have the opportunity to uncover a potential. It is a process of experimentation
with the spontaneous, nonreactive elements that can be experienced in relationship with others.

Working through abandonment depression is a complicated, lengthy, and conflicted process which can be an enormously painful experience in terms of what is remembered and what must be felt. It involves a mourning, a grieving, for the loss of the illusion that the patient had adequate support for the emergence of the real self. Also, it is a mourning for the loss of an identity, the false self, which the person constructed and with which he or she has negotiated much of his or her life. The dismantling of the false self requires a relinquishing the only way of being that the patient has ever known of his interactions with others, an interaction which was better than no stable, organized experience of the self, no matter how false, defensive, or destructive that identity may be.

According to Klein the dismantling of the false self "leaves the impaired real self with the opportunity to convert its potential and its possibilities into actualities." The process of working through brings with it its own unique rewards, of which the most important element in new self-awareness is the growing realization by the individual that they have a fundamental, internal need for relatedness, which they may express in a variety of ways. "Only schizoid patients", suggests Klein, "who have worked through the abandonment depression ... ultimately will believe that the capacity for relatedness and the wish for relatedness are woven into the structure of their beings, that they are truly part of who the patients are and what they contain as human beings. It is this sense that finally allows the schizoid patient to feel the most intimate sense of being connected with humanity more generally, and with another person more personally. For the schizoid patient, this degree of certainty is the most gratifying revelation, and a profound new organizer of the self experience."

**Epidemiology**

SPD is uncommon in clinical settings. It occurs slightly more commonly in males.

SPD is rare compared with other personality disorders. Its prevalence is estimated at less than 1% of the general population.

As an interesting comment on the usual low-prevalence figures for this disorder, Philip Manfield in Split Self, Split Object, Arenson (1992) states that "I believe that the schizoid condition is far more common... comprising perhaps as many as 40 percent of all personality disorders. This huge discrepancy is probably largely because someone with a schizoid disorder is less likely to seek treatment than someone with other axis-II disorders." Manfield backs this claim with a study by Valliant & Drake (1985) who found that over 40% of a particular sample group of inner city males were schizoid.

**History**

The term schizoid was coined in 1908 by Eugen Bleuler to designate a natural human tendency to direct attention toward one’s inner life and away from the external world, a
concept akin to introversion in that it was not viewed in terms of psychopathology. Bleuler also labeled the exaggeration of this tendency the "schizoid personality".

Since then, studies on the schizoid personality have developed along two separate paths; firstly, the descriptive psychiatry tradition which focuses on overtly observable, behavioral, and describable symptoms which finds its clearest exposition in the DSM-IV revised, and secondly, the dynamic psychiatry tradition which includes the exploration of covert or unconscious motivation and character structure as elaborated by classic psychoanalysis and object-relations theory.

The descriptive tradition began in Ernst Kretschmer’s (1925) portrayal of observable schizoid behaviours which he organized into three groups of characteristics:

- unsociability, quietness, reservedness, seriousness, and eccentricity
- timidity, shyness with feelings, sensitivity, nervousness, excitability, and fondness of nature and books
- pliability, kindliness, honesty, indifference, silence, and cold emotional attitudes.

In these characteristics one can see the precursors of the DSM-IV division of schizoid character into three distinct personality disorders, though Kretschmer himself did not conceive of separating these behaviours to the point of radical isolation, considering them instead as simultaneously present as varying potentials in schizoid individuals. For Kretschmer the majority of schizoids are not either oversensitive or cold, but they are oversensitive and cold “at the same time” in quite different relative proportions, with a tendency to move along these dimensions from one behavior to the other.

The second path, that of dynamic psychiatry, began with observations by Eugen Bleuler (1924) who observed that the schizoid person and schizoid pathology were not things to be set apart. In 1940 W. R. D. Fairbairn presented his seminal work on the schizoid personality in which most of what is known today about schizoid phenomena can be found. Here Fairbairn delineated four central schizoid themes; firstly, the need to regulate interpersonal distance as a central focus of concern; secondly, the ability to mobilize self-preservative defenses and self-reliance; thirdly a pervasive tension between the anxiety-laden need for attachment, and the defensive need for distance, which manifests in observable behavior as indifference; and fourthly an overvaluation of the inner world at the expense of the outer world. Following Fairbairn, the dynamic psychiatry tradition has continued to produce rich explorations on the schizoid character, most notably from writers Nannarello (1953); Laing (1960); Winnicott (1965); Guntrip (1969); Khan (1974); Akhtar (1987); Seinfeld (1991); Manfield (1992); and Klein (1995).

**Schizotypal personality disorder**

Schizotypal personality disorder, or simply schizotypal disorder, is a personality disorder that is characterized by a need for social isolation, anxiety in social situations, odd behavior and thinking, and often unconventional beliefs.
Causes

Genetic

Although listed in the DSM-IV-TR on Axis II, schizotypal personality disorder is widely understood to be a "schizophrenia spectrum" disorder. Rates of schizotypal PD are much higher in relatives of individuals with schizophrenia than in the relatives of people with other mental illnesses or in people without mentally ill relatives. Technically speaking, schizotypal PD is an "extended phenotype" that helps geneticists track the familial or genetic transmission of the genes that are implicated in schizophrenia.

There are dozens of studies showing that individuals with schizotypal PD score similar to individuals with schizophrenia on a very wide range of neuropsychological tests. Cognitive deficits in patients with schizotypal PD are very similar to, but somewhat milder than, those for patients with schizophrenia.

Social and environmental

People with schizotypal PD, like patients with schizophrenia, may be quite sensitive to interpersonal criticism and hostility, and there is now evidence to suggest that parenting styles, early separation, and early childhood neglect can lead to the development of schizotypal traits.

Diagnosis

World Health Organization

The World Health Organization's ICD-10 lists schizotypal personality disorder as (F21.) Schizotypal disorder. (Note that in ICD-10, Schizotypal disorder is classified as a mental disorder associated with schizophrenia rather than a personality disorder as with DSM-IV. The DSM-IV designation of schizotypal as a personality disorder is controversial.)

It is characterized as:

A disorder characterized by eccentric behaviour and anomalies of thinking and affect which resemble those seen in schizophrenia, though no definite and characteristic schizophrenic anomalies have occurred at any stage. There is no dominant or typical disturbance, but any of the following may be present:

- Inappropriate or constricted affect (the individual appears cold and aloof);
- Behaviour or appearance that is odd, eccentric, or peculiar;
- Poor rapport with others and a tendency to social withdrawal;
- Odd beliefs or magical thinking, influencing behaviour and inconsistent with subcultural norms;
- Suspiciousness or paranoid ideas;
- Obsessive ruminations without inner resistance, often with dysmorphophobic, sexual or aggressive contents;
- Unusual perceptual experiences including somatosensory (bodily) or other illusions, depersonalization or derealization;
- Vague, circumstantial, metaphorical, overelaborate, or stereotyped thinking, manifested by odd speech or in other ways, without gross incoherence;
- Occasional transient quasi-psychotic episodes with intense illusions, auditory or other hallucinations, and delusion-like ideas, usually occurring without external provocation.

The disorder runs a chronic course with fluctuations of intensity. Occasionally it evolves into overt schizophrenia. There is no definite onset and its evolution and course are usually those of a personality disorder. It is more common in individuals related to people with schizophrenia and is believed to be part of the genetic "spectrum" of schizophrenia.

**Diagnostic Guidelines**

This diagnostic rubric is not recommended for general use because it is not clearly demarcated either from simple schizophrenia or from schizoid or paranoid personality disorders. If the term is used, three or four of the typical features listed above should have been present, continuously or episodically, for at least 2 years. The individual must never have met criteria for schizophrenia itself. A history of schizophrenia in a first-degree relative gives additional weight to the diagnosis but is not a prerequisite.

**Includes**

- borderline schizophrenia
- latent schizophrenia
- latent schizophrenic reaction
- prepsychotic schizophrenia
- prodromal schizophrenia
- pseudoneurotic schizophrenia
- pseudopsychopathic schizophrenia
- schizotypal personality disorder

**Excludes**

- Asperger’s syndrome
- schizoid personality disorder

**Millon’s subtypes**

Theodore Millon identified two subtypes of schizotypal. Any individual schizotypal may exhibit none or one of the following:
insipid schizotypal - a structural exaggeration of the passive-detached pattern. They include schizoid, depressive, dependent features.

- timorous schizotypal - a structural exaggeration of the active-detached pattern. They include avoidant, negativistic (passive-aggressive) features.

**Differential diagnosis**

There is a high rate of comorbidity with other personality disorders. McGlashan et al. (2000) stated that this may be due to overlapping criteria with other personality disorders, such as avoidant personality disorder, paranoid personality disorder and borderline personality disorder.

There are many similarities between the schizotypal and schizoid personalities. Most notable of the similarities is the inability to initiate or maintain relationships (both friendly and romantic). The difference between the two seems to be that those labeled as schizotypal avoid social interaction because of a deep-seated fear of people. The schizoid individuals simply feel no desire to form relationships, because they see no point in sharing their time with others.

**Prevalence (epidemiology)**

Schizotypal personality disorder occurs in 3% of the general population and occurs slightly more commonly in males.

**History**

The specific term schizotype was coined by Sandor Rado in 1956 as an abbreviation of schizophrenic phenotype.

**Schizoid Personality Disorder: Summarized**

**Schizoid Personality Disorder**

Schizoid Personality Disorder is characterized by a long-standing pattern of detachment from social relationships. A person with schizoid personality disorder often has difficulty expression emotions and does so typically in very restricted range, especially when communicating with others.

A person with this disorder may appear to lack a desire for intimacy, and will avoid close relationships with others. They may often prefer to spend time with themselves rather than socialize or be in a group of people. In laypeople terms, a person with schizoid personality disorder might be thought of as the typical "loner."

Individuals with Schizoid Personality Disorder may have particular difficulty expressing anger, even in response to direct provocation, which contributes to the impression that
they lack emotion. Their lives sometimes seem directionless, and they may appear to "drift" in their goals. Such individuals often react passively to adverse circumstances and have difficulty responding appropriately to important life events. Because of their lack of social skills and lack of desire for sexual experiences, individuals with this disorder have few friendships, date infrequently, and often do not marry. Employment or work functioning may be impaired, particularly if interpersonal involvement is required, but individuals with this disorder may do well when they work under conditions of social isolation.

**Symptoms of Schizoid Personality Disorder**

Schizoid personality disorder is characterized by a pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

- Neither desires nor enjoys close relationships, including being part of a family
- Almost always chooses solitary activities
- Has little, if any, interest in having sexual experiences with another person
- Takes pleasure in few, if any, activities
- Lacks close friends or confidants other than first-degree relatives
- Appears indifferent to the praise or criticism of others
- Shows emotional coldness, detachment, or flattened affectivity

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.

Schizoid personality disorder is more prevalent in males than females. Its prevalence in the general population is not known.

Like most personality disorders, schizoid personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

**How is Schizoid Personality Disorder Diagnosed?**

Personality disorders such as schizoid personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose schizoid personality disorder.

Many people with schizoid personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts
to significantly interfere or otherwise impact a person's life. This most often happens when a person's coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for schizoid personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Schizoid Personality Disorder**

Researchers today don't know what causes schizoid personality disorder. There are many theories, however, about the possible causes of schizoid personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual's personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

**Schizoid Personality Disorder Treatment**

**Psychotherapy**

While there are many suggested treatment approaches one could make for this disorder, none of them are likely to be easily effective. As with all personality disorders, the treatment of choice is individual psychotherapy. However, people with this disorder are unlikely to seek treatment unless they are under increased stress or pressure in their life. Treatment will usually be short-term in nature to help the individual solve the immediate crisis or problem. The patient will then likely terminate therapy. Goals of treatment most often are solution-focused using brief therapy approaches.

The development of rapport and a trusting therapeutic relationship will likely be a slow, gradual process that may not ever fully develop as in seeing people with other disorders. Because people who suffer from this disorder often maintain a social distance with people in their lives, even those close to them, the clinician should work to help ensure the client's security in the therapeutic relationship. Acknowledging the client's boundaries are important and the therapist should not look to confront the client on these types of issues.

Long-term psychotherapy should be avoided because of its poor treatment outcomes and the financial hardships inherent in length therapy. Instead, psychotherapy should focus on simple treatment goals to alleviate current pressing concerns or stressors within the individual's life. Cognitive-restructuring exercises may be appropriate for certain types of clear, irrational thoughts which are negatively influencing the patient's behaviors. The therapeutic framework should be clearly defined at the onset. Stability and support are the
keys to good treatment with someone who suffers from schizoid personality disorder. The therapist must be careful not to "smother" the client and be able to tolerate some possible "acting-out" behaviors.

Group therapy may be an alternative treatment modality to examine, although it is usually not a good initial treatment choice. A person who suffers from this disorder who is assigned to group therapy at the onset of therapy will likely terminate treatment prematurely because he or she will be unable to tolerate the effects of being in a social group. If, however, the person is graduating from individual to group therapy, they may have enough minimal social skills and abilities to tolerate group much better. People who suffer from this disorder see little to no reason for social interactions and often will be quite quiet in group, contributing little to others and offering little of themselves. This is to be expected and the individual who has schizoid personality disorder should not be pushed into participating more fully group until he or she is ready and on their own terms. Group leaders must be careful to help protect the individual from criticism from other group members for their lack of participation. Eventually, if the group can tolerate the initially-silent member with this disorder, the individual may gradually participate more and more, although this process will be very slow and drawn out over months.

Clinicians should be wary of too much isolation and introspection on the part of the patient. The goal is not to keep the individual in therapy as long as possible (although they may appreciate, if not fully utilize, therapy). As in group therapy, the individual who suffers from this disorder may engage in long periods of not talking and silence in session. These may be difficult to bear for the clinician. Phillip W. Long, M.D., also notes that the patient may eventually, "reveal a plethora of fantasies, imaginary friends, and fears of unbearable dependency - even of merging with the therapist. Oscillation between fear of clinging to the therapist may be followed by fleeing through fantasy and withdrawal." These types of feelings must be normalized by the clinician and brought into proper focus in the therapeutic relationship.

Medications

Medication is usually not an issue for someone who suffers from this disorder, unless they also have an additional Axis I disorder, such as major depression. Most patients show no additional improvement with the addition of an antidepressant medication, though, unless they are also suffering from suicidal ideation or a major depressive episode. Long-term treatment of this disorder with medication should be avoided; medication should be prescribed only for acute symptom relief. Additionally, prescription of medication may interfere with the effectiveness of certain psychotherapeutic approaches. Consideration of this effect should be taken into account when arriving at a treatment recommendation.

Self-Help

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. The social network provided within a self-help support group can be a very important component of increased,
higher life functioning and a decrease in an inability to function in the face of unexpected stressors. A supportive and non-invasive group can help a person who suffers from schizoid personality disorder overcome fears of closeness and feelings of isolation. Many support groups exist within communities throughout the world which are devoted to helping individuals with this disorder share their commons experiences and feelings.

Patients can be encouraged to try out new coping skills and learn that social attachments to others don’t have to be fraught with fear or rejection. They can be an important part of expanding the individual’s skill set and develop new, healthier social relationships.

**Schizotypal Personality Disorder: Summarized**

**Schizotypal Personality Disorder**

Schizotypal personality disorder is characterized by someone who has great difficulty in establishing and maintaining close relationships with others. A person with schizotypal personality disorder may have extreme discomfort with such relationships, and therefore have less of a capacity for them. Someone with this disorder usually has cognitive or perceptual distortions as well as eccentricities in their everyday behavior.

Individuals with Schizotypal Personality Disorder often have ideas of reference (e.g., they have incorrect interpretations of casual incidents and external events as having a particular and unusual meaning specifically for the person). People with this disorder may be unusually superstitious or preoccupied with paranormal phenomena that are outside the norms of their subculture.

Individuals with Schizotypal Personality Disorder often seek treatment for the associated symptoms of anxiety, depression, or other dysphoric affects rather than for the personality disorder features per se.

**Symptoms of Schizotypal Personality Disorder**

Schizotypal personality disorder is characterized by a pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behavior, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

- Ideas of reference (excluding delusions of reference)
- Odd beliefs or magical thinking that influences behavior, and is inconsistent with subcultural norms (e.g., superstitiousness, belief in clairvoyance, telepathy, or "sixth sense"; in children and adolescents, bizarre fantasies or preoccupations)
- Unusual perceptual experiences, including bodily illusions
- Odd thinking and speech (e.g., vague, circumstantial, metaphorical, overelaborate, or stereotyped)
- Suspiciousness or paranoid ideation
- Inappropriate or constricted affect
- Behavior or appearance that is odd, eccentric, or peculiar
- Lack of close friends or confidants other than first-degree relatives
- Excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.

Schizotypal personality disorder appears in less than 3 percent of the general population.

Like most personality disorders, schizotypal personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

**How is Schizotypal Personality Disorder Diagnosed?**

Personality disorders such as schizotypal personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose schizotypal personality disorder.

Many people with schizotypal personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person’s life. This most often happens when a person’s coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for schizotypal personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Schizotypal Personality Disorder**

Researchers today don’t know what causes schizotypal personality disorder. There are many theories, however, about the possible causes of schizotypal personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual's personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors.
factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

**Schizotypal Personality Disorder Treatment**

**Psychotherapy**

As with most personality disorders, schizotypal personality disorder is best treated with some form of psychotherapy. Individuals with this disorder usually distort reality more so than someone with Schizoid Personality Disorder.

As with Delusional Disorder and Paranoid Personality Disorder, the clinician must exercise care in therapy to not directly challenge delusional or inappropriate thoughts. A warm, supportive, and client-centered environment should be established with initial rapport. As with Avoidant Personality Disorder, the individual lacks an adequate social support system and usually avoids most social interactions because of extreme social anxiety. The patient often reports feelings of being "different" and not "fitting in" with others easily, usually because of their magical or delusion thinking. There is no simple solution to this problem. Social skills training and other behavioral approaches which emphasize the learning of the basics of social relationships and social interactions may be beneficial.

While individual therapy is the preferred modality at the onset of therapy, it may be appropriate to consider group therapy as the client progresses. Such a group should be for this specific disorder, though, which may be difficult to form or find in smaller communities.

**Medications**

Medication can be used for treatment of this disorder's more acute phases of psychosis. These phases are likely to manifest themselves during times of extreme stress or life events with which they cannot adequately cope. Psychosis is usually transitory, though, and should effectively resolve with the prescription of an appropriate anti-psychotic.

**Self-Help**

There are not any self-help support groups or communities that we are aware of that would be conducive to someone suffering from this disorder. Such approaches would likely not be very effective because a person with this disorder is likely to be mistrustful and suspicious of others and their motivations, making group help and dynamics unlikely and possibly harmful.

**Antisocial personality disorder**

Antisocial personality disorder (ASPD) is defined by the American Psychiatric Association's Axis II (personality disorders) of the Diagnostic and Statistical Manual (DSM-IV-TR) as "...a
pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood."

Antisocial personality disorder is sometimes referred to as psychopathy or sociopathy; however, many scholars make distinctions among these terms, though there remains no academic consensus as to their definitions. Currently, for this reason, neither psychopathy nor sociopathy are valid diagnoses described in the Diagnostic and Statistical Manual of Mental Disorders, and the ICD-10 of the World Health Organization also lacks psychopathy as a diagnostic disorder. Psychopathy is normally seen as a subset of the antisocial personality disorder, but Blair believes that the antisocial personality disorder and psychopathy may be separate conditions altogether.

**Diagnosis**

**DSM**

The Diagnostic and Statistical Manual of Mental Disorders fourth edition, DSM IV-TR = 301.7, a widely used manual for diagnosing mental disorders, defines antisocial personality disorder (in Axis II Cluster B) as:

A) There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three or more of the following:

- failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest;
- deception, as indicated by repeatedly lying, use of aliases, or conning others for personal profit or pleasure;
- impulsiveness or failure to plan ahead;
- irritability and aggressiveness, as indicated by repeated physical fights or assaults;
- reckless disregard for safety of self or others;
- consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations;
- lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another;

B) The individual is at least age 18 years.
C) There is evidence of conduct disorder with onset before age 15 years.
D) The occurrence of antisocial behavior is not exclusively during the course of schizophrenia or a manic episode.

New evidence points to the fact that children often develop Antisocial Personality Disorder as a cause of their environment, as well as their genetic line. The individual must be at least 18 years of age to be diagnosed with this disorder (Criterion B), but those commonly diagnosed with ASPD as adults were diagnosed with Conduct Disorder as children. The prevalence of this disorder is 3% in males and 1% from females, as stated from the DSM IV-TR.
Criticism

Researchers debate about whether psychopathy/sociopathy are incorrectly put together under ASPD. These clinicians and researchers who believe that it was incorrect to label the two in the same category are upset that an important distinction has been lost between these two disorders. In other words, the DSM-IV-TR considers ASPD and psychopathy to be the same, or similar. However, they are not the same since antisocial personality disorder is diagnosed via behavior and social deviance, whereas psychopathy also includes affective and interpersonal personality factors.

Other criticisms of ASPD are that it is essentially synonymous with criminality. Nearly 80%-95% of felons will meet criteria for ASPD — thus ASPD predicts nothing in criminal justice populations, whereas psychopathy (using the Hare Psychopathy Checklist-Revised (PCL-R)) is found in only roughly 20% of inmates and PCL-R is considered one of the best predictors of violent recidivism. Also, the DSM-IV field trials never included incarcerated populations.

The official stance of the American Psychiatric Association as presented in the DSM-IV-TR is that "psychopathy" and "sociopathy" are obsolete synonyms. The World Health Organization takes a similar stance in its ICD-10 by referring to psychopathy, antisocial personality, asocial personality, and amoral personality as synonyms for dissocial personality disorder.

WHO

The World Health Organization’s ICD-10 defines a conceptually similar disorder to antisocial personality disorder called (F60.2) Dissocial personality disorder.

It is characterized by at least 3 of the following:

- Callous unconcern for the feelings of others and lack of the capacity for empathy.
- Gross and persistent attitude of irresponsibility and disregard for social norms, rules, and obligations.
- Incapacity to maintain enduring relationships.
- Very low tolerance to frustration and a low threshold for discharge of aggression, including violence.
- Incapacity to experience guilt and to profit from experience, particularly punishment.
- Markedly prone to blame others or to offer plausible rationalizations for the behavior bringing the subject into conflict.
- Persistent irritability.

The criteria specifically rule out conduct disorders. Dissocial personality disorder criteria differ from those for antisocial and sociopathic personality disorders.
It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Millon's subtypes**

Theodore Millon identified five subtypes of antisocial behavior. Any antisocial individual may exhibit none, one or more than one of the following:

- covetous antisocial - variant of the pure pattern where individuals feel that life has not given them their due.
- reputation-defending antisocial - including narcissistic features
- risk-taking antisocial - including histrionic features
- nomadic antisocial - including schizoid, avoidant features
- malevolent antisocial - including sadistic, paranoid features.

**Differential diagnosis**

The following conditions commonly coexist with antisocial personality disorder:

- Anxiety disorders
- Depressive disorder
- Substance-related disorders
- Somatization disorder
- Borderline personality disorder
- Histrionic personality disorder
- Narcissistic personality disorder

When combined with alcoholism, people may show frontal function deficits on neuropsychological tests greater than those associated with each condition.

**Treatment**

To date there have been no controlled studies reported which found an effective treatment for ASPD, although contingency management programs, or a reward system, has been shown moderately effective for behavioral change. Some studies have found that the presence of ASPD does not significantly interfere with treatment for other disorders, such as substance abuse, although others have reported contradictory findings. Schema therapy is being investigated as a treatment for antisocial personality disorder, as well as medicinal marijuana treatments.

**Epidemiology**

Antisocial personality disorder is seen in 3% to 30% of psychiatric outpatients. The prevalence of the disorder is even higher in selected populations, such as people in prisons (who include many violent offenders). Similarly, the prevalence of ASPD is higher among
patients in alcohol or other drug (AOD) abuse treatment programs than in the general population (Hare 1983), suggesting a link between ASPD and AOD abuse and dependence.

Related terms

Psychopathy and sociopathy are terms related to ASPD, considered by many scholars to be obsolete terms. Psychopathy once referred to ASPD in general, but is now (like sociopathy) occasionally classified as a subset of ASPD. No scientific or academic consensus exists as to the specific differences between the three terms.

Psychopathy

Psychopathy (/saɪˈkæpθi/) was, until 1980, the term used for a personality disorder characterized by an abnormal lack of empathy combined with strongly amoral conduct but masked by an ability to appear outwardly normal. The publication of DSM-III changed the name of this mental disorder to Antisocial Personality Disorder and also broadened the diagnostic criteria considerably by shifting from clinical inferences to behavioral diagnostic criteria. However, the DSM-V working party is recommending a revision of Antisocial Personality Disorder to include "Antisocial/Psychopathic Type", with the diagnostic criteria having a greater emphasis on character than on behavior. The ICD-10 diagnostic criteria of the World Health Organization also lacks psychopathy as a personality disorder, its 1992 manual including Dissocial (Antisocial) Personality Disorder, which encompasses amoral, antisocial, asocial, psychopathic, and sociopathic personalities.

Sociopathy

Hare writes that the difference between sociopathy and psychopathy may "reflect the user's views on the origins and determinates of the disorder."

In the preface to the fifth edition of The Mask of Sanity, Cleckly stated, "... revisions of the nomenclature have been made by the American Psychiatric Association. The classification of psychopathic personality was changed to that of sociopathic personality in 1958", suggesting that he did not recognize any difference between the conditions.

David T. Lykken proposed psychopathy and sociopathy are two distinct kinds of antisocial personality disorder. He believed psychopaths are born with temperamental differences such as impulsivity, cortical underarousal, and fearlessness that lead them to risk-seeking behavior and an inability to internalize social norms. On the other hand, he claimed that sociopaths have relatively normal temperaments; their personality disorder being more an effect of negative sociological factors like parental neglect, delinquent peers, poverty, and extremely low or extremely high intelligence. Both personality disorders are the result of an interaction between genetic predispositions and environmental factors, but psychopathy leans towards the hereditary whereas sociopathy tends towards the environmental.
Antisocial Personality Disorder: Summarized

Antisocial Personality Disorder

Antisocial personality disorder is characterized by a long-standing pattern of a disregard for other people’s rights, often crossing the line and violating those rights. It usually begins in childhood or as a teen and continues into their adult lives.

Antisocial personality disorder is often referred to as psychopathy or sociopathy in popular culture.

Individuals with Antisocial Personality Disorder frequently lack empathy and tend to be callous, cynical, and contemptuous of the feelings, rights, and sufferings of others. They may have an inflated and arrogant self-appraisal (e.g., feel that ordinary work is beneath them or lack a realistic concern about their current problems or their future) and may be excessively opinionated, self-assured, or cocky. They may display a glib, superficial charm and can be quite voluble and verbally facile (e.g., using technical terms or jargon that might impress someone who is unfamiliar with the topic). Lack of empathy, inflated self-appraisal, and superficial charm are features that have been commonly included in traditional conceptions of psychopathy and may be particularly distinguishing of Antisocial Personality Disorder in prison or forensic settings where criminal, delinquent, or aggressive acts are likely to be nonspecific. These individuals may also be irresponsible and exploitative in their sexual relationships.

Symptoms of Antisocial Personality Disorder

Antisocial personality disorder is diagnosed when a person's pattern of antisocial behavior has occurred since age 15 (although only adults 18 years or older can be diagnosed with this disorder) and consists of the majority of these symptoms:

- Failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest
- Deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure
- Impulsivity or failure to plan ahead
- Irritability and aggressiveness, as indicated by repeated physical fights or assaults
- Reckless disregard for safety of self or others
- Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations
- Lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it. There should also be evidence of Conduct Disorder in the individual as a child, whether or not it was ever formally diagnosed by a professional.
Antisocial personality disorder is more prevalent in males (3 percent) versus females (1 percent) in the general population.

Like most personality disorders, antisocial personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

**How is Antisocial Personality Disorder Diagnosed?**

Personality disorders such as antisocial personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose antisocial personality disorder.

Many people with antisocial personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person’s life. This most often happens when a person’s coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for antisocial personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Antisocial Personality Disorder**

Researchers today don’t know what causes antisocial personality disorder. There are many theories, however, about the possible causes of antisocial personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual's personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

**Antisocial Personality Disorder Treatment**

**Introduction**
Antisocial personality disorder is often misunderstood by both professionals and laypeople. Confused with the popular terms, "sociopath" or "psychopath," someone who suffers from this disorder can be discriminated against within the mental health system, because of the symptoms of their disorder. Because there is usually a pervasive lack of remorse, and many time any feelings at all, they are assumed not to have any real feelings by many professionals. This can lead to difficulties within treatment.

Psychotherapy is nearly always the treatment of choice for this disorder; medications may be used to help stabilize mood swings or specific and acute Axis I concurrent diagnoses. There is no research that supports the use of medications for direct treatment of antisocial personality disorder, though.

**Psychotherapy**

As with most personality disorders, individuals with this disorder rarely seek treatment on their own, without being mandated to therapy by a court or significant other. Court referrals for assessment and treatment for this disorder are likely the most common referral source. A careful and thorough assessment will ensure that the person that the person has antisocial personality disorder. This can often be confused with simple criminal activity (all criminals do not have this disorder), adult antisocial behavior, and other activities which do not justify the personality disorder diagnosis. As with a thorough assessment of any suspected personality disorder, formal psychological testing should be considered invaluable.

Because many people who suffer from this disorder will be mandated to therapy, sometimes in a forensic or jail setting, motivation on the patient's part may be difficult to find. In a confined setting, it may be nearly impossible and therapy should then focus on alternative life issues, such as goals for when they are released from custody, improvement in social or family relationships, learning new coping skills, etc. In an outpatient setting, the focus of therapy can also be on these types of issues, but a part of the therapy should be devoted to discussing the antisocial behavior and feelings (or lack thereof). Common in the population who suffer from antisocial personality disorder is the lack of connections between feelings and behaviors. Helping the client draw those lines between the two may be beneficial.

Threats are never an appropriate motivating factor in any sort of treatment, and least of all with this disorder. If the only way to motivate the patient is to threaten to report their noncompliance with therapy to the courts or warden, it is highly unlikely the clinician will make any type of gains within therapy anyway. It is appropriate, however, to try and help the individual with this disorder find good reasons that they may want to work on this problem further. For instance, ensuring that they not come into contact with the court system again, be incarcerated, have to submit themselves to additional psychological examinations, etc.

Effective psychotherapy treatment for this disorder is limited. It is likely, though, that intensive, psychoanalytic approaches are inappropriate for this population. Approaches the
reinforce appropriate behaviors and attempting to make connections between the person’s actions and their feelings may be more beneficial. Emotions are usually a key aspect of treatment of this disorder. Patients often have had little or no significant emotionally-rewarding relationships in their lives. The therapeutic relationship, therefore, can be one of the first ones. This can be very scary for the client, initially, and it may become intolerable. A close therapeutic relationship can only occur when a good and solid rapport has been established with the client and he or she can trust the therapist implicitly.

Trust brings up the issue of confidentiality, since often the patient with antisocial personality disorder is mandated to therapy. This means that the clinician may have to occasionally report on the patient’s progress in therapy. While this can usually be done in a very general way which reveals no significant details of the content of therapy, it is still an important issue for the client. He or she may be suspicious and distrustful of the clinician at first, since it will be unclear as to who has the highest priority -- the patient or the court. This fear can only be alleviated with an honest disclosure as to what the therapist will reveal to the courts, and with time, as the client learns that what he says in the therapy session does not become common knowledge. The limitations of therapy should be discussed with the patient up-front, in a clear and matter-of-fact manner, so there are no misunderstandings later.

The content of therapy should focus on the patient’s emotions (or lack thereof). As the individual learns to experience various emotional states, one of the first may be depression. The client will likely be unfamiliar with the feelings associated with depression, and so it is beneficial for the clinician to be supportive and empathetic to the individual during this time. Reinforcing any emotions, outside of anger or frustration, is usually beneficial. Experiencing intense affect is usually a sign of progress in therapy. Staying on "safe issues," and discussing more real-life concerns, while one way of treating this disorder, is not likely to be as effective in long term behavioral change as an approach emphasizing the discovery and labeling of appropriate emotional states.

People who have antisocial personality disorder often experience difficulties with authority figures. The therapist should usually take a neutral stance in this matter, since it is a firmly held belief by the client. The clinician should avoid arguments and taking sides on authority issues and those who hold authority over the client. Their moral and ethical makeup may leave a lot to be desired as well. While this may be an appropriate topic for discussion in therapy, it will also likely be one of little progress. Usually one of the more effective ways for a person with this disorder to learn to change their ineffective behaviors is to have to face up to the consequences of their behavior. This sometimes means dealing with courts and jails, but it can also eventually be a motivating factor in the client’s treatment.

Other modalities of psychotherapy, such as group and family therapy, can be helpful. Often people with this disorder find themselves in a group setting, because they aren’t given any treatment choices. This is usually not conducive to their treatment, since in most groups, the individual can remain emotionally-closed and has little reason to share with others. It also doesn’t help that these groups are often made up of people suffering from a wide range of mental disorders. Groups which are devoted exclusively to this disorder, though rare, are
the best choice. In such a group, the patient is given a greater reason to contribute and share with others. Care must be utilized by group leaders to ensure the group doesn't become a "How-to" course in criminal behavior. Family therapy can be helpful to increase education and understanding among family members. Families often misunderstand and are confused about the cause of the antisocial behaviors and the idea that it is a mental disorder. Phillip W. Long, M.D. adds, "This confusion, guilt, the temptation to make restitution for the patient's criminal acts, and the frustrations of working with someone who is seen to be quite ill but who will not be treated should all be discussed openly with family members."

While there are many theories, as with all personality disorders, research has found little significant causative factors.

**Hospitalization**

Rarely is inpatient care appropriate or necessary for this personality disorder. Like most personality disorders, most people will go through their lives with little realization of the difficulty they have. In this case, though, the person is more likely to be seen as a criminal and have a history of difficulties with the law. Loss of freedom may be more of a motivating factor than in other personality disorders, so some specialized treatment facilities have started to treat people with this disorder.

One such program we've read about is the Patuxent Institute, located in Jessup, Maryland in the U.S. This hospital utilizes a strict behavioral approach of placing patients on a token economy based upon their treatment progress. This is a relatively new and radical approach to this sort of disorder and little research has been conducted to confirm its long-term effectiveness.

As with any treatment, the focus on feelings and connecting antisocial behavior to appropriate feeling states is appropriate. Since inpatient programs tend to be more intensive and expensive, they are rarely sought out by the patient themselves. Community followup and support, either by the hospital or professionals, or with the use of self-help support groups, is imperative to maintaining treatment gains.

**Medications**

Medications should only be utilized to treat clear, acute and serious Axis I concurrent diagnoses. No research has suggested that any medication is effective in the treatment of this disorder.

**Self-Help Strategies**

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. Groups can be especially helpful for people with this disorder, if they are tailored specifically for antisocial personality disorder. Individuals with this disorder typically feel more at ease in discussing
their feelings and behaviors in front of their peers in this type of supportive modality. Leaders of such self-help support groups, though, must be wary of individuals who come to group just to brag about their exploits and who may seek to use the group inappropriately. Usually a group can be very helpful and beneficial to most people with this disorder, once they overcome their initial fears and hesitation to join such a group. Many support groups exist within communities throughout the world which are devoted to helping individuals with this disorder share their commons experiences and feelings.

Borderline personality disorder

Borderline personality disorder (BPD) is a personality disorder described as a prolonged disturbance of personality function in a person (generally over the age of eighteen years, although it is also found in adolescents), characterized by depth and variability of moods.[n 1] The disorder typically involves unusual levels of instability in mood; black and white thinking, or splitting; the disorder often manifests itself in idealization and devaluation episodes, as well as chaotic and unstable interpersonal relationships, self-image, identity, and behavior; as well as a disturbance in the individual's sense of self. In extreme cases, this disturbance in the sense of self can lead to periods of dissociation.

BPD splitting includes a switch between idealizing and demonizing others. This, combined with mood disturbances, can undermine relationships with family, friends, and co-workers. BPD disturbances also may include self-harm. Without treatment, symptoms may worsen, leading (in extreme cases) to suicide attempts.

There is an ongoing debate among clinicians and patients worldwide about terminology and the use of the word borderline, and some have suggested that this disorder should be renamed. The ICD-10 manual has an alternative definition and terminology to this disorder, called Emotionally unstable personality disorder.

There is related concern that the diagnosis of BPD stigmatizes people and supports pejorative and discriminatory practices. It is common for those suffering from borderline personality disorder and their families to feel compounded by a lack of clear diagnoses, effective treatments, and accurate information. This is true especially because of evidence that this disorder originates in the families of those who suffer from it and has a lot to do with psychosocial and environmental factors (Axis IV), rather than belonging strictly in the personality disorders and mental retardation section (Axis II) of the DSM-IV construct. Conceptual, as well as therapeutic, relief may be obtained through evidence that BPD is closely related to traumatic events during childhood and to post-traumatic stress disorder (PTSD), about which much more is known.

Signs and symptoms

Borderline personality disorder is a diagnosis about which many articles and books have been written, yet about which very little is known based on empirical research.
Studies suggest that individuals with BPD tend to experience frequent, strong and long-lasting states of aversive tension, often triggered by perceived rejection, being alone or perceived failure. Individuals with BPD may show lability (changeability) between anger and anxiety or between depression and anxiety and temperamental sensitivity to emotive stimuli.

The negative emotional states specific to BPD may be grouped into four categories: destructive or self-destructive feelings; extreme feelings in general; feelings of fragmentation or lack of identity; and feelings of victimization.

Individuals with BPD can be very sensitive to the way others treat them, reacting strongly to perceived criticism or hurtfulness. Their feelings about others often shift from positive to negative, generally after a disappointment or perceived threat of losing someone. Self-image can also change rapidly from extremely positive to extremely negative. Impulsive behaviors are common, including alcohol or drug abuse, unsafe sex, gambling and recklessness in general. Attachment studies suggest individuals with BPD, while being high in intimacy- or novelty-seeking, can be hyper-alert to signs of rejection or not being valued and tend toward insecure, avoidant or ambivalent, or fearfully preoccupied patterns in relationships. They tend to view the world generally as dangerous and malevolent, and tend to view themselves as powerless, vulnerable, unacceptable and unsure in self-identity.

Individuals with BPD are often described, including by some mental health professionals (and in the DSM-IV), as deliberately manipulative or difficult, but analysis and findings generally trace behaviors to inner pain and turmoil, powerlessness and defensive reactions, or limited coping and communication skills. There has been limited research on family members' understanding of borderline personality disorder and the extent of burden or negative emotion experienced or expressed by family members. However the effect of expressed emotion by family members may actually be opposite (paradoxical) from the anticipated effect on individuals with such illnesses as depressive disorders and schizophrenia. For BPD such effect may be neutral or positive as opposed to negative, a counter-intuitive result.

Parents of individuals with BPD have been reported to show co-existing extremes of over-involvement and under-involvement. BPD has been linked to increased levels of chronic stress and conflict in romantic relationships, decreased satisfaction of romantic partners, abuse and unwanted pregnancy; these links may be general to personality disorder and subsyndromal problems.

Suicidal or self-harming behavior is one of the core diagnostic criteria in DSM IV-TR, and management of and recovery from this can be complex and challenging. The suicide rate is approximately 8 to 10 percent. Self-injury attempts are highly common among patients and may or may not be carried out with suicidal intent. BPD is often characterized by multiple low-lethality suicide attempts triggered by seemingly minor incidents, and less commonly by high-lethality attempts that are attributed to impulsiveness or comorbid major depression, with interpersonal stressors appearing to be particularly common triggers. Ongoing family interactions and associated vulnerabilities can lead to self-destructive
behavior. Stressful life events related to sexual abuse have been found to be a particular trigger for suicide attempts by adolescents with a BPD diagnosis.

**Diagnosis**

Diagnosis is based on a clinical assessment by a qualified mental health professional. The assessment incorporates the patient’s self-reported experiences as well as the clinician's observations. The resulting profile may be supported or corroborated by long-term patterns of behavior as reported by family members, friends or co-workers. The list of criteria that must be met for diagnosis is outlined in the DSM-IV-TR.

Borderline personality disorder was once classified as a subset of schizophrenia (describing patients with borderline schizophrenic tendencies). Today BPD is used more generally to describe individuals who display emotional dysregulation and instability, with paranoid ideation or delusions being only one criterion (criterion #9) of a total of 9 criteria, of which 5, or more, must be present for this diagnosis.

Individuals with BPD are at high risk of developing other psychological disorders such as anxiety and depression. Other symptoms of BPD, such as dissociation, are frequently linked to severely traumatic childhood experiences, which some put forth as one of the many root causes of the borderline personality.

**Adolescence**

Onset of symptoms typically occurs during adolescence or young adulthood. Symptoms may persist for several years, but the majority of symptoms lessen in severity over time, with some individuals fully recovering. The mainstay of treatment is various forms of psychotherapy, although medication and other approaches may also improve symptoms. While borderline personality disorder can manifest itself in children and teenagers, therapists are discouraged from diagnosing anyone before the age of 18, due to adolescence and a still-developing personality.

There are some instances when BPD can be evident and diagnosed before the age of 18. The DSM-IV states: "To diagnose a personality disorder in an individual under 18 years, the features must have been present for at least 1 year." In other words, it is possible to diagnose the disorder in children and adolescents, but a more conservative approach should be taken.

There is some evidence that BPD diagnosed in adolescence is predictive of the disease continuing into adulthood. It is possible that the diagnosis, if applicable, would be helpful in creating a more effective treatment plan for the child or teen.

**Diagnostic and Statistical Manual**
The Diagnostic and Statistical Manual of Mental Disorders fourth edition, DSM IV-TR, a widely used manual for diagnosing mental disorders, defines borderline personality disorder (in Axis II Cluster B) as:

A pervasive pattern of instability of interpersonal relationships, self-image and affects, as well as marked impulsivity, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

- Frantic efforts to avoid real or imagined abandonment. Note: Do not include suicidal or self-injuring behavior covered in Criterion 5
- A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.
- Identity disturbance: markedly and persistently unstable self-image or sense of self.
- Impulsivity in at least two areas that are potentially self-damaging (e.g., promiscuous sex, eating disorders, binge eating, substance abuse, reckless driving). Note: Do not include suicidal or self-injuring behavior covered in Criterion 5
- Recurrent suicidal behavior, gestures, threats or self-injuring behavior such as cutting, interfering with the healing of scars (excoriation) or picking at oneself.
- Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability or anxiety usually lasting a few hours and only rarely more than a few days).
- Chronic feelings of emptiness
- Inappropriate anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).
- Transient, stress-related paranoid ideation, delusions or severe dissociative symptoms

It is a requirement of DSM-IV that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**International Classification of Disease**

The World Health Organization’s ICD-10 defines a conceptually similar disorder to borderline personality disorder called (F60.3) Emotionally unstable personality disorder. It has two subtypes described below.

**F60.30 Impulsive type**

At least three of the following must be present, one of which must be (2):

- marked tendency to act unexpectedly and without consideration of the consequences;
- marked tendency to quarrelsome behaviour and to conflicts with others, especially when impulsive acts are thwarted or criticized;
- liability to outbursts of anger or violence, with inability to control the resulting behavioural explosions;
- difficulty in maintaining any course of action that offers no immediate reward;
- unstable and capricious mood.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**F60.31 Borderline type**

At least three of the symptoms mentioned in F60.30 Impulsive type must be present [see above], with at least two of the following in addition:

- disturbances in and uncertainty about self-image, aims, and internal preferences (including sexual);
- liability to become involved in intense and unstable relationships, often leading to emotional crisis;
- excessive efforts to avoid abandonment;
- recurrent threats or acts of self-harm;
- chronic feelings of emptiness.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Chinese Society of Psychiatry**

The Chinese Society of Psychiatry’s CCMD has a comparable diagnosis of Impulsive Personality Disorder (IPD). A patient diagnosed as having IPD must display "affective outbursts" and "marked impulsive behavior," plus at least three out of eight other symptoms. The construct has been described as a hybrid of the impulsive and borderline subtypes of the ICD-10’s Emotionally Unstable Personality Disorder, and also incorporates six of the nine DSM BPD criteria.

**Millon's subtypes**

Theodore Millon identified four subtypes of borderline. Any individual borderline may exhibit none, or one or more of the following:

- Discouraged borderline — including avoidant, depressive or dependent features
- Impulsive borderline — including histrionic or antisocial features
- Petulant borderline — including negativistic (passive-aggressive) features
- Self-destructive borderline — including depressive or masochistic features

**Differential diagnosis**

Common comorbid (co-occurring) conditions are mental disorders such as substance abuse, depression and other mood and personality disorders.
Borderline personality disorder and mood disorders often appear concurrently. Some features of borderline personality disorder may overlap with those of mood disorders, complicating the differential diagnostic assessment.

Both diagnoses involve symptoms commonly known as "mood swings." In borderline personality disorder, the term refers to the marked lability and reactivity of mood defined as emotional dysregulation. The behavior is typically in response to external psychosocial and intrapsychic stressors, and may arise or subside, or both, suddenly and dramatically and last for seconds, minutes, hours, days, weeks or months.

Bipolar depression is generally more pervasive with sleep and appetite disturbances, as well as a marked nonreactivity of mood, whereas mood with respect to borderline personality and co-occurring dysthymia remains markedly reactive and sleep disturbance not acute.

The relationship between bipolar disorder and borderline personality disorder has been debated. Some hold that the latter represents a subthreshold form of affective disorder, while others maintain the distinctness between the disorders, noting they often co-occur.

Some findings suggest that BPD may lie on a bipolar spectrum, with a number of points of phenomenological and biological overlap between the affective lability criterion of borderline personality disorder and the extremely rapid cycling bipolar disorders. Some findings suggest that the DSM-IV BPD diagnosis mixes up two sets of unrelated items—an affective instability dimension related to Bipolar-II, and an impulsivity dimension not related to Bipolar-II.

It is important to emphasize that medical conditions which cause organic behavioral function may result in a clinical picture that mimics to some degree BPD. Hormonal dysfunction over a long period, or brain dysfunction (e.g. the encephalopathy caused by lyme disease) can result in identity disturbance and mood lability, as can many other chronic medical conditions such as lupus. These conditions may isolate the patient socially and emotionally, and/or cause limbic damage to the brain. However, this is not borderline personality disorder which results, but rather a reaction to the isolating circumstances caused by a medical condition and the possibly coincident struggles of the patient to control his or her mood given damage to the brain's limbic system. Heavy alcohol usage over a long period itself can cause an encephalopathy which may cause limbic damage. Various frontal lobe syndromes can result in disinhibition and impulsive behavior.

Comorbid (co-occurring) conditions in BPD are common. When comparing individuals diagnosed with BPD to those diagnosed with other personality disorders, the former showed a higher rate of also meeting criteria for

- anxiety disorders
- mood disorders (including clinical depression and bipolar disorder)
- eating disorders (including anorexia nervosa and bulimia)
- and, to a lesser extent, somatoform or factitious disorders
dissociative disorders

Substance abuse is a common problem in BPD, whether due to impulsivity or as a coping mechanism, and 50 percent to 70 percent of psychiatric inpatients with BPD have been found to meet criteria for a substance use disorder, especially alcohol dependence or abuse which is often combined with the abuse of other drugs.

Causes

As with other mental disorders, the causes of BPD are complex and not fully understood. One finding is a history of childhood trauma, abuse or neglect, although researchers have suggested diverse possible causes, such as a genetic predisposition, neurobiological factors, environmental factors, or brain abnormalities.

There is evidence that suggests that BPD and post-traumatic stress disorder (PTSD) are closely related. Evidence further suggests that BPD might result from a combination that can involve a traumatic childhood, a vulnerable temperament and stressful maturational events during adolescence or adulthood.

Childhood abuse

Numerous studies have shown a strong correlation between child abuse, especially child sexual abuse, and development of BPD. Many individuals with BPD report to have had a history of abuse and neglect as young children. Patients with BPD have been found to be significantly more likely to report having been verbally, emotionally, physically or sexually abused by caregivers of either gender. There has also been a high incidence of incest and loss of caregivers in early childhood for people with borderline personality disorder. They were also much more likely to report having caregivers (of both genders) deny the validity of their thoughts and feelings. They were also reported to have failed to provide needed protection, and neglected their child’s physical care. Parents (of both sexes) were typically reported to have withdrawn from the child emotionally, and to have treated the child inconsistently. Additionally, women with BPD who reported a previous history of neglect by a female caregiver and abuse by a male caregiver were consequently at significantly higher risk for being sexually abused by a noncaregiver (not a parent). It has been suggested that children who experience chronic early maltreatment and attachment difficulties may go on to develop borderline personality disorder.

Other developmental factors

Some studies suggest that BPD may not necessarily be a trauma-spectrum disorder and that it is biologically distinct from the post-traumatic stress disorder that could be a precursor. The personality symptom clusters seem to be related to specific abuses, but they may be related to more persistent aspects of interpersonal and family environments in childhood.

Otto Kernberg formulated a theory of borderline personality based on a premise of failure to develop in childhood. Writing in the psychoanalytic tradition, Kernberg argued that
failure to achieve the developmental task of psychic clarification of self and other can result
in an increased risk to develop varieties of psychosis, while failure to overcome splitting
results in an increased risk to develop a borderline personality.

**Genetics**

An overview of the existing literature suggested that traits related to BPD are influenced by
genes. A major twin study found that if one identical twin met criteria for BPD, the other
also met criteria in 35 percent of cases. People that have BPD influenced by genes usually
have a close relative with the disorder.

Twin, sibling and other family studies indicate a partially heritable basis for impulsive
aggression, but studies of serotonin-related genes to date have suggested only modest
contributions to behavior.

**Mediators and moderators**

While research has examined variables that predict the development of borderline
personality disorder (BPD), researchers have only recently begun to examine the variables
that mediate and moderate the relationships between these variables and the development
of the disorder. A mediator is a variable that affects how the relationship occurs. Mediation
is said to be present when both the predictor variable and the mediating variable are
significantly correlated with the dependent variable, and when the relationship between
the predictor variable and the outcome variable is significantly reduced when controlling
for the mediating variable. A moderating variable by contrast specifies the conditions
under which a given outcome will occur. Moderation is said to occur when there is an
interaction effect between the predicting variable and the moderating variable on the
dependent variable. More specifically, the effect of the predicting variable is different
depending on the level of the moderating variable.

Research has found statistically significant relationships between BPD symptoms and both
sexual and physical abuse. Other factors including family environment variables also
contribute to the development of the disorder. Bradley et al. found that both child sexual
abuse (CSA) and childhood physical abuse and BPD symptoms were significantly related,
and both CSA and childhood physical abuse were significantly related to family
environment. When family environment and childhood physical abuse were entered
simultaneously into a regression equation, family environment was related to BPD
symptoms and childhood physical abuse was related to BPD symptoms, although the
relationship between BPD symptoms and childhood physical abuse was reduced.
Therefore, CSA and childhood physical abuse both directly influence the development of
BPD symptoms directly and are mediated by family environment.

Other research has examined the relationship between negative affectivity, thought
suppression and BPD symptoms. The results of the mediational models in this study found
that thought suppression mediated the relationship between negative affectivity and BPD
symptoms. While negative affectivity significantly predicted BPD symptoms after
controlling for CSA, this relationship was greatly reduced when thought suppression was introduced into the model. Thus, the relationship of negative affectivity to BPD symptoms is mediated by thought suppression.

Ayduk et al. (2008) found an interaction between rejection sensitivity and executive control in the prediction of BPD symptoms. This study found that BPD features were positively associated with rejection sensitivity (RS) and neuroticism and negatively associated with emotional control (EC). Their statistical analysis indicated that among those low in EC, RS was positively related to BPD features and among those high in RS, EC was negatively associated with BPD. By contrast, among those high in EC, RS was not significantly related to BP features, and among those low in RS, EC was not related to BPD features. In Study 2, BPD features were positively correlated to RS and negatively correlated with executive control. Additionally, the authors found that delay gratification times at age 4 had no significant relationship with BPD features at the time of the current study. Again, as in Study 1, the RS x EC interaction was significant. Among those low in EC, RS was positively related to BPD features, while among those high in EC, the effect of RS was reduced to marginal significance. Moreover, among those high in RS, EC was negatively associated with BPD features, but among those low in RS, EC was unrelated to BPD features.

Parker, Boldero and Bell (2006) indicated that both AI and AO self-discrepancy magnitudes were strongly correlated to each other and to BPD features. Self-complexity was not significantly related to any of the other factors. Among those high in self-complexity, the relationship between AI self-discrepancy magnitudes and BPD features was lower than among those with less self-complexity. Actual-ought self-discrepancy relationship with BPD features was not significantly moderated by self-complexity.

BPD is complex, and several factors have an impact on whether clinical features of BPD are present. None of the prediction factors above are sufficient to be the key factor in the development of BPD features. Increased knowledge of the development of the disorder may help prevent symptom aggravation and identify new treatment strategies. Future research should integrate the knowledge gained from these areas and study these variables simultaneously. Studies in which these variables are simultaneously examined would provide greater specificity in the relationships between the variables. These articles taken together not only increase our knowledge of what factors and variables lead to the development of BPD features and BPD itself but also, when taken together, indicate future lines of research yet to be studied.

Management

Psychotherapy forms the foundation of treatment for borderline personality disorder with medications playing a lesser role. Treatments should be based on individual case presentation, rather than upon the diagnosis of BPD with co-morbid conditions determining medications use, if any. Hospitalization has not been found to improve outcomes or prevent suicide over community care in those with BPD.
Psychotherapy

A number of techniques have been studied for borderline personality disorder including cognitive behavioral therapy, interpersonal therapy, dialectical behavior therapy, and psychodynamic therapy among others. A special problem of psychotherapy with borderline patients is intense projection. It requires the psychotherapist to be flexible in considering negative attributions by the patient rather than quickly interpreting the projection.

Medications

The evidence of benefit for antipsychotics, mood stabilizers, and omega-3 fatty acids is weak. Antidepressants, antipsychotics and mood stabilisers (such as lithium) are regularly used however to treat co-morbid symptoms such as depression.

Services

Individuals with BPD sometimes use mental health services extensively. They accounted for about 20 percent of psychiatric hospitalizations in one survey. The majority of BPD patients continue to use outpatient treatment in a sustained manner for several years, but the number using the more restrictive and costly forms of treatment, such as inpatient admission, declines with time. Experience of services varies. Assessing suicide risk can be a challenge for mental health services (and patients themselves tend to underestimate the lethality of self-injurious behaviours) with typically a chronically elevated risk of suicide much above that of the general population and a history of multiple attempts when in crisis.

Prognosis

The American Psychiatric Association states that recent advancements have led to treatments reaching an 86% remission rate 10 years after treatment.

Particular difficulties have been observed in the relationship between care providers and individuals diagnosed with BPD. A majority of psychiatric staff report finding individuals with BPD moderately to extremely difficult to work with, and more difficult than other client groups. Some clients feel a diagnosis is helpful, allowing them to understand they are not alone, and to connect with others who have BPD and who have developed helpful coping mechanisms. On the other hand, some with the diagnosis of BPD have reported that the term "BPD" felt like a pejorative label rather than a helpful diagnosis, that self-destructive behaviour was incorrectly perceived as manipulative, and that they had limited access to care. Attempts are made to improve public and staff attitudes.

Epidemiology

The prevalence of BPD in the general population ranges from 1 to 2 percent. The diagnosis appears to be several times more common in (especially young) women than in men, by as much as 3:1, according to the DSM-IV-TR, although the reasons for this are not clear.
The prevalence of BPD in the United States has been calculated as 1 percent to 3 percent of the adult population, with approximately 75 percent of those diagnosed being female. It has been found to account for 20 percent of psychiatric hospitalizations.

History

Since the earliest record of medical history, the coexistence of intense, divergent moods within an individual has been recognized by such writers as Homer, Hippocrates and Aretaeus, the last describing the vacillating presence of impulsive anger, melancholia and mania within a single person. After medieval suppression of the concept, it was revived by Swiss physician Théophile Bonet in 1684, who, using the term folie maniaco-mélancolique, noted the erratic and unstable moods with periodic highs and lows that rarely followed a regular course. His observations were followed by those of other writers who noted the same pattern, including writers such as the American psychiatrist C. Hughes in 1884 and J.C. Rosse in 1890, who described "borderline insanity". Kraepelin, in 1921, identified an "excitable personality" that closely parallels the borderline features outlined in the current concept of borderline.

Adolf Stern wrote the first significant psychoanalytic work to use the term "borderline" in 1938, referring to a group of patients with what was thought to be a mild form of schizophrenia, on the borderline between neurosis and psychosis. For the next decade the term was in popular and colloquial use, a loosely conceived designation mostly used by theorists of the psychoanalytic and biological schools of thought. Increasingly, theorists who focused on the operation of social forces were recognized as well.

The 1960s and 1970s saw a shift from thinking of the borderline syndrome as borderline schizophrenia to thinking of it as a borderline affective disorder (mood disorder), on the fringes of manic depression, cyclothymia and dysthymia. In DSM-II, stressing the affective components, it was called cyclothymic personality (affective personality). In parallel to this evolution of the term "borderline" to refer to a distinct category of disorder, psychoanalysts such as Otto Kernberg were using it to refer to a broad spectrum of issues, describing an intermediate level of personality organization between neurotic and psychotic processes.

Standardized criteria were developed to distinguish BPD from affective disorders and other Axis I disorders, and BPD became a personality disorder diagnosis in 1980 with the publication of DSM-III. The diagnosis was formulated predominantly in terms of mood and behavior, distinguished from sub-syndromal schizophrenia which was termed "Schizotypal personality disorder". The final terminology in use by the DSM today was decided by the DSM-IV Axis II Work Group of the American Psychiatric Association.

Society and culture

Film and television
Several films portraying characters either explicitly diagnosed or with traits strongly suggestive of mental illness have been the subject of discussion by certain psychiatrists and film experts. The films Play Misty for Me and Fatal Attraction are two examples, as is the memoir Girl, Interrupted by Susanna Kaysen (and the movie based on it, with Winona Ryder as Kaysen). Each of these films suggests the emotional instability of the disorder; however, the first two cases show a person more aggressive to others than to herself, which in fact is less typical. The 1992 film Single White Female suggests different aspects of the disorder: the character Hedy suffers from a markedly disturbed sense of identity and, as with the last two films, abandonment leads to drastic measures.

The character of Anakin Skywalker/Darth Vader in the Star Wars films has been "diagnosed" as having BPD. Psychiatrists Eric Bui and Rachel Rodgers have argued that the character meets six of the nine diagnostic criteria; Bui also found Anakin a useful example to explain BPD to medical students. In particular, Bui points to the character's abandonment issues, uncertainty over his identity and violent dissociative episodes. Other films attempting to depict characters with the disorder include The Crush, Malicious, Interiors, Notes On a Scandal, The Cable Guy and Cracks. The film Borderline, based on the book of the same name by Marie-Sissi Labrèche, attempts to explore BPD through its main character, Kiki.

**Literature**

The memoir Songs of Three Islands by Millicent Monks is a meditation on how BPD affects several generations of the wealthy Carnegie family.

In Lois McMaster Bujold's science fiction novel Komarr, Tien Vorsoisson has BPD; her disorder drives a large part of the story.

**Awareness**

In early 2008, the United States House of Representatives declared the month of May as Borderline Personality Disorder Awareness Month.

**Controversies**

**Gender**

The diagnosis of BPD has been criticized from a feminist perspective. This is because some of the diagnostic criteria/symptoms of the disorder uphold common gender stereotypes about women. For example, the criteria of "a pattern of unstable personal relationships, unstable self-image, and instability of mood," can all be linked to the stereotype that women are "neither decisive nor constant". The question has also been raised of why women are three times more likely to be diagnosed with BPD than men. Some think that people with BPD commonly have a history of sexual abuse in childhood. One feminist critique suggests that BPD is a stigmatizing diagnosis that can sometimes evoke negative responses from health care providers, and additionally, that women who have survived
sexual abuse in childhood are therefore sometimes re-traumatized by any such abusive mental health service.

Some feminist writers have suggested it would be better to give these women the diagnosis of a post-traumatic disorder as this would acknowledge their abuse, but others have argued that the use of the PTSD diagnosis merely medicalizes abuse rather than addressing the root causes in society. Women may be more likely to receive a personality disorder diagnosis if they reject the female role by being hostile, successful or sexually active; alternatively if a woman presents with psychiatric symptoms but does not conform to a traditional passive sick role, she may be labelled as a "difficult" patient and given the stigmatizing diagnosis of BPD.

Stigma

The features of BPD include emotional instability, intense unstable interpersonal relationships, a need for relatedness and a fear of rejection. As a result, people with BPD often evoke intense emotions in those around them. Pejorative terms to describe persons with BPD such as “difficult,” “treatment resistant,” “manipulative,” “demanding” and “attention seeking” are often used, and may become a self-fulfilling prophecy as the clinician’s negative response triggers further self-destructive behaviour. In psychoanalytic theory, this stigmatization may be thought to reflect countertransference (when a therapist projects their own feelings on to a client), as people with BPD are prone to use defense mechanisms such as splitting and projective identification. Thus the diagnosis “often says more about the clinician’s negative reaction to the patient than it does about the patient … as an expression of counter transference hate, borderline explains away the breakdown in empathy between the therapist and the patient and becomes an institutional epithet in the guise of pseudoscientific jargon” (Aronson, p 217).

This inadvertent counter transference can give rise to inappropriate clinical responses including excessive use of medication, inappropriate mothering and punitive use of limit setting and interpretation. People with BPD are seen as among the most challenging groups of patients, requiring a high degree of skill and training in the psychiatrists, therapists and nurses involved in their treatment. While some clinicians agree with the diagnosis under the name "borderline personality disorder", some would like the name to be changed. One critique says that some who are labeled "Borderline Personality Disorder" feel this name is unhelpful, stigmatizing, and/or inaccurate.

The Treatment and Research Advancements National Association for Personality Disorders (TARA-APD) campaigns to change the name and designation of BPD in DSM-5. The paper How Advocacy is Bringing BPD into the Light reports that "the name BPD is confusing, imparts no relevant or descriptive information, and reinforces existing stigma...".

Terminology

Because of the above concerns, and because of a move away from the original theoretical basis for the term (see history), there is ongoing debate about renaming BPD. Alternative
suggestions for names include emotional regulation disorder or emotional dysregulation disorder. Impulse disorder and interpersonal regulatory disorder are other valid alternatives, according to John Gunderson of McLean Hospital in the United States. Another term (for example, by psychiatrist Carolyn Quadrio) is post traumatic personality disorganization (PTPD), reflecting the condition’s status as (often) both a form of chronic post traumatic stress disorder (PTSD) and a personality disorder in the belief that it is a common outcome of developmental or attachment trauma. Some people do not report any kind of traumatic event.

**Borderline Personality Disorder: Summarized**

**Borderline Personality Disorder**

The main feature of borderline personality disorder (BPD) is a pervasive pattern of instability in interpersonal relationships, self-image and emotions. People with borderline personality disorder are also usually very impulsive.

This disorder occurs in most by early adulthood. The unstable pattern of interacting with others has persisted for years and is usually closely related to the person’s self-image and early social interactions. The pattern is present in a variety of settings (e.g., not just at work or home) and often is accompanied by a similar lability (fluctuating back and forth, sometimes in a quick manner) in a person’s emotions and feelings. Relationships and the person’s emotion may often be characterized as being shallow.

A person with this disorder will also often exhibit impulsive behaviors and have a majority of the following symptoms:

- Frantic efforts to avoid real or imagined abandonment
- A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation
- Identity disturbance, such as a significant and persistent unstable self-image or sense of self
- Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)
- Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior
- Emotional instability due to significant reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)
- Chronic feelings of emptiness
- Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)
- Transient, stress-related paranoid thoughts or severe dissociative symptoms

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.
Borderline personality disorder is more prevalent in females (75 percent of diagnoses made are in females). It is thought that borderline personality disorder affects approximately 2 percent of the general population.

Like most personality disorders, borderline personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

Details about Borderline Personality Disorder Symptoms

Frantic efforts to avoid real or imagined abandonment.

The perception of impending separation or rejection, or the loss of external structure, can lead to profound changes in self-image, emotion, thinking and behavior. Someone with borderline personality disorder will be very sensitive to things happening around them in their environment. They experience intense abandonment fears and inappropriate anger, even when faced with a realistic separation or when there are unavoidable changes in plans. For instance, becoming very angry with someone for being a few minutes late or having to cancel a lunch date. People with borderline personality disorder may believe that this abandonment implies that they are “bad.” These abandonment fears are related to an intolerance of being alone and a need to have other people with them. Their frantic efforts to avoid abandonment may include impulsive actions such as self-mutilating or suicidal behaviors.

Unstable and intense relationships.

People with borderline personality disorder may idealize potential caregivers or lovers at the first or second meeting, demand to spend a lot of time together, and share the most intimate details early in a relationship. However, they may switch quickly from idealizing other people to devaluing them, feeling that the other person does not care enough, does not give enough, is not “there” enough. These individuals can empathize with and nurture other people, but only with the expectation that the other person will “be there” in return to meet their own needs on demand. These individuals are prone to sudden and dramatic shifts in their view of others, who may alternately be seen as beneficial supports or as cruelly punitive. Such shifts other reflect disillusionment with a caregiver whose nurturing qualities had been idealized or whose rejection or abandonment is expected.

Identity disturbance.

There are sudden and dramatic shifts in self-image, characterized by shifting goals, values and vocational aspirations. There may be sudden changes in opinions and plans about career, sexual identity, values and types of friends. These individuals may suddenly change from the role of a needy supplicant for help to a righteous avenger of past mistreatment. Although they usually have a self-image that is based on being bad or evil, individuals with borderline personality disorder may at times have feelings that they do not exist at all. Such
experiences usually occur in situations in which the individual feels a lack of a meaningful relationship, nurturing and support. These individuals may show worse performance in unstructured work or school situations.

**How is Borderline Personality Disorder Diagnosed?**

Personality disorders such as borderline personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose borderline personality disorder.

Many people with borderline personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person’s life. This most often happens when a person’s coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for borderline personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Borderline Personality Disorder**

Researchers today don’t know what causes borderline personality disorder. There are many theories, however, about the possible causes of borderline personality disorder. Most professionals subscribe to a biopsychosocial model of causation — that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual’s personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible — rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be “passed down” to their children.

**Borderline Personality Disorder Treatment**

**Introduction**

Borderline Personality Disorder is experienced in individuals in many different ways. Often, people with this disorder will find it more difficult to distinguish between reality from their own misperceptions of the world and their surrounding environment. While this may seem like a type of delusion disorder to some, it is actually related to their emotions overwhelming regular cognitive functioning.
People with this disorder often see others in “black-and-white” terms. Depending upon the circumstances and situation, for instance, a therapist can be seen as being very helpful and caring toward the client. But if some sort of difficulty arises in the therapy, or in the patient’s life, the person might then begin characterizing the therapist as “bad” and not caring about the client at all. Clinicians should always be aware of this “all-or-nothing” lability most often found in individuals with this disorder and be careful not to validate it.

Therapists and doctors should learn to be like a rock when dealing with a person who has this disorder. That is, the doctor should offer his or her stability to contrast the client’s lability of emotion and thinking. Many professionals are turned-off by working with people with this disorder, because it draws on many negative feelings from the clinician. These occur because of the client’s constant demands on a clinician, the constant suicidal gestures, thoughts, and behaviors, and the possibility of self-mutilating behavior. These are sometimes very difficult items for a therapist to understand and work with.

Psychotherapy is nearly always the treatment of choice for this disorder; medications may be used to help stabilize mood swings. Controversy surrounds overmedicating people with this disorder.

**Psychotherapy**

Like with all personality disorders, psychotherapy is the treatment of choice in helping people overcome this problem. While medications can usually help some symptoms of the disorder, they cannot help the patient learn new coping skills, emotion regulation, or any of the other important changes in a person’s life.

An initially important aspect of psychotherapy is usually contracting with the person to ensure that they do not commit suicide. Suicidality should be carefully assessed and monitored throughout the entire course of treatment. If suicidal feelings are severe, medication and hospitalization should be seriously considered.

The most successful and effective psychotherapeutic approach to date has been Marsha Linehan’s Dialectical Behavior Therapy. Research conducted on this treatment have shown it to be more effective than most other psychotherapeutic and medical approaches to helping a person to better cope with this disorder. It seeks to teach the client how to learn to better take control of their lives, their emotions, and themselves through self-knowledge, emotion regulation, and cognitive restructuring. It is a comprehensive approach that is most often conducted within a group setting. Because the skill set learned is new and complex, it is not an appropriate therapy for those who may have difficulty learning new concepts.

Like all personality disorders, borderline personality disorder is intrinsically difficult to treat. Personality disorders, by definition, are long-standing ways of coping with the world, social and personal relationships, handling stress and emotions, etc. that often do not work, especially when a person is under increased stress or performance demands in their lives.
Treatment, therefore, is also likely to be somewhat lengthy in duration, typically lasting at least a year for most.

Other psychological treatments which have been used, to lesser effectiveness, to treat this disorder include those which focus on social learning theory and conflict resolution. These types of solution-focused therapies, though, often neglect the core problem of people who suffer from this disorder — difficulty in expressing appropriate emotions (and emotional attachments) to significant people in their lives due to faulty cognitions.

Providing a structured therapeutic setting is important no matter which therapy type is undertaken. Because people with this disorder often try and “test the limits” of the therapist or professional when in treatment, proper and well-defined boundaries of your relationship with the client need to be carefully explained at the onset of therapy. Clinicians need to be especially aware of their own feelings toward the patient, when the client may display behavior which is deemed “inappropriate.” Individuals with borderline personality disorder are often unfairly discriminated against within the broad range of mental health professionals because they are seen as “trouble-makers.” While they may indeed need more care than many other patients, their behavior is caused by their disorder. Phillip W. Long, M.D. also notes that:

“The therapeutic alliance should form within the patient's real experiences with the therapist and with the treatment. The therapist must be able to tolerate repeated episodes of primitive rage, distrust, and fear. Uncovering is to be avoided in favor of bolstering of ego defenses, in order to eventually allow the patient to be less anxious about potential fragmentation and loss. The goals of therapy should be in terms of life gains toward independent functioning, and not complete restructuring of the personality.”

Hospitalization

Hospitalization is often a concern with people who suffer from borderline personality disorder because they so often visit hospital emergency rooms and are sometimes seen on inpatient units because of severe depression.

People with this disorder often present in crisis at their local community mental health center, to their therapist, or at the hospital emergency room. While an emergency room is an immediate source of crisis intervention for the patient, it is a costly treatment and regular visits to the E.R. should be discouraged. Instead, patients should be encouraged to find additional social support within their community (including self-help support groups), contact a crisis hotline, or contact their therapist or treating physician directly. Emergency room personnel should be careful not to treat the person with borderline personality disorder in blind conjunction with another set of therapists or doctors who are treating the patient for the same problem at another facility. Every attempt should be made to contact the client’s attending physician or primary therapist as soon as possible, even before the administration of medication which may be contraindicated by the primary treatment provider. Crisis management of the immediate problem is usually the key component to
effective treatment of this disorder when it presents in a hospital emergency room, with discharge to the patient’s usual care provider.

Inpatient treatment often takes the form of medication in conjunction with psychotherapy sessions in groups or individually. This is an appropriate treatment option if the person is experiencing extreme difficulties in living and daily functioning. It is, however, relatively rare to be hospitalized in the U.S. for this disorder. Long-term care of the person suffering from borderline personality disorder within a hospital setting is nearly never appropriate. The typical inpatient stay for someone with borderline personality disorder in the U.S. is about 3 to 4 weeks, depending upon the person’s insurance. Since this treatment is so expensive, it is getting more difficult to obtain. Results of such treatment are also mixed. While it is an excellent way of helping stabilize the client, it is usually too short a time to attain significant changes within the individual’s personality makeup.

Good inpatient care facilities for this disorder should be highly structured environments which seek to expand the individual’s independence. Phillip W. Long, M.D., adds that the goals of such a treatment modality, “include decreasing acting out, clearly identifying and working with inappropriate behaviors and feelings, accepting with the patient the magnitude of the therapeutic task, fostering more effective interpersonal relationships, and working with both real and transference relationships within the hospital.”

Partial hospitalization or a day treatment program is often all that’s needed for people who suffer from borderline personality disorder. This allows the individual to gain support and structure from a safe environment for a short time, or during the day, and returning home in the evening. In times of increased stress or difficulty coping with specific situations, this type of treatment is more appropriate and more healthy for most people than full inpatient hospitalization.

Medications

Phillip W. Long, M.D. has noted:

“During brief reactive psychoses, low doses of antipsychotic drugs may be useful, but they are usually not essential adjuncts to the treatment regimen, since such episodes are most often self-limiting and of short duration.

It is, however, clear that low doses of high potency neuroleptics (e.g., haloperidol) may be helpful for disorganized thinking and some psychotic symptoms. Depression in some cases is amenable to neuroleptics. Neuroleptics are particularly recommended for the psychotic symptoms mentioned above, and for patients who show anger which must be controlled. Dosages should generally be low and the medication should never be given without adequate psychosocial intervention.”

Antidepressant and anti-anxiety agents may be appropriate during particular times in the patient’s treatment, as appropriate. For example, if a client presents with severe suicidal ideation and intent, the clinician may want to seriously consider the prescription of an
appropriate antidepressant medication to help combat the ideation. Medication of this type should be avoided for long-term use, though, since most anxiety and depression is directly related to short-term, situational factors that will quickly come and go in the individual’s life.

**Self-Help**

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. Encouraging the individual with borderline personality disorder to gain additional social support, however, is an important aspect of treatment. Many support groups exist within communities throughout the world which are devoted to helping individuals with this disorder share their commons experiences and feelings.

Patients can be encouraged to try out new coping skills and emotion regulation with people they meet within support groups. They can be an important part of expanding the individual’s skill set and develop new, healthier social relationships.

**Histrionic personality disorder**

Histrionic personality disorder (HPD) is defined by the American Psychiatric Association as a personality disorder characterized by a pattern of excessive emotionality and attention-seeking, including an excessive need for approval and inappropriately seductive behavior, usually beginning in early adulthood. These individuals are lively, dramatic, vivacious, enthusiastic, and flirtatious.

They may be inappropriately sexually provocative, express strong emotions with an impressionistic style, and be easily influenced by others. Associated features may include egocentrism, self-indulgence, continuous longing for appreciation, and persistent manipulative behavior to achieve their own needs.

**Characteristics**

People with this disorder are usually able to function at a high level and can be successful socially and professionally. People with histrionic personality disorder usually have good social skills, but they tend to use these skills to manipulate other people and become the center of attention. Furthermore, histrionic personality disorder may affect a person’s social or romantic relationships or their ability to cope with losses or failures.

People with this disorder lack genuine empathy. They start relationships well but tend to falter when depth and durability are needed, alternating between extremes of idealization and devaluation. They may seek treatment for depression when romantic relationships end, although this is by no means a feature exclusive to this disorder.
They often fail to see their own personal situation realistically, instead tending to dramatize and exaggerate their difficulties. They may go through frequent job changes, as they become easily bored and have trouble dealing with frustration. Because they tend to crave novelty and excitement, they may place themselves in risky situations. All of these factors may lead to greater risk of developing depression.

Additional symptoms include:

- Exhibitionist behavior.
- Constant seeking of reassurance or approval.
- Excessive dramatics with exaggerated displays of emotions, such as hugging someone they have just met or crying uncontrollably during a sad movie (Svrakie & Cloninger, 2005).
- Excessive sensitivity to criticism or disapproval.
- Proud of own personality, unwillingness to change and any change is viewed as a threat.
- Inappropriately seductive appearance or behavior.
- Somatic symptoms, and using these symptoms as a means of garnering attention.
- A need to be the center of attention.
- Low tolerance for frustration or delayed gratification.
- Rapidly shifting emotional states that may appear superficial or exaggerated to others.
- Tendency to believe that relationships are more intimate than they actually are.
- Making rash decisions.

Causes

The cause of this disorder is unknown, but childhood events such as deaths in the immediate family, illnesses within the immediate family which present constant anxiety, divorce of parents and genetics may be involved. Histrionic Personality Disorder is more often diagnosed in women than men; men with some quite similar symptoms are often diagnosed with narcissistic personality disorder.

Little research has been conducted to determine the biological sources, if any, of this disorder. Psychoanalytic theories incriminate authoritarian or distant attitudes by one (mainly mother) or both of the parents of these patients, or love based on expectations from the child that can never be fully met.

Diagnosis

The person's appearance, behavior, and history, along with a psychological evaluation, are usually sufficient to establish the diagnosis. There is no test to confirm this diagnosis. Because the criteria are subjective, some people may be wrongly diagnosed as having the disorder while others with the disorder may not be diagnosed. Treatment is often prompted by depression associated with dissolved romantic relationships. Medication does
little to affect this personality disorder, but may be helpful with symptoms such as depression. Psychotherapy may also be of benefit.

**DSM-IV-TR 301.50**

The Diagnostic and Statistical Manual of Mental Disorders fourth edition, DSM IV-TR, a widely used manual for diagnosing mental disorders, defines histrionic personality disorder (in Axis II Cluster B) as:

A pervasive pattern of excessive emotionality and attention seeking, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

- is uncomfortable in situations in which he or she is not the center of attention
- interaction with others is often characterized by inappropriate sexually seductive or provocative behavior
- displays rapidly shifting and shallow expression of emotions
- consistently uses physical appearance to draw attention to self
- has a style of speech that is excessively impressionistic and lacking in detail
- shows self-dramatization, theatricality, and exaggerated expression of emotion
- is suggestible, i.e., easily influenced by others or circumstances
- considers relationships to be more intimate than they actually are.

It is a requirement of DSM-IV that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**ICD-10**

The World Health Organization’s ICD-10 lists histrionic personality disorder as (F60.4) Histrionic personality disorder.

It is characterized by at least 3 of the following:

- self-dramatization, theatricality, exaggerated expression of emotions;
- suggestibility, easily influenced by others or by circumstances;
- shallow and labile affectivity;
- continual seeking for excitement and activities in which the patient is the center of attention;
- inappropriate seductiveness in appearance or behavior;
- over-concern with physical attractiveness.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Millon’s subtypes**
Theodore Millon identified six subtypes of histrionic. Any individual histrionic may exhibit none or one of the following:

- Theatrical histrionic - especially dramatic, romantic and attention seeking.
- Infantile histrionic - including borderline features.
- Vivacious histrionic - synthesizes the seductiveness of the histrionic with the energy level typical of hypomania.
- Appeasing histrionic - including dependent and compulsive features.
- Tempestuous histrionic - including negativistic (passive-aggressive) features.
- Disingenuous histrionic - antisocial features.

**Mnemonic**

A mnemonic that can be used to remember the criteria for histrionic personality disorder is PRAISE ME:

- P - provocative (or seductive) behavior
- R - relationships, considered more intimate than they are
- A - attention, must be at center of
- I - influenced easily
- S - speech (style) - wants to impress, lacks detail
- E - emotional lability, shallowness

- M - make-up - physical appearance used to draw attention to self
- E - exaggerated emotions - theatrical

**Differential diagnosis**

- Clinical depression
- Anxiety disorders
- Panic disorder
- Somatoform disorders

A person suffering from HPD is highly reactive. If there is another major disorder present, such as delusional disorder, then emotional intensity will create anger, rage, abuse and distance in relationships.

It is important for the therapist and family members to monitor and record all situations that trigger the HPD so that the deep underlying overload of pain can be accessed and released for therapeutic change.

**Treatment**

Because of the lack of research support for work on personality disorders and long-term treatment with psychotherapy, the empirical findings on the treatment of these disorders remain based on the case report method and not on clinical trials. On the basis of case
presentations, the treatment of choice is psychotherapy and/or cognitive-behavioral therapy, aimed at self-development through resolution of conflict and advancement of inhibited developmental lines. Group therapy can assist individuals with HPD to learn to decrease the display of excessively dramatic behaviors, but must be closely monitored because it may provide the person with an audience to play to (perform for), thus giving opportunity to perpetuate histrionic behavior.

- Family therapy
- Medications
- Alternative therapies
- Cognitive behavioral therapy

**Epidemiology**

Histrionic personality disorder shares a divergent history with conversion disorder and somatization disorder. Historically, they are linked to the ancient notion of hysteria, or "wandering womb." (Note, however, that according to the Online Etymology Dictionary, the word "histrionic" derives not from the Greek hystera, but from the Latin histrionicus, "pertaining to an actor.") Ancient Greeks thought that excessive emotionality in women was caused by a displaced uterus and sexual discontent.

Christian ascetics during the Middle Ages blamed women’s mental problems on witchcraft, sexual hunger, moral weakness, and demonic possession. By the 19th century, medical explanations proposed a weakness of women's nervous system related to biological sex. Thus, "hysteria" reflected the stereotype for women as vulnerable, inferior, and emotionally unbalanced. The extent to which the definition of histrionic personality disorder currently reflects gender bias remains the subject of controversy.

"Hysteria" differentiated into conversion hysteria (later to become conversion disorder) and hysterical personality (later to become histrionic personality disorder) in the psychoanalytic literature as well as with the writings of Kraepelin, Schneider, and others. Sigmund Freud wrote primarily about conversion hysteria. Wilhelm Reich wrote about hysteria as a set of personality characteristics and differentiated conversion hysteria as a transient disorder from hysterical character. These early conceptualizations of both kinds of hysteria carried notions of women’s deficiency due to penis envy and feelings of castration. Paul Chodoff has written about the ways in which these diagnoses paralleled the misogynistic sentiment of the times.

The concept of hysterical personality was well developed by the mid-20th century and strongly resembled the current definition of histrionic personality disorder. The first DSM featured a symptom-based category, "hysteria" (conversion) and a personality-based category, "emotionally unstable personality." DSM-II distinguished between hysterical neurosis (conversion reaction and dissociative reaction) and hysterical (histrionic) personality.
In DSM-III, the term hysterical personality changed to histrionic personality disorder to emphasize the histrionic (derived from the Latin word histrio, or actor) behavior pattern and to reduce the confusion caused by the historical links of hysteria to conversion symptoms. The landmark case of Ruth E. helped to fully define and emphasize the characteristics of the current DSM-IV diagnostic. DSM-III-R attempted to reduce the overlap between Histrionic Personality Disorder and borderline personality disorder by dropping three overlapping criteria and adding two criteria that emphasized histrionicity. DSM-IV dropped two more criteria that did not appear to contribute to the consistency of the diagnosis, according to research done by Bruce Fiohl.

Histrionic Personality Disorder: Summarized

Histrionic Personality Disorder

Histrionic personality disorder is characterized by a long-standing pattern of attention seeking behavior and extreme emotionality. Someone with histrionic personality disorder wants to be the center of attention in any group of people, and feel uncomfortable when they are not. While often lively, interesting and sometimes dramatic, they have difficulty when people aren’t focused exclusively on them. People with this disorder may be perceived as being shallow, and may engage in sexually seductive or provocative behavior to draw attention to themselves.

Individuals with Histrionic Personality Disorder may have difficulty achieving emotional intimacy in romantic or sexual relationships. Without being aware of it, they often act out a role (e.g., "victim" or "princess") in their relationships to others. They may seek to control their partner through emotional manipulation or seductiveness on one level, whereas displaying a marked dependency on them at another level.

Individuals with this disorder often have impaired relationships with same-sex friends because their sexually provocative interpersonal style may seem a threat to their friends' relationships. These individuals may also alienate friends with demands for constant attention. They often become depressed and upset when they are not the center of attention.

People with histrionic personality disorder may crave novelty, stimulation, and excitement and have a tendency to become bored with their usual routine. These individuals are often intolerant of, or frustrated by, situations that involve delayed gratification, and their actions are often directed at obtaining immediate satisfaction. Although they often initiate a job or project with great enthusiasm, their interest may lag quickly.

Longer-term relationships may be neglected to make way for the excitement of new relationships.

Symptoms of Histrionic Personality Disorder
A pervasive pattern of excessive emotionality and attention seeking, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

- Is uncomfortable in situations in which he or she is not the center of attention
- Interaction with others is often characterized by inappropriate sexually seductive or provocative behavior
- Displays rapidly shifting and shallow expression of emotions
- Consistently uses physical appearance to draw attention to themself
- Has a style of speech that is excessively impressionistic and lacking in detail
- Shows self-dramatization, theatricality, and exaggerated expression of emotion
- Is highly suggestible, i.e., easily influenced by others or circumstances
- Considers relationships to be more intimate than they actually are

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.

Histrionic personality disorder is more prevalent in females than males. It occurs about 2 to 3 percent in the general population.

Like most personality disorders, histrionic personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

**How is Histrionic Personality Disorder Diagnosed?**

Personality disorders such as histrionic personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose histrionic personality disorder.

Many people with histrionic personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person's life. This most often happens when a person’s coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for histrionic personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Histrionic Personality Disorder**
Researchers today don’t know what causes histrionic personality disorder. There are many theories, however, about the possible causes of histrionic personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual’s personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

Histrionic Personality Disorder Treatment

Psychotherapy

Individuals who suffer from this disorder are usually difficult to treat for a multitude of reasons. As with most personality disorders, people present for treatment only when stress or some other situational factor within their lives has made their ability to function and cope effectively impossible. They are, however (unlike other people who suffer from personality disorders), much quicker to seek treatment and exaggerate their symptoms and difficulties in functioning. Because they also tend to be more emotionally needy, they are often reluctant to terminate therapy.

Psychotherapy, as with most personality disorders, is the treatment of choice. Group and family therapy approaches are generally not recommended, since the individual who suffers from this disorder often draws attention to themselves and exaggerates every action and reaction. People with disorder often come across as "fake" or shallow in their interpersonal relationships with others. Patients often are express all feelings with the same depth of emotion, unaware of the subtleties of their own emotional states and of the vast range available to them.

Therapy should generally be supportive and good rapport will usually be easily established with the patient early on. Clinicians may often find themselves placed in a "rescuer" role, in which the therapist will be asked to constantly reassure and rescue the client from daily problems. Every problem is usually expressed in a dramatic fashion. Many times the therapist will be perceived as sexually attractive to the patient. Boundary issues in relationships and a clear delineation of the therapeutic framework are relevant and important aspects of therapy.

Approaches which take advantage of matter-of-fact and realistic assessment of situations and problems can also be important. Solution-focused therapy is often appropriate with this client. Most therapy approaches should not be focused on the long-term, personality change of the individual, but rather short-term alleviation of difficulties within the person’s life. Few people could afford the time or cost required to "cure" someone of this disorder. This should be explicitly stated up-front at the onset of therapy to dismiss any thoughts the client may have of a "magical" cure for this disorder.
Suicidal behavior is often apparent in a person who suffers from histrionic personality disorder. Suicidality should be assessed on a regular basis and suicidal threats should not be ignored or dismissed. Suicide sometimes occurs when all that was intended was a gesture, so all such thoughts and plans should be taken with the same seriousness as with any other disorder. A suicide contract should be established to specify under what conditions the therapist may be contacted in case the client feels like hurting him or herself. Self-mutilation behavior may also be present in this disorder and should also be taken seriously as an issue of importance to discuss within therapy.

Therapists will find that taking a somewhat skeptical stance within therapy to be useful, due to the usual exaggeration of events and problems by the patient. By following a line of reasoning to its logical conclusion, the client can usually discover the unrealistic expectations and fears associated with many behaviors and thoughts. Since many people who have histrionic personality disorder will emphasize attractiveness ("style over substance") in their lives and relationships, discussing alternatives and trying out new behaviors may be helpful. The therapist can also help by pointing out, in session, when the client is using shallow criteria in which to judge another. The patient should eventually look to be able to do this themselves throughout their lives.

Insight- and cognitive-oriented approaches are generally largely ineffective in treatment of this disorder and should be avoided. People with this disorder are often incapable of examining unconscious motivations and their own thoughts to a degree where it is helpful. While these approaches can be a part of a larger treatment plan, they should not be the focus. Helping the client to examine interactions from a more objective point of view and emphasizing alternative explanations for behavior is likely to be more effective. Examining and clarifying a client's emotions are also important components of therapy.

Clinicians will often experience reactions to treating this disorder, because of the dramatic nature of the patient. Because of this possibility, therapists should be more attuned to their own feelings within the therapy setting and ensure that they are treating the patient fairly and with respect. As with Borderline Personality Disorder, individuals with histrionic personality disorder often find themselves discriminated against by mental health professionals because of the symptoms of their disorder. Clinicians and patients should be aware of this possible discrimination.

**Medications**

As with most personality disorders, medications are not indicated except for the treatment of specific, concurrent Axis I diagnoses. Care should be given when prescribing medications to someone who suffers from histrionic personality disorder, though, because of the potential for using the medication to contribute to self-destructive or otherwise harmful behaviors.

**Self-Help**
There are not any self-help support groups or communities that we are aware of that would be conducive to someone suffering from this disorder. Such approaches would likely not be very effective because a person with this disorder is likely to be very dramatic in their interactions with others, coming across as "artificial" or shallow.

**Narcissistic personality disorder**

Narcissus by Caravaggio. Narcissus gazing at his own reflection.

Narcissistic personality disorder (NPD) is a personality disorder.

The narcissist is described as being excessively preoccupied with issues of personal adequacy, power, prestige and vanity. Narcissistic personality disorder is closely linked to self-centeredness.

**Causes**

The cause of this disorder is unknown, according to Groopman and Cooper. However, they list the following factors identified by various researchers as possibilities.

- An oversensitive temperament at birth is the main symptomatic chronic form
- Being praised for perceived exceptional looks or talents by adults
- Excessive admiration that is never balanced with realistic feedback
- Excessive praise for good behaviors or excessive criticism for poor behaviors in childhood
- Overindulgence and overvaluation by parents
- Severe emotional abuse in childhood
- Unpredictable or unreliable caregiving from parents
- Valued by parents as a means to regulate their own self-esteem

Some narcissistic traits are common and a normal developmental phase. When these traits are compounded by a failure of the interpersonal environment and continue into adulthood, they may intensify to the point where NPD is diagnosed. Some psychotherapists believe that the etiology of the disorder is, in Freudian terms, the result of fixation to early childhood development. If a child does not receive sufficient recognition for their talents during about ages 3–7 they will never mature and continue to be in the narcissistic early development stage.

A 1994 study by Gabbard and Twemlow reports that histories of incest, especially mother-son incest, are associated with NPD in some male patients.

**Theories**

Pathological narcissism occurs in a spectrum of severity. In its more extreme forms, it is narcissistic personality disorder (NPD). NPD is considered to result from a person’s belief that they are flawed in a way that makes them fundamentally unacceptable to others. This belief is held below the person’s conscious awareness; such a person would, if questioned, typically deny thinking such a thing. In order to protect themselves against the intolerably painful rejection and isolation that (they imagine) would follow if others recognised their (perceived) defective nature, such people make strong attempts to control others’ views of them and behavior towards them.

Pathological narcissism can develop from an impairment in the quality of the person's relationship with their primary caregivers, usually their parents, in that the parents were unable to form a healthy and empathic attachment to them. This results in the child's perception of himself/herself as unimportant and unconnected to others. The child typically comes to believe they have some personality defect that makes them unvalued and unwanted.

Narcissistic personality disorder is isolating, disenfranchising, painful, and formidable for those living with it and often those who are in a relationship with them. Distinctions need to be made among those who have NPD because not every person with NPD is the same. Even with similar core issues, the way in which one's individual narcissism manifests itself in his or her relationships varies.

To the extent that people are pathologically narcissistic, they can be controlling, blaming, self-absorbed, intolerant of others’ views, unaware of others’ needs and of the effects of their behavior on others, and insistent that others see them as they wish to be seen.
People who are overly narcissistic commonly feel rejected, humiliated and threatened when criticised. To protect themselves from these dangers, they often react with disdain, rage, and/or defiance to any slight criticism, real or imagined. To avoid such situations, some narcissistic people withdraw socially and may feign modesty or humility. In cases where the narcissistic personality-disordered individual feels a lack of admiration, adulation, attention and affirmation, he/she may also manifest wishes to be feared and to be notorious (narcissistic supply).

Although individuals with NPD are often ambitious and capable, the inability to tolerate setbacks, disagreements or criticism, along with lack of empathy, make it difficult for such individuals to work cooperatively with others or to maintain long-term professional achievements. With narcissistic personality disorder, the individual’s self-perceived fantastic grandiosity, often coupled with a hypomanic mood, is typically not commensurate with his or her real accomplishments.

The exploitativeness, sense of entitlement, lack of empathy, disregard for others, and constant need for attention inherent in NPD adversely affect interpersonal relationships.

**Splitting**

People who are diagnosed with narcissistic personality disorder use splitting (black and white thinking) as a central defense mechanism. They do this to preserve their self-esteem, by seeing the self as purely good and the others as purely bad. The use of splitting also implies the use of other defense mechanisms, namely devaluation, idealization and denial.

**Relationship to shame**

It has been suggested that narcissistic personality disorder may be related to defenses against shame.

Psychiatrist Glen Gabbard suggested NPD could be broken down into two subtypes. He saw the "oblivious" subtype as being grandiose, arrogant, and thick-skinned and the "hypervigilant" subtype as being easily hurt, oversensitive, and ashamed. In his view, the oblivious subtype presents for admiration, envy, and appreciation a powerful, grandiose self that is the antithesis of a weak internalized self, which hides in shame, while the hypervigilant subtype neutralizes devaluation by seeing others as unjust abusers.

Dr. Jeffrey Young, who coined the term "Schema Therapy", a technique originally developed by psychiatrist Aaron T. Beck (1979), also links NPD and shame. He sees the so-called Defectiveness Schema as a core schema of NPD, along with the Emotional Deprivation and Entitlement Schemas.

**Diagnosis**
**DSM-IV-TR 301.81**

The Diagnostic and Statistical Manual of Mental Disorders fourth edition, DSM IV-TR, a widely used manual for diagnosing mental disorders, defines narcissistic personality disorder (in Axis II Cluster B) as:

A pervasive pattern of grandiosity (in fantasy or behavior), need for admiration, and lack of empathy, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

- Has a grandiose sense of self-importance (e.g., exaggerates achievements and talents, expects to be recognized as superior without commensurate achievements)
- Is preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love
- Believes that he or she is "special" and unique and can only be understood by, or should associate with, other special or high-status people (or institutions)
- Requires excessive admiration
- Has a sense of entitlement, i.e., unreasonable expectations of especially favorable treatment or automatic compliance with his or her expectations
- Is interpersonally exploitative, i.e., takes advantage of others to achieve his or her own ends
- Lacks empathy: is unwilling to recognize or identify with the feelings and needs of others
- Is often envious of others or believes others are envious of him or her
- Shows arrogant, haughty behaviors or attitudes

It is also a requirement of DSM-IV that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Proposed removal from DSM-5**

The Personality and Personality Work Group has proposed the elimination of NPD as a distinct disorder in DSM-5 as part of a major revamping of the diagnostic criteria for personality disorders, replacing a categorical with a dimensional approach based on the severity of dysfunctional personality trait domains, raising objections from some clinicians who characterize the new diagnostic system as an "unwieldy conglomeration of disparate models that cannot happily coexist" and may have limited usefulness in clinical practice.

**ICD-10**

The World Health Organization's ICD-10 lists narcissistic personality disorder under (F60.8) Other specific personality disorders.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.
Millon's subtypes

Theodore Millon identified five subtypes of narcissist. Any individual narcissist may exhibit none or one of the following:

- Unprincipled narcissist - including antisocial features. A charlatan - is a fraudulent, exploitative, deceptive and unscrupulous individual.
- Amorous narcissist - including histrionic features. The Don Juan or Casanova of our times - is erotic, exhibitionist.
- Compensatory narcissist - including negativistic (passive-aggressive), avoidant features.
- Elitist narcissist - variant of pure pattern. Corresponds to Wilhelm Reich's "phallic narcissistic" personality type.
- Fanatic narcissist - including paranoid features. An individual whose self-esteem was severely arrested during childhood, who usually displays major paranoid tendencies, and who holds on to an illusion of omnipotence. These people are fighting delusions of insignificance and lost value, and trying to re-establish their self-esteem through grandiose fantasies and self-reinforcement. When unable to gain recognition or support from others, they take on the role of a heroic or worshipped person with a grandiose mission.

Alexander Lowen has also specified five major subtypes from Phallic ("Skirtchasing") to Sociopathic (i.e., dissociative, capable of mayhem and murder) as outlined in his famous book, "Narcissism: Denial of the True Self".

Treatment

Prominent clinical strategies are outlined by Heinz Kohut, Stephen M. Johnson and James F. Masterson, while Johns discusses a continuum of severity and the kinds of therapy most effective in different cases.

Schema Therapy, a form of therapy developed by Jeffrey Young that integrates several therapeutic approaches (psychodynamic, cognitive, behavioral etc.), also offers an approach for the treatment of NPD. It is unusual for people to seek therapy for NPD. Subconscious fears of exposure or inadequacy often cause defensive disdain of therapeutic processes. Pharmacotherapy is rarely effective.

Epidemiology

Lifetime prevalence is estimated at 1% in the general population and 2% to 16% in clinical populations.

History
The history of narcissism predates the discovery of narcissistic personality disorder. The term "narcissistic personality structure" was introduced by Kernberg in 1967 and "narcissistic personality disorder" first proposed by Heinz Kohut in 1968.

Cultural depictions

In the film To Die For, Nicole Kidman’s character wants to appear on television at all costs, even if this involves murdering her husband. A psychiatric assessment of her character noted that she "was seen as a prototypical narcissistic person by the raters: on average, she satisfied 8 of 9 criteria for narcissistic personality disorder... had she been evaluated for personality disorders, she would receive a diagnosis of narcissistic personality disorder."

Robin Quivers, the longtime on-air companion to radio personality Howard Stern, was tested on a narcissism scale by Dr. Drew Pinsky and was scored a 34, which Pinsky noted was the highest he had ever recorded. Quivers denied that she was a narcissist, and Pinsky replied that such a denial is typical of a person scoring so high on the narcissism scale.

Narcissistic Personality Disorder: Summarized

Narcissistic Personality Disorder

Narcissistic Personality Disorder is characterized by a long-standing pattern of grandiosity (either in fantasy or actual behavior), an overwhelming need for admiration, and usually a complete lack of empathy toward others. People with this disorder often believe they are of primary importance in everybody’s life or to anyone they meet. While this pattern of behavior may be appropriate for a king in 16th Century England, it is generally considered inappropriate for most ordinary people today.

People with narcissistic personality disorder often display snobbish, disdainful, or patronizing attitudes. For example, an individual with this disorder may complain about a clumsy waiter’s "rudeness" or "stupidity" or conclude a medical evaluation with a condescending evaluation of the physician.

In laypeople terms, someone with this disorder may be described simply as a "narcissist" or as someone with "narcissism." Both of these terms generally refer to someone with narcissistic personality disorder.

Symptoms of Narcissistic Personality Disorder

In order for a person to be diagnosed with narcissistic personality disorder (NPD) they must meet five or more of the following symptoms:

- Has a grandiose sense of self-importance (e.g., exaggerates achievements and talents, expects to be recognized as superior without commensurate achievements)
- Is preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love
- Believes that he or she is "special" and unique and can only be understood by, or should associate with, other special or high-status people (or institutions)
- Requires excessive admiration
- Has a very strong sense of entitlement, e.g., unreasonable expectations of especially favorable treatment or automatic compliance with his or her expectations
- Is exploitative of others, e.g., takes advantage of others to achieve his or her own ends
- Lacks empathy, e.g., is unwilling to recognize or identify with the feelings and needs of others
- Is often envious of others or believes that others are envious of him or her
- Regularly shows arrogant, haughty behaviors or attitudes

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.

Narcissistic personality disorder is more prevalent in males than females, and is thought to occur in less than 1 percent in the general population.

Like most personality disorders, narcissistic personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

Learn more about the symptoms and characteristics of someone with narcissistic personality disorder.

**How is Narcissistic Personality Disorder Diagnosed?**

Personality disorders such as narcissistic personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose personality disorder.

Many people with narcissistic personality disorder don't seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person's life. This most often happens when a person's coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for narcissistic personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.
Causes of Narcissistic Personality Disorder

Researchers today don’t know what causes narcissistic personality disorder. There are many theories, however, about the possible causes of narcissistic personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual's personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

Narcissistic Personality Disorder Treatment

Medical Treatment

Hospitalization

The hospitalization of patients with severe Narcissistic Personality occurs frequently. For some, such as those who are quite impulsive or self-destructive, or who have poor reality-testing, this is the result of Axis I symptoms which are overlaid upon the personality disorder. Hospitalizations should be brief, and the treatment specific to the particular symptom involved.

Another group of patients for whom hospitalization is indicated, provided long-term residential treatment is available, are those who have poor motivation for outpatient treatment, fragile object relationships, chronic destructive acting out, and chaotic lifestyles. An inpatient program can offer an intensive milieu which includes individual psychotherapy, family involvement, and a specialized residential environment. The structure is physically and emotionally secure enough to sustain the patient with severe ego weakness throughout the course of expressive, conflict-solving psychotherapy.

Small staff-patient groups within the wards, as well as large community meetings, at which feelings are shared and patients' comments taken seriously by staff, and constructive work assignments, recreational activities, and opportunities to sublimate painfully conflictual impulses make the hospital a "holding" environment rather than merely a containing one. The ultimate goals are of effecting a better integrated internal world, more cohesive and modulated self-object representation, and a self-concept less vulnerable to narcissistic injury.

Psychosocial Treatment

Basic Principles
Narcissistic patients try to sustain an image of perfection and personal invincibility for themselves and attempt to project that impression to others as well. Physical illness may shatter this illusion, and a patient may lose the feeling of safety inherent in a cohesive sense of self. This loss precipitates a panicky sensation that "my world is falling to pieces," and the patient feels a sense of personal fragmentation.

The histrionic patient’s idealization of the physician stands in contrast to the narcissistic patient’s frequent contemptuous disregard for the physician, who is denigrated in a defensive effort to maintain a sense of superiority and mastery over illness. Only the most senior physician in a prestigious institution is deemed worthy of respect as the frightened patient seeks an external reflection of his or her own fragile grandeur in the doctor. More junior members of the health care team may be the targets of derision as the patient seeks to establish hierarchical dominance in order to counter the shame and fear triggered by illness.

Health care professionals must convey a feeling of respect and acknowledge the patient’s sense of self-importance so that the patient can reestablish a coherent sense of self, but they must at the same time avoid reinforcing either pathologic grandiosity (which may contribute to denial of illness) or weakness (which frightens the patient). An initial approach of support followed by step-by-step confrontation of the patient’s vulnerabilities may enable the patient to deal with the implications of illness with feelings of greater subjective strength. The increased self-confidence may reduce the patient’s need to attack the health care team in a misguided effort at psychologic self-preservation and eases the pressure to provide perfect care, since the patient’s antagonistic feeling of entitlement (defined by DSM-III as an "expectation of special favors without assuming reciprocal responsibilities") is reduced.

Many of the treatment principles and approaches discussed for this disorder apply as well to Borderline Personality Disorder.

The individual with narcissistic and related personality disorders is likely to present with Axis I symptoms and disorders at various times in his or her life. These should be treated as described elsewhere. Caution should be observed, however, not to overdiagnose psychotic decompensation as Schizophrenia unless all DSM-III criteria are apparent. The same caveat applies to the pharmacologic treatment of depressive symptoms in the absence of clinical signs of Major Affective Disorder. When treating presenting symptoms and Axis I disorders in patients with Narcissistic Personality Disorder and other similar conditions, attention should be paid to the consequences of removing symptoms in a patient whose underlying character is primitive and or fragile.

Some clinicians, suggest that the grandiosity and tendency to idealize and devalue should be interpreted as defensive maneuvers when aspects of early conflictual relationships are played out in adult life. Other clinicians, posit that the emergence of the patient’s grandiosity and tendency to idealize the therapist should initially be viewed supportively. To help the individual develop stronger self-esteem regulation, the therapist then gradually points out the realistic limitations of patient and therapist alike while also offering an
empathic ambience to cushion patients in their efforts to accept and integrate these experiences. Unfortunately, much research will be required to validate the description and course of narcissistic personality disorder before further research can answer which techniques bring about a better response to treatment.

**Individual Psychotherapy**

Most psychiatrists will, as a practical matter, treat most of their severely narcissistic patients for symptoms related to crises and relatively external Axis I diagnoses, rather than in an effort to address the personality disorder itself. The therapist must be aware of the importance of narcissism to the contiguity of the patient’s psyche, refrain from confronting the need for self-aggrandizement, and help the patient use his or her narcissistic characteristics to reconstitute an intact self-image. Positive transference and therapeutic alliance should not be relied upon, since the patient may not be able to acknowledge the real humanness of the therapist but may have to see him/her as either superhuman or devalued.

Those patients who do not terminate treatment after symptom relief has been obtained may wish help for some of the problems related to their personality disorder, such as interpersonal difficulties or depression. The therapist must have a good understanding of the principles of the narcissistic personality style, both for interpretation to the patient and for use in combating countertransference. Goals for ordinary psychotherapy should not be too great, since the source of these patients’ difficulties lies deep in pathological development.

**Group Therapy**

The goals are to help the patient develop a healthy individuality (rather than a resilient narcissism) so that he or she can acknowledge others as separate persons, and to decrease the need for self-defeating coping mechanisms. The first step toward developing a working alliance is empathy with the surprise and hurt that the patient experiences as a result of confrontations within the group. The external structuring group therapy provides can control destructive behavior in spite of ego weakness. In groups, the therapist is less authoritative (and less threatening to the patient’s grandiosity); intensity of emotional experience is lessened; and regression is more controlled, creating a better setting for confrontation and clarification.

Outpatient analytic-expressive group therapy requires a concomitant individual relationship for most patients, which should be somewhat supportive. The need for this additional support, the likelihood of the patient's leaving the group at the first sign of psychic insult, and proneness to disorganized thinking are all found more often in theBorderline patient. The patient with a Narcissistic Personality Disorder does not appear so vulnerable to separation anxieties as the Borderline patient, but is instead involved in issues centered around maintaining a sense of self-worth.
Avoidant personality disorder

Avoidant personality disorder (or anxious personality disorder) is a personality disorder recognized in the Diagnostic and Statistical Manual of Mental Disorders handbook in a person characterized by a pervasive pattern of social inhibition, feelings of inadequacy, extreme sensitivity to negative evaluation, and avoidance of social interaction.

People with Avoidant personality disorder often consider themselves to be socially inept or personally unappealing, and avoid social interaction for fear of being ridiculed, humiliated, rejected, or disliked.

Avoidant personality disorder is usually first noticed in early adulthood. Childhood emotional neglect and peer group rejection are both associated with an increased risk for the development of AvPD.

There is controversy as to whether Avoidant personality disorder is a distinct disorder from generalized social phobia and it is contended by some that they are merely different conceptualisations of the same disorder, where Avoidant personality disorder may represent the more severe form. This is argued as generalized social phobia and Avoidant personality disorder have a similar diagnostic criteria and may share a similar causation, subjective experience, course, treatment, and identical underlying personality features, such as shyness.

Signs and symptoms

People with Avoidant personality disorder are preoccupied with their own shortcomings and form relationships with others only if they believe they will not be rejected. Loss and rejection are so painful that these individuals will choose to be lonely rather than risk trying to connect with others.

- Hypersensitivity to rejection/criticism
- Self-imposed social isolation
- Extreme shyness or anxiety in social situations, though the person feels a strong desire for close relationships
- Avoids physical contact because it has been associated with an unpleasant or painful stimulus
- Avoids interpersonal relationships
- Feelings of inadequacy
- Severe low self-esteem
- Self-loathing
- Mistrust of others
- Emotional distancing related to intimacy
- Highly self-conscious
- Self-critical about their problems relating to others
- Problems in occupational functioning
Lonely self-perception, although others may find the relationship with them meaningful
- Feeling inferior to others
- In some more extreme cases — agoraphobia
- Utilizes fantasy as a form of escapism and to interrupt painful thoughts

**Causes**

Apart from the above, other causes of Avoidant personality disorder are not clearly defined, and may be influenced by a combination of social, genetic, and psychological factors. The disorder may be related to temperamental factors that are inherited. Specifically, various anxiety disorders in childhood and adolescence have been associated with a temperament characterized by behavioral inhibition, including features of being shy, fearful, and withdrawn in new situations. These inherited characteristics may give an individual a genetic predisposition towards AvPD. Childhood emotional neglect and peer group rejection are both associated with an increased risk for the development of AvPD.

**Diagnosis**

**World Health Organization**

The World Health Organization's ICD-10 lists avoidant personality disorder as (F60.6) Anxious (avoidant) personality disorder.

It is characterized by at least four of the following:

- persistent and pervasive feelings of tension and apprehension;
- belief that one is socially inept, personally unappealing, or inferior to others;
- excessive preoccupation with being criticized or rejected in social situations;
- unwillingness to become involved with people unless certain of being liked;
- restrictions in lifestyle because of need to have physical security;
- avoidance of social or occupational activities that involve significant interpersonal contact because of fear of criticism, disapproval, or rejection.

Associated features may include hypersensitivity to rejection and criticism.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfy a set of general personality disorder criteria.

**Millon's subtypes**

Psychologist Theodore Millon identified four subtypes of avoidant personality disorder. Any individual avoidant may exhibit none or one of the following:

- conflicted avoidant - including negativistic features
The conflicted avoidant feels ambivalent towards themselves and others. They can idealize those close to them but under stress they may feel under-appreciated or misunderstood and wish to hurt others in revenge. They may be perceived as petulant or to be sulking.

- **hypersensitive avoidant - including paranoid features**

  The hypersensitive avoidant experiences paranoia, mistrustfulness and fear, but to a lesser extent than an individual with paranoid personality disorder. They may be perceived as petulant or "high-strung".

- **phobic avoidant - including dependent features**

- **self-deserting avoidant - including depressive features**

**Differential diagnosis**

Research suggests that people with Avoidant personality disorder, in common with sufferers of chronic social anxiety disorder (also called social phobia), excessively monitor their own internal reactions when they are involved in social interaction. However, unlike social phobics, who are aware of the irrationality of their phobia yet are unable to control it, people with Avoidant personality disorder are unaware of or reject the idea that their fears are excessive and believe with full conviction that they are inadequate, unlovable, broken, etc.

The extreme tension created by this monitoring may account for the hesitant speech and taciturnity of many people with Avoidant personality disorder; they are so preoccupied with monitoring themselves and others that producing fluent speech is difficult.

Avoidant personality disorder is reported to be especially prevalent in people with anxiety disorders, although estimates of comorbidity vary widely due to differences in (among others) diagnostic instruments. Research suggests that approximately 10–50% of people who have panic disorder with agoraphobia have Avoidant personality disorder, as well as about 20–40% of people who have social phobia (social anxiety disorder).

Some studies report prevalence rates of up to 45% among people with generalized anxiety disorder and up to 56% of those with obsessive-compulsive disorder. Although it is not mentioned in the DSM-IV, earlier theorists have proposed a personality disorder which has a combination of features from borderline personality disorder and Avoidant personality disorder, called "avoidant-borderline mixed personality" (AvPD/BPD).

There is also significant overlap between avoidant personality disorder and autism spectrum disorders.

**Treatment**
Treatment of Avoidant personality disorder can employ various techniques, such as social skills training, cognitive therapy, exposure treatment to gradually increase social contacts, group therapy for practicing social skills, and sometimes drug therapy. A key issue in treatment is gaining and keeping the patient’s trust, since people with Avoidant personality disorder will often start to avoid treatment sessions if they distrust the therapist or fear rejection. The primary purpose of both individual therapy and social skills group training is for individuals with Avoidant personality disorder to begin challenging their exaggerated negative beliefs about themselves.

Epidemiology

According to the DSM-IV-TR, Avoidant personality disorder occurs in approximately 0.5% to 1% of the general population. However, data from the 2001-02 National Epidemiologic Survey on Alcohol and Related Conditions indicates a prevalence rate of the disorder of 2.36% in the American general population. It is seen in about 10% of psychiatric outpatients.

History

The avoidant personality has been described in several sources as far back as the early 1900s, although it was not so named for some time. Swiss psychiatrist Eugen Bleuler described patients who exhibited signs of Avoidant personality disorder in his 1911 work Dementia Praecox: Or the Group of Schizophrenias. Avoidant and schizoid patterns were frequently confused or referred to synonymously until Kretschmer (1921), in providing the first relatively complete description, developed a distinction.

Dependent personality disorder

Dependent personality disorder (DPD), formerly known as asthenic personality disorder, is a personality disorder that is characterized by a pervasive psychological dependence on other people. This personality disorder is a long-term (chronic) condition in which people depend too much on others to meet their emotional and physical needs.

The difference between a 'dependent personality' and a 'dependent personality disorder' is somewhat subjective, which makes diagnosis sensitive to cultural influences such as gender role expectations.

Characteristics

View of others

Individuals with DPD see other people as much more capable to shoulder life’s responsibilities, to navigate a complex world, and to deal with the competitions of life. Other people are powerful, competent, and capable of providing a sense of security and support to individuals with DPD. Dependent individuals avoid situations that require them
to accept responsibility for themselves; they look to others to take the lead and provide continuous support. DPD judgment of others is distorted by their inclination to see others as they wish they were rather than as they are. These individuals are fixated in the past. They maintain youthful impressions; they retain unsophisticated ideas and childlike views of the people toward whom they remain totally submissive. Individuals with DPD view strong caretakers, in particular, in an idealized manner; they believe they will be all right as long as the strong figure upon whom they depend is accessible.

**Self-image**

Individuals with DPD see themselves as inadequate and helpless; they believe they are in a cold and dangerous world and are unable to cope on their own. They define themselves as inept and abdicate self-responsibility; they turn their fate over to others. These individuals will decline to be ambitious and believe that they lack abilities, virtues and attractiveness. The solution to being helpless in a frightening world is to find capable people who will be nurturing and supportive toward those with DPD. Within protective relationships, individuals with DPD will be self-effacing, obsequious, agreeable, docile, and ingratiating. They will deny their individuality and subordinate their desires to significant others. They internalize the beliefs and values of significant others. They imagine themselves to be one with or a part of something more powerful and they imagine themselves to be supporting others. By seeing themselves as protected by the power of others, they do not have to feel the anxiety attached to their own helplessness and impotence. However, to be comfortable with themselves and their inordinate helplessness, individuals with DPD must deny the feelings they experience and the deceptive strategies they employ. They limit their awareness of both themselves and others. Their limited percutiveness allows them to be naive and uncritical. Their limited tolerance for negative feelings, perceptions, or interaction results in the interpersonal and logistical ineptness that they already believe to be true about themselves. Their defensive structure reinforces and actually results in verification of the self-image they already hold.

**Relationships**

Individuals with DPD see relationships with significant others as necessary for survival. They do not define themselves as able to function independently; they have to be in supportive relationships to be able to manage their lives. In order to establish and maintain these life-sustaining relationships, people with DPD will avoid even covert expressions of anger. They will be more than meek and docile; they will be admiring, loving, and willing to give their all. They will be loyal, unquestioning, and affectionate. They will be tender and considerate toward those upon whom they depend.

Dependent individuals play the inferior role to the superior other very well; they communicate to the dominant people in their lives that they are useful, sympathetic, strong, and competent. With these methods, individuals with DPD are often able to get along with unpredictable or isolated people. To further make this possible, individuals with DPD will approach both their own and others’ failures and shortcomings with a saccharine attitude and indulgent tolerance. They will engage in a mawkish minimization, denial, or
distortion of both their own and others’ negative, self-defeating, or destructive behaviors to sustain an idealized, and sometimes fictional, story of the relationships upon which they depend. They will deny their individuality, their differences, and ask for little other than acceptance and support.

Not only will individuals with DPD subordinate their needs to those of others, they will meet unreasonable demands and submit to abuse and intimidation to avoid isolation and abandonment. Dependent individuals so fear being unable to function alone that they will agree with things they believe are wrong rather than risk losing the help of people upon whom they depend. They will volunteer for unpleasant tasks if that will bring them the care and support they need. They will make extraordinary self-sacrifices to maintain important bonds.

It is important to note that individuals with DPD, in spite of the intensity of their need for others, do not necessarily attach strongly to specific individuals, i.e., they will become quickly and indiscriminately attached to others when they have lost a significant relationship. It is the strength of the dependency needs that is being addressed; attachment figures are basically interchangeable. Attachment to others is a self-referenced and, at times, haphazard process of securing the protection of the most readily available powerful other willing to provide nurturance and care. Both DPD and HPD are distinguished from other personality disorders by their need for social approval and affection and by their willingness to live in accord with the desires of others. They both feel paralyzed when they are alone and need constant assurance that they will not be abandoned. Individuals with DPD are passive individuals who lean on others to guide their lives. People with HPD are active individuals who take the initiative to arrange and modify the circumstances of their lives. They have the will and ability to take charge of their lives and to make active demands on others.

Causes

No studies of genetics or of biological traits for dependents have been conducted. Central to their psychodynamic constellation is an insecure form of attachment to others, which may be the result of clinging parental behavior.

DSM-IV

The Diagnostic and Statistical Manual of Mental Disorders fourth edition, DSM IV-TR, a widely used manual for diagnosing mental disorders, defines dependent personality disorder as at least five of the following (in Axis II Cluster C) as:

- has difficulty making everyday decisions without an excessive amount of advice and reassurance from others
- needs others to assume responsibility for most major areas of his or her life
- has difficulty expressing disagreement with others because of fear of loss of support or approval. Note: do not include realistic fears of retribution.
• has difficulty initiating projects or doing things on his or her own (because of a lack of self-confidence in judgment or abilities rather than a lack of motivation or energy)
• goes to excessive lengths to obtain nurturance and support from others, to the point of volunteering to do things that are unpleasant
• feels uncomfortable or helpless when alone because of exaggerated fears of being unable to care for himself or herself
• urgently seeks another relationship as a source of care and support when a close relationship ends
• is unrealistically preoccupied with fears of being left to take care of himself or herself

It is a requirement of DSM-IV that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

Diagnosis

The following questions when assessing individuals for DPD:

• Some people enjoy making decisions. Others prefer to have someone they trust guide them. Which do you prefer?
• Do you seek advice for everyday decisions? (Are the decisions you make understood by the practitioner?)
• Do you find yourself in situations where other people have made decisions about important areas in your life, e.g. what job to take? Symptoms you have they do not understand?
• Is it hard for you to express a different opinion with someone you are close to? What do you think might happen if you did?
• Do you often pretend to agree with others even if you do not? Why? Could it get you into trouble if you disagree?
• Do you often need help to get started on a project?
• Do you ever volunteer to do unpleasant things for others so they will take care of you when you need it?
• Are you uncomfortable when you are alone? Are you afraid you will not be able to take care of yourself?
• Have you found that you are desperate to get into another relationship right away when a close relationship ends? Even if the new relationship might not be the best person for you?
• Do you worry about important people in your life leaving you?

World Health Organization

The World Health Organization’s ICD-10 lists dependent personality disorder as F60.7

Dependent personality disorder:

It is characterized by at least 3 of the following:
encouraging or allowing others to make most of one's important life decisions;
- subordination of one's own needs to those of others on whom one is dependent, and undue compliance with their wishes;
- unwillingness to make even reasonable demands on the people one depends on;
- feeling uncomfortable or helpless when alone, because of exaggerated fears of inability to care for oneself;
- preoccupation with fears of being abandoned by a person with whom one has a close relationship, and of being left to care for oneself;
- limited capacity to make everyday decisions without an excessive amount of advice and reassurance from others.

Associated features may include perceiving oneself as helpless, incompetent, and lacking stamina. Includes:

- asthenic, inadequate, passive, and self-defeating personality (disorder)

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Millon's subtypes**

Psychologist Theodore Millon identified five adult subtypes of dependent personality disorder. Any individual dependent may exhibit none or one of the following:

- disquieted dependant
- including avoidant features
- accommodating dependant
- including histrionic features
- immature dependant
- variant of pure pattern
- ineffectual dependant
- including schizoid features
- selfless dependant
- including masochistic features
Differential diagnosis

The following conditions commonly coexist (comorbid) with dependent personality disorder:

- mood disorders
- anxiety disorders
- adjustment disorder
- borderline personality disorder
- avoidant personality disorder
- histrionic personality disorder

Treatment

Adler suggests that treatment goals for all personality disorders include: preventing further deterioration, regaining an adaptive equilibrium, alleviating symptoms, restoring lost skills, and fostering improved adaptive capacity. Goals may not necessarily include characterological restructuring. The focus of treatment is adaptation, i.e., how individuals respond to the environment. Treatment interventions teach more adaptive methods of managing distress, improving interpersonal effectiveness, and building skills for affective regulation.

For individuals with DPD, the goal of treatment is not independence but autonomy. Autonomy has been defined as the capacity for independence and the ability to develop intimate relationships. Sperry suggests that the basic goal for DPD treatment is self-efficacy. Individuals with DPD must recognize their dependent patterns and the high price they pay to maintain those patterns. This allows them to explore alternatives. The long-range goal is to increase DPD individuals' sense of independence and ability to function. Clients with DPD must build strength rather than foster neediness.

As with other personality disorders, treatment goals should not be in contradiction to the basic personality and temperament of these individuals. They can work toward a more functional version of those characteristics that are intrinsic to their style. Oldham suggests seven traits and behaviors of the "devoted personality style," i.e., the non-personality-disordered version of DPD:

- ability to make commitments;
- enjoyment of intimacy;
- skills as a team player—without need to compete with the leader;
- willingness to seek the opinions and advice of others;
- ability to promote interpersonal harmony;
- thoughtfulness and consideration for others; and,
- willingness to self-correct in response to criticism.

Group psychotherapy
Several reports suggest that group psychotherapy can be successful for the treatment of dependent personality disorder. Montgomery used group therapy for dependent patients who used medications for chronic complaints such as insomnia and nervousness. All but 3 of 30 patients eventually discontinued medications and began to confront their anger at being dependent on the therapist.

Sadoff and Collins administered weekly group psychotherapy to 22 patients who stuttered, most of whom had passive-dependent traits. Although the dropout rate was high, the authors found that the interpretation of passive-dependent behavior and attitudes (e.g., asking for help, believing that others are responsible for helping them) as a defense against recognizing and expressing anger proved helpful. Both stuttering and passive dependency improved in 2 patients who became angry and were able to confront their anger.

Torgersen studied college students who attended a weekend-long encounter group. On follow-up several weeks later, individuals who initially scored high on dependent traits had mixed responses. Although the group experience left them feeling disturbed and anxious, they also reported becoming more accepting of their own feelings and opinions. No other changes were found.

Attrition tends to be higher in group than in individual therapy for personality disorders but may be less of a problem for individuals with dependent personality disorder. Budman et al. reported moderate improvements after an 18-month group for personality disorders (10% with dependent personality disorder), with some changes not beginning until after 6 months.

These reports suggest the usefulness of group psychotherapy for dependent personality disorder. Most clinicians use weekly sessions of an hour to an hour and a half. Treatment generally lasts several years.

**Biological therapies**

Four studies have explored the use of medications in the treatment of dependent personality disorder, and two studies have investigated their use in the treatment of dependent traits. Diagnostic and other limitations of the studies prevent firm conclusions about the efficacy of medications.

Klein and colleagues compared placebo with either imipramine or chlorpromazine in hospitalized patients with passive-aggressive and passive-dependent personality disorders that had been diagnosed according to DSM criteria. None of the patients showed a positive drug response.

Patients with major depressive disorder and an anxious-cluster personality disorder, many with dependent personality disorder, showed significant improvement in depression with imipramine or psychotherapeutic treatment. Fewer patients with Cluster C disorders fully recovered, however, and social adjustment problems remained.
Tyrer et al. drew a similar conclusion after studying patients with "general neurotic syndrome," which includes mixed anxiety-depression and dependent or obsessive personality. Although such patients initially appeared to be as responsive as others to 10-week treatments, including dothiepin (an antidepressant), diazepam, placebo, cognitive-behavioral therapy, or self-help, at 2-year follow-up, they had greater symptom levels and did significantly worse than other outpatients.

Ekselius and von Knorring studied 145 depressed patients, 61% of whom scored in the personality disorder range by self-report questionnaire, who received sertraline or citalopram for 24 weeks. From baseline to termination, the percentage above the cutoff score for dependent personality disorder improved significantly (21% versus 8%) as did the mean number of dependent personality disorder criteria met by the whole sample (3.3 versus 2.3). The self-reported change in dependent personality disorder criteria was significant, even after controlling for change in observer-rated depressive symptoms. Although the comparison across two different measurement perspectives complicates these findings, self-reported dependent symptoms seem to improve with 24 weeks of selective serotonin reuptake inhibitor treatment. Whether this generalizes to observer-rated improvement in life functioning is unknown.

**Residential and day treatment therapies**

Although hospitalization is sometimes necessary for the treatment of an Axis I disorder in individuals with dependent personality disorder, residential treatments are generally not indicated. However, residential and day treatment may provide support necessary to allow definitive psychotherapy to continue, when dependent personality disorder is complicated by recurrent depression, severe anxiety disorders, repetitive suicide attempts, other more severe personality disorders (such as borderline personality) or overwhelming life stress.

Several day treatment and residential programs for severe personality disorders have included individuals with dependent personality disorder. Active treatment days varied from 4 to 5 days per week over a range of 17–30 weeks and usually involved both group and individual sessions, most within a dynamic framework. All had moderate to large effect sizes. Piper et al. (1993) conducted a randomized controlled trial and found significantly greater changes in the day treatment than in the control groups. These data suggest a valuable role for these modalities when dependent personality disorder is not responsive to other outpatient therapies.

**Medication**

There is little evidence to suggest that the use of medication will result in long-term benefits in the personality functioning of individuals with DPD. DPD is not amenable to pharmacological measures; treatment relies upon verbal therapies. It is recommended that target symptoms rather than specific personality disorders be medicated. One of these target symptoms of particular importance is dysphoria -- marked by low energy, leaden fatigue, and depression. Dysphoria can also be associated with a craving for chocolate and
for stimulants, e.g. cocaine. DPD is one of the most vulnerable personality disorders to
dysphoria and some individuals with DPD respond well to antidepressant medications.

People with DPD are prone to both depressive and anxiety disorders. Stone suggests that
these individuals may respond well to benzodiazepines in a crisis. However, clients with
DPD are likely to abuse anxiolytics and their use should be limited and monitored with
cautions.

Unfortunately, individuals with DPD tend to be appealing clients. They are not inclined to
be demanding and provocative. This can be precisely why they are given benzodiazepines
by psychiatrists who may feel both benevolent and protective. Their inclination to use
denial and escape to manage their lives makes the use of sedative-hypnotics familiar and
pleasant. Iatrogenic addiction is a serious concern.

**Epidemiology**

Dependent personality disorder occurs in about 0.5% of the general population. It is more
frequent in females.

**History**

Clinical interest in dependent personality disorder has existed since Karl Abraham first
described it. As a disorder, the personality type first appeared in a United States
Department of War technical bulletin in 1945 and later in the first edition of the Diagnostic
and Statistical Manual in 1952 (American Psychiatric Association, 1952) as a subtype of
passive-aggressive personality disorder. Since then, a surprising number of studies have
upheld the descriptive validity of dependent personality traits, viewed as submissiveness,
oral character traits, oral dependence, or passive dependence, or as a constellation of both
pathological and adaptive traits under the rubric

**Dependent Personality Disorder: Summarized**

**Dependent Personality Disorder**

Dependent personality disorder is characterized by a long-standing need for the person to
be taken care of and a fear of being abandoned or separated from important individuals in
his or her life. This leads the person to engage in dependent and submissive behaviors that
are designed to elicit care-giving behaviors in others. The dependent behavior may be see
as being "clingy" or "cling on" to others, because the person fears they can't live their
lives without the help of others.

Individuals with Dependent Personality Disorder are often characterized by pessimism and
self-doubt, tend to belittle their abilities and assets, and may constantly refer to themselves
as "stupid." They take criticism and disapproval as proof of their worthlessness and lose
faith in themselves. They may seek overprotection and dominance from others.
Occupational functioning may be impaired if independent initiative is required. They may avoid positions of responsibility and become anxious when faced with decisions. Social relations tend to be limited to those few people on whom the individual is dependent.

Chronic physical illness or Separation Anxiety Disorder in childhood or adolescence may predispose an individual to the development of dependent personality disorder.

**Symptoms of Dependent Personality Disorder**

Dependent personality disorder is characterized by a pervasive fear that leads to "clinging behavior" and usually manifests itself by early adulthood. It includes a majority of the following symptoms:

- Has difficulty making everyday decisions without an excessive amount of advice and reassurance from others
- Needs others to assume responsibility for most major areas of his or her life
- Has difficulty expressing disagreement with others because of fear of loss of support or approval
- Has difficulty initiating projects or doing things on his or her own (because of a lack of self-confidence in judgment or abilities rather than a lack of motivation or energy)
- Goes to excessive lengths to obtain nurturance and support from others, to the point of volunteering to do things that are unpleasant
- Feels uncomfortable or helpless when alone because of exaggerated fears of being unable to care for himself or herself
- Urgently seeks another relationship as a source of care and support when a close relationship ends
- Is unrealistically preoccupied with fears of being left to take care of himself or herself

As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.

Dependent personality disorder is the most commonly diagnosed personality disorder in mental health clinics.

Like most personality disorders, dependent personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

**How is Dependent Personality Disorder Diagnosed?**

Personality disorders such as dependent personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician
about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose dependent personality disorder.

Many people with dependent personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person’s life. This most often happens when a person’s coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for dependent personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

**Causes of Dependent Personality Disorder**

Researchers today don’t know what causes dependent personality disorder. There are many theories, however, about the possible causes of dependent personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual's personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.

**Dependent Personality Disorder Treatment**

**Introduction**

Individuals with dependent personality disorder are usually quite needy, for attention, valuation, and social contact. Clients with disorder usually don't present in a dramatic fashion, but will often make repeated requests for attention to their complaints, whether these complaints are about their lifestyle, social relationships, lack of meaning in life, medical, or education. People who suffer from this disorder are often outwardly compliant with clinicians’ suggestion for treatment, and will usually be passive in their overall treatment, no matter what form it takes. However, real gains in therapy may not be made easily, because the client's compliance (due to the disorder) is often only surface-deep. While the individual may be one of the easiest to see week after week or month after month in therapy, they may also be one of the most difficult because of their strong need for constant reassurance and support. Dependency upon the clinician specifically and therapy in general should be carefully monitored and avoided.

Psychiatrists and physicians should be aware that individuals with dependent personality disorder will often present with a number of physical or somatic complaints. While
appropriate medications need to be prescribed for these as necessary, the clinician should carefully monitor medication intake and maintenance to ensure the patient is not abusing it. Physical complaints should not be minimized or dismissed, as is often the case with someone who suffers from this disorder, but they must not also be encouraged. A simple, matter-of-fact approach works best in this case.

Clinicians in general should be wary of the therapeutic relationship with a person suffering from dependent personality disorder. The needs of the individual can be great and overwhelming at times, and the patient will often try to test the limits of the frame set for therapy. Burnout among therapists treating this disorder is common, because of the client’s demands for constant reassurance and attention, especially between therapy sessions. A clear explanation at the onset of therapy about how treatment is to be conducted, including a discussion of appropriate times and needs for contacting the clinician in-between sessions, is vitally important. While rapport and a close, therapeutic relationship must be established, the boundaries in therapy must also be constantly and clearly delineated.

**Psychotherapy**

As with all personality disorders, psychotherapy is the treatment of choice. Treatment is likely to be sought by individuals suffering from this disorder when stress or other complications within their life have led to decreased efficiency in life functioning. As with all other personality disorders as well, they may present with a clear Axis I diagnosis and the personality disorder may only become apparent after a few sessions of therapy.

The most effective psychotherapeutic approach is one which is focuses on solutions to specific life problems the patient is presently experiencing. Long-term therapy, while ideal for many personality disorders, is contra-indicated in this instance since it reinforces a dependent relationship upon the therapist. While some form of dependency will exist no matter the length of therapy, the shorter the better in this case. Termination issues will likely be of extreme importance and will virtually be a litmus test of how effective the therapy has been. If the individual cannot end therapy successfully and move on to become more self-reliant, it should not be seen as a therapeutic failure. Rather, the individual was not likely seeking life-changing therapy in the first instance but instead solution-focused therapy.

Examining the client’s faulty cognitions and related emotions (of lack of self-confidence, autonomy versus dependency, etc.) can be an important component of therapy. Assertiveness training and other behavioral approaches have been shown to be most effective in helping treat individuals with this disorder. Group therapy can also be helpful, although care should be utilized to ensure that the patient doesn’t use groups to enhance existing or new dependent relationships. Challenging dependent relationships the client has with others that may be unhealthy for the client should generally be avoided at the onset of therapy. As therapy progresses, these challenges can occur but must be done carefully; restraint must be used if the individual is not ready to give up these unhealthy relationships.
Termination of therapy with a person who has this disorder is an extremely important issue to consider. While termination should always be a joint decision between the clinician and the client, people with this disorder often don't know "how much is enough" therapy. The therapist, therefore, may need to prod the patient toward ending therapy. As the end of therapy approaches, the patient is likely to re-experience feelings of insecurity, lack of self-confidence, increased anxiety and perhaps even depression. This can be typical of individuals with this disorder terminating therapy and should be treated appropriately. The clinician should not allow the patient to use these new symptoms, though, as a way of prolonging the current therapy. The goal is to end a relationship at an agreed-upon time and way. The client should be reinforced for the positive gains made in therapy and encouraged to explore their new-found autonomy or improved management of their anxious feelings.

Medications

As with all personality disorders, medications should only be prescribed for specific problems suffered by the individual. Sedative drug abuse and overdose is common in this population and should be prescribed with additional caution. Anti-anxiety agents and antidepressants should be prescribed only when there is a clear Axis I diagnosis in conjunction with the personality disorder. Physicians should resist the temptation to overprescribe to someone with this disorder, because they often present with multiple physical complaints or anxiety. The anxiety in this instance is clearly situationally-related and medication may actually interfere with effective psychotherapeutic treatment.

Giving any individual with a personality or mental disorder a placebo drug for its perceived value by the patient is ethically questionable. Doctors rarely have need to prescribe a vitamin or other non-psychoactive substance unless a patient's medical condition clearly indicates it. When such a prescription is made, it should be made with the clear understanding what it is being prescribed for. Any indirect suggestion that such a medication will help an individual overcome their feelings of insecurity, inadequacy, need for dependence, etc. should be avoided. A medication should not be prescribed because of its "magical" effects, and more expensive medications should not be prescribed over less-expensive medications just because they are "newer." Prescriptions should always be written for a specific medication because of the research suggesting its effectiveness with the patient’s specific medical complaint or diagnosed mental disorder and avoidance of intolerable side-effects.

Self-Help

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. Suggesting such a support group later in treatment, to help put some of their new skill sets to use in a group setting, may be helpful. Many support groups exist within communities throughout the world which are devoted to helping individuals with this disorder share their commons experiences and feelings.
Individuals should likely avoid using a support group as the only means of treatment for this disorder, since it is likely to encourage additional dependent relationships.

**Obsessive-compulsive personality disorder**

Obsessive-compulsive personality disorder (OCPD) is a personality disorder characterized by a pervasive pattern of preoccupation with orderliness, perfectionism, and mental and interpersonal control at the expense of flexibility, openness, and efficiency.

**Signs and symptoms**

The primary symptoms of OCPD can include preoccupation with remembering and paying attention to minute details and facts, following rules and regulations, compulsion to make lists and schedules, as well as rigidity/inflexibility of beliefs, and/or exhibition of perfectionism that interferes with task-completion. Symptoms may cause extreme distress and interfere with a person’s occupational and social functioning. According to the National Institute for Mental Health:

OCPD has some of the same symptoms as obsessive-compulsive disorder (OCD). However, people with OCD have unwanted thoughts, while people with OCPD believe that their thoughts are correct.

Most patients spend their early life avoiding symptoms and developing techniques to not deal with these issues.

**Obsession**

Some, but not all, patients with OCPD show an obsessive need for cleanliness. This OCPD trait is not to be confused with domestic efficiency, and, in fact, over-attention to related details may make these (and other) activities of daily living difficult to accomplish. Though obsessive behavior is in part a way of controlling anxiety, tension often remains. In the case of a hoarder, attention effectively to clean the home may be hindered by the amount of clutter that the hoarder resolves later to organize.

Whilst there are superficial similarities between the list-making and obsessive aspects of Asperger’s syndrome and OCPD, the former is different from OCPD especially regarding affective behaviours, including (but not limited to) empathy, social coping, and general social skills.

Perception of own and other's actions and beliefs tend to be polarised (i.e., "right" or "wrong", with little or no margin between the two) for people with this disorder. As might be expected, such rigidity places strain on interpersonal relationships, with frustration sometimes turning into anger and even violence. This is known as disinhibition. People with OCPD often tend to general (pessimism) and/or underlying form(s) of depression. This can at times become so serious that suicide is a risk. Indeed, one study suggests that
personality disorders are a significant substrate to psychiatric morbidity. They may cause more problems in functioning than a major depressive episode.

**Causes**

Research into the familial tendency of OCPD may be illuminated by DNA studies. Two studies suggest that people with a particular form of the DRD3 gene are highly likely to develop OCPD and depression, particularly if they are male. Genetic concomitants, however, may lie dormant until triggered by events in the lives of those who are predisposed to OCPD. These events could include trauma faced during childhood, such as physical, emotional or sexual abuse, or other types of psychological trauma.

**Diagnosis**

**DSM**

The Diagnostic and Statistical Manual of Mental Disorders fourth edition, (DSM IV-TR = 301.4), a widely used manual for diagnosing mental disorders, defines obsessive-compulsive personality disorder (in Axis II Cluster C) as:

A pervasive pattern of preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency, beginning by early adulthood and present in a variety of contexts. It is a requirement of DSM-IV that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Criticism**

Since DSM IV-TR was published in 2000, some studies have found fault with its OCPD coverage. A 2004 study challenged the usefulness of all but three of the criteria: perfectionism, rigidity and stubbornness, and miserliness. A study in 2007 found that OCPD is etiologically distinct from avoidant and dependent personality disorders, suggesting it is incorrectly categorized as a Cluster C disorder.

**WHO**

The World Health Organization’s ICD-10 defines a conceptually similar disorder to obsessive-compulsive personality disorder called (F60.5) Anankastic personality disorder.

It is characterized by at least three of the following:

- feelings of excessive doubt and caution;
- preoccupation with details, rules, lists, order, organization or schedule;
- perfectionism that interferes with task completion;
- excessive conscientiousness, scrupulousness, and undue preoccupation with productivity to the exclusion of pleasure and interpersonal relationships;
excessive pedantry and adherence to social conventions;
rigidity and stubbornness;
unreasonable insistence by the individual that others submit exactly to his or her way of doing things, or unreasonable reluctance to allow others to do things;
intrusion of insistent and unwelcome thoughts or impulses.

Includes:

- compulsive and obsessional personality (disorder)
- obsessive-compulsive personality disorder

Excludes:

- obsessive-compulsive disorder

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

**Millon's subtypes**

Theodore Millon identified five subtypes of compulsive. Any individual compulsive may exhibit none or one of the following:

- conscientious compulsive—including dependent features
- puritanical compulsive—including paranoid features
- bureaucratic compulsive—including narcissistic features
- parsimonious compulsive—including schizoid features. Resembles Fromm's hoarding orientation
- bedeviled compulsive—including negativistic (passive-aggressive) features

**Differential diagnosis**

Obsessive–compulsive personality disorder is often confused with obsessive–compulsive disorder (OCD). Despite the similar names, they are two distinct disorders, although some OCPD individuals also suffer from OCD, and the two are sometimes found in the same family, sometimes along with eating disorders. People experiencing OCPD do not generally feel the need to repeatedly perform ritualistic actions—a common symptom of OCD—and usually find pleasure in perfecting a task, whereas OCD patients are often more distressed after their actions.

OCPD is often thought to be the same as mood disorders, such as depression and generalized anxiety disorder.

**Treatment**
Treatment for OCPD normally involves psychotherapy and self-help. However, in some cases, there can be an impediment to change in that the patient does not accept that they have OCPD, and/or believes (at least at some level) that their thoughts and/or behaviours are in some sense "correct" and therefore should not be changed. Medication in isolation is generally not indicated for this personality disorder, but fluoxetine has been prescribed with success. Anti-anxiety medication may reduce feelings of fear while SSRIs (anti-depressants) can ease frustration, reducing stubbornness and negative rumination.

**Psychotherapy**

**Cognitive behavioral therapy**

- Behavior therapy: Discussing with a psychotherapist ways of changing compulsions into healthier, productive behaviors. An effective form of this therapy has been found to be cognitive analytic therapy.
- Psychotherapy: Discussion with a trained counsellor or psychotherapist who understands the condition.
- Psychopharmacology: A psychiatrist may be able to prescribe medication to facilitate self-management and also enable more productive participation in other therapies.

**Epidemiology**

Obsessive-compulsive personality disorder occurs in about 1 percent of the general population. It is seen in 3-10 percent of psychiatric outpatients. It is twice as common in males as females.

**History**

In 1908, Sigmund Freud named what is now known as obsessive-compulsive or anankastic personality disorder "anal retentive character". He identified the main strands of the personality type as a preoccupation with orderliness, parsimony (frugality), and obstinacy (rigidity and stubbornness). The concept fits his theory of psychosexual development.

Since the early 1990s, considerable new research continues to emerge into OCPD and its characteristics, including the tendency for it to run in families along with eating disorders and even to appear in childhood.

**Obsessive Compulsive Personality Disorder: Summarized**

**Obsessive-Compulsive Personality Disorder**

Obsessive-Compulsive Personality Disorder is characterized by a preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency.
When rules and established procedures do not dictate the correct answer, decision making may become a time-consuming, often painful process. Individuals with Obsessive-Compulsive Personality Disorder may have such difficulty deciding which tasks take priority or what is the best way of doing some particular task that they may never get started on anything.

They are prone to become upset or angry in situations in which they are not able to maintain control of their physical or interpersonal environment, although the anger is typically not expressed directly. For example, a person may be angry when service in a restaurant is poor, but instead of complaining to the management, the individual ruminates about how much to leave as a tip. On other occasions, anger may be expressed with righteous indignation over a seemingly minor matter.

People with this disorder may be especially attentive to their relative status in dominance-submission relationships and may display excessive deference to an authority they respect and excessive resistance to authority that they do not respect.

Individuals with this disorder usually express affection in a highly controlled or stilted fashion and may be very uncomfortable in the presence of others who are emotionally expressive. Their everyday relationships have a formal and serious quality, and they may be stiff in situations in which others would smile and be happy (e.g., greeting a lover at the airport). They carefully hold themselves back until they are sure that whatever they say will be perfect. They may be preoccupied with logic and intellect.

Symptoms of Obsessive-Compulsive Personality Disorder

A pervasive pattern of preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

- Is preoccupied with details, rules, lists, order, organization, or schedules to the extent that the major point of the activity is lost
- Shows perfectionism that interferes with task completion (e.g., is unable to complete a project because his or her own overly strict standards are not met)
- Is excessively devoted to work and productivity to the exclusion of leisure activities and friendships (not accounted for by obvious economic necessity)
- Is overconscientious, scrupulous, and inflexible about matters of morality, ethics, or values (not accounted for by cultural or religious identification)
- Is unable to discard worn-out or worthless objects even when they have no sentimental value
- Is reluctant to delegate tasks or to work with others unless they submit to exactly his or her way of doing things
- Adopts a miserly spending style toward both self and others; money is viewed as something to be hoarded for future catastrophes
- Shows significant rigidity and stubbornness
As with all personality disorders, the person must be at least 18 years old before they can be diagnosed with it.

Obsessive-Compulsive personality disorder is approximately twice as prevalent in males than females, and occurs in about 1 percent of the general population.

Like most personality disorders, Obsessive-Compulsive personality disorder typically will decrease in intensity with age, with many people experiencing few of the most extreme symptoms by the time they are in the 40s or 50s.

How is Obsessive-compulsive Personality Disorder Diagnosed?

Personality disorders such as obsessive-compulsive personality disorder are typically diagnosed by a trained mental health professional, such as a psychologist or psychiatrist. Family physicians and general practitioners are generally not trained or well-equipped to make this type of psychological diagnosis. So while you can initially consult a family physician about this problem, they should refer you to a mental health professional for diagnosis and treatment. There are no laboratory, blood or genetic tests that are used to diagnose obsessive-compulsive personality disorder.

Many people with obsessive-compulsive personality disorder don’t seek out treatment. People with personality disorders, in general, do not often seek out treatment until the disorder starts to significantly interfere or otherwise impact a person’s life. This most often happens when a person’s coping resources are stretched too thin to deal with stress or other life events.

A diagnosis for obsessive-compulsive personality disorder is made by a mental health professional comparing your symptoms and life history with those listed here. They will make a determination whether your symptoms meet the criteria necessary for a personality disorder diagnosis.

Causes of Obsessive-compulsive Personality Disorder

Researchers today don't know what causes obsessive-compulsive personality disorder. There are many theories, however, about the possible causes of obsessive-compulsive personality disorder. Most professionals subscribe to a biopsychosocial model of causation -- that is, the causes of are likely due to biological and genetic factors, social factors (such as how a person interacts in their early development with their family and friends and other children), and psychological factors (the individual’s personality and temperament, shaped by their environment and learned coping skills to deal with stress). This suggests that no single factor is responsible -- rather, it is the complex and likely intertwined nature of all three factors that are important. If a person has this personality disorder, research suggests that there is a slightly increased risk for this disorder to be "passed down" to their children.
Obsessive-Compulsive Personality Disorder Treatment

Introduction

Individuals who suffer from this personality disorder often are characterized by their lack of openness and flexibility in not only their daily routines, but also with interpersonal relationships and expectations. The overwhelming preoccupation with orderliness, perfectionism and control of their lives and relationships means that most types of treatment are going to be, at best, difficult. Treatment options which do not fit within the client’s cognitive schema will likely be quickly rejected rather than attempted.

Individuals who suffer from this disorder have difficulty in incorporating new and changing information into their lives, so new learning takes place only over a great deal of time and with as much effort on both the clinician’s and client’s part. Their ability to work with others is equally affected, since they see the world as black and white -- their way of doing things and the wrong way of doing things. Naturally, this faulty logic will also be translated into their therapeutic relationship with the clinician and their treatment. It is therefore unlikely the clinician will have much success in using techniques or treatment modalities which haven’t first been approved by the patient for use. Sometimes this may be done simply by stating the effectiveness of a given treatment for a specific problem, citing relevant research studies. More often, though, this technique won’t be effective.

When this disorder is combined with the presentation of a medical illness, physicians should expect a logical and coherent presentation of troubling symptoms with little emotionality attached to their physical discomfort. Treatment is most effective when the nature of the disease process is first discussed with the individual, as well as typical and accepted treatments. A physician in this instance is best sticking with the facts of the presenting problem and underlying disorder rather than offering vague impressions of their opinion. Since the individual with this disorder tends to be meticulous and concerned with details, the treatment regimen -- once accepted -- will likely be adhered to rigorously, without incident.

Psychotherapy

As with most personality disorders, individuals seek treatment for items in their life which have become overwhelming to their existing coping skills. These skills may be somewhat limited, in the first place, because of their disorder. While they may be generally effective enough in most instances to shield the client from stress and emotional difficulties, during times of increased stress, work pressure, family problems, etc. the underlying disorder will become more evident in day-to-day behaviors.

As with most personality disorders, treatment is often focused on short-term symptom relief and the support of existing coping mechanisms while teaching new ones. Long-term or substantive work on personality change is usually beyond most clinician’s skill levels, and patient’s budgets. Obsessive-compulsive personality disorder is especially resistant to such changes, because of the basic makeup of this disorder.
Short-term therapy will be most likely to be beneficial when the patient’s current support system and coping skills are examined. Those skills which are not currently working could be reinforced with additional skill sets. Social relationships can also be examined, reinforcing strong, positive relationships while having the client re-examine negative or harmful relationships. One important aspect is to try and have the individual examine and properly identify their feeling states, rather than just intellectualizing or distancing themselves from their emotions. This can be accomplished through a variety of techniques, such as feeling identification (e.g., the “feeling faces”) at the onset of every therapy session. Homework might include writing feelings down in a journal, especially as they notice them. Proper identification and realization of feelings can bring about much change in and of itself.

Individuals suffering from obsessive-compulsive personality disorder often are not in touch with their emotional states as much as their thoughts. Leading the client away from describing situations, events, and daily happenings and to talking about how such situations, events and daily happenings made them feel may be helpful. Sometimes the patient may complain he or she doesn’t remember or know how he or she felt at the time; the journal becomes a useful tool at this point.

Therapy with people who have this disorder can sometimes be trying, since they can see the world in a very "all-or-nothing" manner. Beck’s cognitive therapy doesn't seem to be all that effective in treatment, and cognitive approaches in general probably aren’t useful in this case. Clinicians must be willing to undergo verbal attacks on their professionalism and knowledge, as such skepticism about a therapist's treatment approach from the client with this disorder can be expected. Clinicians should also be careful about engaging the client within these verbal attacks or intellectual discussions, as they continue to distance the patient from his or her feelings. And take the focus off of the client and onto unrelated matters (e.g., a therapist’s professional training).

Most people who suffer from this personality disorder (and the different, but related, obsessive-compulsive disorder) lead relatively normal lives, may have a family, friends, and work regularly. Clinicians should be careful not to overgeneralize psychopathology and look to change aspects of the patient’s personality he or she is not ready or willing to change. This means, in effect, that if the way they relate to others in their environment (which a clinician might characterize as a personality disorder) is working for them, a clinician should not seek to change it 180 degrees without the client’s purposeful consent. Therapy will most often be most effective when it focuses on correcting short-term difficulties currently being experienced. It will become increasingly less effective when the goal of therapy is complex, long-term personality change.

Although a group therapy modality may be helpful and an effective treatment option, most people who suffer from this disorder will not be able to withstand the minimum social contact necessary to gain a healthy group dynamic. They may quickly become ostracized by the group for pointing out other people’s deficits and "wrong-headed" ways of doing things.
Hospitalization

Hospitalization is rarely needed for people who suffer from this disorder, unless an extreme or severe stressor or stressful life event occurs which increases the compulsive behaviors to an extent where regular daily activities are halted or present possible risks of harm to the patient. Hospitalization may also be needed when the obsessive thoughts do not allow the individual to conduct any usual activities, paralyzing them in bed or with their accompanying compulsive behaviors.

Medications

In most cases, medication for this disorder is not indicated unless the individuals is also suffering from a clearly delineated Axis I diagnosis as well. However, newer medications such as Prozac, an SSR, have been approved for the treatment of obsessive-compulsive disorder and may provide some relief to individuals with the personality disorder. Long-term use, though, is rarely indicated, appropriate, or beneficial.

Self-Help

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. Support groups, though, offer an excellent adjunct to continuing medication check-ups once a month, and a way to gain emotional and social support through the community. These groups also allow others to ensure the client is doing well and promotes the client’s independence and stability. Many support groups exist within communities throughout the world which are devoted to helping individuals with this disorder share their commons experiences and feelings.

Such support groups are recommended to individuals suffering from this disorder, especially if they have found therapy unhelpful or too expensive.

Depressive personality disorder

Depressive Personality Disorder (also known as melancholic personality disorder) is a controversial psychiatric diagnosis that denotes a personality disorder with depressive features.

Originally included in the American Psychiatric Association’s DSM-II, depressive personality disorder was removed from the DSM-III and DSM-III-R. Recently, it has been reconsidered for reinstatement as a diagnosis. Depressive personality disorder is currently described in Appendix B in the DSM-IV-TR as worthy of further study. Although no longer listed in the manual’s personality disorder category, the diagnosis is included under the section “personality disorder not otherwise specified”.

While depressive personality disorder shares some similarities with mood disorders such as dysthymic disorder, it also shares many similarities with personality disorders including
avoidant personality disorder. Some researchers argue that depressive personality disorder is sufficiently distinct from these other conditions so as to warrant a separate diagnosis.

**Characteristics**

The DSM-IV defines depressive personality disorder as "a pervasive pattern of depressive cognitions and behaviors beginning by early adulthood and occurring in a variety of contexts." Depressive personality disorder occurs before, during, and after major depressive episodes, making it a distinct diagnosis not included in the definition of either major depressive episodes or dysthymic disorder. Specifically, five or more of the following must be present most days for at least two years in order for a diagnosis of depressive personality disorder to be made:

- Usual mood is dominated by dejection, gloominess, cheerlessness, joylessness, and unhappiness
- Self-concept centers around beliefs of inadequacy, worthlessness, and low self-esteem
- Is critical, blaming, and derogatory towards the self
- Is brooding and given to worry
- Is negativistic, critical, and judgmental toward others
- Is pessimistic
- Is prone to feeling guilty or remorseful

People with depressive personality disorder have a generally gloomy outlook on life, themselves, the past and the future. They are plagued by issues developing and maintaining relationships. In addition, studies have found that people with depressive personality disorder are more likely to seek psychotherapy than people with Axis I depression spectrum diagnoses.

Recent studies have concluded that people with depressive personality disorder are at a greater risk of developing dysthymic disorder than a comparable group of people without depressive personality disorder. These findings lead to the fact that depressive personality disorder is a potential precursor to dysthymia or other depression spectrum diagnoses. If included in the DSM-V, depressive personality disorder would be included as a warning sign for potential development of more severe depressive episodes.

Researchers at McLean Hospital in Massachusetts looked at the comorbidity of depressive personality disorder and a variety of other disorders. It was found that subjects with depressive personality disorder were more likely than the subjects without depressive personality disorder to currently have major depression and an eating disorder. Subjects with and without depressive personality disorder were statistically equally likely to have any of the other disorders examined.

**Millon's subtypes**
Theodore Millon, a former professor of Harvard Medical School and University of Miami and a respected psychiatrist, identified five subtypes of depression. Any individual depressive may exhibit none, or one or more of the following:

- Ill-humored depressive, including negativistic (passive-aggressive) features. Patients in this subtype are often hypochondriacal, cantankerous and irritable, and guilt-ridden and self-condemning. In general, ill-humored depressives are down on themselves and think the worst of everything.
- Voguish depressive, including histrionic, narcissistic features. Voguish depressives see unhappiness as a popular and stylish mode of social disenchanted, personal depression as self-glorifying, and suffering as ennobling. The attention from friends, family, and doctors is seen as a positive aspect of the voguish depressive’s condition.
- Self-derogating depressive, including dependent features. Patients who fall under this subtype are self-deriding, discrediting, odious, dishonorable, and disparage themselves for weaknesses and shortcomings. These patients blame themselves for not being good enough.
- Morbid depressive, including masochistic features. Morbid depressives experience profound dejection and gloom, are highly lugubrious, and often feel drained and oppressed.
- Restive depressive, including avoidant features. Patients who fall under this subtype are consistently unsettled, agitated, wrought in despair, and perturbed. This is the subtype most likely to commit suicide in order to avoid all the despair in life.

Not all patients with a depressive disorder fall into a subtype. These subtypes are multidimensional in that patients usually experience multiple subtypes, instead of being limited to fitting into one subtype category. Currently, this set of subtypes is associated with melancholic personality disorders. All depression spectrum personality disorders are melancholic and can be looked at in terms of these subtypes.

**Criticisms of the Inclusion of Depressive Personality Disorder in the DSM-V**

**Similarities to Dysthymic Disorder**

Much of the controversy surrounding the potential inclusion of depressive personality disorder in the DSM-V stems from its apparent similarities to dysthymic disorder, a diagnosis already included in the DSM-IV. Dysthymic disorder is characterized by a variety of depressive symptoms, such as hypersomnia or fatigue, low self esteem, poor appetite, or difficulty making decisions, for over two years, with symptoms never numerous or severe enough to qualify as major depressive disorder. Patients with dysthymic disorder may experience social withdrawal, pessimism, and feelings of inadequacy at higher rates than other depression spectrum patients. Early-onset dysthymia is the diagnosis most closely related to depressive personality disorder.

The key difference between dysthymic disorder and depressive personality disorder is the focus of the symptoms used to diagnose. Dysthymic disorder is diagnosed by looking at the somatic senses, the more tangible senses. Depressive personality disorder is diagnosed by
looking at the cognitive and intrapsychic symptoms. The symptoms of dysthymic disorder and depressive personality disorder may look similar at first glance, but the way these symptoms are considered distinguish the two diagnoses.

Comorbidity with Other Disorders

Many researchers believe that depressive personality disorder is so highly comorbid with other depressive disorders, manic-depressive episodes and dysthymic disorder, that it is redundant to include it as a distinct diagnosis. Recent studies however, have found that dysthymic disorder and depressive personality disorder are not as comorbid as previously thought. It was found that almost two thirds of the test subjects with depressive personality disorder did not have dysthymic disorder, and 83% did not have early-onset dysthymia. The comorbidity with Axis I depressive disorders is not as high as had been assumed. An experiment conducted by American psychologists showed that depressive personality disorder shows a high comorbidity rate with major depression experienced at some point in a lifetime and with any mood disorders experienced at any point in a lifetime. A high comorbidity rate with these disorders is expected of many diagnoses. As for the extremely high comorbidity rate with mood disorders, it has been found that essentially all mood disorders are comorbid with at least one other, especially when looking at a lifetime sample size.

Changes to Cluster C

If depressive personality disorder were added to the DSM-V, it would be included in the Cluster C personality disorders, anxious and fearful personality disorders. At this time, those include avoidant, obsessive-compulsive, and dependent personality disorders. The make-up of Cluster C would have to be rethought, as the figure shown below could no longer represent all of the disorders if depressive personality disorder were to be included. The relation shown in the Venn diagram has been accepted for years and would have to be rethought and redesigned if depressive personality disorder were to be added. Further studies are in progress looking into the comorbidity of Cluster C disorders and depressive personality disorder, as well as how these disorders interact with each other in patients diagnosed with multiple Cluster C disorders.

Passive–aggressive behavior

Passive–aggressive behavior, a personality trait, is passive, sometimes obstructionist resistance to following through with expectations in interpersonal or occupational situations. It is a personality trait marked by a pervasive pattern of negative attitudes and passive, usually disavowed resistance in interpersonal or occupational situations.

It can manifest itself as learned helplessness, procrastination, stubbornness, resentment, sullenness, or deliberate/repeated failure to accomplish requested tasks for which one is (often explicitly) responsible.
Signs and symptoms

The book Living with the Passive-Aggressive Man lists 11 responses that may help identify passive-aggressive behavior:

- Ambiguity or speaking cryptically: a means of creating a feeling of insecurity in others or of disguising one's own insecurities.
- Chronically being late and forgetting things: another way to exert control or to punish.
- Fear of competition
- Fear of dependency
- Fear of intimacy as a means to act out anger: The passive-aggressive often cannot trust. Because of this, they guard themselves against becoming intimately attached to someone.
- Making chaotic situations
- Making excuses for non-performance in work teams
- Obstructionism
- Procrastination
- Sulking
- Victimization response: instead of recognizing one's own weaknesses, tendency to blame others for own failures.

A passive-aggressive person may not display all of these behaviors, and may have other non-passive-aggressive traits.

Diagnostic criteria (DSM-IV Appendix B) and personality disorder

Passive-aggressive personality disorder was listed as an Axis II personality disorder in the DSM-III-R, but was moved in the DSM-IV to Appendix B ("Criteria Sets and Axes Provided for Further Study") because of controversy and the need for further research on how to also categorize the behaviors in a future edition. As an alternative, the diagnosis personality disorder not otherwise specified may be used instead.

The DSM-IV Appendix B definition is as follows:

A pervasive pattern of negativistic attitudes and passive resistance to demands for adequate performance, beginning by early adulthood and present in a variety of contexts, as indicted by four (or more) of the following:

- passively resists fulfilling routine social and occupational tasks
- complains of being misunderstood and unappreciated by others
- is sullen and argumentative
- unreasonably criticizes and scorns authority
- expresses envy and resentment toward those apparently more fortunate
- voices exaggerated and persistent complaints of personal misfortune
- alternates between hostile defiance and contrition
Does not occur exclusively during major depressive episodes and is not better accounted for by dysthmic disorder.

Diagnostic criteria (ICD-10)

The World Health Organization's ICD-10 lists passive-aggressive personality disorder under (F60.8) Other specific personality disorders.

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria.

Millon's subtypes

Theodore Millon identified four subtypes of negativist (passive-aggressive). Any individual negativist may exhibit none or one of the following:

- circuitous negativist – including dependent features
- abrasive negativist – including sadistic features
- discontented negativist – including depressive features
- vacillating negativist – including borderline features

Causes

Passive-aggressive disorder may stem from a specific childhood stimulus (e.g., alcohol/drug addicted parents) in an environment where it was not safe to express frustration or anger. Families in which the honest expression of feelings is forbidden tend to teach children to repress and deny their feelings and to use other channels to express their frustration.

Children who sugarcoat their hostility may fail to ever grow beyond such behavior. Never developing better coping strategies or skills for self-expression, they can become adults who, beneath a seductive veneer, harbor vindictive intent. Martin Kantor suggests three areas that contribute to passive-aggressive anger in individuals: conflicts about dependency, control, and competition.

Treatment

Kantor suggests a treatment approach using psychodynamic, supportive, cognitive, behavioral and interpersonal therapeutic methods. These methods apply to both the passive aggressive person and their target victim.

History

Passive aggressive behavior was first defined clinically by Colonel William Menninger during World War II in the context of men's reaction to military compliance. But
noncompliance is not indicative of true passive-aggressive behavior, which is the manifestation of emotions that have been repressed based on a self-imposed need for acceptance.

In the first version of the Diagnostic and Statistical Manual of Mental Disorders, DSM-I, in 1952, the passive-aggressive was defined in a narrow way, grouped together with the passive-dependent.

**Sadistic personality disorder**

Sadistic personality disorder is a diagnosis which only appeared in an appendix of the revised third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R). The current version of the DSM (DSM-IV) does not include it, so it is no longer considered a valid diagnostic category. As an alternative, the diagnosis Personality disorder not otherwise specified may be used instead. However, the disorder is still studied for research purposes.

**Causes (etiology)**

There appears to be a genetic component to the disorder.

**Diagnosis**

**Proposed DSM III-R criteria**

Sadistic personality disorder is:

A) A pervasive pattern of cruel, demeaning, and aggressive behavior, beginning by early adulthood, as indicated by the repeated occurrence of at least four of the following:

- Has used physical cruelty or violence for the purpose of establishing dominance in a relationship (not merely to achieve some noninterpersonal goal, such as striking someone in order to rob him/her).
- Humiliates or demeans people in the presence of others.
- Has treated or disciplined someone under his/her control unusually harshly.
- Is amused by, or takes pleasure in, the psychological or physical suffering of others (including animals).
- Has lied for the purpose of harming or inflicting pain on others (not merely to achieve some other goal).
- Gets other people to do what he/she wants by frightening them (through intimidation or even terror).
- Restricts the autonomy of people with whom he or she has a close relationship, e.g., will not let spouse leave the house unaccompanied or permit teenage daughter to attend social functions.
- Is fascinated by violence, weapons, injury, or torture.
B) The behavior in A has not been directed toward only one person (e.g., spouse, one child) and has not been solely for the purpose of sexual arousal (as in sexual sadism).

**Exclusion from DSM-IV**

This disorder was dropped from DSM-IV for two reasons:

- because of scientific concerns, such as the relatively low prevalence rate of the disorder in many settings
- for political reasons - sadistic personalities are most often male and it was felt that any such diagnosis might have the paradoxical effect of legally excusing cruel behaviour.

Sexual sadism that "causes clinically significant distress or impairment in social, occupational, or other important areas of functioning" is still in DSM-IV.

**Millon's subtypes**

Theodore Millon identified four subtypes of sadist. Any individual sadist may exhibit none, one or many of the following:

- explosive sadist - including borderline features
- tyrannical sadist - including negativistic (passive-aggressive) features
- enforcing sadist - including compulsive features
- spineless sadist - including avoidant features.

**Self-defeating personality disorder**

Self-defeating personality disorder (also known as masochistic personality disorder) is a personality disorder that was never formally admitted into the Diagnostic and Statistical Manual of Mental Disorders (DSM). It was discussed in an appendix of the manual’s revised third edition (DSM-III-R). As an alternative, the diagnosis Personality disorder not otherwise specified may be used instead. Some researchers and theorists continue to use its criteria. It has an official code number, 301.90.

**Diagnosis**

**Proposed DSM III-R**

Self-defeating personality disorder is:
A) A pervasive pattern of self-defeating behavior, beginning by early adulthood and present in a variety of contexts. The person may often avoid or undermine pleasurable experiences, be drawn to situations or relationships in which he or she will suffer, and prevent others from helping him, as indicated by at least five of the following:

- chooses people and situations that lead to disappointment, failure, or mistreatment even when better options are clearly available
- rejects or renders ineffective the attempts of others to help him or her
- following positive personal events (e.g., new achievement), responds with depression, guilt, or a behavior that produces pain (e.g., an accident)
- incites angry or rejecting responses from others and then feels hurt, defeated, or humiliated (e.g., makes fun of spouse in public, provoking an angry retort, then feels devastated)
- rejects opportunities for pleasure, or is reluctant to acknowledge enjoying himself or herself (despite having adequate social skills and the capacity for pleasure)
- fails to accomplish tasks crucial to his or her personal objectives despite demonstrated ability to do so, e.g., helps fellow students write papers, but is unable to write his or her own
- is uninterested in or rejects people who consistently treat him or her well, e.g., is unattracted to caring sexual partners
- engages in excessive self-sacrifice that is unsolicited by the intended recipients of the sacrifice

B) The behaviors in A do not occur exclusively in response to, or in anticipation of, being physically, sexually, or psychologically abused.

C) The behaviors in A do not occur only when the person is depressed.

Exclusion from DSM-IV

Historically, masochism has been associated with feminine submissiveness. This disorder became politically awkward when associated with domestic violence which is mostly caused by males. However a number of studies suggest that the disorder is common. In spite of its exclusion from DSM-IV, it continues to enjoy widespread currency amongst clinicians as a construct that explains a great many facets of human behaviour.

Sexual masochism that "causes clinically significant distress or impairment in social, occupational, or other important areas of functioning" is still in DSM-IV.

Millon's subtypes

Theodore Millon identified four subtypes of masochist. Any individual masochist may exhibit none or one of the following:

- self-undoing masochist - including avoidant features
- possessive masochist - including negativistic (passive-aggressive) features
- oppressed masochist - including depressive features
- virtuous masochist - including histrionic features.

**Substance use disorder**

Substance use disorders include substance abuse and substance dependence. In DSM-IV, the conditions are formally diagnosed as one or the other, but it has been proposed that DSM-5 combine the two into a single condition called "Substance-use disorder".

**Terminology and usage**

Although the term substance can refer to any physical matter, "substance abuse" has come to refer to the overindulgence in and dependence of a drug or other chemical leading to effects that are detrimental to the individual’s physical and mental health, or the welfare of others.

The disorder is characterized by a pattern of continued pathological use of a medication, non-medically indicated drug or toxin, which results in repeated adverse social consequences related to drug use, such as failure to meet work, family, or school obligations, interpersonal conflicts, or legal problems. There are on-going debates as to the exact distinctions between substance abuse and substance dependence, but current practice standard distinguishes between the two by defining substance dependence in terms of physiological and behavioral symptoms of substance use, and substance abuse in terms of the social consequences of substance use.

Substance abuse may lead to addiction or substance dependence. Medically, physiologic dependence requires the development of tolerance leading to withdrawal symptoms. Both abuse and dependence are distinct from addiction which involves a compulsion to continue using the substance despite the negative consequences, and may or may not involve chemical dependency. Dependence almost always implies abuse, but abuse frequently occurs without dependence, particularly when an individual first begins to abuse a substance. Dependence involves physiological processes while substance abuse reflects a complex interaction between the individual, the abused substance and society.

Substance abuse is sometimes used as a synonym for drug abuse, drug addiction, and chemical dependency, but actually refers to the use of substances in a manner outside sociocultural conventions. All use of controlled drugs and all use of other drugs in a manner not dictated by convention (e.g. according to physician's orders or societal norms) is abuse according to this definition, however there is no universally accepted definition of substance abuse.

The physical harm for twenty drugs was compared in an article in the Lancet (see diagram, above right). Physical harm was assigned a value from 0 to 3 for acute harm, chronic harm and intravenous harm. Shown is the mean physical harm. Not shown, but also evaluated, was the social harm.
Substance use may be better understood as occurring on a spectrum from beneficial to problematic use. This conceptualization moves away from the ill-defined binary antonyms of "use" vs. "abuse" (see diagram, lower right) towards a more nuanced, public health-based understanding of substance use.

**Mediators & Moderators**

When a predictor variable and an outcome variable have a significant relationship, which is, in turn, influenced by a third variable, the relationship is said to be mediated by the third variable. In this relationship the predictor variable influences the mediating variable in a causal manner. This mediating variable then leads to the outcome, creating the relationship between the predictor and outcome. It is only because of this mediating variable that a relationship between the predictor and outcome exists. Also, quasi-causal inferences may be drawn from mediated relationships. Mediation is best illustrated by the following model:

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X ----> Z ----> Y
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As demonstrated by the chart below, numerous studies have examined factors which mediate substance abuse or dependence. In these examples, the predictor variables lead to the mediator which in turn leads to the outcome, which is always substance abuse or dependence. For example, research has found that being raised in a single-parent home can lead to increased exposure to stress and that increased exposure to stress, not being raised in a single-parent home, leads to substance abuse or dependence. The following are some, but by no means all, of the possible mediators of substance abuse.

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>Mediator Variables</th>
<th>Outcome Variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single-parent Home</td>
<td>Exposure to Stress, Association w/ Deviant Peers</td>
<td>Substance Abuse or Dependence</td>
</tr>
<tr>
<td>Child Abuse/Neglect</td>
<td>PTSD symptoms, Stressful Life Events, Criminal Behavior</td>
<td>Substance Abuse or Dependence</td>
</tr>
<tr>
<td>Parental Substance Abuse</td>
<td>Physical/Sexual Abuse, Delinquency Status</td>
<td>Substance Abuse or Dependence</td>
</tr>
<tr>
<td>Witnessing Violence</td>
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When a variable indicates the conditions under which a specific effect occurs as well as displays how the direction or strength varies within a given relationship, the variable is said to moderate the relationship. Another explanation is that a moderator variable indicates that an effect only occurs under specific conditions. Unlike a relationship containing a mediator variable, the impact of the predictor variable on the outcome is
dependent on the value of the moderating variable. Also unlike a relationship involving mediation, no causal inferences can be drawn from a moderated relationship; relationships can only be described as correlated. However, moderated relationships do identify interaction effects between predictor and moderator variables. Moderation is best illustrated by the following model:

![Moderator Model](image)

As demonstrated by the chart below, numerous studies have examined factors which moderate substance abuse or dependence. In these examples, the moderator variable impacts the level to which the strength of the relationship varies between a given predictor variable and the outcome of substance abuse or dependence. For example, there is a significant relationship between psychobehavioral risk factors, such as tolerance of deviance, rebelliousness, achievement, perceived drug risk, familism, family church attendance and other factors, and substance abuse and dependence. That relationship is moderated by familism which means that the strength of the relationship is increased or decreased based on the level of familism present in a given individual.

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>Moderator Variables</th>
<th>Outcome Variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychobehavioral Risk</td>
<td>Familism, Family Church Attendance</td>
<td>Substance Abuse or Dependence</td>
</tr>
<tr>
<td>Victimization Effects</td>
<td>Race/Ethnicity Physical/Sexual Abuse</td>
<td>Substance Abuse or Dependence</td>
</tr>
<tr>
<td>Family History of Alcoholism</td>
<td>Gender</td>
<td>Substance Abuse or Dependence</td>
</tr>
</tbody>
</table>

Mediation and moderation research continues to inform the field’s knowledge and understanding of a pervasive and dangerous threat to public health, substance abuse and dependence. As the relationships between various predictor variables and the factors which influence them are more closely scrutinized, clinicians and researchers are provided with the necessary information to create more sophisticated and relevant methods of prevention and intervention. While these factors are important to the development of SUDs, there are plenty of other factors both known and unknown that influence the development of this disorder. As such, continued research is both necessary and invaluable.

**Additional Mediators and Moderators of Substance Abuse**

Mediators and Moderators Defined: Baron and Kenny (1986) define a moderator as, “a qualitative (e.g., sex, race, class) or quantitative (e.g., level of reward) variable that affects the direction and/or strength of the relation between and independent or predictor variable and a dependent or criterion variable” (p. 1174). Moderators may operate as
protective factors, decreasing the strength of the relationship between the predictor variable and the outcome. Conversely, moderators may heighten risk levels and strengthen the effects of the predictor on the outcome. In either instance, moderators do not explain why the connection exists, but rather affect the strength and direction of the relationship between the variables.

A mediator, as defined by Baron and Kenny (1986), “represents the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest” (p. 1173). Unlike moderators, mediators can explain the relationship between the predictor variable and outcome. Holmbeck (1997) elaborated on Baron and Kenny’s definition by adding, “the nature of the mediated relationship is such that the independent variable influences the mediator which, in turn, influences the outcome” (p. 600). Examples of mediators and moderators in empirical research:

Examples of mediators and moderators can be found in several empirical studies. For example, Pilgrim et al.’s hypothesized mediation model posited that school success and time spent with friends mediated the relationship between parental involvement and risk-taking behavior with substance use (2006). More specifically, the relationship between parental involvement and risk-taking behavior is explained via the interaction with third variables, school success and time spent with friends. In this example, increased parental involvement led to increased school success and decreased time with friends, both of which were associated with decreased drug use. Another example of mediation involved risk-taking behaviors. As risk-taking behaviors increased, school success decreased and time with friends increased, both of which were associated with increased drug use. A second example of a mediating variable is depression. In a study by Lo and Cheng (2007), depression was found to mediate the relationship between childhood maltreatment and subsequent substance abuse in adulthood. In other words, childhood physical abuse is associated with increased depression, which in turn, is associated with increased drug and alcohol use in young adulthood. More specifically, depression helps to explain how childhood abuse is related to subsequent substance abuse in young adulthood.

A third example of a mediating variable is an increase of externalizing symptoms. King and Chassin (2008) conducted research examining the relationship between stressful life events and drug dependence in young adulthood. Their findings identified problematic externalizing behavior on subsequent substance dependency. In other words, stressful life events are associated with externalizing symptoms, such as aggression or hostility, which can lead to peer alienation or acceptance by socially deviant peers, which could lead to increased drug use. The relationship between stressful life events and subsequent drug dependence however exists via the presence of the mediation effects of externalizing behaviors.

An example of a moderating variable is level of cognitive distortion. An individual with high levels of cognitive distortion might react adversely to potentially innocuous events, and may have increased difficulty reacting to them in an adaptive manner (Shoal & Giancola, 2005). In their study, Shoal and Giancola investigated the moderating effects of cognitive distortion on adolescent substance use. Individuals with low levels of cognitive distortion
may be more apt to choose more adaptive methods of coping with social problems, thereby potentially reducing the risk of drug use. Individuals with high levels of cognitive distortions, because of their increased misperceptions and misattributions, are at increased risk for social difficulties. Individuals may be more likely to react aggressively or inappropriately, potentially alienating themselves from their peers, thereby putting them at greater risk for delinquent behaviors, including substance use and abuse. In this study, social problems are a significant risk factor for drug use when moderated by high levels of cognitive distortions.

**Terminology**

In the United States, physical dependence, abuse of, and withdrawal from drugs and other substances is outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV TR). It does not use the word 'addiction' at all. It has instead a section about Substance dependence:

"Substance dependence When an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. This, along with Substance Abuse are considered Substance Use Disorders..."

Terminology has become quite complicated in the field. Pharmacologists continue to speak of addiction from a physiologic standpoint (some call this a physical dependence); psychiatrists refer to the disease state as psychological dependence; most other physicians refer to the disease as addiction. The field of psychiatry is now considering, as they move from DSM-IV to DSM-V, transitioning from "substance dependence" to "addiction" as terminology for the disease state.

Addiction is now narrowly defined as "uncontrolled, compulsive use"; if there is no harm being suffered by, or damage done to, the patient or another party, then clinically it may be considered compulsive, but to the definition of some it is not categorized as 'addiction'. In practice, the two kinds of addiction are not always easy to distinguish. Addictions often have both physical and psychological components.

There is also a lesser known situation called pseudo-addiction. A patient will exhibit drug-seeking behavior reminiscent of psychological addiction, but they tend to have genuine pain or other symptoms that have been under-treated. Unlike true psychological addiction, these behaviors tend to stop when the pain is adequately treated. Physical and psychological dependency

The medical community now makes a careful theoretical distinction between physical dependence (characterized by symptoms of withdrawal) and psychological dependence (or simply addiction).
The DSM definition of addiction can be boiled down to compulsive use of a substance (or engagement in an activity) despite ongoing negative consequences—this is also a summary of what used to be called "psychological dependency." Physical dependence, on the other hand, is simply needing a substance to function. Humans are all physically dependent on oxygen, food and water. A drug can cause physical dependence and not addiction (for example, some blood pressure medications, which can produce fatal withdrawal symptoms if not tapered) and can cause addiction without physical dependence (the withdrawal symptoms associated with cocaine are all psychological, there is no associated vomiting or diarrhea as there is with opiate withdrawal).

**Physical dependency**

Physical dependence on a substance is defined by the appearance of characteristic withdrawal symptoms when the substance is suddenly discontinued. Opiates, benzodiazepines, barbiturates, alcohol and nicotine induce physical dependence. On the other hand, some categories of substances share this property and are still not considered addictive: cortisone, beta blockers and most antidepressants are examples. So, while physical dependency can be a major factor in the psychology of addiction and most often becomes a primary motivator in the continuation of an addiction, the initial primary attribution of an addictive substance is usually its ability to induce pleasure, although with continued use the goal is not so much to induce pleasure as it is to relieve the anxiety caused by the absence of a given addictive substance, causing it to become used compulsively.

Some substances induce physical dependence or physiological tolerance - but not addiction — for example many laxatives, which are not psychoactive; nasal decongestants, which can cause rebound congestion if used for more than a few days in a row; and some antidepressants, most notably venlafaxine, paroxetine and sertraline, as they have quite short half-lives, so stopping them abruptly causes a more rapid change in the neurotransmitter balance in the brain than many other antidepressants. Many non-addictive prescription drugs should not be suddenly stopped, so a doctor should be consulted before abruptly discontinuing them.

The speed with which a given individual becomes addicted to various substances varies with the substance, the frequency of use, the means of ingestion, the intensity of pleasure or euphoria, and the individual’s genetic and psychological susceptibility. Some people may exhibit alcoholic tendencies from the moment of first intoxication, while most people can drink socially without ever becoming addicted. Opioid dependent individuals have different responses to even low doses of opioids than the majority of people, although this may be due to a variety of other factors, as opioid use heavily stimulates pleasure-inducing neurotransmitters in the brain. Nonetheless, because of these variations, in addition to the adoption and twin studies that have been well replicated, much of the medical community is satisfied that addiction is in part genetically moderated. That is, one’s genetic makeup may regulate how susceptible one is to a substance and how easily one may become psychologically attached to a pleasurable routine.
Eating disorders are complicated pathological mental illnesses and thus are not the same as addictions described in this article. Eating disorders, which some argue are not addictions at all, are driven by a multitude of factors, most of which are highly different than the factors behind addictions described in this article. It has been reported, however, that patients with eating disorders can successfully be treated with the same non-pharmacological protocols used in patients with chemical addiction disorders. Gambling is another potentially addictive behavior with some biological overlap. Conversely gambling urges have emerged with the administration of Mirapex (pramipexole), a dopamine agonist.

The obsolete term physical addiction is deprecated, because of its connotations. In modern pain management with opioids physical dependence is nearly universal. While opiates are essential in the treatment of acute pain, the benefit of this class of medication in chronic pain is not well proven. Clearly, there are those who would not function well without opiate treatment; on the other hand, many states are noting significant increases in non-intentional deaths related to opiate use. High-quality, long-term studies are needed to better delineate the risks and benefits of chronic opiate use.

**Psychological dependency**

In the now outdated conceptualization of the problem, psychological dependency leads to psychological withdrawal symptoms (such as cravings, irritability, insomnia, depression, anorexia, etc). Addiction can in theory be derived from any rewarding behaviour, and is believed to be strongly associated with the dopaminergic system of the brain’s reward system (as in the case of cocaine and amphetamines). Some claim that it is a habitual means to avoid undesired activity, but typically it is only so to a clinical level in individuals who have emotional, social, or psychological dysfunctions (psychological addiction is defined as such), replacing normal positive stimuli not otherwise attained.

A person who is physically dependent, but not psychologically dependent can have their dose slowly dropped until they are no longer physically dependent. However, if that person is psychologically dependent, they are still at serious risk for relapse into abuse and subsequent physical dependence.

Psychological dependence does not have to be limited only to substances; even activities and behavioural patterns can be considered addictions, if they become uncontrollable, e.g. problem gambling, Internet addiction, computer addiction, sexual addiction / pornography addiction, overeating, self-injury, compulsive buying, or work addiction.

**Management**

Early editions of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders (DSM) described addiction as a physical dependency to a substance that resulted in withdrawal symptoms in its absence. Recent editions, including DSM-IV, have moved toward a diagnostic instrument that classifies such conditions as dependency, rather than addiction.
Addiction severity index

Some medical systems, including those of at least 15 states of the United States, refer to an Addiction Severity Index to assess the severity of problems related to substance use. The index assesses problems in six areas: medical, employment/support, alcohol and other drug use, legal, family/social, and psychiatric.

Detoxification

Early treatment of acute withdrawal often includes medical detoxification, which can include doses of anxiolytics or narcotics to reduce symptoms of withdrawal. An experimental drug, ibogaine, is also proposed to treat withdrawal and craving.

Neurofeedback therapy has shown statistically significant improvements in numerous researches conducted on alcoholic as well as mixed substance abuse population. In chronic opiate addiction, a surrogate drug such as methadone is sometimes offered as a form of opiate replacement therapy. But treatment approaches universal focus on the individual’s ultimate choice to pursue an alternate course of action.

Tailoring treatment

Therapists often classify patients with chemical dependencies as either interested or not interested in changing.

Treatments usually involve planning for specific ways to avoid the addictive stimulus, and therapeutic interventions intended to help a client learn healthier ways to find satisfaction. Clinical leaders in recent years have attempted to tailor intervention approaches to specific influences that affect addictive behavior, using therapeutic interviews in an effort to discover factors that led a person to embrace unhealthy, addictive sources of pleasure or relief from pain.

Treatment modality matrix

<table>
<thead>
<tr>
<th>Behavioral pattern</th>
<th>Intervention</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low self-esteem, anxiety, verbal hostility</td>
<td>Relationship therapy, client centered approach</td>
<td>Increase self esteem, reduce hostility and anxiety</td>
</tr>
<tr>
<td>Defective personal constructs, ignorance of interpersonal means</td>
<td>Cognitive restructuring including directive and group therapies</td>
<td>Insight</td>
</tr>
<tr>
<td>Focal anxiety such as fear of crowds</td>
<td>Desensitization</td>
<td>Change response to same cue</td>
</tr>
<tr>
<td>Undesirable behaviors, lacking appropriate behaviors</td>
<td>Aversive conditioning, operant conditioning, counter conditioning</td>
<td>Eliminate or replace behavior</td>
</tr>
<tr>
<td>Lack of information</td>
<td>Provide information</td>
<td>Have client act on information</td>
</tr>
<tr>
<td>---------------------</td>
<td>---------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Difficult social circumstances</td>
<td>Organizational intervention, environmental manipulation, family counseling</td>
<td>Remove cause of social difficulty</td>
</tr>
<tr>
<td>Poor social performance, rigid interpersonal behavior</td>
<td>Sensitivity training, communication training, group therapy</td>
<td>Increase interpersonal repertoire, desensitization to group functioning</td>
</tr>
<tr>
<td>Grossly bizarre behavior</td>
<td>Medical referral</td>
<td>Protect from society, prepare for further treatment</td>
</tr>
</tbody>
</table>

Adapted from: Essentials of Clinical Dependency Counseling, Aspen Publishers

From the applied behavior analysis literature and the behavioral psychology literature, several evidenced-based intervention programs have emerged (1) behavioral marital therapy (2) community reinforcement approach (3) cue exposure therapy and (4) contingency management strategies. In addition, the same author suggests that social skills training adjunctive to inpatient treatment of alcohol dependence is probably efficacious.

Causes

Several explanations (or "models") have been presented to explain addiction. These divide, more or less, into the models which stress biological or genetic causes for addiction, and those which stress social or purely psychological causes. Of course there are also many models which attempt to see addiction as both a physiological and a psycho-social phenomenon.

Psycho-social

The free-will model or "life-process model" proposed by Thomas Szasz and later refined by Jeffrey Schaler questions the very concept of "addiction". Free-will model theorists argue that addiction cannot be a disease, because drug-taking is a behavior, and all behaviors are choices. Szasz views addiction as a metaphor, and that the only reason to make the distinction between habit and addiction "is to persecute somebody." Free-will model theorists believe that individuals are capable of deliberate action in pursuit of chosen goals, and that physiology alone can never determine whether a person will take a drug, or how often they will take it. Although the free-will model of addiction has received much research support, it is opposed by groups like the American Psychiatric Association and the National Institute of Mental Health.

The pleasure model proposed by professor Nils Bejerot. Addiction "is an emotional fixation (sentiment) acquired through learning, which intermittently or continually expresses itself in purposeful, stereotyped behavior with the character and force of a natural drive, aiming at a specific pleasure or the avoidance of a specific discomfort.""
pleasure mechanism may be stimulated in a number of ways and give rise to a strong fixation on repetitive behavior. Stimulation with drugs is only one of many ways, but one of the simplest, strongest, and often also the most destructive" "If the pleasure stimulation becomes so strong that it captivates an individual with the compulsion and force characteristic of natural drives, then there exists...an addiction" The pleasure model is used as one of the reason for zero tolerance for use of illicit drugs.

The experiential model devised by Stanton Peele argues that addictions occur with regard to experiences generated by various involvements, whether drug-induced or not. This model is in opposition to the disease, genetic, and neurobiological approaches. Among other things, it proposes that addiction is both more temporary or situational than the disease model claims, and is often outgrown through natural processes.

The opponent-process model generated by Richard Soloman states that for every psychological event A will be followed by its opposite psychological event B. For example, the pleasure one experiences from heroin is followed by an opponent process of withdrawal, or the terror of jumping out of an airplane is rewarded with intense pleasure when the parachute opens. This model is related to the opponent process color theory. If you look at the color red then quickly look at a gray area you will see green. There are many examples of opponent processes in the nervous system including taste, motor movement, touch, vision, and hearing. Opponent-processes occurring at the sensory level may translate "down-stream" into addictive or habit-forming behavior.

The allostatic (stability through change) model generated by George Koob and Michel LeMoal is a modification of the opponent process theory where continued use of a drug leads to a spiralling of uncontrolled use, negative emotional states and withdrawal and a shift into use to new allostatic set point which is lower than that maintained before use of the drug.

The cultural model recognizes that the influence of culture is a strong determinant of whether or not individuals fall prey to certain addictions. For example, alcoholism is rare among Saudi Arabians, where obtaining alcohol is difficult and using alcohol is prohibited. In North America, on the other hand, the incidence of gambling addictions soared in the last two decades of the 20th century, mirroring the growth of the gaming industry. Half of all patients diagnosed as alcoholic are born into families where alcohol is used heavily, suggesting that familiar influence, genetic factors, or more likely both, play a role in the development of addiction. What also needs to be noted is that when people don't gain a sense of moderation through their development they can be just as likely, if not more, to abuse substances than people born into alcoholic families.

The moral model states that addictions are the result of human weakness, and are defects of character. Those who advance this model do not accept that there is any biological basis for addiction. They often have scant sympathy for people with serious addictions, believing either that a person with greater moral strength could have the force of will to break an addiction, or that the addict demonstrated a great moral failure in the first place by starting the addiction. The moral model is widely applied to dependency on illegal substances,
perhaps purely for social or political reasons, but is no longer widely considered to have any therapeutic value. Elements of the moral model, especially a focus on individual choices, have found enduring roles in other approaches to the treatment of dependencies.

Similarly, the **rational addiction model** hypothesizes that addictions (to heroin, tobacco, television, etc.) can be usefully modeled as specific kinds of rational, forward-looking, optimal consumption plans. In other words, addiction is perceived as a rational response to individual and/or environmental factors.

The **chemical model** : Nearly all drugs, directly or indirectly, target the brain’s reward system by flooding the circuit with dopamine. As a person continues to overstimulating the “reward circuit”, the brain adapts to the overwhelming surges in dopamine by producing less of the hormones or by reducing the number of receptors in the reward circuit. As a result, the chemical’s impact on the reward circuit is lessened, reducing the abuser’s ability to enjoy the things that previously brought pleasure. This decrease compels those addicted to dopamine to increase the drug consumption in order to attempt to bring their "feel-good" hormone level back to normal —an effect known as tolerance. Development of dopamine tolerance can eventually lead to profound changes in neurons and brain circuits, with the potential to severely compromise the long-term health of the brain. Modern antipsychotics are designed to block dopamine function. Unfortunately, this blocking can also cause relapses in depression, and can increase addictive behaviors.

Finally, the **blended model** attempts to consider elements of all other models in developing a therapeutic approach to dependency. It holds that the mechanism of dependency is different for different individuals, and that each case must be considered on its own merits.

**Physiological**

The **disease model of addiction** holds that addiction is a disease, coming about as a result of either the impairment of neurochemical or behavioral processes, or of some combination of the two. Within this model, addictive disease is treated by specialists in Addiction Medicine. Within the clinical field, the American Medical Association, National Association of Social Workers, and American Psychological Association all have policies which are predicated on the theory that addictive processes represent a disease state. Most treatment approaches, as well, are based on the idea that dependencies are behavioral dysfunctions, and, therefore, contain, at least to some extent, elements of physical or mental disease. Organizations such as the American Society of Addiction Medicine believe the research-based evidence for addiction’s status as a disease is overwhelming.

The **genetic model** posits a genetic predisposition to certain behaviors. It is frequently noted that certain addictions "run in the family," and while researchers continue to explore the extent of genetic influence, many researchers argue that there is strong evidence that genetic predisposition is often a factor in dependency.
The development of addiction is thought to involve a simultaneous process of 1) increased focus on and engagement in a particular behavior and 2) the attenuation or "shutting down" of other behaviors. For example, under certain experimental circumstances such as social deprivation and boredom, animals allowed the unlimited ability to self-administer certain psychoactive drugs will show such a strong preference that they will forgo food, sleep, and sex for continued access. The neuro-anatomical correlate of this is that the brain regions involved in driving goal-directed behavior grow increasingly selective for particular motivating stimuli and rewards, to the point that the brain regions involved in the inhibition of behavior can no longer effectively send "stop" signals. A good analogy is to imagine flooring the gas pedal in a car with very bad brakes. In this case, the limbic system is thought to be the major "driving force" and the orbitofrontal cortex is the substrate of the top-down inhibition.

A specific portion of the limbic circuit known as the mesolimbic dopaminergic system is hypothesized to play an important role in translation of motivation to motor behavior- and reward-related learning in particular. It is typically defined as the ventral tegmental area (VTA), the nucleus accumbens, and the bundle of dopamine-containing fibers that are connecting them. This system is commonly implicated in the seeking out and consumption of rewarding stimuli or events, such as sweet-tasting foods or sexual interaction. However, its importance to addiction research goes beyond its role in "natural" motivation: while the specific site or mechanism of action may differ, all known drugs of abuse have the common effect in that they elevate the level of dopamine in the nucleus accumbens. This may happen directly, such as through blockade of the dopamine re-uptake mechanism (see cocaine). It may also happen indirectly, such as through stimulation of the dopamine-containing neurons of the VTA that synapse onto neurons in the accumbens (see opiates). The euphoric effects of drugs of abuse are thought to be a direct result of the acute increase in accumbal dopamine.

The human body has a natural tendency to maintain homeostasis, and the central nervous system is no exception. Chronic elevation of dopamine will result in a decrease in the number of dopamine receptors available in a process known as downregulation. The decreased number of receptors changes the permeability of the cell membrane located post-synaptically, such that the post-synaptic neuron is less excitable- i.e.: less able to respond to chemical signaling with an electrical impulse, or action potential. It is hypothesized that this dulling of the responsiveness of the brain's reward pathways contributes to the inability to feel pleasure, known as anhedonia, often observed in addicts. The increased requirement for dopamine to maintain the same electrical activity is the basis of both physiological tolerance and withdrawal associated with addiction.

Downregulation can be classically conditioned. If a behavior consistently occurs in the same environment or contingently with a particular cue, the brain will adjust to the presence of the conditioned cues by decreasing the number of available receptors in the absence of the behavior. It is thought that many drug overdoses are not the result of a user taking a higher dose than is typical, but rather that the user is administering the same dose in a new environment.
In cases of physical dependency on depressants of the central nervous system such as opioids, barbiturates, or alcohol, the absence of the substance can lead to symptoms of severe physical discomfort. Withdrawal from alcohol or sedatives such as barbiturates or benzodiazepines (valium-family) can result in seizures and even death. By contrast, withdrawal from opioids, which can be extremely uncomfortable, is rarely if ever life-threatening. In cases of dependence and withdrawal, the body has become so dependent on high concentrations of the particular chemical that it has stopped producing its own natural versions (endogenous ligands) and instead produces opposing chemicals. When the addictive substance is withdrawn, the effects of the opposing chemicals can become overwhelming. For example, chronic use of sedatives (alcohol, barbiturates, or benzodiazepines) results in higher chronic levels of stimulating neurotransmitters such as glutamate. Very high levels of glutamate kill nerve cells, a phenomenon called excitatory neurotoxicity.

Epidemiology

Addiction and drug control legislation

Most countries have legislation which brings various drugs and drug-like substances under the control of licensing systems. Typically this legislation covers any or all of the opiates, amphetamines, cannabinoids, cocaine, barbiturates, hallucinogens (tryptamines, LSD, phencyclidine, and psilocybin) and a variety of more modern synthetic drugs, and unlicensed production, supply or possession may be a criminal offense.

Usually, however, drug classification under such legislation is not related simply to addictiveness. The substances covered often have very different addictive properties. Some are highly prone to cause physical dependency, whilst others rarely cause any form of compulsive need whatsoever.

Also, although the legislation may be justifiable on moral grounds to some, it can make addiction or dependency a much more serious issue for the individual. Reliable supplies of a drug become difficult to secure as illegally produced substances may have contaminants. Withdrawal from the substances or associated contaminants can cause additional health issues and the individual becomes vulnerable to both criminal abuse and legal punishment. Criminal elements that can be involved in the profitable trade of such substances can also cause physical harm to users.

Opposition to common views

Thomas Szasz denies that addiction is a psychiatric problem. In many of his works, he argues that addiction is a choice, and that a drug addict is one who simply prefers a socially taboo substance rather than, say, a low risk lifestyle. In Our Right to Drugs, Szasz cites the biography of Malcolm X to corroborate his economic views towards addiction: Malcolm claimed that quitting cigarettes was harder than shaking his heroin addiction. Szasz postulates that humans always have a choice, and it is foolish to call someone an ‘addict’
just because they prefer a drug induced euphoria to a more popular and socially welcome lifestyle.

Professor John Booth Davies at the University of Strathclyde has argued in his book The Myth of Addiction that 'people take drugs because they want to and because it makes sense for them to do so given the choices available' as opposed to the view that 'they are compelled to by the pharmacology of the drugs they take'. He uses an adaptation of attribution theory (what he calls the theory of functional attributions) to argue that the statement 'I am addicted to drugs' is functional, rather than veridical. Stanton Peele has put forward similar views.

Experimentally, Bruce K. Alexander used the classic experiment of Rat Park to show that 'addicted' behaviour in rats only occurred when the rats had no other options. When other options and behavioural opportunities were put in place, the rats soon showed far more complex behaviours.

**Substance abuse**

Substance abuse, also known as drug abuse, refers to a maladaptive pattern of use of a substance that is not considered dependent. The term "drug abuse" does not exclude dependency, but is otherwise used in a similar manner in nonmedical contexts. The terms have a huge range of definitions related to taking a psychoactive drug or performance enhancing drug for a non-therapeutic or non-medical effect. All of these definitions imply a negative judgment of the drug use in question (compare with the term responsible drug use for alternative views). Some of the drugs most often associated with this term include alcohol, amphetamines, barbiturates, benzodiazepines (particularly temazepam, nimetazepam, and flunitrazepam), cocaine, methaqualone, and opioids. Use of these drugs may lead to criminal penalty in addition to possible physical, social, and psychological harm, both strongly depending on local jurisdiction. Other definitions of drug abuse fall into four main categories: public health definitions, mass communication and vernacular usage, medical definitions, and political and criminal justice definitions.

Substance abuse is a form of substance-related disorder.

**Classification**

**Public health definitions**

Source: A Public Health Approach to Drug Control in Canada, Health Officers Council of British Columbia, 2005

Public health practitioners have attempted to look at drug abuse from a broader perspective than the individual, emphasizing the role of society, culture and availability. Rather than accepting the loaded terms alcohol or drug "abuse," many public health
professionals have adopted phrases such as "substance and alcohol type problems" or "harmful/problematic use" of drugs.

The Health Officers Council of British Columbia — in their 2005 policy discussion paper, A Public Health Approach to Drug Control in Canada — has adopted a public health model of psychoactive substance use that challenges the simplistic black-and-white construction of the binary (or complementary) antonyms "use" vs. "abuse". This model explicitly recognizes a spectrum of use, ranging from beneficial use to chronic dependence (see diagram to the right).

Medical definitions

In the modern medical profession, the two most used diagnostic tools in the world, the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM) and the World Health Organization's International Statistical Classification of Diseases and Related Health Problems (ICD), no longer recognize 'drug abuse' as a current medical diagnosis. Instead, DSM has adopted substance abuse as a blanket term to include drug abuse and other things. ICD refrains from using either "substance abuse" or "drug abuse", instead using the term "harmful use" to cover physical or psychological harm to the user from use. Physical dependence, abuse of, and withdrawal from drugs and other miscellaneous substances is outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR). Its section Substance dependence begins with:

"Substance dependence When an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. These, along with Substance Abuse are considered Substance Use Disorders...."

However, other definitions differ; they may entail psychological or physical dependence, and may focus on treatment and prevention in terms of the social consequences of substance uses.

Drug misuse

Drug misuse is a term used commonly for prescription medications with clinical efficacy but abuse potential and known adverse effects linked to improper use, such as psychiatric medications with sedative, anxiolytic, analgesic, or stimulant properties. Prescription misuse has been variably and inconsistently defined based on drug prescription status, the uses that occur without a prescription, intentional use to achieve intoxicating effects, route of administration, co-ingestion with alcohol, and the presence or absence of abuse or dependence symptoms. Tolerance relates to the pharmacological property of substances in which chronic use leads to a change in the central nervous system, meaning that more of the substance is needed in order to produce desired effects. Stopping or reducing the use of this substance would cause withdrawal symptoms to occur.
As a value judgment

Harm Caused by Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Harm to others</th>
<th>Harm to users</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>10</td>
<td>70</td>
</tr>
<tr>
<td>Heroin</td>
<td>0</td>
<td>50</td>
</tr>
<tr>
<td>Crack Cocaine</td>
<td>20</td>
<td>40</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>30</td>
<td>30</td>
</tr>
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<td>Cocaine</td>
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</tr>
<tr>
<td>Tobacco</td>
<td>50</td>
<td>10</td>
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</table>

*With a maximum possible harm rating of 100

Adapted from "Drug harms in the UK: a multi-criteria decision analysis", by David Nutt, Leslie King and Lawrence Phillips, on behalf of the Independent Scientific Committee on Drugs. The Lancet.

Legal drugs are not necessarily safer. A study in 2010 asked drug-harm experts to rank various illegal and legal drugs. Alcohol was found to be the most dangerous by far.

Philip Jenkins points out that there are two issues with the term "drug abuse". First, what constitutes a "drug" is debatable. For instance, GHB, a naturally occurring substance in the central nervous system is considered a drug, and is illegal in many countries, while nicotine is not officially considered a drug in most countries. Second, the word "abuse" implies a recognized standard of use for any substance. Drinking an occasional glass of wine is considered acceptable in many Western countries, while drinking several bottles is seen as an abuse. Strict temperance advocates, which may or may not be religiously motivated, would see drinking even one glass as an abuse, and some groups even condemn caffeine use in any quantity. Similarly, adopting the view that any (recreational) use of marijuana or...
amphetamines constitutes drug abuse implies that we have already decided that substance is harmful even in minute quantities.

**Signs and symptoms**

Depending on the actual compound, drug abuse including alcohol may lead to health problems, social problems, morbidity, injuries, unprotected sex, violence, deaths, motor vehicle accidents, homicides, suicides, physical dependence or psychological addiction.

There is a high rate of suicide in alcoholics and other drug abusers. The reasons believed to cause the increased risk of suicide include the long-term abuse of alcohol and other drugs causing physiological distortion of brain chemistry as well as the social isolation. Another factor is the acute intoxicating effects of the drugs may make suicide more likely to occur. Suicide is also very common in adolescent alcohol abusers, with 1 in 4 suicides in adolescents being related to alcohol abuse. In the USA approximately 30 percent of suicides are related to alcohol abuse. Alcohol abuse is also associated with increased risks of committing criminal offences including child abuse, domestic violence, rapes, burglaries and assaults.

Drug abuse, including alcohol and prescription drugs can induce symptomatology which resembles mental illness. This can occur both in the intoxicated state and also during the withdrawal state. In some cases these substance induced psychiatric disorders can persist long after detoxification, such as prolonged psychosis or depression after amphetamine or cocaine abuse. A protracted withdrawal syndrome can also occur with symptoms persisting for months after cessation of use. Benzodiazepines are the most notable drug for inducing prolonged withdrawal effects with symptoms sometimes persisting for years after cessation of use. Abuse of hallucinogens can trigger delusional and other psychotic phenomena long after cessation of use and cannabis may trigger panic attacks during intoxication and with use it may cause a state similar to dysthymia. Severe anxiety and depression are commonly induced by sustained alcohol abuse which in most cases abates with prolonged abstinence. Even moderate alcohol sustained use may increase anxiety and depression levels in some individuals. In most cases these drug induced psychiatric disorders fade away with prolonged abstinence.

Drug abuse makes central nervous system (CNS) effects, which produce changes in mood, levels of awareness or perceptions and sensations. Most of these drugs also alter systems other than the CNS. Some of these are often thought of as being abused. Some drugs appear to be more likely to lead to uncontrolled use than others.

Traditionally, new pharmacotherapies are quickly adopted in primary care settings, however; drugs for substance abuse treatment have faced many barriers. Naltrexone, a drug originally marketed under the name "ReVia," and now marketed in intramuscular formulation as "Vivitrol" or in oral formulation as a generic, is a medication approved for the treatment of alcohol dependence. This drug has reached very few patients. This may be due to a number of factors, including resistance by Addiction Medicine specialists and lack of resources.
The ability to recognize the signs of drug use or the symptoms of drug use in family members by parents and spouses has been affected significantly by the emergence of home drug test technology which helps identify recent use of common street and prescription drugs with near lab quality accuracy.

Epidemiology

The initiation of drug and alcohol use is most likely to occur during adolescence, and some experimentation with substances by older adolescents is common. For example, results from Monitoring the Future (2008), a nationwide study on rates of substance use, show that 47% of 12th graders report having used an illicit drug at some point in their lives. In 2009 in the United States about 21% of high school students have taken prescription drugs without a prescription. And earlier in 2002, the World health Organization estimated that around 140 million people were alcohol dependent and another 400 million suffered alcohol-related problems. Thankfully, the large majority of adolescents will phase out of drug use before it becomes problematic. Thus, although rates of overall use are high, the percentage of adolescents who meet criteria for substance abuse is significantly lower (close to 5%). According to BBC, "Worldwide, the UN estimates there are more than 50 million regular users of morphine diacetate (heroin), cocaine and synthetic drugs."

Total recorded alcohol per capita consumption (15+), in liters of pure alcohol

History

APA, AMA, and NCDA

In 1932, the American Psychiatric Association created a definition that used legality, social acceptability, and cultural familiarity as qualifying factors:

...as a general rule, we reserve the term drug abuse to apply to the illegal, nonmedical use of a limited number of substances, most of them drugs, which have properties of altering the mental state in ways that are considered by social norms and defined by statute to be inappropriate, undesirable, harmful, threatening, or, at minimum, culture-alien."

In 1966, the American Medical Association's Committee on Alcoholism and Addiction defined abuse of stimulants (amphetamines, primarily) in terms of 'medical supervision':

...'use' refers to the proper place of stimulants in medical practice; 'misuse' applies to the physician's role in initiating a potentially dangerous course of therapy; and 'abuse' refers to self-administration of these drugs without medical supervision and particularly in large doses that may lead to psychological dependency, tolerance and abnormal behavior.

In 1973 the National Commission on Marijuana and Drug Abuse stated:
...drug abuse may refer to any type of drug or chemical without regard to its pharmacologic actions. It is an eclectic concept having only one uniform connotation: societal disapproval. ... The Commission believes that the term drug abuse must be deleted from official pronouncements and public policy dialogue. The term has no functional utility and has become no more than an arbitrary codeword for that drug use which is presently considered wrong.

**DSM**

In the first edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (published in 1952) grouped alcohol and drug abuse under Sociopathic Personality Disturbances, which were thought to be symptoms of deeper psychological disorders or moral weakness.

The third edition, published in 1980, was the first to recognize substance abuse (including drug abuse) and substance dependence as conditions separate from substance abuse alone, bringing in social and cultural factors. The definition of dependence emphasised tolerance to drugs, and withdrawal from them as key components to diagnosis, whereas abuse was defined as "problematic use with social or occupational impairment" but without withdrawal or tolerance.

In 1987 the DSM-III-R category "psychoactive substance abuse," which includes former concepts of drug abuse is defined as "a maladaptive pattern of use indicated by...continued use despite knowledge of having a persistent or recurrent social, occupational, psychological or physical problem that is caused or exacerbated by the use (or by) recurrent use in situations in which it is physically hazardous." It is a residual category, with dependence taking precedence when applicable. It was the first definition to give equal weight to behavioural and physiological factors in diagnosis.

By 1988, the DSM-IV defines substance dependence as "a syndrome involving compulsive use, with or without tolerance and withdrawal"; whereas substance abuse is "problematic use without compulsive use, significant tolerance, or withdrawal." Substance abuse can be harmful to your health and may even be deadly in certain scenarios.

By 1994, The fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) issued by the American Psychiatric Association, the DSM-IV-TR, defines substance dependence as "when an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed." followed by criteria for the diagnose

**DSM-IV-TR defines substance abuse as:**

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following, occurring within a 12-month period:
Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences or poor work performance related to substance use; substance-related absences, suspensions or expulsions from school; neglect of children or household)

Recurrent substance use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use)

Recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct)

Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication, physical fights)

B. The symptoms have never met the criteria for Substance Dependence for this class of substance.

The fifth edition of the DSM (DSM-5), planned for release in 2013, is likely to have this terminology revisited yet again. Under consideration is a transition from the abuse/dependence terminology. At the moment, abuse is seen as an early form or less hazardous form of the disease characterized with the dependence criteria. However, the APA's 'dependence' term, as noted above, does not mean that physiologic dependence is present but rather means that a disease state is present, one that most would likely refer to as an addicted state. Many involved recognize that the terminology has often led to confusion, both within the medical community and with the general public. The American Psychiatric Association requests input as to how the terminology of this illness should be altered as it moves forward with DSM-5 discussion.

Society and culture

Legal approaches

Most governments have designed legislation to criminalize certain types of drug use. These drugs are often called "illegal drugs" but generally what is illegal is their unlicensed production, distribution, and possession. These drugs are also called "controlled substances". Even for simple possession, legal punishment can be quite severe (including the death penalty in some countries). Laws vary across countries, and even within them, and have fluctuated widely throughout history.

Attempts by government-sponsored drug control policy to interdict drug supply and eliminate drug abuse have been largely unsuccessful. In spite of the huge efforts by the U.S., drug supply and purity has reached an all time high, with the vast majority of resources spent on interdiction and law enforcement instead of public health. In the United States, the number of nonviolent drug offenders in prison exceeds by 100,000 the total incarcerated population in the EU, despite the fact that the EU has 100 million more citizens.

Compiled by Amit Shekhar Email: numerons@gmail.com Contact: +91-9560344245
Despite drug legislation (or perhaps because of it), large, organized criminal drug cartels operate worldwide. Advocates of decriminalization argue that drug prohibition makes drug dealing a lucrative business, leading to much of the associated criminal activity.

Cost

The UK Home Office estimated that the social and economic cost of drug abuse to the UK economy in terms of crime, absenteeism and sickness is in excess of £20 billion a year.

However, it does not estimate what portion of those crimes are unintended consequences of drug prohibition (crimes to sustain expensive drug consumption, risky production and dangerous distribution), nor what is the cost of enforcement. Those aspects are necessary for a full analysis of the economics of prohibition.

The Home Office has a recent history of taking a hard line on controlled drugs, including those with no known fatalities and even medical benefits, in direct opposition to the scientific community.

Treatment

Treatment for binge drinking and other forms of substance abuse is critical for many around the world. Often a formal intervention is necessary to convince the substance abuser to submit to any form of treatment. Behavioral interventions and medications exist that have helped many people reduce, or discontinue, their substance abuse. From the applied behavior analysis literature, the behavioral psychology literature, and from randomized clinical trials, several evidenced based interventions have emerged:

- Behavioral Marital Therapy
- Motivational Interviewing
- Community Reinforcement Approach
- Exposure therapy
- Contingency Management
- Pharmacological therapy

A number of medications have been approved for the treatment of substance abuse. These include replacement therapies such as buprenorphine and methadone as well as antagonist medications like disulfiram and naltrexone in either short acting, or the newer long acting form (under the brand name Vivitrol). Several other medications, often ones originally used in other contexts, have also been shown to be effective including bupropion (Zyban or Wellbutrin), Modafinil (Provigil) and more.

In children and adolescents, cognitive behavioral therapy (CBT) and family therapy currently have the most research evidence for the treatment of substance abuse problems. These treatments can be administered in a variety of different formats, each of which has varying levels of research support.
It has been suggested that social skills training adjunctive to inpatient treatment of alcohol dependence is probably efficacious.

**Substance dependence**

The section about substance dependence in the Diagnostic and Statistical Manual of Mental Disorders (more specifically, the 2000 "text revision", the DSM-IV-TR) does not use the word addiction at all. It explains:

When an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. This, along with Substance abuse are considered Substance Use Disorders....

This is far from the only way of defining the relevant terms, however (see "Defining terms" section below).

**Brief overview**

Discussions about drug addiction involve much discussion about compulsive behaviours and disorders.

Doug Sellman at the National Addiction Center offers what he calls "The 10 most important things to know about addiction". He offers the following points, before explaining them in more detail (although even his full paper does not presume to be able to discuss all the important facts about addiction). First, Sellman says that the most important thing to know about addiction may be that addiction is "fundamentally about compulsive behaviour" (see also Obsessive compulsive disorder)". Such habits, he goes on to warn, originate outside of consciousness (i.e. from the unconscious mind). The compulsive sequence of behaviours are so practiced that they can be extremely difficult to avoid initiating, and even harder to interrupt. Sellman maintains, thirdly, that addiction is 50% heritable. In other words, family background and genetics play a large role (see also Nature versus Nurture).

The fourth most important thing is that people with addictions often have other psychiatric problems (e.g. psychiatric disorders), which can complicate matters. Next, Sellman explains that addiction is characterized by frequent relapse, and that one should not expect to overcome addiction on the first try. He describes how the different forms of psychotherapy all produce similar results that may be based on what is common between them (i.e. a strong bond with a trusted friend). His seventh most important thing about addiction is that 'come back when you're motivated' is an inappropriate approach to addiction. Individuals have very specific problems, and so it is important to find ways to engage the addicted individual (Sellman describes the important of empathy, for example). His next point expands on this idea: Sellman says that doctors should apply as broad an approach to the individual as possible. This means combining various rejuvenating approaches,
including prescription drugs, family therapy, social and legal support, providing accommodations, and more. Another important thing about addiction is that epiphanies are rare - even though they are the most popular kind of story to spread.

The tenth, and final important thing that Sellman explains is that change takes time (months or years of failing and trying more). He advocates for the importance of patience and persistence in practicing new behaviours over long periods of time. He concludes by appealing to all professionals involved in combating addiction; he asks that they all work together - because the combined knowledge of all fields is what is required.

**Defining terms**

According to the current Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), substance dependence is defined as:

> When an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. This, along with Substance Abuse are considered Substance Use Disorders....

Substance dependence can be diagnosed with physiological dependence, evidence of tolerance or withdrawal, or without physiological dependence.

By the American Society of Addiction Medicine definition, drug addiction differs from drug dependence and drug tolerance. It is, both among scientists and other writers, quite usual to allow the concept of drug addiction to include persons who are not drug abusers according to the definition of the American Society of Addiction Medicine. The term drug addiction is then used as a category which may include the same persons who, under the DSM-IV, can be given the diagnosis of substance dependence or substance abuse. (See also DSM-IV Codes)

The terms abuse and addiction have been defined and re-defined over the years. The 1957 World Health Organization (WHO) Expert Committee on Addiction-Producing Drugs defined addiction and habituation as components of drug abuse:

> Drug addiction is a state of periodic or chronic intoxication produced by the repeated consumption of a drug (natural or synthetic). Its characteristics include: (i) an overpowering desire or need (compulsion) to continue taking the drug and to obtain it by any means; (ii) a tendency to increase the dose; (iii) a psychic (psychological) and generally a physical dependence on the effects of the drug; and (iv) detrimental effects on the individual and on society.

Drug habituation (habit) is a condition resulting from the repeated consumption of a drug. Its characteristics include (i) a desire (but not a compulsion) to continue taking the drug for the sense of improved well-being which it engenders; (ii) little or no tendency to
increase the dose; (iii) some degree of psychic dependence on the effect of the drug, but absence of physical dependence and hence of an abstinence syndrome [withdrawal], and (iv) detrimental effects, if any, primarily on the individual.

In 1964, a new WHO committee found these definitions to be inadequate, and suggested using the blanket term "drug dependence":

The definition of addiction gained some acceptance, but confusion in the use of the terms addiction and habituation and misuse of the former continued. Further, the list of drugs abused increased in number and diversity. These difficulties have become increasingly apparent and various attempts have been made to find a term that could be applied to drug abuse generally. The component in common appears to be dependence, whether psychic or physical or both. Hence, use of the term "drug dependence", with a modifying phase linking it to a particular drug type in order to differentiate one class of drugs from another, had been given most careful consideration. The Expert Committee recommends substitution of the term "drug dependence" for the terms "drug addiction" and "drug habituation".

The committee did not clearly define dependence, but did go on to clarify that there was a distinction between physical and psychological ("psychic") dependence. It said that drug abuse was "a state of psychic dependence or physical dependence, or both, on a drug, arising in a person following administration of that drug on a periodic or continued basis." Psychic dependence was defined as a state in which "there is a feeling of satisfaction and psychic drive that requires periodic or continuous administration of the drug to produce pleasure or to avoid discomfort" and all drugs were said to be capable of producing this state:

There is scarcely any agent which can be taken into the body to which some individuals will not get a reaction satisfactory or pleasurable to them, persuading them to continue its use even to the point of abuse – that is, to excessive or persistent use beyond medical need.

The 1957 and 1964 definitions of addiction, dependence and abuse persist to the present day in medical literature. It should be noted that at this time (2006) the Diagnostic Statistical Manual (DSM-IV-TR) now spells out specific criteria for defining abuse and dependence. (DSM-IV-TR) uses the term substance dependence instead of addiction; a maladaptive pattern of substance abuse, leading to clinically significant impairment or distress, as manifested by three (or more) specified criteria, occurring at any time in the same 12-month period. This definition is also applicable on drugs with smaller or nonexistent physical signs of withdrawal, e.g., cannabis.

In 2001, the American Academy of Pain Medicine, the American Pain Society, and the American Society of Addiction Medicine jointly issued "Definitions Related to the Use of Opioids for the Treatment of Pain", which defined the following terms:

Addiction is a primary, chronic, neurobiologic disease, with genetic, psychosocial, and environmental factors influencing its development and manifestations. It is characterized
by behaviors that include one or more of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving.

Physical dependence is a state of being that is manifested by a drug class specific withdrawal syndrome that can be produced by abrupt cessation, rapid dose reduction, decreasing blood level of the drug, and/or administration of an antagonist.

Tolerance is the body's physical adaptation to a drug; greater amounts of the drug are required over time to achieve the initial effect as the body "gets used to" and adapts to the intake.

Pseudo addiction is a term which has been used to describe patient behaviors that may occur when pain is undertreated. Patients with unrelieved pain may become focused on obtaining medications, may "clock watch," and may otherwise seem inappropriately "drug seeking." Even such behaviors as illicit drug use and deception can occur in the patient's efforts to obtain relief. Pseudoaddiction can be distinguished from true addiction in that the behaviors resolve when pain is effectively treated.

A definition of addiction proposed by professor Nils Bejerot:

An emotional fixation (sentiment) acquired through learning, which intermittently or continually expresses itself in purposeful, stereotyped behavior with the character and force of a natural drive, aiming at a specific pleasure or the avoidance of a specific discomfort.

Causes

Drugs known to cause addiction include both legal and illegal drugs as well as prescription or over-the-counter drugs, according to the definition of the American Society of Addiction Medicine.

Stimulants (psychic addiction, moderate to severe; withdrawal is purely psychological and psychosomatic):

- Amphetamine and methamphetamine
- Cocaine
- Nicotine
- Caffeine
- Sedatives and hypnotics (psychic addiction, mild to severe, and physiological addiction, severe; abrupt withdrawal may be fatal):
  - Alcohol
  - Barbiturates
  - Benzodiazepines, particularly flunitrazepam, triazolam, temazepam, and nimetazepam Z-drugs like Zimovane have a similar effect in the body to Benzodiazepines.
  - Methaqualone and the related quinazolinone sedative-hypnotics
- Opiate and opioid analgesics (psychic addiction, mild to severe, physiological addiction, mild to severe; abrupt withdrawal is unlikely to be fatal):
  - Morphine and codeine, the two naturally occurring opiate analgesics
  - Semi-synthetic opiates, such as heroin (diacetylmorphine; morphine diacetate), oxycodone, buprenorphine, and hydromorphone
  - Fully synthetic opioids, such as fentanyl, meperidine/pethidine, and methadone

Addictive drugs also include a large number of substrates that are currently considered to have no medical value and are not available over the counter or by prescription.

Several theories of drug addiction exist, some of the main ones being genetic predisposition, the self-medication theory, and factors involved with social/economic development. It has long been established that genetic factors along with social and psychological factors are contributors to addiction. A common theory along these lines is the self-medication hypotheses. Epidemiological studies estimate that genetic factors account for 40-60% of the risk factors for alcoholism. Similar rates of heritability for other types of drug addiction have been indicated by other studies (Kendler,1994). Knestler hypothesized in 1964 that a gene or group of genes might contribute to predisposition to addiction in several ways. For example, altered levels of a normal protein due to environmental factors could then change the structure or functioning of specific brain circuits during development. These altered brain circuits could change the susceptibility of an individual to an initial drug use experience. In support of this hypothesis, animal studies have shown that environmental factors such as stress can affect an animal’s genotype.

**Addictive potential**

The addictive potential of a drug varies from substance to substance, and from individual to individual. Dose, frequency, pharmacokinetics of a particular substance, route of administration, and time are critical factors for developing a drug addiction.

An article in The Lancet compared the harm and addiction of 20 drugs, using a scale from 0 to 3 for physical addiction, psychological addiction, and pleasure to create a mean score for addiction. A caffeine control was not included in the study. Selected results can be seen in the chart below.

<table>
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</table>

Self-medication hypotheses

Espoused by both psychoanalysts and biological researchers, self-medication hypotheses predict that certain individuals abuse drugs in an attempt to self-medicate their unique and seemingly intolerable states of mind. The self-medication theory has a long history. Freud in 1884, first raised this concept in noting the anti-depressing properties of cocaine. Stress has long been recognized as a major contributor for drug cravings and relapse and is therefore supportive of the self-medication theory. In line with this theory, a person's use of a particular drug of choice is not an accident, but rather it is chosen for its pharmacological effect in relieving stressful symptoms or unwanted feelings. Research has shown that people who survive disasters are prone to stress-related disorders such as posttraumatic stress disorder (PTSD) and depression. People who experience major trauma in their life experiences may self-medicate with alcohol or other drugs to relieve the symptoms of PTSD and depression.

Social development

Social development and adjustment factors also play a role in drug abuse and addiction. An assumption of the developmental perspective, as mentioned by Thornberry 1987, is that the course of one's life is a process in which life circumstances change, milestones are met or missed and new social roles are created while old ones are abandoned. There are well known and widely accepted norms about when certain developmental events should happen in a person's life. Studies of the social factors involved in drug use have mostly focused either on adolescence or young adulthood, but surprisingly a significant amount of cocaine users may not initiate use until middle adulthood. The majority of people enter into adult social roles on schedule. However, some people enter these roles earlier or later than their same-age peers. The developmental perspective predicts that this will lead to less than satisfactory adjustment and possibly negative consequences including drug and alcohol dependence.

Pathophysiology

Researchers have conducted numerous investigations using animal models and functional brain imaging on humans in order to define the mechanisms underlying drug addiction in the brain. This intriguing topic incorporates several areas of the brain and synaptic changes, or neuroplasticity, which occurs in these areas.

Acute effects

Acute (or recreational) use of most psychoactive drugs causes the release and prolonged action of dopamine and serotonin within the reward circuit. Different types of drugs produce these effects by different methods. Dopamine (DA) appears to harbor the largest
effect and its action is characterized. DA binds to the D1 receptor, triggering a signaling cascade within the cell. cAMP-dependent protein kinase (PKA) phosphorylates cAMP response element binding protein (CREB), a transcription factor, which induces the transcription of certain genes including C-Fos.

**Reward circuit**

When examining the biological basis of drug addiction, one must first understand the pathways in which drugs act and how drugs can alter those pathways. The reward circuit, also referred to as the mesolimbic system, is characterized by the interaction of several areas of the brain.

- The ventral tegmental area (VTA) consists of dopaminergic neurons which respond to glutamate. These cells respond when stimuli indicative of a reward are present. The VTA supports learning and sensitization development and releases dopamine (DA) into the forebrain. These neurons also project and release DA into the nucleus accumbens, through the mesolimbic pathway. Virtually all drugs causing drug addiction increase the dopamine release in the mesolimbic pathway, in addition to their specific effects.
- The nucleus accumbens (NAc) consists mainly of medium-spiny projection neurons (MSNs), which are GABA neurons. The NAcc is associated with acquiring and eliciting conditioned behaviors and involved in the increased sensitivity to drugs as addiction progresses.
- The prefrontal cortex, more specifically the anterior cingulate and orbitofrontal cortices, is important for the integration of information which contributes to whether a behavior will be elicited. It appears to be the area in which motivation originates and the salience of stimuli are determined.
- The basolateral amygdala projects into the NAcc and is thought to be important for motivation as well.
- More evidence is pointing towards the role of the hippocampus in drug addiction because of its importance in learning and memory. Much of this evidence stems from investigations manipulating cells in the hippocampus alters dopamine levels in NAcc and firing rates of VTA dopaminergic cells.

**Role of dopamine**

Nearly all addictive drugs, directly or indirectly, target the brain’s reward system by flooding the circuit with dopamine. As a person continues to overstimulate the “reward circuit”, the brain adapts to the overwhelming surges in dopamine by producing less of the hormones or by reducing the number of receptors in the reward circuit. As a result, the chemical’s impact on the reward circuit is lessened, reducing the abuser’s ability to enjoy the things that previously brought pleasure. This decrease compels those addicted to dopamine to increase the drug consumption in order to attempt to bring their "feel-good" hormone level back to normal—an effect known as tolerance. Development of dopamine tolerance can eventually lead to profound changes in neurons and brain circuits, with the potential to severely compromise the long-term health of the brain. Modern antipsychotics
are designed to block dopamine function. Unfortunately, this blocking can also cause relapses in depression, and can increase addictive behaviors.

**Stress response**

In addition to the reward circuit, it is hypothesized that stress mechanisms also play a role in addiction. Koob and Kreek have hypothesized that during drug use, the corticotropin-releasing factor (CRF) activates the hypothalamic-pituitary-adrenal axis (HPA) and other stress systems in the extended amygdala. This activation influences the dysregulated emotional state associated with drug addiction. They have found that as drug use escalates, so does the presence of CRF in human cerebrospinal fluid (CSF). In rat models, the separate use of CRF antagonists and CRF receptor antagonists both decreased self-administration of the drug of study. Other studies in this review showed a dysregulation in other hormones associated with the HPA axis, including enkephalin which is an endogenous opioid peptide that regulates pain. It also appears that the µ-opioid receptor system, which enkephalin acts on, is influential in the reward system and can regulate the expression of stress hormones.

**Behavior**

Understanding how learning and behavior work in the reward circuit can help understand the action of addictive drugs. Drug addiction is characterized by strong, drug seeking behaviors in which the addict persistently craves and seeks out drugs, despite the knowledge of harmful consequences. Addictive drugs produce a reward, which is the euphoric feeling resulting from sustained dopamine concentrations in the synaptic cleft of neurons in the brain. Operant conditioning is exhibited in drug addicts as well as laboratory mice, rats, and primates; they are able to associate an action or behavior, in this case seeking out the drug, with a reward, which is the effect of the drug. Evidence shows that this behavior is most likely a result of the synaptic changes which have occurred due to repeated drug exposure. The drug seeking behavior is induced by glutamatergic projections from the prefrontal cortex to the NAc. This idea is supported with data from experiments showing the drug seeking behavior can be prevented following the inhibition of AMPA glutamate receptors and glutamate release in the NAc.

**Allostasis**

Allostasis is the process of achieving stability through changes in behavior as well as physiological features. As a person progresses into drug addiction, he or she appears to enter a new allostatic state, defined as divergence from normal levels of change which persist in a chronic state. Addiction to drugs can cause damage to a brain and body as an organism enters the pathological state; the cost stemming from damage is known as allostatic load. The dysregulation of allostasis gradually occurs as the reward from the drug decreases and the ability to overcome the depressed state following drug use begins to decrease as well. The resulting allostatic load creates a constant state of depression relative to normal allostatic changes. What pushes this decrease is the propensity of drug users to take the drug before the brain and body have returned to original allostatic levels,
producing a constant state of stress. Therefore, the presence of environmental stressors may induce stronger drug seeking behaviors.

**Neuroplasticity**

Neuroplasticity is the putative mechanism behind learning and memory. It involves physical changes in the synapses between two communicating neurons, characterized by increased gene expression, altered cell signaling, and the formation of new synapses between the communicating neurons. When addictive drugs are present in the system, they appear to hijack this mechanism in the reward system so that motivation is geared towards procuring the drug rather than natural rewards. Depending on the history of drug use, excitatory synapses in the nucleus accumbens (NAc) experience two types of neuroplasticity: long-term potentiation (LTP) and long-term depression (LTD). Using mice as a model, Kourrich et al. showed that chronic exposure to cocaine increases the strength of synapses in NAc after a 10-14 day withdrawal period, while strengthened Synapses did not appear within a 24 hour withdrawal period after repeated cocaine exposure. A single dose of cocaine did not elicit any attributes of a strengthened synapse. When drug-experienced mice were challenged with one dose of cocaine, synaptic depression occurred. Therefore, it seems the history of cocaine exposure along with withdrawal times affects the direction of glutamatergic plasticity in the NAc.

Once a person has transitioned from drug use to addiction, behavior becomes completely geared towards seeking the drug, even though addicts report the euphoria is not as intense as it once was. Despite the differing actions of drugs during acute use, the final pathway of addiction is the same. Another aspect of drug addiction is a decreased response to normal biological stimuli, such as food, sex, and social interaction. Through functional brain imaging of patients addicted to cocaine, scientists have been able to visualize increased metabolic activity in the anterior cingulate and orbitofrontal cortex (areas of the prefrontal cortex) in the brain of these subjects. The hyperactivity of these areas of the brain in addicted subjects is involved in the more intense motivation to find the drug rather than seeking natural rewards, as well as an addict's decreased ability to overcome this urge. Brain imaging has also shown cocaine-addicted subjects to have decreased activity, as compared to non-addicts, in their prefrontal cortex when presented with stimuli associated with natural rewards. The transition from recreational drug use to addiction occurs in gradual stages and is produced by the effect of the drug of choice on the neuroplasticity of the neurons found in the reward circuit. During events preceding addiction, cravings are produced by the release of dopamine (DA) in the prefrontal cortex. As a person transitions from drug use to addiction, the release of DA in the NAc becomes unnecessary to produce cravings; rather, DA transmission decreases while increased metabolic activity in the orbitofrontal cortex contributes to cravings. At this time a person may experience the signs of depression if cocaine is not used. Before a person becomes addicted and exhibits drug-seeking behavior, there is a time period in which the neuroplasticity is reversible. Addiction occurs when drug-seeking behavior is exhibited and the vulnerability to relapse persists, despite prolonged withdrawal; these behavioral attributes are the result of neuroplastic changes which are brought about by repeated exposure to drugs and are relatively permanent.
The exact mechanism behind a drug molecule’s effect on synaptic plasticity is still unclear. However, neuroplasticity in glutamatergic projections seems to be a major result of repeated drug exposure. This type of synaptic plasticity results in LTP, which strengthens connections between two neurons; onset of this occurs quickly and the result is constant. In addition to glutamatergic neurons, dopaminergic neurons present in the VTA respond to glutamate and may be recruited earliest during neural adaptations caused by repeated drug exposure. As shown by Kourrich, et al., history of drug exposure and the time of withdrawal from last exposure appear to play an important role in the direction of plasticity in the neurons of the reward system.

An aspect of neuron development that may also play a part in drug-induced neuroplasticity is the presence of axon guidance molecules such as semaphorins and ephrins. After repeated cocaine treatment, altered expression (increase or decrease dependent on the type of molecule) of mRNA coding for axon guidance molecules occurred in rats. This may contribute to the alterations in the reward circuit characteristic of drug addiction.

**Neurogenesis**

Drug addiction also raises the issue of potential harmful effects on the development of new neurons in adults. Eisch and Harburg raise three new concepts they have extrapolated from the numerous recent studies on drug addiction. First, neurogenesis decreases as a result of repeated exposure to addictive drugs. A list of studies show that chronic use of opiates, psychostimulants, nicotine, and alcohol decrease neurogenesis in mice and rats. Second, this apparent decrease in neurogenesis seems to be independent of HPA axis activation. Other environmental factors other than drug exposure such as age, stress and exercise, can also have an effect on neurogenesis by regulating the hypothalamic-pituitary-adrenal (HPA) axis. Mounting evidence suggests this for 3 reasons: small doses of opiates and psychostimulants increase corticosterone concentration in serum but with no effect of neurogenesis; although decreased neurogenesis is similar between self-administered and forced drug intake, activation of HPA axis is greater in self-administration subjects; and even after the inhibition of opiate induced increase of corticosterone, a decrease in neurogenesis occurred. These, of course, need to be investigated further. Last, addictive drugs appear to only affect proliferation in the subgranular zone (SGZ), rather than other areas associated with neurogenesis. The studies of drug use and neurogenesis may have implications on stem cell biology.

**Psychological drug tolerance**

The reward system is partly responsible for the psychological part of drug tolerance.

The CREB protein, a transcription factor activated by cyclic adenosine monophosphate (cAMP) immediately after a high, triggers genes that produce proteins such as dynorphin, which cuts off dopamine release and temporarily inhibits the reward circuit. In chronic drug users, a sustained activation of CREB thus forces a larger dose to be taken to reach the same effect. In addition it leaves the user feeling generally depressed and dissatisfied, and
unable to find pleasure in previously enjoyable activities, often leading to a return to the drug for an additional "fix".

A similar mechanism, interfering also with the dopamine system, but relying on a different transcription factor, CEBPB, has also been proposed. In this case dopamine release onto the nucleus accumbens neurons would trigger the increased synthesis of substance P which, in turn, would increase the dopamine synthesis in the VTA. The effect of this positive feedback is suggested to be dampened by repeated substance abuse.

**Sensitization**

Sensitization is the increase in sensitivity to a drug after prolonged use. The proteins delta FosB and regulator of G-protein Signaling 9-2 (RGS9-2) are thought to be involved:

A transcription factor, known as delta FosB, is thought to activate genes that, counter to the effects of CREB, actually increase the user's sensitivity to the effects of the substance. Delta FosB slowly builds up with each exposure to the drug and remains activated for weeks after the last exposure—long after the effects of CREB have faded. The hypersensitivity that it causes is thought to be responsible for the intense cravings associated with drug addiction, and is often extended to even the peripheral cues of drug use, such as related behaviors or the sight of drug paraphernalia. There is some evidence that delta FosB even causes structural changes within the nucleus accumbens, which presumably helps to perpetuate the cravings, and may be responsible for the high incidence of relapses that occur in treated drug addicts.

Regulator of G-protein Signaling 9-2 (RGS9-2) has recently been the subject of several animal knockout studies. Animals lacking RGS9-2 appear to have increased sensitivity to dopamine receptor agonists such as cocaine and amphetamines; over-expression of RGS9-2 causes a lack of responsiveness to these same agonists. RGS9-2 is believed to catalyze inactivation of the G-protein coupled D2 receptor by enhancing the rate of GTP hydrolysis of the G alpha subunit which transmits signals into the interior of the cell.

**Individual mechanisms of effect**

The basic mechanisms by which different substances activate the reward system are as described above, but vary slightly among drug classes.

**Depressants**

Depressants such as alcohol, barbiturates, and benzodiazepines work by increasing the affinity of the GABA receptor for its ligand: GABA. Narcotics such as morphine and heroin work by mimicking endorphins—chemicals produced naturally by the body which have effects similar to dopamine—or by disabling the neurons that normally inhibit the release of dopamine in the reward system. These substances (sometimes called "downers") typically facilitate relaxation and pain relief.
Stimulants

Stimulants such as amphetamines, nicotine, and cocaine increase dopamine signaling in the reward system either by directly stimulating its release, or by blocking its absorption (see "Reuptake"). These substances (sometimes called "uppers") typically cause heightened alertness and energy. They cause a pleasant feeling in the body and euphoria, known as a high. Once this high wears off, the user may feel depressed. This makes them want another dose of the drug, and can worsen the addiction.

Management

Addiction is a complex but treatable condition. It is characterized by compulsive drug craving, seeking, and use that persist even in the face of severe adverse consequences. For most people, addiction becomes chronic, with relapses possible even after long periods of abstinence. As a chronic, recurring illness, addiction may require continued treatments to increase the intervals between relapses and diminish their intensity. Through treatment tailored to individual needs, people with drug addiction can recover and lead fulfilling lives. The ultimate goal of addiction treatment is to enable an individual to achieve lasting abstinence, but the immediate goals are to reduce substance abuse, improve the patient’s ability to function, and minimize the medical and social complications of substance abuse and their addiction. Like people with diabetes or heart disease, people in treatment for addiction will need to change behavior to adopt a more healthful lifestyle.

Treatments for addiction vary widely according to the types of drugs involved, amount of drugs used, duration of the drug addiction, medical complications and the social needs of the individual. Determining the best type of recovery program for an addicted person depends on a number of factors, including: personality, drug(s) of choice, concept of spirituality or religion, mental or physical illness, and local availability and affordability of programs.

Many different ideas circulate regarding what is considered a "successful" outcome in the recovery from addiction. It is widely accepted that abstinence from addictive substances is a successful outcome. However, abstinence is difficult to achieve in practice. Programs that emphasize controlled drinking exist for alcohol addiction. Opiate replacement therapy has been a medical standard of treatment for opioid addiction for many years.

Treatments and attitudes toward addiction vary widely among different countries. In the USA and developing countries, the goal of treatment for drug dependence is generally total abstinence from all drugs. While ideal, this is in practice very difficult to achieve. Other countries, particularly in Europe, argue the aims of treatment for drug dependence are more complex, with treatment aims including reduction in use to the point that drug use no longer interferes with normal activities such as work and family commitments; shifting the addict away from more dangerous routes of drug administration such as injecting to safer routes such as oral administration; reduction in crime committed by drug addicts; and treatment of other comorbid conditions such as AIDS, hepatitis and mental health disorders. These kinds of outcomes can be achieved without eliminating drug use...
completely. Drug treatment programs in Europe often report more favourable outcomes than those in the USA because the criteria for measuring success are functional rather than abstinence-based. The supporters of programs with total abstinence from drugs as a goal stress that enabling further drug use just means prolonged drug use and risks an increase in addiction and complications from addiction.

It is, of course, sometimes difficult to convince people with substance dependencies to engage in any form of treatment. Family Interventions have been highly successful in helping these people accept help they need.

**Residential**

Residential drug treatment can be broadly divided into two camps: 12 step programs or Therapeutic Communities. 12 step programs have the advantage of coming with an instant social support network, though some find the spiritual context not to their taste. In the UK drug treatment is generally moving towards a more integrated approach with rehabs offering a variety of approaches. These other programs may use a Cognitive-Behavioral Therapy approach, such as SMART Recovery, that looks at the relationship between thoughts, feelings and behaviors, recognizing that a change in any of these areas can affect the whole. CBT sees addiction as a behavior rather than a disease and subsequently curable, or rather, unlearnable. CBT programs recognize that for some individuals controlled use is a more realistic possibility.

One of many recovery methods is the 12 step recovery program, with prominent examples including Alcoholics Anonymous, Narcotics Anonymous, Drug Addicts Anonymous and Pills Anonymous. They are commonly known and used for a variety of addictions for the individual addicted and the family of the individual. Substance-abuse rehabilitation (or "rehab") centers frequently offer a residential treatment program for the seriously addicted in order to isolate the patient from drugs and interactions with other users and dealers. Outpatient clinics usually offer a combination of individual counseling and group counseling. Frequently a physician or psychiatrist will assist, with prescriptions, the side effects of the addiction. Medications can help immensely with anxiety and insomnia, can treat underlying mental disorders (cf. Self-medication hypothesis, Khantzian 1997) such as (manic-)depression, and can help reduce or eliminate withdrawal symptomology when withdrawing from physiologically addictive drugs. Some examples are using benzodiazepines for alcohol detoxification, which prevents delirium tremens and complications; using a slow taper of benzodiazepines or a taper of phenobarbital, sometimes including another antiepileptic agent such as gabapentin, pregabalin, or valproate, for withdrawal from barbiturates or benzodiazepines; using drugs such as baclofen to reduce cravings and propensity for relapse amongst addicts to any drug, especially effective in stimulant users, and alcoholics (in which it is nearly as effective as benzodiazepines in preventing complications); using clonidine, a benzodiazepine, and loperamide for opioid detoxification, for first-time users or those who wish to attempt an abstinence-based recovery (90% of opioid users relapse to active addiction within 8 months and/or are "multiple relapse patients"); or replacing an opioid that is interfering with or destructive to a user's life, such as illicitly-obtained heroin, Dilaudid, or oxycodone,
with an opioid that can be administered legally, reduces or eliminates drug cravings, and
does not produce a high, such as methadone or buprenorphine - opioid replacement
therapy - which is the gold standard for treatment of opioid dependence in developed
countries, reducing the risk and cost to both user and society more effectively than any
other treatment modality (for opioid dependence), and shows the best short-term and
long-term gains for the user, with the greatest longevity, least risk of fatality, greatest
quality of life, and lowest risk of relapse and/or legal issues including arrest and
incarceration.

In a survey of treatment providers from three separate institutions (the National
Association of Alcoholism and Drug Abuse Counselors, Rational Recovery Systems and the
Society of Psychologists in Addictive Behaviors) measuring the treatment provider's
responses on the Spiritual Belief Scale (a scale measuring belief in the four spiritual
characteristics AA identified by Ernest Kurtz); the scores were found to explain 41% of the
variance in the treatment provider’s responses on the Addiction Belief Scale (a scale
measuring adherence to the disease model or the free-will model addiction).

**Anti-addictive drugs**

Other forms of treatment include replacement drugs such as suboxone/subutex (both
containing the active ingredient buprenorphine), and methadone, are all used as substitutes
for illicit opiate drugs. Although these drugs perpetuate physical dependence, the goal of
opiate maintenance is to provide a clinically supervised, stable dose of a particular opioid
in order to provide a measure of control to both pain and cravings. This provides a chance
for the addict to function normally and to reduce the negative consequences associated
with obtaining sufficient quantities of controlled substances illicitly, by both reducing
opioid cravings and withdrawal symptomology. Once a prescribed dosage is stabilized,
treatment enters maintenance or tapering phases. In the United States, opiate replacement
therapy is tightly regulated in methadone clinics and under the DATA 2000 legislation.
In some countries, other opioid derivatives such as levomethadyl acetate, dihydrocodeine,
dihydroetorphine and even heroin are used as substitute drugs for illegal street opiates,
with different drugs being used depending on the needs of the individual patient. Baclofen
has been shown successful in attenuating cravings for most drugs of abuse - stimulants,
ethanol, and opioids - and also attenuates the actual withdrawal syndrome of ethanol.
Many patients have stated they "became indifferent to alcohol" or "indifferent to cocaine"
overnight after starting baclofen therapy. It is possible that one of the best, albeit relatively
unexplored, treatment modalities for opioid addiction - notoriously the most difficult
addiction to treat (and to recover from), having relapse rates of around 60% at four weeks
and 97% at twelve months if not on maintenance therapy with a mu-opioid agonist - would
be to combine an opioid maintenance agent, such as methadone or buprenorphine, to block
withdrawal symptomology, with baclofen, to attenuate cravings and the desire to use, in
people who find that they are still using or still craving drugs while on methadone or
buprenorphine maintenance.

Substitute drugs for other forms of drug dependence have historically been less successful
than opioid substitute treatment, but some limited success has been seen with drugs such
as dextroamphetamine to treat stimulant addiction, and clomethiazole to treat alcohol addiction. Bromocriptine and desipramine have been reported to be effective for treatment of cocaine but not amphetamine addiction.

Other pharmacological treatments for alcohol addiction include drugs like naltrexone, disulfiram, acamprosate and topiramate, but rather than substituting for alcohol, these drugs are intended to reduce the desire to drink, either by directly reducing cravings as with acamprosate and topiramate, or by producing unpleasant effects when alcohol is consumed, as with disulfiram. These drugs can be effective if treatment is maintained, but compliance can be an issue as alcoholic patients often forget to take their medication, or discontinue use because of excessive side effects. Additional drugs acting on glutamate neurotransmission such as modafinil, lamotrigine, gabapentin and memantine have also been proposed for use in treating addiction to alcohol and other drugs.

Opioid antagonists such as naltrexone and nalmefene have also been used successfully in the treatment of alcohol addiction, which is often particularly challenging to treat. Some have also attempted to use these drugs for maintenance treatment of former opiate addicts with little success. They cannot be started until the patient has been abstinent for an extended period - unlikely with opioid addicts who are not on maintenance with a full or partial mu-opioid agonist - or they will trigger acute opioid withdrawal symptoms. No study has found them to be efficacious treatments in preventing relapse. They do nothing to block craving, and block endorphin and enkephalin, two natural neurotransmitters that regulate one’s sense of well-being. An addict must discontinue the drug for just eighteen hours in order to use again.

Treatment of stimulant addiction can often be difficult, with substitute drugs often being ineffective, although newer drugs such as nocaine, vanoxerine and modafinil may have more promise in this area, as well as the GABAB agonist baclofen. Another strategy that has recently been successfully trialled used a combination of the benzodiazepine antagonist flumazenil with hydroxyzine and gabapentin for the treatment of methamphetamine addiction.

Another area in which drug treatment has been widely used is in the treatment of nicotine addiction. Various drugs have been used for this purpose such as bupropion, mecamylamine and the more recently developed varenicline. The cannabinooid antagonist rimonabant has also been trialled for treatment of nicotine addiction but has not been widely adopted for this purpose.

Ibogaine is a hallucinogen (psychotomimetic) that some claim interrupts addiction and reduces or eliminates withdrawal syndromes, specifically in regards to opioids. Its mechanism of action is unknown but likely linked to nAChR α3/64 antagonism. In one animal trial, it was shown to slightly reduce self-administration of cocaine. Another uncontrolled trial showed it reduced tremor by a mild to moderate degree during morphine withdrawal in rats. These findings can not be extrapolated to human beings with any certainty. Research is complicated by the fact that ibogaine is illegal in many developed countries, and a Schedule I substance in the US, and as a result no controlled human trials
have ever been performed. A semi-synthetic analogue of ibogaine, 18-methoxycoronaridine was developed, in an attempt to reduce the toxic (ibogaine is significantly cardiotoxic, and several deaths have been reported from its use; because of its illegal, underground nature, it is impossible to know how toxic the drug is) and psychotomimetic effects of the drug.

**Behavioral programming**

Behavioral programming is considered critical to helping those with addictions achieve abstinence. From the applied behavior analysis literature and the behavioral psychology literature several evidenced based intervention programs have emerged (1) behavioral marital therapy; (2) community reinforcement approach; (3) cue exposure therapy; and (4) contingency management strategies. In addition, the same author suggest that Social skills training adjunctive to inpatient treatment of alcohol dependence is probably efficacious. Community reinforcement has both efficacy and effectiveness data. In addition, behavioral treatment such as community reinforcement and family training (CRAFT) have helped family members to get their loved ones into treatment.

**Alternative therapies**

Alternative therapies, such as acupuncture, are used by some practitioners to alleviate the symptoms of drug addiction. In 1997, the American Medical Association (AMA) adopted as policy the following statement after a report on a number of alternative therapies including acupuncture:

There is little evidence to confirm the safety or efficacy of most alternative therapies. Much of the information currently known about these therapies makes it clear that many have not been shown to be efficacious. Well-designed, stringently controlled research should be done to evaluate the efficacy of alternative therapies.

Acupuncture has been shown to be no more effective than control treatments in the treatment of opiate dependence. Acupuncture, acupressure, laser therapy and electrostimulation have no demonstrated efficacy for smoking cessation.

**Epidemiology**

Disability-adjusted life year for drug use disorders per 100,000 inhabitants in 2002.

The most common drug addictions are to legal substances such as:

- Nicotine in the form of tobacco, particularly cigarettes
- Alcohol
- Caffeine

**History**
The phenomenon of drug addiction has occurred to some degree throughout recorded history (see "Opium"). Modern agricultural practices, improvements in access to drugs, advancements in biochemistry, and dramatic increases in the recommendation of drug usage by clinical practitioners have exacerbated the problem significantly in the 20th century. Improved means of active biological agent manufacture and the introduction of synthetic compounds, such as methamphetamine are also factors contributing to drug addiction.

**Society and culture**

**Legislation**

Depending on the jurisdiction, addictive drugs may be legal only as part of a government sponsored study, illegal to use for any purpose, illegal to sell, or even illegal to merely possess.

Most countries have legislation which brings various drugs and drug-like substances under the control of licensing systems. Typically this legislation covers any or all of the opiates, amphetamines, cannabinoids, cocaine, barbiturates, benzodiazepines, anesthetics, hallucinogens, derivatives and a variety of more modern synthetic drugs. Unlicensed production, supply or possession is a criminal offence.

Usually, however, drug classification under such legislation is not related simply to addictiveness. The substances covered often have very different addictive properties. Some are highly prone to cause physical dependency, while others rarely cause any form of compulsive need whatsoever. Also, under legislation specifically about drugs, alcohol, caffeine and nicotine are not usually included.

Although the legislation may be justifiable on moral or public health grounds, it can make addiction or dependency a much more serious issue for the individual: reliable supplies of a drug become difficult to secure, and the individual becomes vulnerable to both criminal abuse and legal punishment.

It is unclear whether laws against illegal drug use do anything to stem usage and dependency. In jurisdictions where addictive drugs are illegal, they are generally supplied by drug dealers, who are often involved with organized crime. Even though the cost of producing most illegal addictive substances is very low, their illegality combined with the addict’s need permits the seller to command a premium price, often hundreds of times the production cost. As a result, addicts sometimes turn to crime to support their habit.

**Physical dependence**

Physical dependence refers to a state resulting from chronic use of a drug that has produced tolerance and where negative physical symptoms of withdrawal result from abrupt discontinuation or dosage reduction. Physical dependence can develop from low-
dose therapeutic use of certain medications such as benzodiazepines, opioids, antiepileptics and antidepressants, as well as misuse of recreational drugs such as alcohol, opioids and benzodiazepines. The higher the dose used, the greater the duration of use, and the earlier age use began are predictive of worsened physical dependence and thus more severe withdrawal syndromes. Acute withdrawal syndromes can last days, weeks or months, and protracted withdrawal syndrome, also known as "post-acute withdrawal syndrome" or "PAWS" - a low-grade continuation of some of the symptoms of acute withdrawal, typically in a remitting-relapsing pattern, that often results in relapse in to active addiction and prolonged disability of a degree to preclude the possibility of lawful employment - can last for months, years, or, in relatively common to extremely rare cases, depending on individual factors, indefinitely. Protracted withdrawal syndrome is noted to be most often caused by benzodiazepines, but is also present in a majority of cases of alcohol and opioid addiction, especially that of a long-term, high-dose, adolescent-beginning, or chronic-relapsing nature (viz. a second or third addiction after withdrawal from the self-same substance of dependence). Withdrawal response will vary according to the dose used, the type of drug used, the duration of use, the age of the patient, the age of first use, and the individual person.

**Symptoms**

Physical dependence can manifest itself in the appearance of both physical and psychological symptoms but which are caused by physiological adaption in the central nervous system and the brain due to chronic exposure to a substance. Symptoms which may be experienced during withdrawal or reduction in dosage include increased heart rate and/or blood pressure, sweating, and tremors. More serious withdrawal symptoms such as confusion, seizures, and visual hallucinations indicate a serious emergency and the need for immediate medical care. Sedative hypnotic drugs such as alcohol, benzodiazepines, and barbiturates are the only commonly available substances that can be fatal in withdrawal due to their propensity to induce withdrawal convulsions. Abrupt withdrawal from other drugs, such as opioids, can cause an extremely physiologically and psychologically painful withdrawal that is very rarely fatal in patients of general good health and with medical treatment, but is more often fatal in patients with weakened cardiovascular systems; toxicity is generally caused by the often-extreme increases in heart rate and blood pressure (which can be treated with clonidine), or due to arrhythmia due to electrolyte imbalance caused by the inability to eat, and constant diarrhea and vomiting (which can be treated with loperamide and ondansetron respectively) associated with acute opioid withdrawal, especially in longer-acting substances where the diarrhea and emesis can continue unabated for weeks, although life-threatening complications are extremely rare, and nearly non-existent with proper medical management. Dependence itself and chronic intoxication on psychostimulants can cause mild-to-moderate neurotoxic effects due to hyperthermia and generation of free radicals; this is treated with discontinuation; life-threatening complications are nonexistent.

**Treatment**
Treatment for physical dependence depends upon the drug being withdrawn and often includes administration of another drug, especially for substances that can be dangerous when abruptly discontinued. Physical dependence is usually managed by a slow dose reduction over a period of weeks, months or sometimes longer depending on the drug, dose and the individual. A physical dependence on alcohol is often managed with a cross tolerant drug, such as long acting benzodiazepines to manage the alcohol withdrawal symptoms.

**Drugs that cause physical dependence**

- All μ-opioids with any (even slight) agonist effect, such as (partial list) morphine, heroin, oxycodone, buprenorphine, nalbuphine, methadone, and fentanyl, but not agonists specific to non-μ opioid receptors, such as salvinorin A (a κ-opioid agonist), nor opioid antagonists or inverse agonists, such as naltrexone (a universal opioid inverse agonist)
- All GABA agonists and positive allosteric modulators of both the GABA-A ionotropic receptor and GABA-B metabotropic receptor subunits, of which the following drugs are examples (partial list):
  - barbiturates such as phenobarbital, sodium thiopental and secobarbital
  - benzodiazepines such as diazepam (Valium), lorazepam (Ativan), and alprazolam (Xanax) (see benzodiazepine dependence and benzodiazepine withdrawal syndrome)
  - nonbenzodiazepines (z-drugs) such as zopiclone and zolpidem.
  - ethyl alcohol (alcoholic beverage) (cf. alcohol dependence, alcohol withdrawal, delirium tremens)
  - gamma-hydroxybutyric acid (GHB) and 1,4-butanediol
  - carisoprodol (Soma) and related carbamates (tybamate and meprobamate)
  - baclofen (Lioresal) and its non-chlorinated analogue phenibut
  - chloral hydrate
  - glutethimide
  - clomethiazole
  - methaqualone (Quaalude)
  - gabapentin (Neurontin) and pregabalin (Lyrica), calcium channel modifiers that affect GABA
  - antiepileptic drugs such as valproate, lamotrigine, tiagabine, vigabatrin, carbamazepine and oxcarbazepine, and topiramate
  - possibly neuroleptic drugs such as clozapine, risperidone, olanzapine, haloperidol, thioridazine, etc.
  - commonly prescribed antidepressants such as the selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) (cf. SSRI/SNRI withdrawal syndrome)
  - Nicotine
  - blood pressure medications, including beta blockers such as propanolol and alpha-adrenergic agonists such as clonidine
  - androgenic-anabolic steroids
  - glucocorticoids
Drugs such as amphetamines (including methylamphetamine and methylenedioxyamphetamine (MDMA), cocaine, cathinone, hallucinogens (such as LSD, psilocin, and mescaline), cannabis (tetrahydrocannabinol) do not cause physical dependency/physical addiction, but range from extremely psychologically addictive (coca and methylamphetamine) to mildly psychologically addictive (MDMA).

**Rebound syndrome**

A wide range of drugs whilst not causing a true physical dependence can still cause withdrawal symptoms or rebound effects during dosage reduction or especially abrupt or rapid withdrawal. These can include caffeine, stimulants, steroidal drugs and antiparkinsonian drugs. It is debated if the entire antipsychotic drug class causes true physical dependency, if only a subset do, or if none do, but all, if discontinued too rapidly, cause an acute withdrawal syndrome. Drugs like cocaine, marijuana, amphetamines, and hallucinogens can be associated with minimal physical dependence but can still cause withdrawal or rebound symptoms. However, with sustained and heavy cocaine abuse signs of physiological dependence may occur. When talking about illicit drugs rebound withdrawal is, especially with stimulants, sometimes referred to as "coming down" or "crashing".

Some drugs, like anticonvulsants and antidepressants, describe the drug category and not the mechanism. The individual agents and drug classes in the anticonvulsant drug category act at many different receptors and it is not possible to generalize their potential for physical dependence or incidence or severity of rebound syndrome as a group so need to be looked at individually. Anticonvulsants as a group however are known to cause tolerance to the anti-seizure effect. SSRI drugs, which have an important use as antidepressants, are considered to cause physical dependence, although it is considered mild compared to drugs like opioids and GABA modulators, but they engender a discontinuation syndrome, which was originally called "SSRI withdrawal" until a 1997 symposium sponsored by Pfizer and Eli Lilly (the producers of several anti-depressants including Prozac and Effexor) was held, with the drug representative attendees concluding that "discontinuation syndrome" sounded less threatening than "withdrawal"; however, "SSRI discontinuation syndrome" is a withdrawal syndrome upon discontinuation of SSRI/SNRI drugs, just as "heroin discontinuation syndrome" is a synonym for "heroin withdrawal". Due to this, in Europe these drugs cannot be advertised as "non-habit forming". There have been case reports of dependence with venlafaxine (Effexor).

**Alcohol abuse**

Alcohol abuse, as described in the DSM-IV, is a psychiatric diagnosis describing the recurring use of alcoholic beverages despite negative consequences. Alcohol abuse is sometimes referred to by the less specific term alcoholism. However, many definitions of alcoholism exist, and only some are compatible with alcohol abuse. Binge drinking is
another form of alcohol abuse. Frequent binge drinking or getting severely drunk more than twice is classed as alcohol misuse.

**Definitions**

Alcohol abuse is a pattern of drinking that results in harm to one's health, interpersonal relationships, or ability to work. According to Gelder, Mayou & Geddes (2005) alcohol abuse is linked with suicide. They state the risk of suicide is high in older men who have a history of drinking, also if a person is suffering from depression. Certain manifestations of alcohol abuse include failure to fulfill responsibilities at work, school or home; drinking in dangerous situations, such as while driving; legal problems associated with alcohol use; and continued drinking despite problems that are caused or worsened by drinking. Alcohol abuse can lead to alcohol dependence.

Alcohol abuse has both short-term and long-term risks. If a person has driven while drunk or regularly binge drinks (more than 5 or 6 standard drinks in one drinking session), they are considered to have been involved in alcohol abuse. Short-term abuses of alcohol include, but are not limited to, violence, injuries, unprotected sexual activities and additionally social and financial problems.

**Binge Drinking**

In the USA, binge drinking is defined as consuming more than 5 drinks in men and 4 drinks in women. It increases chances for vandalism, fights, injuries, drunk driving, trouble with police, and negative health, social, economic, or legal consequences to occur. Binge drinking is also associated with neurocognitive deficits of frontal lobe processing and impaired working memory as well as delayed auditory and verbal memory deficits. Binge drinking combine with the stress of returning to work is a contributing factor to Monday deaths from heart attacks.

**Symptoms & Signs**

Symptoms are the defining characteristic of alcohol abuse and are reviewed above in Definitions. Patient will often complain of difficulty with interpersonal relationships, problems at work or school, and legal problems. Additionally patients do complain of irritability and insomnia.

Signs of alcohol abuse are related to alcohol's effects on organ systems. However, while these findings are often present, they are not necessary to make a diagnosis of alcohol abuse. Signs of alcohol's effects on the central nervous system acutely include inebriation and poor judgment; chronic anxiety, irritability, and insomnia often feature. Alcohol's effects on the liver include elevated liver function tests (classically AST is at least twice as high as ALT). Prolonged use leads to cirrhosis and failure of the liver. With cirrhosis patients develop an inability to process hormones and toxins. The skin of a patient with alcoholic cirrhosis can feature cherry angiomas, palmar erythema and - in fulminent liver
failure - jaundice and ascities. The derrangements of the endocrine system lead to
gynecomastia. Inability to process toxins leads to hepatic encephalopathy.

Binge drinking is associated with individuals reporting fair to poor health compared to
non-binge drinking individuals and which may progressively worsen over time. Binge
drinking is associated with alcohol poisoning, unintentional injuries, suicide, hypertension,
pancreatitis, sexually transmitted diseases, and meningitis, among other disorders. Other
negative consequences include social costs (including interpersonal violence), drunk
driving, and lost economic productivity. Impairments in neurophysiological and
neurocognitive function can result from binge drinking. A substantial proportion of
alcohol-related deaths are due to binge drinking.

Causes

Peer pressure influences individuals to abuse alcohol; however most of the influence of
peers is due to inaccurate perceptions of the risks of alcohol abuse. According to Gelder,
Mayou and Geddes (2005) easy accessibility of alcohol is one of the reasons people engage
in alcohol abuse as this substance is easily obtained in shops.

Prevention

Preventing or reducing the harm has been called for via increased taxation of alcohol,
stricter regulation of alcohol advertising and the provision of brief Interventions. Brief
Interventions for alcohol abuse reduce the incidence of unsafe sex, sexual violence,
unplanned pregnancy and, likely, STD transmission. Information and education on social
norms and the harms associated with alcohol abuse delivered either via the internet or face
to face has been found to result in a decrease in harmful drinking behaviours in young
people.

Epidemiology

Alcohol abuse is said to be most common in people aged between 15 and 24 years: however this particular study of 7275 college students in England collected no data about
other age groups or other countries.

Societal and economic costs

Alcohol abuse is associated with many accidents, fights, driving offenses and unprotected
sex. Alcohol is responsible in the world for 1.8 million deaths and results in disability in
approximately 58.3 million people. Approximately 40 percent of the 58.3 million people
disabled through alcohol abuse are disabled due to alcohol related neuropsychiatric
disorders. In South Africa, where HIV infection is epidemic, alcohol abusers exposed
themselves to double the risk of this infection. Additionally, alcohol abuse increases the
risk of individuals either being the victim of sexual violence or perpetrating sexual violence.
Alcohol misuse costs the National Health Service (UK) 3 billion pounds sterling per year and the cost to employers is 6.4 billion pounds sterling per year. These figures do not include the crime and social problems associated with alcohol misuse. The number of women regularly drinking alcohol has almost caught up with men.

**Prognosis**

Alcohol abuse during adolescence, especially early adolescence (i.e. before age 15), may lead to long-term changes in the brain which leaves them at increased risk of alcoholism in later years; genetic factors also influence age of onset of alcohol abuse and risk of alcoholism. For example, about 40 percent of those who begin drinking alcohol before age 15 develop alcohol dependence in later life, whereas only 10 percent of those who did not begin drinking until 20 years or older developed an alcohol problem in later life. It is not entirely clear whether this association is causal, and some researchers have been known to disagree with this view.

College/university students who are heavy binge drinkers (3 or more times in the past 2 weeks) are 19 times more likely to be diagnosed with alcohol dependence, and 13 times more likely to be diagnosed with alcohol abuse compared to non-heavy episodic drinkers, though the direction of causality remains unclear. Occasional binge drinkers (one or two times in past 2 weeks), were found to be 4 times more likely to be diagnosed with alcohol abuse or dependence compared to non-heavy episodic drinkers.

**Alcopops**

The introduction of alcopops, which are flavoured alcoholic drinks which have a sweet and pleasant taste was responsible in Sweden for half of the increase in alcohol abuse in 15-16 year olds in a survey. In the case of girls the alcopops, which disguise the taste of alcohol, were responsible for two thirds of the increase. The introduction of alcopops to Sweden was a result of Sweden joining the European Union and adopting the entire European Union law. Alcohol abuse is highly associated with adolescent suicide. Adolescents who abuse alcohol are 17 times more likely to commit suicide than adolescents who don’t drink.

**Association with violence**

Alcohol abuse is significantly associated with suicide and violence. Alcohol is the most significant health concern in Native American communities because of very high rates of alcohol dependence and abuse; up to 80 percent of suicides and 60 percent of violent acts are a result of alcohol abuse in Native American communities.

The overuse of alcohol has led to 16% of intoxicated people that have abused children. Abusing children can also come in the form of verbal abuse while under the influence, as well as physical abuse. Alcohol can impair one’s judgment and make emotions more easily emphasized, such as anger towards a minor topic, which can cause them to become dangerous.
Alcohol dependence

Alcohol dependence, as described in the DSM-IV, is a psychiatric diagnosis (a substance related disorder DSM-IV) describing an entity in which an individual uses alcohol despite significant areas of dysfunction, evidence of physical dependence, and/or related hardship.

Definition and diagnosis

According to the DSM-IV criteria for alcohol dependence, at least three out of seven of the following criteria must be manifest during a 12 month period:

- Tolerance
- Withdrawal symptoms or clinically defined Alcohol Withdrawal Syndrome
- Use in larger amounts or for longer periods than intended
- Persistent desire or unsuccessful efforts to cut down on alcohol use
- Time is spent obtaining alcohol or recovering from effects
- Social, occupational and recreational pursuits are given up or reduced because of alcohol use
- Use is continued despite knowledge of alcohol-related harm (physical or psychological)

History and epidemiology

About 12% of American adults have had an alcohol dependence problem at some time in their life. Alcohol dependence is acknowledged by the American Medical Association as a disease because it has a characteristic set of signs and symptoms and a progressive course.

The contemporary definition of alcohol dependence is still based upon early research. There has been considerable scientific effort over the past several decades to identify and understand the core features of alcohol dependence. This work began in 1976 when the British psychiatrist Griffith Edwards and his American colleague Milton M. Gross collaborated to produce a formulation of what had previously been understood as ‘alcoholism’ – the alcohol dependence syndrome.

The alcohol dependence syndrome was seen as a cluster of seven elements that concur. It was argued that not all elements may be present in every case, but the picture is sufficiently regular and coherent to permit clinical recognition. The syndrome was also considered to exist in degrees of severity rather than as a categorical absolute. Thus, the proper question is not ‘whether a person is dependent on alcohol’, but ‘how far along the path of dependence has a person progressed’.

Screening tools

The Alcohol Use Disorders Identification Test (AUDIT) is the most accurate alcohol screening tool for identifying potential alcohol misuse, including dependence. It was
developed by the World Health Organisation, designed initially for use in primary healthcare settings with supporting guidance. Its use has replaced older screening tools such as CAGE but there are many shorter alcohol screening tools, mostly derived from the AUDIT. The Severity of Alcohol Dependence Questionnaire (SAD-Q) is a more specific twenty item inventory for assessing the presence and severity of alcohol dependence.

Comparisons with other alcohol-related disorders

Because only 3 of the 7 DSM-IV criteria for alcohol dependence are required, not all patients meet the same criteria and therefore not all have the same symptoms and problems related to drinking. Not everyone with alcohol dependence, therefore, experiences physiological dependence. Alcohol dependence is differentiated from alcohol abuse by the presence of symptoms such as tolerance and withdrawal. Both alcohol dependence and alcohol abuse are sometimes referred to by the less specific term alcoholism. However, many definitions of alcoholism exist, and only some are compatible with alcohol abuse. There are two major differences between alcohol dependence and alcoholism as generally accepted by the medical community.

- Alcohol dependence refers to an entity in which only alcohol is the involved addictive agent. Alcoholism refers to an entity in which alcohol or any cross-tolerant addictive agent is involved.
- In alcohol dependence, remission as defined within DSM-IV can be attained despite continued use of alcohol. That is, a patient can be in full sustained remission yet still be drinking alcohol so long as the patient does not meet the noted criteria. In alcoholism, patients are generally not presumed to be in remission unless they are abstinent from alcohol.

The following elements are the template for which the degree of dependence is judged:

- Narrowing of the drinking repertoire.
- Increased salience of the need for alcohol over competing needs and responsibilities.
- An acquired tolerance to alcohol.
- Withdrawal symptoms.
- Relief or avoidance of withdrawal symptoms by further drinking.
- Subjective awareness of compulsion to drink.
- Reinstatement after abstinence.

Opioid dependence

Opioid dependency is a medical diagnosis characterized by an individual’s inability to stop using opioids (morphine/heroin, codeine, oxycodone, hydrocodone, etc.) even when objectively it is in his or her best interest to do so. In 1964 the WHO Expert Committee on Drug Dependence introduced "dependence" as “A cluster of physiological, behavioural and cognitive phenomena of variable intensity, in which the use of a psychoactive drug (or
drugs) takes on a high priority. The necessary descriptive characteristics are preoccupation with a desire to obtain and take the drug and persistent drug-seeking behaviour. Determinants and problematic consequences of drug dependence may be biological, psychological or social, and usually interact”. The core concept of the WHO definition of “drug dependence” requires the presence of a strong desire or a sense of compulsion to take the drug; and the WHO and DSM-IV-TR clinical guidelines for a definite diagnosis of “dependence” require that three or more of the following six characteristic features be experienced or exhibited:

- A strong desire or sense of compulsion to take the drug;
- Difficulties in controlling drug-taking behaviour in terms of its onset, termination, or levels of use;
- A physiological withdrawal state when drug use is stopped or reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms;
- Evidence of tolerance, such that increased doses of the drug are required in order to achieve effects originally produced by lower doses;
- Progressive neglect of alternative pleasures or interests because of drug use, increased amount of time necessary to obtain or take the drug or to recover from its effects;
- Persisting with drug use despite clear evidence of overtly harmful consequences, such as harm to the liver, depressive mood states or impairment of cognitive functioning.

The Walid-Robinson Opioid-Dependence (WROD) Questionnaire was designed based on these guidelines. According to position papers on the treatment of opioid dependence published by the United Nations Office on Drugs and Crime and the World Health Organization, care providers should not mistake opioid dependence for a weakness of character or will. Accordingly, detoxification alone does not constitute adequate treatment.

Causes

Studies show that most opioid dependent patients suffer from at least one severe psychiatric comorbidity. Since opioids used in pain therapy rarely cause any of these conditions, they are assumed to have existed prior to the development of dependence. Opioids are known to have strong antidepressive, anxiolytic and antipsychotic effects and thus opioid dependence often develops as a result of self medication.

Material used for intravenous injection of opiates

Furthermore some studies suggest a permanent dysregulation of the endogenous opioid receptor system after chronic exposure to opiates. A recent study has shown that an increase in BDNF, brain-derived neurotrophic factor, in the ventral tegmental area (VTA) in rats can cause opiate-naive rats to begin displaying opiate-dependent behavior, including
withdrawal and drug-seeking behavior. It has been shown that when an opiate-naive person begins using opiates at levels inducing euphoria, this same increase in BDNF occurs.

Another recent study concluded to have shown "a direct link between morphine abstinence and depressive-like symptoms" and postulates "that serotonin dysfunction represents a main mechanism contributing to mood disorders in opiate abstinence".

**Symptoms of withdrawal**

Symptoms of withdrawal from opiates include, but are not limited to,

**Physical Symptoms**

- Extreme Pain
- Tremors
- Cramps
- Chills
- Perspiration
- Priapism
- Tachycardia
- Itch
- Restless legs syndrome
- Flu-like symptoms
- Rhinitis
- Yawning
- Sneezing
- Vomiting
- Diarrhea
- Weakness

**Psychological Symptoms**

- Dysphoria
- Malaise
- Cravings
- Anxiety/Panic Attacks
- Paranoia
- Insomnia
- Dizziness
- Nausea
- Depression

Other rare symptoms but more serious are cardiac arrhythmias, strokes, seizures, dehydration and suicide attempts.
Depending on the quantity, type, frequency, and duration of opioid use, the physical withdrawal symptoms last for as little as forty-eight to seventy-two hours (for short-acting opioids such as hydromorphone [Dilaudid] and oxycodone after short duration lower-dose use), and as long as thirty to sixty days for long-acting opioids such as buprenorphine and methadone, respectively, after extended high-dose use. When long acting opioids like methadone (Methadose, Physeptone) or buprenorphine (Suboxone [buprenorphine in a 4:1 ratio to naloxone] and Subutex [single-agent buprenorphine]) are used for an extended period, physical withdrawal symptoms can last up to six weeks. This initial withdrawal is characterized by the body regaining physical homeostasis.

Treatment

Opioid dependence is a complex health condition that often requires long-term treatment and care. The treatment of opioid dependence is important to reduce its health and social consequences and to improve the well-being and social functioning of people affected. The main objectives of treating and rehabilitating persons with opioid dependence are to reduce dependence on illicit drugs; to reduce the morbidity and mortality caused by the use of illicit opioids, or associated with their use, such as infectious diseases; to improve physical and psychological health; to reduce criminal behaviour; to facilitate reintegration into the workforce and education system and to improve social functioning. The ultimate achievement of a drug free state is the ideal and ultimate objective but this is unfortunately not feasible for all individuals with opioid dependence, especially in the short term.

As no single treatment is effective for all individuals with opioid dependence, diverse treatment options are needed, including psychosocial approaches and pharmacological treatment.

Relapse following detoxification alone is extremely common, and therefore detoxification rarely constitutes an adequate treatment of substance dependence on its own. However, it is a first step for many forms of longer-term abstinence-based treatment. Both detoxification with subsequent abstinence-oriented treatment and substitution maintenance treatment are essential components of an effective treatment system for people with opioid dependence.

Methadone Treatment

MMT (Methadone Maintenance Treatment), a form of opioid replacement therapy, reduces and/or eliminates the use of illicit opiates, the criminality associated with opiate use, and allows patients to improve their health and social productivity. In addition, enrollment in methadone maintenance has the potential to reduce the transmission of infectious diseases associated with opiate injection, such as hepatitis and HIV. The principal effects of methadone maintenance are to relieve narcotic craving, suppress the abstinence syndrome, and block the euphoric effects associated with opiates. Methadone maintenance has been found to be medically safe and non-sedating. It is also indicated for pregnant women addicted to opiates.
Buprenorphine Treatment

Buprenorphine sublingual preparations are often used in the management of opioid dependence (that is, dependence on heroin, oxycodone, hydrocodone, morphine, oxymorphone, fentanyl or other opioids). The Suboxone and Subutex preparations were approved for this indication by the United States Food and Drug Administration in October 2002. This was only possible due to the Drug Addiction Treatment Act of 2000 which overturned a series of 1914–1920 Supreme Court rulings that had found that maintenance and detox treatments were not a form of medical treatment. Although the rulings had the power of legal precedent prior to 2000, it is likely that they were not the intended interpretation of the laws passed originally by congress.

Diamorphine Treatment

In Switzerland, Germany, the Netherlands, and the United Kingdom, longterm injecting drug users that do not benefit from methadone and other medication options are being treated with pure injectable diamorphine that is administered twice daily under the supervision of medical staff. For this group of patients, diamorphine treatment has proven superior in improving their social and health situation. Studies show that even after years of homelessness and delinquency and despite severe comorbidities, about half of the patients find employment within the first year of treatment.

Experimental Treatments

- Ibogaine
- Dextromethorphan
- Ketamine
- Apomorphine
- Medical Cannabis

Barbiturate dependence

With regular use of barbiturates, barbiturate dependence develops. This in turn may lead to a need for increasing doses of the drug to get the original desired pharmacological or therapeutic effect. Barbiturate use can lead to both addiction and physical dependence, and as such they have a high potential for abuse. Psychological addiction to barbiturates can develop quickly. The GABAA receptor, one of barbiturates’ main sites of action, is thought to play a pivotal role in the development of tolerance to and dependence on barbiturates, as well as the euphoric "high" that results from their abuse. The mechanism by which barbiturate tolerance develops is believed to be different than that of ethanol or benzodiazepines, even though these drugs have been shown to exhibit cross-tolerance with each other. The management of a physical dependence on barbiturates is stabilisation on the long-acting barbiturate phenobarbital followed by a gradual titration down of dose. The slowly eliminated phenobarbital lessens the severity of the withdrawal syndrome and reduces the chances of serious barbiturate withdrawal effects such as seizures.
Antipsychotics are not recommended for barbiturate withdrawal (or other CNS depressant withdrawal states) especially clozapine, olanzapine or low potency phenothiazines e.g. chlorpromazine as they lower the seizure threshold and can worsen withdrawal effects; if used extreme caution is required.

**Cocaine dependence**

Cocaine dependence (or addiction) is psychological dependency on the regular use of cocaine. It can result in cardiovascular and brain damage, specifically in the central nervous system.

The use of cocaine, depending of the severity, can cause mood swings, paranoia, insomnia, psychosis, high blood pressure, tachycardia, panic attacks, cognitive impairments and drastic changes in the personality that can lead to aggressive, compulsive, criminal and/or erratic behaviors.

The symptoms of cocaine withdrawal (also known as comedown or crash) range from moderate to severe: dysphoria, depression, anxiety, psychological and physical weakness, pain and compulsive craving.

Historically, the addiction was known as cocainism.

**Risk**

According to a study of 1081 US residents aged over 11 years who had used cocaine for the first time within 24 months prior to assessment, the risk of becoming cocaine-dependent within 2 years of first use (recent-onset) is 5-6%; after 10 years, it increases to 15-16%. These are the aggregate rates for all types of use considered, i.e., smoking, snorting, injecting. Among recent-onset users, the relative rates are higher for smoking (3.4 times) and much higher for injecting. They also vary, based on other characteristics, such as sex: among recent-onset users, women are 3.3 times more likely to become addicted, compared with men; age: among recent-onset users, those who started using at ages 12 or 13 were 4 times as likely to become addicted, compared with those who started between ages 18 and 20.

However, a study of non-deviant users in Amsterdam found "relative absence of destructive and compulsive use patterns over a ten year period" and concluded that cocaine users can and do exercise control. "Our respondents applied two basic types of controls to themselves: 1) restricting use to certain situations and to emotional states in which cocaine's effects would be most positive, and 2) limiting mode of ingestion to snorting of modest amounts of cocaine, staying below 2.5 grams a week for some, and below 0.5 grams a week for most. Nevertheless, those whose use level exceeded 2.5 grams a week all returned to lower levels."

Louis Lewin wrote in Phantastica (1924) of the craving:
During recent years I have seen among men of science frightful symptoms due to the craving for cocaine. Those who believe they can enter the temple of happiness through the gate of pleasure purchase their momentary delights at the cost of body and soul. They speedily pass through the gate of unhappiness into the night of the abyss.

**Presentation**

The immediate craving of the addict for more soon after use is due to the short-lived high that usually subsides within an hour, leading to prolonged, multi-dose binge use. When administration stops after binge use, it is followed by a "crash" (also known as a "come down"), the onset of severely dysphoric mood with escalating exhaustion until sleep is achieved, which is sometimes accomplished by taking sleeping medications, or sedatives, a popular one being Seroquel, or by combination use of alcohol and cannabis. Resumption of use may occur upon awakening or may not occur for several days, but the intense euphoria of such use can, as it has in many users, produce intense craving and develop rather quickly into addiction.

Many habitual abusers develop a transient manic-like condition similar to amphetamine psychosis and schizophrenia, whose symptoms include aggression, severe paranoia, and tactile hallucinations (including the feeling of insects under the skin, or "coke bugs, also known as formication.") during binges.

Cocaine has positive reinforcement effects, which refers to the effect that certain stimuli have on behavior. Good feelings become associated with the drug, causing a frequent user to take the drug as a response to bad news or mild depression. This activation strengthens the response that was just made. If the drug was taken by a fast acting route such as injection or inhalation, the response will be the act of taking more cocaine, so the response will be reinforced. Powder cocaine, being a club drug, is mostly consumed in the evening and night hours. Because cocaine is a stimulant, a user will often drink large amounts of alcohol during and after usage or smoke cannabis to dull "crash" or "come down" effects and hasten slumber. Benzodiazepines (e.g., temazepam, alprazolam, clonazepam) are also used for this purpose. Other drugs such as heroin and various pharmaceuticals are often used to amplify reinforcement or to minimize such negative effects, further increasing addiction potential and harmfulness.

**Mechanism of dependence**

Positron Emission Tomography scans showing the average level of dopamine receptors in six primates' brains. Red is high- and blue is low-concentration of dopamine receptors. The higher the level of dopamine, the fewer receptors there will be.

It is speculated that cocaine's intense addictive properties stem partially from its DAT-blocking effects (in particular, increasing the dopaminergic transmission from ventral tegmental area neurons). However, a study has shown that mice with no dopamine transporters still exhibit the rewarding effects of cocaine administration. Later work
demonstrated that a combined DAT/SERT knockout eliminated the rewarding effects. The rewarding effects of cocaine are influenced by circadian rhythms, possibly by involving a set of genes termed "clock genes".

There is a correlation between a variant of the CAMK4 gene and cocaine addiction; a German study found that addicts were 25% more likely to have a variant of the gene than people who did not use cocaine.

However, chronic cocaine addiction is not solely due to cocaine reward. Chronic repeated use is needed to produce cocaine-induced changes in brain reward centers and consequent chronic dysphoria. Dysphoria magnifies craving for cocaine because cocaine reward rapidly, albeit transiently, improves mood. This contributes to continued use and a self-perpetuating, worsening condition, since those addicted usually cannot appreciate that long-term effects are opposite those occurring immediately after use.

**Treatment**

A study published in May, 2008, in the journal Molecular Psychiatry, detailed the effect of long-term cocaine intake on the amount and activity of thousands of proteins in monkeys. The researchers used “proteomic” technology, which enables the simultaneous analysis of thousands of proteins, to compare the "proteome" (all proteins expressed at a given time) between a group of monkeys that self-administered cocaine and a group that did not receive the drug. The study provides a comprehensive assessment of biochemical changes occurring in the cocaine-addicted brain. The profound changes in structure, metabolism and signaling of neurons may explain why relapse occurs and why it is difficult to reverse these changes after the drug use is discontinued.

On February 14, 2011 two Swiss psychologists, Dr. Geneva Francois Crespo and Dr. Sylvie Petit Jean, published two years of trials and research which demonstrates that gambling along with psychotherapy is the best method to break the cocaine addiction cycle. According to Dr. Crespo - "After cocaine is used, it activates the reward center inside the brain. The brain released euphoric hormones and neurotransmitters such as dopamine, oxytocin and adrenaline which act as a mood enhancer. The same occurs with gambling: the brain perceives a reward from winning or the potential of a reward. The reward causes intense moments of happiness, which compensate for the desire to use cocaine". Further research is being conducted on long term relapse rates.

**Withdrawal symptoms**

After taking cocaine on a regular basis, the user will almost inevitably become addicted. When the drug is discontinued immediately, the user will experience what has come to be known as a "crash" along with a number of other cocaine withdrawal symptoms, including paranoia, depression, exhaustion, anxiety, itching, mood swings, irritability, fatigue, insomnia, delirium tremens, an intense craving for more cocaine, and in some cases nausea and vomiting. Some cocaine users also report having similar symptoms to schizophrenia patients and feel that their mind is lost. Some users also report feeling a crawling sensation
on the skin known as "coke bugs". These symptoms can last for weeks or, in some cases, months. Even after most withdrawal symptoms dissipate most users feel the need to continue using the drug; this feeling can last for years. About 30-40% of cocaine addicts will turn to other substances such as medication and alcohol after giving up cocaine. There are various medications on the market to ease cocaine withdrawal symptoms.

**Therapy**

Cognitive Behavioral Therapy (CBT) combined with Motivational Therapy (MT) have proven to be effective to treat drug and alcohol addictions. Cocaine vaccines are on trial that will stop desirable effects from the drug. The National Institutes of Health of US, particularly National Institute on Drug Abuse (NIDA) is researching modafinil, a narcolepsy drug and mild stimulant, as a potential cocaine treatment. Twelve-step programs such as Cocaine Anonymous (modeled on Alcoholics Anonymous) are claimed by participants to be helpful in achieving long-term abstinence; however, the 12 step based programs have no statistically-measurable effect and does not release any quantifiable measure of its success rates. Cocaine addiction continues to be the most difficult to manage behind heroin, and according to some scientists, addiction to cocaine may be almost impossible to stop.

**Medications**

Medications that have been investigated include Acetylcysteine, Baclofen, Bupropion, Vanoxerine, and Vigabatrin.

**See also**

SB-277011-A - a dopamine D3 receptor antagonist, used in the study of cocaine addiction. Where cocaine reduces the threshold for brain electrical self-stimulation in rats, an indication of cocaine’s rewarding effects, SB-277011-A completely reverses this effect.

**Cannabis dependence**

Cannabis dependence is a condition defined in DSM-IV applying the general concept of substance dependence to cannabis.

Despite cannabis being one of the most widely used illicit drugs in the world, controlled trials for cannabis use disorder have only been reported in literature in the last 15 years. Although cannabis dependence is physically non-existent (many clinicians continue to conclude that the relatively mild withdrawal syndrome associated with cannabis indicates that dependence is unlikely and treatment unnecessary), research has shown that some cannabis users may develop cannabis-related problems, including mild dependency.

Evidence for cannabis dependence comes from a number of sources including epidemiological surveys, studies of long-term users, clinical trials of people seeking treatment, controlled experiments on withdrawal and tolerance and laboratory studies on
cannabis brain mechanisms. Budney et al. state that "clinical and epidemiological studies indicate that cannabis dependence is a relatively common phenomenon associated with significant psychosocial abnormality. Basic research has identified a neurobiological system specific to the actions of cannabinoids. Human and non-human studies have demonstrated a valid withdrawal syndrome that is relatively common among heavy marijuana users". In addition, clinical trials evaluating treatments for cannabis dependence indicate that, among other substance dependencies, cannabis dependency is responsive to intervention.

**Worldwide data on cannabis use and dependence**

The Australian National Survey of Mental Health and Wellbeing indicated that approximately 300,000 people (or 2.2% of the adult population of Australia) either used or were dependent on cannabis. Swift et al. estimate that this equates to roughly one in three individuals having used cannabis in the past 12 months. According to Swift, Hall and Teeson (2001), the top four symptoms reported by dependent adults were: withdrawal or using cannabis as withdrawal relief (88.8%); persistent desire or unsuccessful attempts to control use (86.9%); tolerance (72.6%); and using cannabis in larger amounts or for a longer time than intended (62.8%).

Only a minority of cannabis users seek medical help, but demand for treatment for cannabis use disorder is increasing internationally. Gates et al. report that 1.5% of Australians (approximately 200,000) are considered dependent on cannabis and research indicates that 1 in 10 people who try cannabis feel dependent. Hall et al. provide evidence that suggests that among those who have used cannabis more than a few times the risk of developing dependence is in the range of from 1 in 5 to 1 in 3; the more often cannabis has been used, and the longer it has been used, the higher the risk of the feeling of dependence. In addition, the majority of 'dependent' users continue to use cannabis without seeking treatment.

Agosti and Levin (2004) indicate that cannabis-dependent users are more likely to seek professional treatment for dependency if they had previously sought treatment or suffered from alcohol dependence. However, only 1/10 – 1/3 cannabis dependent users will seek treatment within a year. And the percentage of cannabis-dependent users who entered treatment is the lowest of all illicit drugs.

An Australian study showed that of individuals presenting for interventions for cannabis problems, many had been using on an almost daily basis for an average of 14 years and were suffering serious health and psychological consequences from cannabis use. In addition, Arendt and Munk-Jorgensen (2004) report that cannabis-dependent users entering treatment for cannabis dependency were found to have previously suffered from depression, schizophrenia and personality disorders more than people dependent on other drugs. This research indicates that these psychological problems are among the main reasons for seeking treatment for cannabis use.
Studies of long-term and regular cannabis users have found that a variety of cannabis-related problems are reported. For example, among a sample of heavy cannabis users in rural Australia, three in four people reported experiencing a persistent desire for cannabis and frequent intoxication during daily activities. Over half of the survey group (54%) reported tolerance while 5% reported suffering withdrawal symptoms. Swift et al. surveyed long-term cannabis users in Sydney, Australia and found that 78% reported withdrawal and 76% reported tolerance. More than a third (39%) reported using cannabis to relieve withdrawal symptoms.

Hathaway reports that in a study of regular cannabis users in Canada the symptoms most frequently reported for the 12 months prior to the study were using cannabis in larger amounts of for longer than intended (32%) and a persistent desire to cut down or unsuccessful attempts to do so (24%). One in ten (10%) respondents reported giving up or reducing social, recreational or work activities due to cannabis use.

Diagnostic criteria

According to Hall et al. who quotes the Diagnostic and Statistical Manual of the American Psychiatric Association, "the essential feature of Substance Dependence is a cluster of cognitive, behavioral and physiologic symptoms indicating that the individual continues use of the substance despite significant substance-related problems". Accordingly, a diagnosis of substance dependence is made if three or more of the following criteria occur at any time in the same 12-month period:

- Tolerance, as defined by either or all of the following:
  - A need for markedly increased amounts of the substance to achieve intoxication or the desired effect
  - A markedly diminished effect on the user with continued use of the same amount of the substance
- Withdrawal, as manifested by either of the following:
  - Characteristic withdrawal symptoms from the substance, such as insomnia, restlessness, loss of appetite, depression, irritability, and anger.
  - The same or closely related substance is taken to relieve or avoid withdrawal symptoms
  - The substance is often taken in larger amounts or over a longer period than was intended
  - There is a persistent desire to cut back or control substance use, or unsuccessful attempts to do so
  - Considerable time is spent obtaining the substance
  - Social, occupational or recreational activities are given up or reduced because of use of the substance
  - The substance is used despite knowledge of persistent or recurrent physical or psychological problems caused by the substance.

Evidence suggests that cannabis users can develop tolerance to the effects of THC and experience withdrawal symptoms. Tolerance to the behavioral and psychological effects of
THC has been demonstrated in humans and animals. The mechanisms that create this tolerance to THC are thought to involve changes in cannabinoid receptor function.

Addiction potential

Research has shown the overall addiction potential for cannabis to be much less than for tobacco, alcohol, cocaine or heroin, but slightly higher than that for psilocybin, mescaline, LSD, and MDMA. There is some evidence that dependence on cannabis can exist in some heavy users. One study with 500 heavy users of cannabis showed that when trying to cease consumption, some experience one or more symptoms such as insomnia, restlessness, loss of appetite, depression, irritability, and anger. Cannabis Dependence has been recognized as a clinical entity in the DSM-IV. Prolonged marijuana use produces both pharmacokinetic changes (how the drug is absorbed, distributed, metabolized, and excreted) and pharmacodynamic changes (how the drug interacts with target cells) to the body. These changes require the user to consume higher doses of the drug to achieve a common desirable effect (known as a higher tolerance), and reinforce the body’s metabolic systems for synthesizing and eliminating the drug more efficiently. It is clear that cannabis ultimately acts through the mesolimbic dopaminergic system of reinforcement, just as all other addictive substances act.

Preliminary research, published in the April 2006 issue of the Journal of Consulting and Clinical Psychology, indicates that cannabis addiction can be offset by a combination of cognitive-behavioral therapy and motivational incentives. Participants in the study (previously diagnosed with marijuana dependence) received either vouchers as incentives to stay drug free, cognitive-behavioral therapy, or both over a 14-week period. At the end of 3 months, 43 percent of those who received both treatments were no longer using marijuana, compared with 40 percent of the voucher group, and 30 percent of the therapy group. At the end of a 12-month follow-up, 37 percent of those who got both treatments remained abstinent, compared with 17 percent of the voucher group, and 23 percent of the therapy group.

A 1998 French governmental report commissioned by Health Secretary of State Bernard Kouchner, and directed by Dr. Pierre-Bernard Roques, classed drugs according to addictiveness and neurotoxicity. It placed heroin, cocaine and alcohol in the most addictive and lethal categories; benzodiazepine, hallucinogens and tobacco in the medium category, and cannabis in the last category. The report stated that "Addiction to cannabis does not involve neurotoxicity such as it was defined in chapter 3 by neuroanatomical, neurochemical and behavioral criteria. Thus, former results suggesting anatomic changes in the brain of chronic cannabis users, measured by tomography, were not confirmed by the accurate modern neuro-imaging techniques. Moreover, morphological impairment of the hippocampus [which plays a part in memory and navigation] of rats after administration of very high doses of THC (Langfield et al., 1988) was not shown (Slikker et al., 1992)." Health Secretary Bernard Kouchner concluded that: "Scientific facts show that, for cannabis, no neurotoxicity is demonstrated, to the contrary of alcohol and cocaine."
In treating marijuana use, Dr. David McDowell of Columbia University found that there is a
need for the clinician to differentiate in the spectrum between a casual user who still has
difficulty with drug screens, and a daily, heavy user. McDowell found that the sedating and
anxiolytic properties of THC in some users might make the use of cannabis an attempt to
self-medicate personality or psychiatric disorders.

Risk factors for developing cannabis dependency

Hall et al. conclude that around one in ten people who ever try cannabis will become
dependent at some point. For those who use cannabis several times the chance is increased
from one in five to one in three and daily users are considered at the greatest risk of
dependence with about a one in two chance.

Certain factors are considered to heighten the risk of developing cannabis dependence and
longitudinal studies over a number of years have enabled researchers to track aspects of
social and psychological development concurrently with cannabis use. Increasing evidence
is being shown for the elevation of associated problems by the frequency and age at which
cannabis is used, with young and frequent users being at most risk.

Cross-sectional studies that examine the association between conduct disorder and
attention deficit hyperactivity disorder have reported a significant association in
community and in treatment populations with cannabis use and dependence among
adolescents. Although early cannabis initiation is considered a strong predictor of later
cannabis-related problems, findings that early cannabis initiators are a group already
facing social problems have been supported by longitudinal research in Australia. Coffey et
al., in a study of 2032 secondary school students in Victoria, found that mid-school
cannabis use was associated with factors including daily cigarette smoking, peer cannabis
use and anti-social behaviour. The study also found that regular use at an early age
predicted persistence in use from mid- to late-school, with potentially harmful late-school
use occurring in 12% of mid-school initiators. A recent follow-up of this group at age 20-21
found that one in five adolescent users experienced later cannabis dependence.

According to Copeland, Gerber and Swift, the main factors related to a heightened risk for
developing problems with cannabis use include frequent use at a young age; personal
maladjustment; emotional distress; poor parenting; school drop-out; affiliation with drug-
using peers; moving away from home at an early age; daily cigarette smoking; and ready
access to cannabis. The researchers conclude there is emerging evidence that positive
experiences to early cannabis use are a significant predictor of late dependence and that
gene tic predisposition plays a role in the development of problematic use.

Groups at higher risk of developing cannabis dependence

A number of groups have been identified as being at greater risk of developing cannabis
dependence and include adolescent populations, Aboriginal and Torres Strait Islanders (in
Australia) and people suffering from mental health conditions.
Adolescent populations

In their review of the literature, McLaren and Mattick indicate that young people are at greater risk of developing cannabis dependency because of the association between early initiation into substance use and subsequent problems such as dependence, and the risks associated with using cannabis at a developmentally vulnerable age. In addition there is evidence that cannabis use during adolescence, at a time when the brain is still developing, may have deleterious effects on neural development and later cognitive functioning.

Aboriginal and Torres Strait Islanders (an Australian perspective)

There is evidence that cannabis use occurs at higher rates among Aboriginal and Torres Strait Islander peoples when compared to the general population in Australia. This is part of a broader picture of poor health and wellbeing, stemming from the alienation and dispossession experienced by this population over time. Many of the social determinants of harmful substance use are disproportionately present in Aboriginal and Torres Strait Islander communities.

Psychiatric disorders

McLaren and Mattick show a correlation between populations who suffer from a mental disorder such as schizophrenia and a worsening of these symptoms with cannabis use. In addition, people who are vulnerable to developing psychosis, such as people with a family history of the disorder or with a genetic predisposition, may be at risk of developing a psychotic disorder following frequent cannabis use. Hall and Solowij indicate that given this risk the finding that cannabis use if higher among those with mental health problems than those who do not suffer from such problems is of concern and this population should be treated as a group at risk of adverse effects from cannabis use. This is an especially challenging group to engage and retain in treatment and "clinician’s recommendations for the management of substance use in the context of severe and persistent mental illness rests with integrated shared care or dual diagnosis services, in which the critical components are assertive outreach, motivational interventions, skilled counseling, social support interventions, a comprehensive and long-term perspective and cultural sensitivity and competence".

Treatment

Demand for treatment for cannabis dependency is increasing internationally. Cannabis is responsible for most illicit drug admissions in the USA, with a 32% increase in the proportion of admissions for cannabis-related problems from 12% in 1996 to 16% in 2006. The most commonly accessed forms of treatment, according to Copeland and Swift, were 12-step programmes, physicians, rehabilitation programmes, and detox services, with inpatient and outpatient services equally accessed (each approximately 10%). In the EU approximately 20% of all primary admissions and 29% of all new drug clients in 2005, had primary cannabis problems. And in all countries that reported data between 1999-2005 the number of people seeking treatment for cannabis use increased. In Australia between
2006 and 2007, cannabis was the second most common principal drug of concern for which treatment was sought after alcohol, accounting for 23% of closed treatment episodes. Among 10-19-year-old clients, cannabis represented 47% of episodes compared to 29% for alcohol. Stephens et al. describe the symptoms reported by 382 people who signed up for treatment for cannabis dependency. These included: "an inability to stop using (93%), feeling bad about using cannabis (87%), procrastinating (86%), loss of self-confidence (76%), memory loss (67%) and withdrawal symptoms (51%).

Treatment options for cannabis dependence are far fewer than for opiate or alcohol dependence. Most treatment falls into the categories of psychological or psychotherapeutic, intervention, pharmacological intervention or treatment through peer support and environmental approaches. McRae, Budney and Brady postulate that, as with alcohol research, the therapeutic effects of pharacotherapy and psychotherapy may be synergistic, with greatest treatment efficacy seen when medications are combined with psychotherapy, as per the research of Anton et al., 1999. Degenhardt et al. indicate that screening and brief intervention sessions can be given in a variety of settings, particularly at doctor’s surgeries, which is of importance as most cannabis users seeking help will do so from their general practitioner rather than a drug treatment service agency. Hall and Swift conclude that brief intervention sessions should involve the provision of personalized advice about the client’s cannabis use, information about cannabis use and dependence and self-help materials. Evidence suggests that there is value in brief sessions, even for highly dependent cannabis users, and treatment outcomes for cannabis-dependent individuals is considered comparable to those suffering from dependence on other substances.

**Psychological intervention**

Psychological intervention is most commonly Cognitive Behavioral Therapy (CBT) or Motivational Interviewing (MI). According to Copeland et al., while CBT examines the interplay between thoughts, behaviour and environment, the main aim of MI is to enhance the motivation of the participant to change. Stevens et al. conducted the first psychological intervention study in the US, with a sample of 212 heavy cannabis users. Participants were assigned to either a 10-week social support group or a 10-week relapse prevention group with a CBT focus. The support group took part in discussions that centered on issues such as: the giving and receiving of support; dealing with denial; and affiliating with friends who still used cannabis The 10-week relapse prevention CBT group included planned exercises, homework and formalised quit contracts undertaken between participant and counsellor. A 12 month follow up revealed similar rates of reduction in cannabis use for both groups (15.2% remained abstinent in the CBT group; 18.1% in the social support group) and one in five people from both groups were judged to have improved, either reporting a 50% less usage than pre-treatment levels or no cannabis related problems.

Later follow up of this study saw Stevens et al. introduce a delayed treatment condition, offered four months later than in the active treatment groups, a 14-week CBT relapse prevention group and a brief two-session MI interview (to create a control group for comparison). Results showed that at the follow-up participants in the active treatment groups had a significantly lower number of dependence symptoms and fewer cannabis-
related problems compared to the delayed treatment group. Abstinence rates at four months were 37% for both active groups, compared to 9% for the delayed treatment condition.

An Australian study undertaken by Copeland, Swift, Roffman and Stevens further supported the effectiveness of brief interventions for cannabis use. 229 cannabis users were allocated treatment in either six sessions of CBT, one session of CBT or a delayed treatment control group. The CBT interventions incorporated elements of MI and thereby compared two matching therapies, the only difference being in length. At follow up it was found that 15.1% of participants in the six-session CBT group had achieved continuous abstinence as compared to 4.9% in the single session CBT group and 0% in the delayed treatment group. In addition, those in the active treatment groups were considered to be less severely dependent than before the study and reported "higher levels of control over their cannabis use and fewer cannabis-related problems compared to those in the delayed treatment group"

Budney et al. conducted a smaller study that tested the effect of a voucher system, whereby heavy cannabis users were offered vouchers that could be exchanged for retail items in exchange for abstinence.

Participants had to provide cannabinoid-free urine samples to be eligible for the vouchers, the rationale of the study being that previous research indicated that voucher incentives, in conjunction with behavioral interventions, improved the treatment outcome of cocaine-dependent individuals. Results showed that the group receiving motivational enhancement (ME), CBT and voucher incentives (as opposed to ME; ME and CBT) were more likely to have been abstinent during the last week of treatment (35% as opposed to ME/ CBT: 10%; MET: 5%). Furthermore, at 30 days post treatment although all groups reported using substantially less cannabis than before treatment, there was a higher reduction for the voucher group. A recent study with cannabis users referred from probation strengthens these findings further. Participants were involved in either three sessions of motivational enhancement therapy, or this same therapy with added vouchers for attendance. Results showed that significant reductions in cannabis use were noted in both groups, however, more participants in the voucher group completed the treatment. Research undertaken by Copeland and the Cannabis Centre at the University of NSW indicates that although relatively brief, CBT has the strongest evidence of success for adults with cannabis dependence, among adolescents involved in the juvenile justice system and those with severe persistent mental illness.

**Pharmacological intervention**

Research that relates to pharmacological intervention for cannabis dependency is in its infancy. Carl Hart, in the journal Drug and Alcohol Dependence reviews data from recent research on cannabinoids. The discussion considers the findings from studies that have assessed the ability of medications to ameliorate cannabis-related symptoms in laboratory animals and human research participants. In addition, "data from studies that have investigated the effects of pharmacological agents on cannabis self-administration are also
reviews because these data may provide information critical for informing relapse prevention medication development efforts”. A number of small-scale trials have examined the impact of mood-altering substances on cannabis withdrawal, and the impact of drugs that block the acute effects of cannabis. Drugs such as Bupropion, Nefazodone and Lithium Carbonate have all been tested with variable results. Studies that consider the effects of oral THC maintenance for cannabis craving and withdrawal also produced mixed results. Hanley et al. proved that the administration of oral THC had no significant effect on the frequency at which participants chose to smoke cannabis. A 2001 study published in the Journal of Neuroscience, however, indicated that the effects of a mood stabilizer (divalproex) and oral THC on cannabis cravings and withdrawal symptoms effectively reduced cannabis craving and very low doses of oral THC were effective in decreasing all measured withdrawal symptoms in addition to craving. The use of antagonistic pharmacotherapies, agents that block the effects of drugs by binding to receptors in the brain, is used in the treatment of opiates, alcohol and nicotine. One drug that shows promise in this field is CB1 cannabinoid receptor antagonist SR141716A (Rimonabant), which inhibits signs of THC intoxications in monkeys, rats and pigeons. A human clinical trial undertaken in 2002 found that SR141716 blocked the acute effects of smoked cannabis.

Columbia University, in collaboration with the National Institute on Drug Abuse (NIDA), is undertaking a clinical trial that looks at the effects of combined pharmacotherapy on cannabis dependency, to see if Lofexidine in combination with Marinol is superior to placebo in achieving abstinence, reducing cannabis use and reducing withdrawal in cannabis-dependent patients seeking treatment for their marijuana use. 180 men and women between the ages of 18-60 who met DSM-IV criteria for current marijuana dependence were enrolled in a 12 week trial that started in January 2010.

Peer support and environmental approaches

Self-help groups that strongly endorse the therapeutic potential of peer support, such as Narcotics Anonymous (NA), are increasingly used as an approach to cannabis dependency. The only requirement for membership at NA is a 'desire to stop using drugs'. Twelve step programs such as NA view addiction as a disease, with complete abstinence the only option for recovery; the support of a former addict helping another is at the core of the program's philosophy and people who become a part of the NA program acquire a 'sponsor', someone who provides personal support and helps recovering addicts implement the 12 steps. These steps include belief in a higher power and keeping a fearless moral inventory of oneself.

Evaluations of Marijuana Anonymous programs, modelled on the 12-step lines of Alcoholics Anonymous and Narcotics Anonymous, have shown small beneficial effects for general drug use reduction.

Barriers to cannabis treatment
Research that looks at barriers to cannabis treatment frequently cites a lack of interest in treatment, lack of motivation and knowledge of treatment facilities, an overall lack of facilities, costs associated with treatment, difficulty meeting program eligibility criteria and transport difficulties. According to Marlatt et al., the most frequently reported social barrier to treatment entry is the stigma associated with being labelled as an illicit drug user and associated concerns over privacy. A recent technical report compiled by Australia’s National Cannabis Centre

**Quitting Cannabis**

Cannabis is the most widely used illicit drug in the Western world and there is little, if any evidence that describes the addictive nature of cannabis. There is plenty of documented evidence to suggest a need for users to find ways to assist them to stop using cannabis and the demand for treatment for cannabis dependency is increasing internationally. There are a number of ways to quit cannabis and increasing evidence-based treatments for cannabis users wishing to change the patterns of their use. Luckily withdrawal symptoms from prolonged cannabis use are mild with most users experiencing no symptoms.

**Hallucinogen persisting perception disorder**

Hallucinogen persisting perception disorder (HPPD) is a disorder characterized by a continual presence of visual disturbances that are reminiscent of those generated by the ingestion of hallucinogenic substances. Previous use of hallucinogens by the person is needed, though not sufficient, for diagnosing someone with the disorder. For an individual to be diagnosed with HPPD, the symptoms cannot be due to another medical condition. HPPD is distinct from flashbacks by reason of its relative permanence; while flashbacks are transient, HPPD is persistent. HPPD is a DSM-IV diagnosis with diagnostic code 292.89.

**Symptoms**

There are a number of perceptual changes that can accompany HPPD. Typical symptoms of the disorder include: halos or auras surrounding objects, trails following objects in motion, difficulty distinguishing between colors, apparent shifts in the hue of a given item, the illusion of movement in a static setting, air assuming a grainy or textured quality (visual snow or static, by popular description, which may also be caused by the normal "blue field entoptic phenomenon"), distortions in the dimensions of a perceived object, and a heightened awareness of floaters. The visual alterations experienced by those with HPPD are not homogeneous and there appear to be individual differences in both the number and intensity of symptoms.

Visual aberrations can occur periodically in healthy individuals - e.g. afterimages after staring at a light, noticing floaters inside the eye, or seeing specks of light in a darkened room. However, in people with HPPD, symptoms are typically so severe that the individual cannot ignore them and HPPD is associated with new visual disturbances. It does not appear to merely increase those already in existence.
It also should be noted that the visuals do not constitute true hallucinations in the clinical sense of the word; people with HPPD recognize the visuals to be illusory, or pseudohallucinations, and thus demonstrate no inability to determine what is real (in contrast to, e.g., Schizophrenia).

**Prevalence of HPPD**

The probability of developing HPPD after consuming a hallucinogen is unknown. In their review article, John Halpern and Harrison Pope write that "the data do not permit us to estimate, even crudely, the prevalence of 'strict' HPPD." These authors noted that they had not encountered it in their evaluation of 500 Native American Church members who had taken the hallucinogenic cactus peyote on at least 100 occasions. In a presentation of preliminary results from ongoing research, Matthew Baggott and colleagues from University of California Berkeley found that HPPD-like symptoms occurred in 4.1% of participants (107 of 2,679) in a web-based survey of hallucinogen users. These people reported visual problems after drug use that were serious enough that they considered seeking professional help. This number may over-estimate the prevalence of HPPD since people with visual problems may have been more interested in completing the researchers' questionnaire. The authors reported that 16,192 people viewed the study information but did not complete the questionnaire. If all these people had used hallucinogens without developing visual problems, then the prevalence of serious visual problems in this larger group would be 0.66%. Since these people were not formally diagnosed in person (and may have had visual problems caused by other disorders), this number may provide a reasonable upper limit on the prevalence of HPPD.

It is possible the prevalence of HPPD has been underestimated by authorities because many people with visual problems relating to drug use either do not seek treatment or, when they do seek treatment, do not admit to having used illicit drugs. In the sample of Baggott, only 16 of the 107 people with possible HPPD had sought help and two of these people had been diagnosed with HPPD. Thus, it may be that HPPD occurs more often than is detected by the health care system.

**Causes**

The cause(s) of HPPD are not yet known. The most current neurological research indicates that HPPD symptoms may manifest from abnormalities in CNS function, following the hallucinogen use. One theory derived from this research is that inhibitory mechanisms involved with sensory gating are disrupted.

In some cases, HPPD appears to have a sudden onset after a single drug experience, strongly suggesting the drug played a direct role in triggering symptoms. But in other cases, people report gradual worsening of symptoms with ongoing drug use. Drugs that have been associated with HPPD include LSD, 2C-E, 2C-I, 5-MeO-DIPT, MDMA, Psilocybin, Mescaline, diphenhydramine, PCP and high doses of dextromethorphan.
Co-existing problems

The visual problems of HPPD can occur along with other mental ailments. Of these, the most prominent are anxiety, panic attacks, depersonalization disorder, and depression. In the sample of Baggott and colleagues, hallucinogen users with persisting and severe visual problems were significantly more likely to report anxiety and depression diagnoses than hallucinogen users without serious visual problems. For example, 25.9% of hallucinogen users with visual problems reported current or past diagnosis of depression. While it is difficult, if not impossible, to establish a clear relationship between the visual and mental symptoms, those with HPPD often testify that a connection indeed exists. For example, anxiety can cause the visuals to become more prominent and vice-versa. Anecdotal wisdom thus maintains that there is a synergistic link between the two. However, there appear to be people with 'pure' cases of HPPD in which no other disorders co-exist.

Treatment

As of yet, there is no cure available for HPPD. The principal treatments seek to reduce symptoms and distress without treating underlying causes. Benzodiazepines including clonazepam (Klonopin), diazepam (Valium) and alprazolam (Xanax) are prescribed with a fair amount of success. Some medications have been contraindicated on the basis of their effects on HPPD or the concurrent mental issues. The atypical antipsychotic Risperidone is reported to worsen symptoms of HPPD during the drug’s duration in some people.

Those with HPPD are often advised to discontinue all drug use, many of which are thought to increase visuals in the short-term. There are also less concrete factors that may be generally detrimental to those with HPPD. For example, sleep deprivation and stress are thought to increase HPPD symptoms. However, no published studies have investigated whether any of these recommendations are helpful.

There is no universal time course of HPPD recovery. The adverse psychological effects of HPPD (assuming these effects appeared at all) appear to lessen more rapidly than the visuals; quality of life often returns as a person adjusts. Recovery may be facilitated by a psychological habituation to the visuals, which, in effect, reduces the victim's inclination to attend to and react negatively to them. The deleterious consequences of the visuals can therefore be reduced even if the HPPD does not disappear.

There is currently little reliable information on how often people fully recover from HPPD. There have been reports of HPPD victims having normal perception totally return. The small number of cases of HPPD that have been studied in depth make it difficult to determine how often and under what conditions the visual symptoms of HPPD resolve.

Current Research

A HPPD research program is under development at McLean Hospital, a teaching affiliate of Harvard Medical School. David Kozin, consulting with multiple investigators, is involved in designing HPPD-related research.
Other disorders with similar symptoms

It must be emphasized that individuals without HPPD will sometimes notice visual abnormalities. These include floaters (material floating in the eye fluid that appears as black/dark objects floating in front of the eyes and are particularly visible when looking at the bright sky or on a white wall) and the white blood cells of the retinal blood vessels (seen as tiny, fast-moving and quickly disappearing white specks). Likewise, bright lights in an otherwise dark environment may generate trails and halos. Most people don’t notice these effects, because they are so used to them. A person fearful of having acquired HPPD may be much more conscious about any visual disturbance, including those that are normal. In addition, visual problems can be caused by brain infections or lesions, epilepsy, and a number of mental disorders (e.g., delirium, dementia, schizophrenia, Parkinson’s disease). For an individual to be diagnosed with HPPD, these other potential causes must be ruled out.

Inhalant abuse

Inhalants are a broad range of drugs whose volatile vapors are taken in via the nose and trachea.

Inhalants are taken by volatilization, and do not include drugs that are inhaled after burning or heating. For example, amyl nitrite and toluene are considered inhalants, but tobacco, marijuana, and crack are not.

While some inhalant drugs are used for medical purposes, as in the case of nitrous oxide (a dental anaesthetic), this article focuses on inhalant abuse as recreational drugs that are used for their intoxicating effect. Inhaling volatile substances because of their intoxicating effect is called huffing.

Classification

Inhalants can be classified by the intended function. Most inhalant drugs that are used non-medically are ingredients in household or industrial chemical products that are not intended to be concentrated and inhaled. A small number of recreational inhalant drugs are pharmaceutical products that are used illicitly.

Inhalants can also be classified by chemical structure. Classes include:

- **aliphatic hydrocarbons** - petroleum products (gasoline and kerosene), propane, butane
- **aromatic hydrocarbons** - toluene, xylene
- **ketones** - acetone (nail polish remover)
- **haloalkanes** - hydrofluorocarbons, chlorofluorocarbon, trichloroethylene, 1,1,1-
  Trichloroethane (including many aerosols and propellants) 1,1,1-
  trichloroethane-
- **nitrites** - alkyl nitrites (poppers such as amyl nitrite), nitrous oxide

It is also possible to classify inhalants by the effect they have on the body. Many inhalants act primarily as asphyxiating gases, with their primary effect due to oxygen deprivation. Other agents may have more direct effects at receptors.

**Inhalant users**

The most serious inhalant abuse occurs among children and teens who "...live on the streets completely without family ties." Inhalant users inhale vapor or aerosol propellant gases using plastic bags held over the mouth or by breathing from a solvent-soaked rag or an open container. The effects of inhalants range from an alcohol-like intoxication and intense euphoria to vivid hallucinations, depending on the substance and the dosage. Some inhalant users are injured due to the harmful effects of the solvents or gases or due to other chemicals used in the products that they are inhaling. As with any recreational drug, users can be injured due to dangerous behavior while they are intoxicated, such as driving under the influence. In some cases, users have died from hypoxia (lack of oxygen), pneumonia, cardiac failure or arrest, or aspiration of vomit.

**Administration and effects**

Inhalant users inhale vapors or aerosol propellant gases using plastic bags held over the mouth or by breathing from an open container of solvents, such as gasoline or paint thinner. Nitrous oxide gases from whipped cream aerosol cans, aerosol hairspray or non-stick frying spray are sprayed into plastic bags. When inhaling non-stick cooking spray or other aerosol products, some users may filter the aerosolized particles out with a rag. Some gases, such as propane and butane gases, are inhaled directly from the canister. Once these solvents or gases are inhaled, the extensive capillary surface of the lungs rapidly absorb the solvent or gas, and blood levels peak rapidly. The intoxication effects occur so quickly that the effects of inhalation can resemble the intensity of effects produced by intravenous injection of other psychoactive drugs.

The effects of solvent intoxication can vary widely depending on the dose and what type of solvent or gas is inhaled. A person who has inhaled a small amount of rubber cement or paint thinner vapor may be impaired in a manner resembling alcohol inebriation. A person who has inhaled a larger quantity of solvents or gases, or a stronger chemical, may experience stronger effects such as distortion in perceptions of time and space, hallucinations, and emotional disturbances.

In the short term, many users experience headache, nausea and vomiting, slurred speech, loss of motor coordination, and wheezing. A characteristic "glue sniffer's rash" around the nose and mouth is sometimes seen after prolonged use. An odor of paint or solvents on
clothes, skin, and breath is sometimes a sign of inhalant abuse, and paint or solvent residues can sometimes emerge in sweat.

Computer-cleaning dusters are dangerous to inhale, because the gases expand and cool rapidly upon being sprayed.

**Mechanisms of action**

Inhalants are a large class of drugs and therefore exhibit a variety of mechanisms of action. The mechanisms of action of many non-medical inhalants have not been well elucidated. Anesthetic gases used for surgery, such as nitrous oxide or enflurane, are believed to induce anesthesia primarily by acting as NMDA receptor antagonists, open channel blockers that bind to the inside of the calcium channels on the outer surface of the neuron, and provide high levels of NMDA receptor blockade for a short period of time.

This makes inhaled anesthetic gases different from other NMDA antagonists, such as ketamine, which bind to a regulatory site on the NMDA-sensitive calcium transporter complex and provide slightly lower levels of NMDA blockade, but for a longer and much more predictable duration. This makes a deeper level of anesthesia achievable more easily using anesthetic gases but can also make them more dangerous than other drugs used for this purpose.

Alcohol is known to act as a GABA agonist. The solvent diethyl ether has seen historical episodes of both inhalation and drinking and produces effects suggestive of both NMDA- and GABA-mediated activity.

**Dangers and health problems**

Statistics on deaths caused by inhalant abuse are difficult to determine. It may be severely underreported, because death is often attributed to a discrete event such as a stroke or a heart attack, even if the event happened because of inhalant abuse. Inhalant use or abuse was mentioned on 144 death certificates in Texas during the period 1988–1998 and was reported in 39 deaths in Virginia between 1987 and 1996 from acute voluntary exposure to abused inhalants.

**General risks of inhalants**

Regardless of which inhalant is used, improper administration can lead to death or injury. One major risk is hypoxia, which can occur due to inhaling fumes from a plastic bag, or from using proper equipment but not adding oxygen or room air. When a gas that was stored under high pressure is released, it cools abruptly and can cause frostbite if it is inhaled directly from the container. Finally, many inhalants are volatile organic chemicals and can catch fire or explode. As with many other drugs, users may also injure themselves due to loss of coordination or impaired judgment, especially if they attempt to drive.
Solvents have many potential risks in common, including pneumonia, cardiac failure or arrest, and aspiration of vomit. The inhaling of some solvents can cause hearing loss, limb spasms, and damage to the central nervous system and brain. Serious but potentially reversible effects include liver and kidney damage and blood-oxygen depletion. Death from inhalants is generally caused by a very high concentration of fumes. Deliberately inhaling solvents from an attached paper or plastic bag or in a closed area greatly increases the chances of suffocation. Brain damage is typically seen with chronic long-term use as opposed to short-term exposure.

Female inhalant users who are pregnant may have adverse effects on the fetus, and the baby may be smaller when it is born and may need additional health care (similar to those seen with alcohol - Fetal Alcohol Syndrome). There is some evidence of birth defects and disabilities in babies born to women who sniffed solvents such as gasoline.

In the short term, death from solvent abuse occurs most commonly from aspiration of vomit while unconscious or from a combination of respiratory depression and hypoxia, the second cause being especially a risk with heavier-than-air vapors such as butane or gasoline vapor. Deaths typically occur from complications related to excessive sedation and vomiting. Actual overdose from the drug does occur, however, and inhaled solvent abuse is statistically more likely to result in life-threatening respiratory depression than intravenous use of opiates such as heroin. Most deaths from solvent abuse could be prevented if individuals were resuscitated quickly when they stopped breathing and their airway cleared if they vomited. However, most inhalant abuse takes place when people inhale solvents by themselves or in groups of people who are intoxicated. Certain solvents are more hazardous than others, such as gasoline.

In contrast, a few inhalants like amyl nitrate and diethyl ether have medical applications and are less harmful, though they are still dangerous when used recreationally. Nitrous oxide is thought to be particularly non-toxic, though long-term use can lead to a variety of serious health problems linked to destruction of vitamin B12 and folic acid.

**Risks associated with specific agents**

The hypoxic effect of inhalants can cause damage to many organ systems (particularly the brain, which has a very low tolerance for oxygen deprivation), but there can also be additional toxicity resulting from either the physical properties of the compound itself or additional ingredients present in a product.

- Methylene chloride, after being metabolized, can cause carbon monoxide poisoning.
- Gasoline sniffing can cause lead poisoning, though this is less common where leaded gas is banned.
- Ingestion of alkyl nitrites can cause methemoglobinemia, although inhalation does not.
- Carbon tetrachloride can cause significant damage to multiple systems, but its association with liver damage is so strong that it is used in animal models to induce liver injury.
- Use of butane and propane can create a risk of burns.
- Benzene use can cause bone marrow depression.
- Toluene can damage myelin.

Toxicity may also result from the pharmacological properties of the drug; excess NMDA antagonism can completely block calcium influx into neurons and provoke cell death through apoptosis, although this is more likely to be a long-term result of chronic solvent abuse than a consequence of short-term use.

"Sudden sniffing death"

Inhaling butane gas can cause drowsiness, narcosis, asphyxia, cardiac arrhythmia and frostbite. Butane is the most commonly misused volatile solvent in the UK and caused 52% of solvent-related deaths in 2000. By spraying butane directly into the throat, the jet of fluid can cool rapidly to ~20°C by adiabatic expansion, causing prolonged laryngospasm. Some inhalants can also indirectly cause sudden death by cardiac arrest, in a syndrome known as "sudden sniffing death." The anesthetic gases present in the inhalants appear to sensitize the user to adrenaline. In this state, a sudden surge of adrenaline (e.g., from a frightening hallucination or run-in with the law), can cause a fatal cardiac arrhythmia.

Furthermore, the inhalation of any gas that is capable of displacing oxygen in the lungs (especially gasses heavier than oxygen) carries the risk of hypoxia due to the mechanism by which breathing is triggered. Since reflexive breathing results from elevated carbon dioxide levels, rather than depressed oxygen in the blood, breathing a concentrated, relatively inert gas (such as the computer-duster tetrafluoroethane or nitrous oxide) will allow for adequate elimination of carbon dioxide from the blood, meaning that there are no outward signs of suffocation even when the brain is undergoing hypoxia. By the time the full symptoms of hypoxia appear, it may be too late to breathe without assistance, especially if the gas is heavy enough to reside in the lungs for extended periods. Even completely inert gasses, such as argon, can have this effect if oxygen is largely excluded (e.g., via a mask).

Socioeconomic factors

Inhalant drugs are often used by children, teenagers, incarcerated or institutionalized people, and impoverished people, because these solvents and gases are ingredients in hundreds of legally available, inexpensive products, such as deodorant sprays, hair spray, and aerosol air fresheners. However, most users tend to be "...adolescents (between the ages of 12 and 17)." In some countries, chronic, heavy inhalant use is concentrated in marginalized, impoverished communities. Young people who become chronic, heavy
inhalant abusers are also more likely to be those who are isolated from their families and community. The article "Epidemiology of Inhalant Abuse: An International Perspective" notes that "[t]he most serious form of obsession with inhalant use probably occurs in countries other than the United States where young children live on the streets completely without family ties. These groups almost always use inhalants at very high levels (Leal et al. 1978). This isolation can make it harder to keep in touch with the sniffer and encourage him or her to stop sniffing."

The article also states that "...high [inhalant use] rates among barrio Hispanics almost undoubtedly are related to the poverty, lack of opportunity, and social dysfunction that occur in barrios" and states that the "...same general tendency appears for Native-American youth" because "...Indian reservations are among the most disadvantaged environments in the United States; there are high rates of unemployment, little opportunity, and high rates of alcoholism and other health problems." There are a wide range of social problems associated with inhalant use, such as feelings of distress, anxiety and grief for the community; violence and damage to property; violent crime; stresses on the juvenile justice system; and stresses on youth agencies and support services.

Solvent abuse in developing countries attacks youths in their most productive years. The ease of access of solvents and aerosols in developing countries could have economic ties as it may be related to the introduction of Structural Adjustment Programs (SAP) in the country. Since SAPs may cause poverty, as well as education suffering financial support, this could explain why the youths in countries such as those in Latin America have an ease of access to and abuse solvents.

**Patterns of non-medical usage**

**Africa and Asia**

Glue and gasoline sniffing is also a problem in parts of Africa, especially with street children, and South Asia. Three of the most widely abused inhalants are the Dendrite brand and other forms of contact adhesives and rubber cements manufactured in Kolkata, toluenes in paint thinners, and Iodex—a muscle-stress-relieving balm. Another very common inhalant is Erase-X, a correction fluid that contains toluene. It has become very common for school and college students to use it, because it is easily available in stationery shops in India. This fluid is also used by street and working children in Delhi.

**Europe and North America**

In the UK, marginalized youth use a number of inhalants, such as solvents and propellants. As well, in the UK rave culture, inhalants such as nitrous oxide "whippets" and amyl nitrite poppers are used to enhance the effect of the electronic dance music. In Russia and Eastern Europe, gasoline sniffing became common on Russian ships following attempts to limit the supply of alcohol to ship crews in the 1980s. The documentary Children Underground depicts the huffing of a solvent called Aurolac (a product used in chroming) by Romanian homeless children.
In Canada, Native children in the isolated Northern Labrador community of Davis Inlet were the focus of national concern in 1993, when many were found to be sniffing gasoline. The federal Canadian and provincial Newfoundland and Labrador governments intervened on a number of occasions, sending many children away for treatment. Despite being moved to the new community of Natuashish in 2002, serious inhalant abuse problems have continued. Similar problems were also reported in Sheshatshiu in 2000. In Mexico, the inhaling of a mixture of gasoline and/or industrial solvents, known locally as "Activo" or "Chemo", has risen in popularity among the homeless and among the street children of Mexico City in recent years. The mixture is poured onto a handkerchief and inhaled while held in one’s fist.

In the US, ether was used as a recreational drug during the 1930s Prohibition era, when alcohol was made illegal. Ether was either sniffed or drunk and, in some towns, replaced alcohol entirely. However, the risk of death from excessive sedation or overdose is greater than that with alcohol, and ether drinking is associated with damage to the stomach and gastrointestinal tract. Use of glue, paint and gasoline became more common after the 1950s. Abuse of aerosol sprays became more common in the 1980s, as older propellants such as CFCs were phased out and replaced by more environmentally friendly compounds such as propane and butane. Most inhalant solvents and gases are not regulated under drug laws such as the United States' Controlled Substances Act. However, many US states and Canadian cities have placed restrictions on the sale of some solvent-containing products to minors, particularly for products widely associated with sniffing, such as model cement. The practice of inhaling such substances is sometimes colloquially referred to as huffing, sniffing (or glue sniffing), dusting, or chroming.

Australia

Australia has long faced a petrol (gasoline) sniffing problem, in isolated and impoverished aboriginal communities. Although some sources argue that sniffing was introduced by United States servicemen stationed in the nation's Top End during World War II or through experimentation by 1940s-era Cobourg Peninsula sawmill workers, other sources claim that inhalant abuse (such as glue inhalation) emerged in Australia in the late 1960s. Chronic, heavy petrol sniffing appears to occur among remote, impoverished indigenous communities, where the ready accessibility of petrol has helped to make it a common substance for abuse.

In Australia, petrol sniffing now occurs widely throughout remote Aboriginal communities in the Northern Territory, Western Australia, northern parts of South Australia and Queensland. The number of people sniffing petrol goes up and down over time as young people experiment or sniff occasionally, "Boss" or chronic sniffers may move in and out of communities; they are often responsible for encouraging young people to take it up.

A 1983 survey of 4,165 secondary students in New Lydiate showed that solvents and aerosols ranked just after analgesics (e.g., codeine pills) and alcohol for drugs that were abused. This 1983 study did not find any common usage patterns or social class factors.
The causes of death for inhalant users in Australia included pneumonia, cardiac failure/arrest, aspiration of vomit, and burns. In 1985, there were 14 communities in Central Australia reporting young people sniffing. In July 1997, it was estimated that there were around 200 young people sniffing petrol across 10 communities in Central Australia. Approximately 40 were classified as chronic sniffers. There have been reports of young Aboriginal people sniffing petrol in the urban areas around Darwin and Alice Springs.

In 2005, the Government of Australia and BP Australia began the usage of opal fuel in remote areas prone to petrol sniffing. Opal is a non-sniffable fuel (which is much less likely to cause a high) and has made a difference in some indigenous communities.

Popular culture references

Music and musical culture

Inhalant use, especially glue sniffing, is widely associated with the late-1970s punk youth subculture in the UK and North America. Raymond Cochrane and Douglas Carroll claim that when glue sniffing became widespread in the late 1970s, it was "...adopted by punks because public [negative] perceptions of sniffing fitted in with their self-image" as rebels against societal values. While punks at first used inhalants "experimentally and as a cheap high, adult disgust and hostility [to the practice] encouraged punks to use glue sniffing as a way of shocking society." As well, using inhalants was a way of expressing their anti-corporatist DIY (Do It Yourself) credo; by using inexpensive household products as inhalants, punks did not have to purchase industrially manufactured liquor or beer. One history of the punk subculture argues that "substance abuse was often referred to in the music and did become synonymous with the genre, glue sniffing especially" because the youths' "...faith in the future had died and that the youth just didn't care anymore" due to the "awareness of the threat of nuclear war and a pervasive sense of doom." In a BBC interview with a person who was a punk in the late 1970s, they said that "there was a real fear of imminent nuclear war - people were sniffing glue knowing that it could kill them, but they didn't care because they believed that very soon everybody would be dead anyway."

A number of 1970s punk rock and 1980s hardcore punk songs refer to inhalant use. The Ramones, an influential early US punk band, referred to inhalant use in several of their songs. The song "Now I Want to Sniff Some Glue" describes adolescent ennui, and the song "Carbona not Glue" states, "My brain is stuck from shooting glue." An influential punk fanzine about the subculture and music took its name (Sniffin' Glue) from the Ramones song. The 1980s punk band The Dead Milkmen wrote a song, "Life is Shit" from their album Beelzebubba, about two friends hallucinating after sniffing glue. Punk band-turned hip hop group the Beastie Boys penned a song "Hold It Now...Hit It," which includes the line "cause I'm beer drinkin', breath stinkin', sniffing glue." Pop punk band Sum 41 wrote a song, "Fat Lip", which refers to a character who does not "... make sense from all the gas you be huffing..."
Inhalants are also referred to by bands from other genres, including several grunge bands—an early 1990s genre that was influenced by punk rock. The 1990s grunge band Nirvana, which was influenced by punk music, penned a song, "Dumb", in which Kurt Cobain sings "my heart is broke/But I have some glue/ help me inhale /And mend it with you". L7, an all-female grunge band, penned a song entitled "Scrap" about a skinhead who inhales spray-paint fumes until his mind "starts to gel". Also in the 1990s, the Britpop band Suede had a UK hit with their song Animal Nitrate whose title is clearly a thinly veiled reference to Amyl Nitrate. The Beck song "Fume" from his "Fresh Meat and Old Slabs" release is about inhaling nitrous oxide. Another Beck song, "Cold Ass Fashion", contains the line "O.G. – Original Gluesniffer!" The band Primus's 1998 song "Lacquer Head" is about adolescents who use inhalants to get high. Hip hop performer Eminem wrote a song, "Bad Meets Evil", which refers to breathing "...ether in three lethal amounts." The Brian Jonestown Massacre, a retro-rock band from the 1990s, has a song entitled "Hyperventilation", which is about sniffing model-airplane cement.

Films

A number of films have depicted or referred to the use of solvent inhalants. In the 1980 comedy film Airplane!, the character of McCroskey (Lloyd Bridges) refers to his inhalant use when he states, "I picked the wrong week to quit sniffing glue." In the 1996 film Citizen Ruth, the character Ruth, a homeless drifter, is depicted inhaling patio sealant from a paper bag in an alleyway. In the tragi-comedy Love Liza, the main character, played by Philip Seymour Hoffman, plays a man who takes up building remote-controlled airplanes as a hobby to give him an excuse to sniff the fuel in the wake of his wife’s suicide. Harmony Korine’s 1997 film Gummo depicts adolescent boys inhaling contact cement for a high. Edet Belzberg’s 2001 documentary Children Underground chronicles the lives of Romanian street children addicted to inhaling paint. Yet another film that depicts the drug use is The Basketball Diaries, in which a group of boys are huffing carbona cleaning liquid at 3 minutes and 27 seconds into the movie, and, further into the movie, a boy is reading a diary describing the experience of sniffing the cleaning liquid.

In the David Lynch film Blue Velvet, the bizarre and manipulative character played by Dennis Hopper uses a mask to inhale amyl nitrite. In Little Shop of Horrors, Steve Martin’s character dies from nitrous oxide inhalation. The 1999 independent film Boys Don't Cry depicts two young low-income women inhaling aerosol computer cleaner (Canned Air) for a buzz. In The Cider House Rules, Michael Caine’s character is addicted to inhaling ether vapors. In Thirteen, the main character, a teen, uses a can of aerosol computer cleaner to get high. In the action movie Shooter, an ex-serviceman on the run from the law (Mark Wahlberg) inhales nitrous oxide gas from a number of Whip-It! whipped cream canisters until he becomes unconscious. The film Fear and Loathing in Las Vegas describes how the two main characters inhale diethyl ether and amyl nitrite. The South African film The Wooden Camera also depicts the use of inhalants by one of the main characters, a homeless teen, and their use in terms of socio-economic stratification. The titular characters in Samson and Delilah sniff petrol; in Samson’s case, possibly causing brain damage.
In the 2004 film Taxi, Queen Latifah and Jimmy Fallon are trapped in a room with a burst tank containing nitrous oxide. Queen Latifah’s character curses at Fallon while they both laugh hysterically. Fallon’s character asks if it is possible to die from nitrous oxide, to which Queen Latifah’s character responds with "It’s laughing gas, stupid!" Neither of them suffered any side effects other than their voices becoming much deeper while in the room.

Books

The science fiction story "Waterspider" by Philip K. Dick (first published in January 1964 in If magazine) contains a scene in which characters from the future are discussing the culture of the early 1950s. One character says: "You mean he sniffed what they called ‘airplane dope’? He was a ‘glue-sniffer’?", to which another character replies: "Hardly. That was a mania among adolescents and did not become widespread in fact until a decade later. No, I am speaking about imbibing alcohol."

Poly drug use

Polydrug use refers to the use of two or more psychoactive drugs in combination to achieve a particular effect. In many cases one drug is used as a base or primary drug, with additional drugs to leaven or compensate for the side effects of the primary drug and make the experience more enjoyable with drug synergy effects, or to supplement for primary drug when supply is low.

Risks

Poly drug use often carries with it more risk than use of a single drug, due to an increase in side effects, and drug synergy. The potentiating effect of one drug on another is sometimes considerable and here the licit drugs and medicines – such as alcohol, nicotine and antidepressants – have to be considered in conjunction with the controlled psychoactive substances. The risk level will depend on the dosage level of both substances. Concerns exist about a number of pharmacological pairings: alcohol and cocaine increase cardiovascular toxicity; alcohol or depressant drugs, when taken with opioids, lead to an increased risk of overdose; and opioids or cocaine taken with ecstasy or amphetamines also result in additional acute toxicity. Temazepam, a powerful hypnotic benzodiazepine, is notorious for causing death when mixed with other CNS depressants such as opioids, alcohol, or barbiturates.

Scheduling

Within the general concept of multiple drug use, several specific meanings of the term must be considered. At one extreme is planned use. On the other hand, the use of several substances in an intensive and chaotic way, simultaneously or consecutively, in many cases each drug substituting for another according to availability.
Happiness Disposition and Positive Health

Psychological resilience

"Resilience" in psychology is the positive capacity of people to cope with stress and adversity. This coping may result in the individual “bouncing back” to a previous state of normal functioning, or using the experience of exposure to adversity to produce a “steeling effect” and function better than expected (much like an inoculation gives one the capacity to cope well with future exposure to disease). Resilience is most commonly understood as a process, and not a trait of an individual.

Recently there has also been evidence that resilience can indicate a capacity to resist a sharp decline in functioning even though a person temporarily appears to get worse. A child, for example, may do poorly during critical life transitions (like entering junior high) but experience problems that are less severe than would be expected given the many risks the child faces.

There is also controversy about the indicators of good psychological and social development when resilience is studied across different cultures and contexts. The American Psychological Association’s Task Force on Resilience and Strength in Black Children and Adolescents, for example, notes that there may be special skills that these young people and families have that help them cope, including the ability to resist racial prejudice. People who cope may also show "hidden resilience" when they don’t conform with society’s expectations for how someone is supposed to behave (in some contexts, aggression may be required to cope, or less emotional engagement may be protective in situations of abuse).

In all these instances, resilience is best understood as a process. It is often mistakenly assumed to be a trait of the individual, an idea more typically referred to as “resiliency”. Most research now shows that resilience is the result of individuals interacting with their environments and the processes that either promote well-being or protect them against the overwhelming influence of risk factors. These processes can be individual coping strategies, or may be helped along by good families, schools, communities, and social policies that make resilience more likely to occur. In this sense "resilience" occurs when there are cumulative "protective factors". These factors are likely to play a more and more important role the greater the individual’s exposure to cumulative "risk factors". The phrase "risk and resilience" in this area of study is quite common.

Commonly used terms, which are closely related within psychology, are "psychological resilience", "emotional resilience", "hardiness", "resourcefulness", and "mental toughness". The earlier focus on individual capacity which Anthony described as the “invulnerable child” has evolved into a more multilevel ecological perspective that builds on theory developed by Uri Bronfenbrenner (1979), and more recently discussed in the work of Michael Ungar (2004, 2008), Ann Masten (2001), and Michael Rutter (1987, 2008). The focus in research has shifted from "protective factors" toward protective "processes";
trying to understand how different factors are involved in both promoting well-being and protecting against risk.

**Definition of resilience**

Resilience is a dynamic process that individuals exhibit positive behavioral adaptation when they encounter significant adversity, trauma, tragedy, threats, or even significant sources of Stress (biology). It is different from strengths or developmental assets which are a characteristic of an entire population, regardless of the level of adversity they face. Under adversity, assets function differently (a good school, or parental monitoring, for example, have a great deal more influence in the life of a child from a poorly resourced background than one from a wealthy home with other options for support, recreation, and self-esteem).

Resilience is a two-dimensional construct concerning the exposure of adversity and the positive adjustment outcomes of that adversity. This two-dimensional construct implies two judgments: one about a "positive adaptation" and the other about the significance of risk (or adversity). One point of view about adversity could define it as any risks associated with negative life conditions that are statistically related to adjustment difficulties, such as poverty, children of mothers with schizophrenia, or experiences of disasters. Positive adaptation, on the other hand, is considered in a demonstration of manifested behaviour on social competence or success at meeting any particular tasks at a specific life stage, such as the absence of psychiatric distress after the September 11th terrorism attacks on the United States. Ungar argues that this standard definition of resilience could be problematic because it does not adequately account for cultural and contextual differences in how people in other systems express resilience. Through collaborative mixed methods research in eleven countries, Ungar and his colleagues at the Resilience Research Centre have shown that cultural and contextual factors exert a great deal of influence on the factors that affect resilience among a population of youth-at-risk.

Resilience has been shown to be more than just the capacity of individuals to cope well under adversity. Resilience is better understood as both the capacity of individuals to navigate their way to the psychological, social, cultural, and physical resources that sustain their well-being, and their capacity individually and collectively to negotiate for these resources to be provided and experienced in culturally meaningful ways. Studies of demobilized child soldiers, high school drop-outs, urban poor, immigrant youth, and other populations at risk are showing these patterns. Among adults, these same themes emerge, as detailed in the work of Zautra, Hall and Murray (2010).

**History of research on resilience**

Garmezy (1973) published the first research findings on resilience. He used epidemiology, which is the study of who gets ill, who doesn't, and why, to uncover the risks and the protective factors that now help define resilience. Garmezy and Streitman (1974) then created tools to look at systems that support development of resilience.
Emmy Werner (1982) was one of the early scientists to use the term resilience in the 1970s. She studied a cohort of children from Kauai, Hawaii. Kauai was quite poor and many of the children in the study grew up with alcoholic or mentally ill parents. Many of the parents were also out of work. Werner noted that of the children who grew up in these very bad situations, two-thirds exhibited destructive behaviors in their later teen years, such as chronic unemployment, substance abuse, and out-of-wedlock births (in case of teenage girls). However one-third of these youngsters did not exhibit destructive behaviours. Werner called the latter group 'resilient'. Resilient children and their families had traits that made them different from non-resilient children and families.

Resilience emerged as a major theoretical and research topic from the studies of children of schizophrenic mothers in the 1980s. In Masten’s (1989) study, the results showed that children with a schizophrenic parent may not obtain comforting caregiving compared to children with healthy parents, and such situations had an impact on children’s development. However, some children of ill parents thrived well and were competent in academic achievement, and therefore led researchers to make efforts to understand such responses to adversity.

In the onset of the research on resilience, researchers have been devoted to discovering the protective factors that explain people’s adaptation to adverse conditions, such as maltreatment, catastrophic life events, or urban poverty. The focus of empirical work then has been shifted to understand the underlying protective processes. Researchers endeavor to uncover how some factors (e.g. family) may contribute to positive outcomes.

**Expressions of resilience**

Resilience can be described by viewing:

- good outcomes regardless of high-risk status,
- constant competence under stress,
- recovery from trauma, and
- using challenges for growth that makes future hardships more tolerable.

Resilience describes people who are expected to adapt successfully even though they experience risk factors that ‘stack the odds’ against them experiencing good development. Risk factors are related to poor or negative outcomes. For example, poverty, low socioeconomic status, and mothers with schizophrenia are coupled with lower academic achievement and more emotional or behavioral problems. Risk factors may be cumulative, carrying additive and exponential risks when they co-occur. When these risk factors happen, according to a study conducted on children, resilient children are capable of resulting in no behavioural problems and developing well. Additionally, they are more active and socially responsive. These positive outcomes are attributed to some protective factors, such as good parenting or positive school experiences.

Resilience is also treated as an effective coping mechanism when people are under stress, such as divorce. In this context, resilience is relevant with sustained competence exhibited
by individuals who experience challenging conditions. Most research built on this perspective focuses on the children’s response to parents’ divorce in terms of gender. Boys show more conduct problems than do girls; girls obtain more support from mothers and are less exposed to family conflict than boys. Although divorce may have some negative impacts on children’s development, it may help children in single households to become more responsible than those in dual-parents households because of helping with chores. Some protective factors attributing to resilient children in single-family, for example, are adults caring for children during or after major stressors (e.g., divorce), or self-efficacy for motivating endeavor at adaptation.

Finally, resilience can be viewed as the phenomenon of recovery from a prolonged or severe adversity, or from an immediate danger or stress. In this case, resilience is not related to vulnerability. People who experience acute trauma, for example, may show extreme anxiety, sleep problems, and intrusive thoughts. Over time, these symptoms decrease and recovery is likely. This realm of research shows that age and the supportive qualities of the family influence the condition of recovery. The Buffalo Creek dam disaster, for example, had longer effects on older children than on younger. Additionally, children with supportive families show fewer symptoms (e.g., dreams of personal death) than children from troubled families, as revealed by a study on victims of the 1976 Chowchilla bus kidnapping.

Factors related to resilience

Several factors are found to modify the negative effects of adverse life situations. Many studies show that the primary factor is to have relationships that provide care and support, create love and trust, and offer encouragement, both within and outside the family. Additional factors are also associated with resilience, like the capacity to make realistic plans, having self-confidence and a positive self image, developing communications skills, and the capacity to manage strong feelings and impulses.

Another protective factor is related to moderating the negative effects of environmental hazards or a stressful situation in order to direct vulnerable individuals to optimistic paths, such as external social support. More specifically, Werner (1995) distinguished three contexts for protective factors: (1) personal attributes, including outgoing, bright, and positive self-concepts; (2) the family, such as having close bonds with at least one family member or an emotionally stable parent; and (3) the community, like receiving support or counsel from peers.

Besides the above distinction on resilience, research has also been devoted to discovering the individual differences in resilience. Self-esteem, ego-control, and ego-resiliency are related to behavioral adaptation. For example, maltreated children who feel good about themselves may process risk situations differently by attributing different reasons to the environments they experience and, thereby, avoid producing negative internalized self-perceptions. Ego-control is "the threshold or operating characteristics of an individual with regard to the expression or containment" (Block & Block, 1980, p. 43) of their impulses, feelings, and desires. Ego-resilience refers to "dynamic capacity......to modify his or her
model level of ego-control, in either direction, as a function of the demand characteristics of the environmental context" (Block & Block, 1980, p. 48).

Maltreated children, who experienced some risk factors (e.g., single parenting, limited maternal education, or family unemployment), showed lower ego-resilience and intelligence than nonmaltreated children (Cicchetti et al., 1993). Furthermore, maltreated children are more likely than nonmaltreated children to demonstrate disruptive-aggressive, withdraw, and internalized behavior problems (Cicchetti et al., 1993). Finally, ego-resiliency, and positive self-esteem were predictors of competent adaptation in the maltreated children (Cicchetti et al., 1993).

Demographic information (e.g., gender) and resources (e.g., social support) are also used to predict resilience. Examining people's adaptation after the 9/11 attacks (Bonanno, Galea Bucciarelli, & Vlahov, 2007) showed women were associated with less likelihood of resilience than men. Also, individuals who were less involved in affinity groups and organisations showed less resilience. King, King, Fairbank, Keane, and Adams (1998) studied resilience in Vietnam War veterans and found social support to be a major factor contributing to resilience.

Schnurr, Lunney, and Sengupta (2004) found that several protective factors among those were the following factors protecting against the development of PTSD:

- Japanese-American ethnicity, high school degree or college education, older age at entry to war, higher socioeconomic status, and a more positive paternal relationship as premilitary factors
- Social support at homecoming and current social support as postmilitary factors

and the following factors protecting among the maintenance of PTSD

- Native Hawaiian or Japanese-American ethnicity and college education as premilitary factors
- Current social support as postmilitary factor

A number of other factors that promote resilience have been identified:

- The ability to cope with stress effectively and in a healthy manner
- Having good problem-solving skills
- Seeking help
- Holding the belief that there is something one can do to manage your feelings and cope
- Having social support
- Being connected with others, such as family or friends
- Self-disclosure of the trauma to loved ones
- Spirituality
- Having an identity as a survivor as opposed to a victim
- Helping others
Finding positive meaning in the trauma

Certain aspects of religions/spirituality may, hypothetically, promote or hinder certain psychological virtues that increase resilience. Research has not established connection between spirituality and resilience. According to the 4th edition of Psychology of Religion by Hood, et al., "The study of positive psychology is a relatively new development...there has not yet been much direct empirical research looking specifically at the association of religion and ordinary strengths and virtues." In a review of the literature on the relationship between religiosity/spirituality and PTSD, amongst the significant findings, about half of the studies showed a positive relationship and half showed a negative relationship between measures of religiosity/spirituality and resilience. The United States Army has received criticism for promoting spirituality in its new [Comprehensive Soldier Fitness] program as a way prevent PTSD, due to the lack of conclusive supporting data.

An emerging field in the study of resilience is the neurobiological basis of resilience to stress. For example, neuropeptide Y (NPY) and 5-Dehydroepiandrosterone (5-DHEA) are thought to limit the stress response by reducing sympathetic nervous system activation and protecting the brain from the potentially harmful effects of chronically elevated cortisol levels respectively. In addition, the relationship between social support and stress resilience is thought to be mediated by the oxytocin system's impact on the hypothalamic-pituitary-adrenal axis.

Resilience building

The American Psychological Association suggests “10 Ways to Build Resilience”, which are:

(1) maintaining good relationships with close family members, friends and others;
(2) to avoid seeing crises or stressful events as unbearable problems;
(3) to accept circumstances that cannot be changed;
(4) to develop realistic goals and move towards them;
(5) to take decisive actions in adverse situations;
(6) to look for opportunities of self-discovery after a struggle with loss;
(7) developing self-confidence;
(8) to keep a long-term perspective and consider the stressful event in a broader context;
(9) to maintain a hopeful outlook, expecting good things and visualizing what is wished;
(10) to take care of one's mind and body, exercising regularly, paying attention to one's own needs and feelings and engaging in relaxing activities that one enjoys. Learning from the past and maintaining flexibility and balance in life are also cited.

The Young Foundation’s work on wellbeing in the UK emphasises 'subjective wellbeing', what people feel about the quality of their life. A key element of this is 'resilience', how people bounce back from adversity. Their work includes:

Working with Lord Richard Layard from the London School of Economics, the IDEa and three leading local authorities, Hertfordshire, Manchester and South Tyneside, as the lead partner in the Local Wellbeing Project to look at the different ways in which local
government and its local partners can promote wellbeing. The State of Happiness, the final Local Wellbeing Project report, brings together three years of groundbreaking work in the three partner local authority areas as well as other national and international developments in this field.

**Emotional Resilience for Gangs** - commissioned by Harrow Metropolitan Police to develop and pilot an emotional resilience programme targeting 14-19 year olds who are offending or at risk of offending, and are associated with gang activity. The Young Foundation is working in collaboration with Dr Ilona Boniwell, one of Europe's leading positive psychologists, to develop this new programme, training professionals in Harrow from Youth Services, the Anti-social Behaviour Unit, Safer Neighbourhoods Team and the Wealdstone Anti-social Behaviour Partnership. Training of professionals and the delivery of the pilot will take place in early 2011 with a report to follow.

**Resilience and social programs**

Head Start was shown to promote resilience. So was the Big Brothers Big Sisters Programme, the Abecedarian Early Intervention Project, and social programs for youth with emotional or behavioral difficulties.

**Children and resilience**

Resilience in children refers to individuals who are doing better than expected, given a history that includes risk or adverse experience. Simply put, resilience requires two conditions to be met:

1. the child must have experienced some sort of risk or adversity that has been linked with poor outcomes, and
2. the child is generally doing okay despite being exposed to that risk or adversity; they are not showing that poor outcome.

Resilience is a description of a group of children. It is not a trait or something that some children 'just have.' There is no such thing as an 'invulnerable child' who can overcome any obstacle that life throws at her (although some children may seem that way!). Resilience is not a rare and magical quality. In fact, it is quite common. Resilience is the product of a large number of developmental processes over time that has allowed children who experience some sort risk to continue to develop competently (while other children have not). Research on 'protective factors' has helped developmental scientists to understand what matters most for resilient children. Protective factors are characteristics of children or situations that particularly help children in the context of risk. There are many different protective factors that are important for resilient children. Two that have emerged time and again in studies of resilient children are good cognitive functioning (like cognitive self-regulation and IQ) and positive relationships (especially with competent adults, like parents). Children who have protective factors in their lives tend to do better in some risky contexts when compared to children without protective factors in the same contexts.
However, this is not a justification to expose any child to risk. Children do better when not exposed to high levels of risk or adversity.

When it comes to children, there are still many scientific debates with respect to resilience. One debate involves differing opinions about what constitutes 'doing okay.' There is considerable agreement that child competence can be defined and measured in a way that can indicate whether or not the child is doing well. Called 'age-salient developmental tasks,' these are things that are generally expected of children of a certain age, in a certain culture, of a certain time or point in history. Developmental tasks can span all areas or domains of a person's life. For example, in many cultures (but certainly not all) 24 month old children are expected to be able to show the beginnings of spoken language, early motor coordination that allows them to start walking, able to form an attachment relationship with a primary caregiver, etc. These tasks certainly change with age; generally children are expected to show increasingly sophisticated cognitive and social abilities as they grow older: 5 year olds are expected to show a higher degree of independence and self-regulation skills (for example), compared to a 2 year old. Resilient children can be thought of as those who show competence in age-salient developmental tasks even though they have experienced some risk or adversity that threatened that competence. Others have focused on different criteria for 'doing okay,' such as the absence of mental health problems like depression or conduct problems. Still others have focused on happiness or the experience of positive emotions.

**Building resilience in the classroom**

Resilient children as described by Garmezy, as working and playing well and holding high expectations, have often been characterized using constructs such as locus of control, self-esteem, self-efficacy, and autonomy. Benard concluded that resilient children have high expectations, a meaning for life, goals, personal agency, and inter-personal problem-solving skills. All of these things work together to prevent the debilitating behaviors that are associated with learned helplessness. Chess identified “adaptive distancing” as the psychological process whereby an individual can stand apart from distressed family members and friends in order to accomplish constructive goals and advance his or her psychological development. Moving away to college after high school is a way of practicing adaptive distancing. Classrooms in which students are given an opportunity to respond, an engaging cooperative learning environment, a participating role in setting goals, and a high expectation for student achievement. All of these characteristics help students develop a sense of belonging and involvement. These two characteristics help to reduce the feelings of alienation and disengagement. With that kind of connection in the school, students will have more of a protective shield against the adverse circumstances that life throws at them.

**The role a community has in fostering resilience in a child**

Communities play a huge role in fostering resilience. Benard identifies three characteristics of those types of communities (1) availability of social organizations that provide an array of resources to residents, (2) consistent expression of social norms so that community members understand what constitutes desirable behavior, (3) and
opportunities for children and youth to participate in the life of the community as valued members. The clearest sign of a cohesive and supportive community is the presence of social organizations that provide healthy human development. Services are unlikely to be used unless there is good communication concerning them. Community-school relationships are very important to give extra resources to meet even basic psychological needs of students and families.

**The role a family has in fostering resilience in a child**

Fostering resilience in children requires family environments that are caring and structured, hold high expectations for children's behavior, and encourage participation in the life of the family. Most resilient children have a strong relationship with at least one adult, not always a parent, and this relationship helps to diminish risk associated with family discord. Benard found that even though divorce produces stress, the availability of social support from family and community can reduce stress and yield positive outcomes. Any family that emphasizes the value of assigned chores, caring for brothers or sisters, and the contribution of part-time work in supporting the family helps to foster resilience.

**The role religion plays in fostering resilience**

When youths from problem neighbourhoods join a church their academic performance improves. The poorer a neighbourhood is, the more church attendance helps kids academically. Improving academic performance seems to flow more from attending church than from merely believing. The church's social life influences youth from poor communities more than doctrine does. Church attendance also improves the physical, social, and emotional health of students. According to Glen Elder: "What you have in the role of the religious community is a selected group of people who share values and are committed to the success of the child". This pattern is likely the result of many protective processes that take place inside a religious institution. Ungar and his colleagues identified seven aspects of resilience across many different cultures. Each depends on the other. These seven aspects include:

1. Access to material resources - Availability of financial, educational, medical and employment assistance and/or opportunities, as well as access to food, clothing and shelter

2. Access to supportive relationships - Relationships with significant others, peers and adults within one's family and community

3. Development of a desirable personal identity - Desirable sense of one's self as having a personal and collective sense of purpose, ability for self-appraisal of strengths and weaknesses, aspirations, beliefs and values, including spiritual and religious identification

4. Experiences of power and control - Experiences of caring for one's self and others, the ability to affect change in one's social and physical environment in order to access health resources
5. Adherence to cultural traditions - Adherence to, or knowledge of, one’s local and/or global cultural practices, values and beliefs

6. Experiences of social justice - Experiences related to finding a meaningful role in one’s community that brings with it acceptance and social equality

7. Experiences of a sense of cohesion with others - Balancing one’s personal interests with a sense of responsibility to the greater good; feeling a part of something larger than one’s self socially and spiritually

For example, attending a church has been shown to increase a child’s social network, provide a feeling of cohesion and belonging in her community, even promote a sense of personal control and sense of social justice when threatened. It is this complexity and multilevel nature of resilience that explains how people use the internal and external resources (assets) that are both available and accessible to overcome adversity.

**Resilience and emotion**

Some studies confirmed the association between resilience and positive emotion (e.g., Ong, Bergeman, Bisconti, & Wallace, 2006; Tugade et al., 2004).

Examining the role positive emotion plays in resilience, Ong et al. (2006) found that widows with high levels of resilience experience more positive (e.g., peaceful) and negative (e.g., anxious) emotions than those with low levels. The former group shows high emotional complexity which is the capacity to maintain the differentiation of positive and negative emotional states while underlying stress.

Ong et al. (2006) further suggest that the adaptive consequence of resilience is a function of an increase in emotional complexity while stress is present.

Moreover, high resilient widows showed the likelihood of controlling their positive emotional experiences to recover and bounce back from daily stress. Indeed, positive emotions were found to disrupt the experience of stress and help high resilient individuals to recover efficiently from daily stress (Fredrickson et al., 2003). In this case, some studies argue (e.g., Fredrickson et al., 2003; Tugade et al., 2004) that positive emotion helps resilient people to construct psychological resources that are necessary for coping successfully with significant catastrophe, such as the September 11th attacks. As a result, positive emotion experienced by resilient people functions as a protective factor to moderate the magnitude of adversity to individuals and assists them to cope well in the future (Tugade et al., 2004).

In addition to the above findings, a study (Fredrickson et al., 2003) further suggests that positive emotions are active elements within resilience.

By examining people’s emotional responses to the September 11th, Fredrickson et al. (2003) suggests that positive emotions are critical elements in resilience and as a mediator
that buffer people from depression after the crises. Moreover, high resilient people were more likely to notice positive meanings within the problems they faced (e.g., felt grateful to be alive), endured fewer depressive symptoms, and experienced more positive emotions than low resilient people after terrorism attacks (Fredrickson et al., 2003). Similar results were obtained in another study regarding the effects of 911 attacks on resilient individuals' healthy adjustment (Bonanno et al., 2007).

People with high levels of resilience are likely to show low levels of depression, and less likely to smoke cigarettes or use marijuana (Bonanno et al., 2007). Moreover, low resilient people exhibit the difficulties of regulating negative emotions and demonstrate sensitive reaction to daily stressful life events (e.g., the loss of loved one) (Ong et al., 2006). They are likely to believe that there is no end for the unpleasant experience of daily stressors and may have higher levels of stress. In general, resilient people are believed to possess positive emotions, and such emotions in turn influence their responses to adversity.

Resilient groups

Psychological Resilience has been studied in a number of groups. Among those are the children of European Jews in the United States, the children of the Vietnamese boat people in the United States. Middle class families in times of the great depression, children of farmers in times of economical crisis, children of Spanish and Vietnamese immigrants in Germany, adoptive children, who went through trauma and malnutrition.

The children of poor Vietnamese parents in the U.S.A. and Germany

Nathan Caplan studied the children of poor Vietnamese parents in the US. Most of these parents were refugees. In many cases they did not own anything but the clothes they were wearing when they arrived. Most did not speak English. Half of the parents had less than five years of formal schooling. The refugees studied by Caplan lived in the worst neighborhoods of big cities. Yet their children turned out to be academically more successful than American middle class children.

Why?

Caplan et al. found out the Vietnamese stress the value of education. Parents wanted their children to enjoy a better education than they did themselves. The Vietnamese children spend an average of 3 hours and 10 minutes per day doing their homework and reading for school, while American middle class students just spend an average of 1 hour and 30 minutes per day with these activities.

Nathan Caplan also found out the older siblings were supposed to help their younger siblings. That way the younger ones did not only learn facts but also attitudes towards school and learning from their older siblings. The more siblings a child of Vietnamese parentage has, the more likely is he or she to achieve in school.
Germany is a multi-ethnic society. 8% of the population and 25% of the 15 year olds are born abroad themselves or have at least one parent born abroad. In Germany Vietnamese families started arriving as foreign workers during the 1980s and they are still coming in great numbers to search for a better life. As a rule children of immigrants are not as successful academically as children of native Germans. However it is not true for children of Asian parentage. The Vietnamese are the biggest Asian group in Germany and also one of the poorest ethnic groups. It has been found that Vietnamese parents value education and that Vietnamese students spend a lot more time learning than their German counterparts.

Children of American farmers

Elder and Conger examined data from several Iowa counties to see how the farm crisis of the 1980s and 1990s affected children growing up in rural parts of the state. They found that a large number of those young people were on paths to successful development and life achievement. Most children of those children grew up to be academically successful and law-abiding.

Elder was able to identify five resource mechanisms:

- strong intergenerational bonds, joint activity between parents and children
- being socialized into productive roles in work and social leadership; stressing non-material goals
- a network of positive engagement in church, school, and community life
- close ties with grandparents, support from grandparents
- strong family connections with the community

Children in times of the Great Depression

Elder studied the life of men who were children during the Great Depression of 1929-1939 and came to maturity at the outset of World War II. When these children came of age Elder found them to be healthy, law abiding, well adapted and bright.

One stunning finding was that poverty had slight positive effects on children from the middle classes. Once they reached adulthood those men earned a college degree as often as men from nondeprived middle class homes. In later life they did a little better in terms of economic success than their nondeprived middle class peers.

Men of working class background did not do as well as men from middle class homes. However many of them were upwardly mobile and on most measures they did do just as well as men from never-deprived working class backgrounds.

Spaniards in Germany

In the 1970s, Spain was a dictatorship under the rule of Francisco Franco. Many Spaniards fled to Germany in search of a better life. Most of those immigrants were poor and only few
were able to speak proper German. Today their children do as well as German children when it comes to educational success and Spaniard adults do as well as German adults when it comes to occupational success.

Quality of life

The term quality of life is used to evaluate the general well-being of individuals and societies. The term is used in a wide range of contexts including the fields of international development, healthcare, and politics. Quality of life should not be confused with the concept of standard of living, which is based primarily on income. Instead, standard indicators of the quality of life include not only wealth and employment, but also the built environment, physical and mental health, education, recreation and leisure time, and social belonging.

According to ecological economist Robert Costanza:

While Quality of Life (QOL) has long been an explicit or implicit policy goal, adequate definition and measurement have been elusive. Diverse "objective" and "subjective" indicators across a range of disciplines and scales, and recent work on subjective well-being (SWB) surveys and the psychology of happiness have spurred renewed interest.

Also frequently related are concepts such as freedom, human rights, and happiness. However, since happiness is subjective and hard to measure, other measures are generally given priority. It has also been shown that happiness, as much as it can be measured, does not necessarily increase correspondingly with the comfort that results from increasing income. As a result, standard of living should not be taken to be a measure of happiness.

International development

Quality of life is an important concept in the field of international development, since it allows development to be analyzed on a measure broader than standard of living. Within development theory, however, there are varying ideas concerning what constitutes desirable change for a particular society, and the different ways that quality of life is defined by institutions therefore shapes how these organizations work for its improvement as a whole.

Organizations such as the World Bank, for example, declare a goal of "working for a world free of poverty", with poverty defined as a lack of basic human needs, such as food, water, shelter, freedom, access to education, healthcare, or employment. In other words, poverty is defined as a low quality of life. Using this definition, the World Bank works towards improving quality of life through neoliberal means, with the stated goal of lowering poverty and helping people afford a better quality of life.

Other organizations, however, may also work towards improved global quality of life using a slightly different definition and substantially different methods. Many NGOs do not focus
at all on reducing poverty on a national or international scale, but rather attempt to improve quality of life for individuals or communities. One example would be sponsorship programs that provide material aid for specific individuals. Although many organizations of this type may still talk about fighting poverty, the methods are significantly different.

Because of these differences in the theory and practice of development, there is also a wide range of quantitative measures used to describe quality of life.

**Quantitative measurement**

Unlike per capita GDP or standard of living, both of which can be measured in financial terms, it is harder to make objective or long-term measurements of the quality of life experienced by nations or other groups of people. Researchers have begun in recent times to distinguish two aspects of personal well-being: Emotional well-being, in which respondents are asked about the quality of their everyday emotional experiences—the frequency and intensity of their experiences of, for example, joy, stress, sadness, anger, and affection— and life evaluation, in which respondents are asked to think about their life in general and evaluate it against a scale. Such and other systems and scales of measurement have been in use for some time.

**Human Development Index**

Perhaps the most commonly used international measure of development is the Human Development Index (HDI), which combines measures of life expectancy, education, and standard of living, in an attempt to quantify the options available to individuals within a given society. The HDI is used by the United Nations Development Programme in their Human Development Report.

**Other measures**

The Physical Quality of Life Index (PQLI) is a measure developed by sociologist Morris David Morris in the 1970s, based on basic literacy, infant mortality, and life expectancy. Although not as complex as other measures, and now essentially replaced by the Human Development Index, the PQLI is notable for Morris's attempt to show a "less fatalistic pessimistic picture" by focussing on three areas where global quality of life was generally improving at the time, and ignoring Gross National Product and other possible indicators that were not improving.

The Happy Planet Index, introduced in 2006, is unique among quality of life measures in that, in addition to standard determinants of well-being, it uses each country's ecological footprint as an indicator. As a result, European and North American nations do not dominate this measure. The 2009 list is instead topped by Costa Rica, the Dominican Republic, and Jamaica.

Gallup researchers trying to find the world's happiest countries found Denmark to be at the top of the list.
A 2010 study by two Princeton University professors looked at 1,000 randomly selected U.S. residents over an extended period. It concludes that their life evaluations - that is, their considered evaluations of their life against a stated scale of one to ten - rise steadily with income. On the other hand, their reported quality of emotional daily experiences (their reported experiences of joy, affection, stress, sadness, or anger) levels off after a certain income level (approximately $75,000 per year); income above $75,000 does not lead to more experiences of happiness nor to further relief of unhappiness or stress. Below this income level, respondents reported decreasing happiness and increasing sadness and stress, implying the pain of life’s misfortunes, including disease, divorce, and being alone, is exacerbated by poverty.

Livability

The term quality of life is also used by politicians and economists to measure the liveability of a given city or nation. Two widely known measures of liveability are the Economist Intelligence Unit's quality-of-life index and Mercer's Quality of Living Reports. These two measures calculate the liveability of countries and cities around the world, respectively, through a combination of subjective life-satisfaction surveys and objective determinants of quality of life such as divorce rates, safety, and infrastructure. Such measures relate more broadly to the population of a city, state, or country, not to individual quality of life.

Crimes

Some crimes against property (e.g., graffiti and vandalism) and some "victimless crimes" have been referred to as "quality-of-life crimes." American sociologist James Q. Wilson encapsulated this argument as the Broken Window Theory, which asserts that relatively minor problems left unattended (such as litter, graffiti, or public urination by homeless individuals) send a subliminal message that disorder in general is being tolerated, and as a result, more serious crimes will end up being committed (the analogy being that a broken window left unrepaired shows an image of general dilapidation). Wilson's theories have been used to justify the implementation of zero tolerance policies by many prominent American mayors, most notably Oscar Goodman in Las Vegas, Richard Riordan in Los Angeles, Rudolph Giuliani in New York City and Gavin Newsom in San Francisco. Such policies do not tolerate even minor crimes, it is argued, in order to improve the quality of life of local residents. However, critics of zero tolerance policies believe that such policies neglect investigation on a case-by-case basis and may lead to unreasonably harsh penalties for crimes.

Popsicle index

The Popsicle Index is a quality of life measurement coined by Catherine Austin Fitts as the percentage of people in a community who believe that a child in their community can safely leave their home, walk to the nearest possible location to buy a popsicle, and walk back to their homes.
Healthcare

Within the field of healthcare, quality of life is often regarded in terms of how it is negatively affected, on an individual level, a debilitating illness that is not life-threatening, life-threatening illness that is not terminal, terminal illness, the predictable, natural decline in the health of an elder, an unforeseen mental/physical decline of a loved one, chronic, end-stage disease processes. Researchers at the University of Toronto's Quality of Life Research Unit define quality of life as “The degree to which a person enjoys the important possibilities of his or her life” (UoT). Their Quality of Life Model is based on the categories “being”, “belonging”, and “becoming”, respectively who one is, how one is connected to one's environment, and whether one achieves one's personal goals, hopes, and aspirations.

Self-Perceived Quality of Life Scale

Self-Perceived Quality of Life Scale is a psychological assessment instrument which is based on a comprehensive theory of the Self-Perceived Quality of Life (SPQL) and provides a multi-faceted measurement of health-related and non-health-related aspects of well-being. The scale has become an instrument of choice for monitoring quality of life in some clinical populations, for example, it was adopted by the Positively Sound network for women living with HIV.

The improvement of mental disorders may have an effect on multiple domains of an individual's life which could be captured only through a comprehensive measurement. For example, the treatment of a phobia may reduce fear (mental health index), which could lead to the improvement of social relations (social relations index) and, in turn, performance at work, resulting in an increase in salary (financial index). Hence, in order to detect all implications of a treatment (e.g., for a phobia), a comprehensive measurement across multiple domains of an individual's life is needed. The SPQL scale can provide such a comprehensive measurement.

The scale is designed in an electronic format. The software calculates scores automatically; this allows for advanced quantification methods. The automatic calculations and quantification methods allowed undertaking a comprehensive approach for assessing SPQL from multiple facets. A multi-facet approach, in turn, provided a comprehensive evaluation of the effectiveness of mental health interventions (through pre and post tests).

The scale emerged from synthesis of existing theories including: (a) subjective well-being, (b) developmental life-stages, (c) different categories of human needs, (d) quality of life, and (e) subjective evaluation processes. The scale consists of three axes: Subjective well-being, positive and negative affect, and fulfillment of needs. See a model diagram below.

The scale can (a) identify possible side effects of psychiatric or psychological interventions which could occur in multiple domains of an individual's life, (b) detect the occurrence of relapses, (c) assist in evaluating the progress of recovery, (d) measure the effects of various non-normative positive and negative events (e.g., divorce, promotion at work, becoming a
parent) on an individual’s life as a whole and trace the course of their development, (e) evaluate an individual’s SPQL throughout the lifespan, (f) predict depression, anxiety, and mood, and (g) assess the effectiveness of interventions intended to enhance well-being and improve quality of life on an individual level.

This scale could be used by individual mental health professionals to evaluate the progress of treatment. This is useful for clients as well because they themselves would be able to compare their initial scores with scores after intervention. Because the scale is available online, clients are able to complete the questionnaire outside of the therapy sessions. The scale also could be used in medical settings to assess how medical treatment affects a patient’s life overall and in specific aspects overtime, as well as allow detecting psychological side effects. The scale could be of use to insurers because it would help in evaluating the effectiveness of mental health interventions.

**SPQL Model**
Theory

It is safe to postulate that all people want to have a good life. Although the meaning of "a good life" may vary from culture to culture and from individual to individual, this meaning revolves around the same aspects of life across cultures. What actually varies between cultures and individuals is the availability of certain aspects of a good life, the subjective significance people assign to these aspects, and the way people evaluate these aspects of a good life.

Everything we do or do not do, wish or do not wish, and have or do not have has an explicit or an implicit relevance to how good or not good we perceive our lives to be. Because the preference for a good life over a bad life underlies all facets of our lives, understanding
what constitutes and influences a good life on an individual level has a significant value for all people.

During the past several decades researchers investigated the concept of “the good life” based on three theoretical approaches: (a) focusing on quality of life (QOL) on a population and on individual levels by considering objective and/or subjective factors present or absent in people’s lives (Power, 2004; World Health Organization Quality of Life [WHOQOL] Group, 1995); (b) focusing on subjective well-being (SWB) by considering an individual’s level of overall happiness and life satisfaction (Corey, Keyes, & Magyar-Moe, 2004; Diener, Suh, Lucas, & Smith, 1999; Watson, 2000); and (c) focusing on an individual’s level of functionality across social, psychological, and health factors (Keyes, 1998; Ryff, 1989; Ware & Sherbourne, 1992).

The comprehensive scale of the good life, the Self-Perceived Quality of Life (SPQL) scale, overcame the limitations of prior approaches (Hagerty et al., 2001; Rapley, 2003) by integrating measurements of SWB, QOL, and functionality on an individual level, and by utilizing innovative quantification methods. The scale focused on how individuals evaluate their lives and compare these measurements with the average good life of others. The SPQL scale includes well-being, emotions, and physical and mental health indices. The SPQL scale has implications for evaluating the effectiveness of a wide range of interventions intended to improve mental health and well-being.

**Conceptual Model**

The SPQL construct consists of three axes (see SPQL model diagram): Subjective well-being (SWB), subjective affective experiences (SAE), and fulfillment of needs and preferences. Each axis is compounded from several variables (see SPQL model diagram). SWB consists of its baseline, which is the average of overall happiness/unhappiness, and transient deviations, which are measures of frequency and intensity of nonnormative transient experiences of happiness/unhappiness. Subjective affective experiences (SAE) consist of the average of overall positive and negative affect. Fulfillment of needs consists of a product of strength and fulfillment of a wide range of needs and preferences.

Because fluctuations within SPQL are likely to occur over time, a single-occasion measurement will not provide a comprehensive assessment (Diener, 2000; Lyubomirsky, Sheldon, & Schkade, 2005). In order to capture a more comprehensive picture of SPQL, the SWB variable (axis) was measured retrospectively throughout three major life stages of adult human development: Early-adulthood, mid-adulthood, and late-adulthood (see SPQL model diagram).

**Transitions Between Life Stages**

As people approach a life stage in their development, they face developmental tasks that they need to master in order for the transition to the next life stage to be successful (Erikson, 1968; Harter, 1998, 1999). The cycle of transition from one life stage to another is marked by three phases: (a) Mastering or failing a task; (b) consequential reevaluation of
life circumstances, values, and self-concept; and (c) adjustment and adaptation to new values and circumstances. To a lesser degree, cycles of transitions occur continually within major life stages on annual and even on daily bases.

A curve of SWB throughout the lifespan can reflect the experience of an individual’s good life. See Figure 2 for an example of the curve of a 69-year-old person. Ideally, all three SPQL axes should be evaluated for each life stage. However, this would make a questionnaire too long. Although the SPQL scale measures only one SPQL axis (SWB) for each life stage, the developed theoretical framework discusses the evaluation of all three SPQL axes throughout the major life stages. Thus, the framework can support the development of a next version of the scale that would accomplish this goal. Future research could explore possibilities for reducing the number of the evaluated scale items and include questions that will evaluate all three SPQL axes throughout the major life stages.

![Figure 2. Lifespan curve sample](image)

**SPQL Axes**

Participants’ responses on the inventories for each of the three SPQL axes (see Appendixes) provided the data for the psychometric validation of the scale and for the quantitative analyses that allowed measuring the good life. The theoretical framework for the first two axes was based on the existing theories of SWB, positive affect and negative affect, and mood (Diener et al., 1999; Fredrickson, 1998; Watson, 2000; Watson & Clark, 1994). The theoretical framework for the third axis was based on theories that conceptually differentiate between different categories of needs (Maslow, 1970; Panksepp, 2000). Different categories of needs, in turn, are sorted into four general categories of needs composing the third axis. The measurement of an individual’s level of functionality across social, psychological, and health factors was integrated in the third axis. This integration was accomplished through evaluating the strength and fulfillment of an individual’s needs for optimal functioning across these factors.

**Axis I: Subjective Well-Being (SWB)**
The SWB baseline is maintained by psychological and biological homeostasis (Brickman & Campbell, 1971; Cummins & Lau, 2004). Measurement of overall happiness determined the SWB baseline. A higher SWB baseline indicates a higher SPQL. People who have experienced more positive and less negative intense experiences during their lives (i.e., transient deviations), have a higher SPQL. Intense experiences were assessed through measuring the frequency and intensity of nonnormative transient subjective experiences of happiness/unhappiness that deviate from the SWB baseline throughout time.

**Axis II: Subjective Affective Experiences (SAE)**

People who have experienced more positive and less negative subjective affective experiences (SAE) during their lives (i.e., transient deviations), have a higher SPQL. The average of positive and negative SAE was used to measure overall SAE.

**Axis III: Fulfillment of Needs**

Individuals with the same score on SWB can differ in their evaluations of standards of living even if their objective life circumstances are alike. Accordingly, their self-perceived QOL may vary. Hence, in order to capture a more accurate measurement of SPQL, the strength and degree of fulfillment of a wide range of human needs and preferences for life circumstances was evaluated. However, felt needs are not the only kind of needs that a person may have (Maslow, 1970). If a need is satisfied it may not be felt as intensely as an unsatisfied need of lesser importance in terms of overall happiness. Thus, the strength with which a need is felt at a certain point in time does not necessarily indicate that it makes a greater contribution to the overall SPQL than other needs, which are felt less intensely or unfelt at all at that point in time because they are satisfied. Hence, the strength of individual preferences and needs was evaluated not only through questions such as "how important is fulfillment of this need to your overall happiness?" but also with questions such as "if this need were unfulfilled, how would it affect your overall happiness?"

Conceptual model for axis III fulfillment of needs. In order to measure fulfillment of needs, a broad range of human needs was sorted into four conceptually distinct categories (see Table 2) that are (a) contingent on corresponding stages of cognitive and moral development (Kohlberg, Levine, & Hewer, 1983; Piaget, 1952), (b) constitute major components of self-concept (Marcia, 1980), and (c) correspond to the neural activity in different clusters of anatomical brain regions (Berger, 2004; Lewis, 2000a). Because sometimes the same anatomical brain regions are involved in different ways in neural activity associated with the four categories of needs, implicated brain regions will be distinguished based on their dominance in related processes, and based on the chronological maturation of the dominant regions.
### Table 2. Categories of Needs by Self-Concept, Cognitive Development, Moral Development, and Corresponding Anatomical Brain Regions

<table>
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<tr>
<th>Categories of needs</th>
<th>Components of the self-concept</th>
<th>Cognitive development (Piaget)</th>
<th>Moral development (Kohlberg)</th>
<th>Corresponding anatomical brain regions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Instinctual needs</td>
<td>Instinctual self-concept</td>
<td>Prenatal through Sensorimotor</td>
<td>Preconventional</td>
<td>Brainstem; Lower functions</td>
</tr>
<tr>
<td>Self-centered emotional needs</td>
<td>Self-centered emotional self-concept</td>
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<td>Humanistic &amp; spiritual emotional needs</td>
<td>Humanistic &amp; spiritual emotional self-concept</td>
<td>Early Formal operational through Formal Operational</td>
<td>Conventional through Postconventional</td>
<td>Frontal lobes; Cortical association zones</td>
</tr>
<tr>
<td>Cognitive needs</td>
<td>Cognitive self-concept</td>
<td>Formal operational through Postformal Operational</td>
<td>Postconventional</td>
<td>Frontal lobes; Limbic system amygdala</td>
</tr>
</tbody>
</table>

**Four categories of needs on axis III.**

1. Instinctual needs include (a) sensory stimulation needs that became linked with positive or negative affect without involvement of cognitive evaluations; (b) physiological needs such as hunger, thirst, and sex; and (c) other physiological needs, such as those related to digestion, fluid balance, body temperature, and blood pressure, which could elicit positive or negative affect depending on whether the needs are met.

2. Self-centered emotional needs include (a) needs for safety and security (e.g., financial stability, home), love and belonging (e.g., affectionate relationships, sense of community), esteem (e.g., recognition, confidence); (b) ego-centered self-conscious needs (underpinned by self-conscious emotions, that is, emotions which require self-conscious awareness and evaluations), such as pride and honor (e.g., from personal accomplishments vs. nurturing), and guilt and embarrassment (with a focus on how one’s status has been affected vs. focusing on how others have been affected); and (c) spiritual/religious needs motivated by ego inflation or belonging emotional needs.

3. Humanistic and spiritual emotional needs include (a) a higher order of self-conscious altruistic needs that are not self-centered, such as pride and honor (e.g., from nurturing vs. from personal accomplishments), and guilt and embarrassment (with a focus on how others have been affected vs. focusing on how one’s status has been affected); (b) humanistic, self-actualization needs, characterized by a desire to fulfill one’s potential, which are not ego-centered (e.g., desiring truth over dishonesty); and (c) spiritual/religious needs which are not motivated by ego inflation or belonging emotional needs.
4. Cognitive needs include needs for harmony, organization, and coherence in (a) aesthetics (e.g., art, architecture, poetry, and music) and (b) intellect (sciences, information, and skills).

**Measuring axis III fulfillment of needs.**

Because according to the SPQL theory an individual’s motivations ensue from the idiosyncratic cluster of the four categories of needs, these four categories are proposed to compound an individual’s motivational framework (MF). In the following discussion, disparate preferences and needs will be referred to as motivational units (MU). Motivational units have two dimensions, importance of MU to the SPQL (strength) and the degree of fulfillment (see Figure 3). The strength of a motivational unit (MU) was determined by evaluating the capacity for the fulfillment or unfulfillment of the MU to skew the SWB baseline.

![Figure 2. Motivational units.](image)

**Happiness**

The smiley face is a well-known symbol of happiness
Happiness is a mental state of well-being characterized by positive emotions ranging from contentment to intense joy. A variety of biological, psychological, religious, and philosophical approaches have striven to define happiness and identify its sources.

Positive psychology endeavors to apply the scientific method to answer questions about what "happiness" is, and how we might attain it.

Philosophers and religious thinkers often define happiness in terms of living a good life, or flourishing, rather than simply as an emotion. Happiness in this older sense was used to translate the Greek Eudaimonia, and is still used in virtue ethics.

Happiness economics suggests that measures of public happiness should be used to supplement more traditional economic measures when evaluating the success of public policy.

**Scientific views**

Martin Seligman asserts that happiness is not just external, momentary pleasures. Flow (engagement) and general life satisfaction are parts of happiness too, for example.

Hotei, god of happiness in East Asian folklore
Happiness is a very fuzzy concept and can mean many things to many people. Part of the challenge of the science of happiness is to identify all the different uses of the word "happiness", or else to understand its various components.

Studies have found that things like money, education, or the weather do not affect happiness the way one might expect. There are various habits that have been correlated with happiness. Psychologist Martin Seligman provides the acronym PERMA to summarize many of Positive Psychology’s findings: humans seem happiest when they have Pleasure (tasty foods, warm baths, etc.), Engagement (or flow, the absorption of an enjoyed yet challenging activity), Relationships (social ties have turned out to be extremely reliable indicator of happiness), Meaning (a perceived quest or belonging to something bigger), and finally Accomplishments (having realized tangible goals).

There is evidence suggesting that people can improve their happiness. Mood disorders like depression are often understood through a biopsychosocial model, meaning biological, psychological, and social factors all contribute to mood (i.e. there is no single cause). The diathesis–stress model further argues that a diathesis (a biological vulnerability due to genes) to certain moods are worsened or improved by the environment and upbringing. The idea is that individuals with high vulnerability, especially if their early environment worsened depressive tendencies, may need antidepressants. Furthermore, the model suggests that everyone can benefit, to varying degrees, from the various habits and practices identified by positive psychology.

There have also been some studies of religion as it relates to happiness, as well as religious or generally philosophical notions of happiness. Research has generally found that religion
may help make people happier by providing various important components (e.g. PERMA) in countries where there are many who share that religion.

**Religious perspectives**

**Buddhism**

Happiness forms a central theme of Buddhist teachings. For ultimate freedom from suffering, the Noble Eightfold Path leads its practitioner to Nirvana, a state of everlasting peace. Ultimate happiness is only achieved by overcoming craving in all forms. More mundane forms of happiness, such as acquiring wealth and maintaining good friendships, are also recognized as worthy goals for lay people (see sukha). Buddhism also encourages the generation of loving kindness and compassion, the desire for the happiness and welfare of all beings.

**Catholicism**

In Catholicism, the ultimate end of human existence consists in felicity (Latin equivalent to the Greek eudaimonia), or "blessed happiness", described by the 13th-century philosopher-theologian Thomas Aquinas as a Beatific Vision of God's essence in the next life.

**Philosophical views**

The Chinese Confucian thinker Mencius, who 2300 years ago sought to give advice to the ruthless political leaders of the warring states period, was convinced that the mind played a mediating role between the "lesser self" (the physiological self) and the "greater self" (the moral self) and that getting the priorities right between these two would lead to sage-hood. He argued that if we did not feel satisfaction or pleasure in nourishing one's "vital force" with "righteous deeds", that force would shrivel up (Mencius,6A:15 2A:2). More specifically, he mentions the experience of intoxicating joy if one celebrates the practice of the great virtues, especially through music.

Al-Ghazali (1058–1111) the Muslim Sufi thinker wrote the Alchemy of Happiness, a manual of spiritual instruction throughout the Muslim world and widely practiced today.

The Hindu thinker Patanjali, author of the Yoga Sutras, wrote quite exhaustively on the psychological and ontological roots of bliss.

In the Nicomachean Ethics, written in 350 BCE, Aristotle stated that happiness (also being well and doing well) is the only thing that humans desire for its own sake, unlike riches, honor, health or friendship. He observed that men sought riches, or honor, or health not only for their own sake but also in order to be happy. Note that eudaimonia, the term we translate as "happiness", is for Aristotle an activity rather than an emotion or a state. Happiness is characteristic of a good life, that is, a life in which a person fulfills human nature in an excellent way. People have a set of purposes which are typically human: these belong to our nature. The happy person is virtuous, meaning they have outstanding
abilities and emotional tendencies which allow him or her to fulfill our common human ends. For Aristotle, then, happiness is "the virtuous activity of the soul in accordance with reason": happiness is the practice of virtue.

Many ethicists make arguments for how humans should behave, either individually or collectively, based on the resulting happiness of such behavior. Utilitarians, such as John Stuart Mill and Jeremy Bentham, advocated the greatest happiness principle as a guide for ethical behavior.

**Economic views**

![Newly commissioned officers celebrate their new positions by throwing their midshipmen covers into the air as part of the U.S. Naval Academy class of 2005 graduation and commissioning ceremony.](image)

Common market health measures such as GDP and GNP have been used as a measure of successful policy. On average richer nations tend to be happier than poorer nations, but this effect seems to diminish with wealth. This has been explained by the fact that the dependency is not linear but logarithmic, i.e., the same percentual increase in the GNP produces the same increase in happiness for wealthy countries as for poor countries.

Economic freedom correlates strongly with happiness preferably within the context of a western mixed economy, with free press and a democracy. "Socialist" East European countries were less happy than Western ones, even less happy than other equally poor countries. It would be inaccurate to consider the ex-Soviet states as socialist, however, as socialism indicates that the workers own the means of production, which under the Soviet Union was not the case.

It has been argued that happiness measures could be used not as a replacement for more traditional measures, but as a supplement. According to professor Edward Glaeser, people
constantly make choices that decrease their happiness, because they have also more important aims. Therefore, the government should not decrease the alternatives available for the citizen by patronizing them but let the citizen keep a maximal freedom of choice.

It has been argued that happiness at work is the one of the driving forces behind positive outcomes at work, rather than just being a resultant product.

**Happiness economics**

Happiness economics is the quantitative study of happiness, positive and negative affect, well-being, quality of life, life satisfaction and related concepts, typically combining economics with other fields such as psychology and sociology. It typically treats such happiness-related measures, rather than wealth, income or profit, as something to be maximized. The field has grown substantially since the late 20th century, for example by the development of methods, surveys and indices to measure happiness and related concepts.

**Metrology**

Given its very nature, reported happiness is subjective. It is difficult to compare one person’s happiness with another. It can be especially difficult to compare happiness across cultures. However, many happiness economists believe they have solved this comparison problem. Cross-sections of large data samples across nations and time demonstrate consistent patterns in the determinates of happiness.

Happiness is typically measured using subjective measures - e.g. surveys - and/or objective measures. One concern has always been the accuracy and reliability of people’s responses to happiness surveys. Objective measures such as lifespan, income and education, are often used as well as or instead of subjectively reported happiness, though this assumes that they generally produce happiness, which while plausible may not necessarily be the case. The terms quality of life or well-being are often used to encompass these more objective measures.

Some scientists claim that happiness can be measured both subjectively and objectively by observing the joy center of the brain lit up with advanced imaging, although this raises philosophical issues, for example about whether this can be treated as more reliable than reported subjective happiness.

Micro-econometric happiness equations have the standard form: \( W_{it} = \alpha + \beta x_{it} + \epsilon_{it} \). In this equation \( W \) is the reported well-being of individual \( i \) at time \( t \), and \( x \) is a vector of known variables, which include socio-demographic and socioeconomic characteristics.

**Determinants**
Maslow's hierarchy of needs

Abraham Maslow theorized that human happiness is the outcome of meeting a set of needs. He listed these in order of priority, leading to a pyramid called Maslow's hierarchy of needs. The set of needs includes physiological, safety, love/belonging, esteem, and self-actualization needs. These needs can be used as a basis for evaluating the overall happiness level of individuals.

Money

GDP and GNP

Typically national financial measures, such as GDP and GNP, have been used as a measure of successful policy. Although on average richer nations tend to be happier than poorer nations, some studies have indicated that beyond an average GDP per capita of about $15,000 (most of the world’s nations have less than this), the average income in a nation makes little difference to the average self-reported happiness. Other economists have disputed the accuracy of these studies, finding a logarithmic correlation between GDP per capita and self-reported happiness.

However, a free market think tank Cato Institute points out that since life expectancy has continued to increase in nations wealthier than this, often partly attributed to economic growth, Happy Life Years have continued to increase.

It has been argued that happiness measures could be used not as a replacement for more traditional measures but as a supplement.

Individual income

Historically, economists have said that well-being is a simple function of income. However, it has been found that once wealth reaches a subsistence level, its effectiveness as a generator of well-being is greatly diminished. This paradox has been referred to as the Easterlin paradox and may result from a "hedonic treadmill." This means that aspirations increase with income; after basic needs are met, relative rather than absolute income levels influence well-being. Happiness economists hope to change the way governments view well-being and how to most effectively govern and allocate resources given this paradox. However, other research suggests that no paradox exists, and happiness is linearly related to the logarithm of absolute (real, PPP-adjusted) income, with little or no relative income component.

Money correlates with happiness, but the rate diminishes with more money. In 2010, Daniel Kahneman and Angus Deaton found that higher earners generally reported better life satisfaction, but people’s day-to-day emotional well-being only rose with earnings until a threshold annual income of $75,000. Other factors have been suggested as making people happier than money. A short term course of psychological therapy is 32 times more cost
effective at increasing happiness than simply increasing income. One study, when corrected for social status, showed no correlation between income and happiness.

Social security

Professor Ruut Veenhoven showed that social security payments do not seem to add to happiness. This may be due to the fact that non-self-earned income (e.g., from a lottery) does not add to happiness in general either. Happiness may be the minds reward to a useful action. However, Johan Norberg of CFS, a free enterprise economy think tank, presents a hypothesis that as people who think that they themselves control their lives are more happy, paternalist institutions may decrease happiness.

An alternative perspective focuses on the role of the welfare state as an institution that improves quality of life not only by increasing the extent to which basic human needs are met, but also by promoting greater control of one's life by limiting the degree to which individuals find themselves at the mercy of impersonal market forces that are indifferent to the fate of individuals. This is the argument suggested by the U.S. political scientist Benjamin Radcliff, who has presented a series of papers in peer reviewed scholarly journals demonstrating that a more generous welfare state contributes to higher levels of life satisfaction, and does so to rich and poor alike.

Employment

Work is important to happiness. It creates a sense of purpose, beneficial relationships with co-workers, and also earns money. Losing one's job can be a great source of unhappiness.

Relationships and children

Relationships, particularly those with women, are important to the happiness of both sexes.

Children tend to decrease parental happiness, at least until they leave home, although in terms of a broader life narrative the opposite may be true. Some research shows that at some ages (toddlers and teenagers) they decrease parental happiness, whereas at others they increase it, averaging out to no overall change. Married people are happier, but it is unclear if this is due to the marriage or if happy people are more likely to marry.

Marriage, children and how happy they make us, provide a perfect case study for these questions. Gildert's writes that prospective parents know that raising children will be laborious, yet they believe it will make them very happy. In fact, studies show it does just the opposite, and that levels of parental happiness don't rise until kids leave for college (so much for the empty-nest theory). Still, if happiness is thought of in terms of a broader life narrative, rather than just specific moments of teething, diaper changing and petty-cash culling, it's pretty clear that kids do add value. Happiness politicians know that welfare states need more kids to plug the coming labor shortage — but should they actively encourage something that will make people unhappy, at least in the short run? Likewise marriage—married couples test happier, but it's unclear if that's because happy people
marry. Whether or not politicians back policies that support marriage and having kids doesn’t really matter, because people embrace these happiness myths quite willingly. "We are the product of our genes and our societies," says Gilbert. Traditions will trump the empirical evidence that money and kids won’t make us happy.

**Freedom and control**

There is a significant correlation between feeling in control of one’s own life and happiness levels.

A study conducted at the University of Zurich suggested that democracy and federalism bring well-being to individuals. It concluded that the more direct political participation possibilities available to citizens raises their subjective well-being. Two reasons were given for this finding. First, a more active role for citizens enables better monitoring of professional politicians by citizens, which leads to greater satisfaction with government output. Second, the ability for citizens to get involved in and have control over the political process, independently increases well-being.

According to a free market think tank Cato Institute, higher economic freedom, as measured by both the Heritage and the Fraser indices, correlates strongly with higher self-reported happiness.

**Leisure**

The amount of spare time people have, as well as their control over how much spare time they have, correlates with happiness.

Whereas leisure pursuits increase happiness, watching television is an anomaly, as it seems to correlate with lower happiness. This may be because people who watch a lot of television are lacking in better sources of happiness, such as relationships and other leisure pursuits; that is, people watch television if they don’t have anything better to do.

**Health**

Happiness appears to be inversely related to levels of stress, allergy, asthma and other chronic conditions.

**Indices**

The idea that happiness is important to a society is not new. Thomas Jefferson put the “pursuit of happiness” on the same level as life and liberty in the United States Jeremy Bentham believed that public policy should attempt to maximize happiness, and he even attempted to estimate a "hedonic calculus". Many other prominent economists and philosophers throughout history, including Aristotle, incorporated happiness into their work.
The Satisfaction with Life Index is an attempt to show the average self-reported happiness in different nations. This is an example of a recent trend to use direct measures of happiness, such as surveys asking people how happy they are, as an alternative to traditional measures of policy success such as GDP or GNP. Some studies suggest that happiness can be measured effectively. The Inter-American Development Bank (IDB), published in November 2008 a major study on happiness economics in Latin America and the Caribbean ("Beyond Facts: Measuring Quality of Life", http://www.iadb.org/idd/docs/1776308.pdf).

There are also several examples of measures that includes self-reported happiness as one variable. Happy Life Years, a concept brought by Dutch sociologist Ruut Veenhoven, combines self-reported happiness with life expectancy. The Happy Planet Index combines it with life expectancy and ecological footprint.

Gross national happiness (GNH) is a concept introduced by the King of Bhutan in 1972 as an alternative to GDP. Several countries have already developed or are in the process of developing such an index. Bhutan’s index has led that country to limit the amount of deforestation it will allow and to require that all tourists to its nation must spend US$200. Allegedly, extensive tourism and deforestation lead to unhappiness.

After the military coup of 2006, Thailand also instituted an index. The stated promise of the new Prime Minister Surayud Chulanont is to make the Thai people not only richer but happier as well. Much like GDP results, Thailand releases monthly GNH data. The Thai GNH index is based on a 1-10 scale with 10 being the most happy. As of May 13, 2007, the Thai GNH measured 5.1 points. The index uses poll data from the population surveying various satisfaction factors such as, security, public utilities, good governance, trade, social justice, allocation of resources, education and community problems.

Australia, Canada, China, France and the United Kingdom are also coming up with indexes to measure national happiness. North Korea also announced an international Happiness Index in 2011 through Korean Central Television. North Korea itself came in second, behind #1 China.

Ecuador's and Bolivia's new constitutions state the indigenous concept of "good life" ("buen vivir" in Spanish, "sumak kawsay" in Quichua, and "suma qamaña" in Aymara) as the goal of sustainable development.

**Neo-classical economics**

Under neo-classical economic theory happiness, subjectively defined, has long been the standard of measurement used interchangeably with utility as well as the general welfare.

Modern classical economics no longer attempts to quantify happiness or satisfaction through measurements in consumption and profits. Instead, modern neoclassical framework argue that individual’s preference is revealed through choice. Therefore, if an individual decided to purchase an apple over orange, the satisfaction one derived from...
apple is revealed to be greater than an orange. Similarly, modern economics also consider that work/leisure balance is also matter of individual choice.

The idea that modern neoclassical economics define happiness on the basis of consumption is widely disputed. The basis of utility has been defined as revealed preference.

The assumption within neoclassic economics that satisfactions are highly subjective found expression in the work of Vilfredo Pareto, whose definition of optimal allocation in the nineteenth century was a crucial contribution that allowed further development of the mathematical precision of the discipline. Pareto argued that because satisfactions are subjective, we cannot know for certain that we have increased the amount of satisfaction in the system if we take a dollar from a billionaire and give it to a starving person to buy food; for all we know, the billionaire might have derived as much satisfaction from that dollar as the starving person does in spending it on food.

This counter-intuitive result is the cornerstone of Pareto Optimality: a system is in Pareto Optimality when no one can be made better off (in their own estimation) without making someone worse off (in their own estimation). In practice, "better off" and "worse off" are defined by consumption: by definition, it is always better to consume more. Thus, Pareto Optimality led to the bias in standard economics toward perpetual growth models—models that are increasingly being called into question, as being impractical (and dangerously destructive) in a finite world.

**Criticism**

Some have suggested that establishing happiness as a metric is only meant to serve political goals. Recently there has been concern that happiness research could be used to advance authoritarian aims. As a result, some participants at a happiness conference in Rome have suggested that happiness research should not be used as a matter of public policy but rather used to inform individuals.

In addition, survey findings can lead to subjective interpretations. For example, a happiness study conducted in Russia during the 1990s indicated that as unemployment grew, the well-being of both those employed and unemployed rose. The interpretation of this could be that it resulted from diminished expectations and respondents who were less critical of their own situation when many around them were unemployed, or it could be interpreted as being the result of everyone benefitting from the unpaid work that the unemployed were able to do for their families and communities with their increased time resource.

**Health realization**

Health realization (HR) is a resiliency approach to personal and community psychology first developed in the 1980s by Roger C. Mills and George Pransky, and based on ideas and insights these psychologists elaborated from attending the lectures of philosopher and author Sydney Banks. HR first became known for its application in economically and
socially marginalized communities living in highly stressful circumstances (see Community Applications below).

HR focuses on the nature of thought and how it affects one's experience of the world. Students of HR are taught that they can change how they react to their circumstances by becoming aware that they are creating their own experience as they respond to their thoughts, and by connecting to their "innate health" and "inner wisdom."

HR also goes under the earlier names "Psychology of Mind" and "Neo-Cognitive Psychology," and it is closely related to "Innate Health" and the "Three Principles" understanding.

**The health realization model**

In the health realization ("HR") model, all psychological phenomena, from severe disorder to glowing health, are presented as manifestations of three operative "principles" first formulated as principles of human experience by Sydney Banks:

- **Mind** - the universal energy that animates all of life, the source of innate health and well-being.
- **Consciousness** - the ability to be aware of one's life.
- **Thought** - the power to think and thereby to create one's experience of reality.

"Mind" has been likened to the electricity running a movie projector, and "Thought" to the images on the film. "Consciousness" is likened to the light from the projector that throws the images onto the screen, making them appear real.

According to HR, people experience their reality and their circumstances through the constant filter of their thoughts. Consciousness makes that filtered reality seem "the way it really is." People react to it as if this were true. But, when their thinking changes, reality seems different and their reactions change. Thus, according to HR, people are constantly creating their own experience of reality via their thinking.

People tend to experience their reality as stressful, according to HR, when they are having insecure or negative thoughts. But HR suggests that such thoughts do not have to be taken seriously. When one chooses to take them more lightly, according to HR, the mind quiets down and positive feelings emerge spontaneously. Thus, HR also teaches that people have health and well-being already within them (in HR this is known as "innate health"), ready to emerge as soon as their troubled thinking calms down. When this happens, according to HR, people also gain access to common sense, and they can tap into the universal capacity for creative problem solving or "inner wisdom." Anecdotal reports suggest that, when a person grasps the understanding behind HR in an experiential way, an expansive sense of emotional freedom and well-being can result.

**Health realization as therapy**
In contrast to psychotherapies that focus on the content of the clients' dysfunctional thinking, HR focuses on "innate health" and the role of "Mind, Thought, and Consciousness" in creating the clients' experience of life.

The HR counselor does not attempt to get clients to change their thoughts, "think positive," or "reframe" negative thoughts to positive ones. According to HR, one's ability to control one's thoughts is limited and the effort to do so can itself be a source of stress. Instead, clients are encouraged to consider that their "minds are using thought to continuously determine personal reality at each moment."

HR characterizes feelings and emotions as indicators of the quality of one's thinking. Within the HR model, unpleasant feelings or emotions, or stressful feelings, indicate that one's thinking is based on insecurity, negative beliefs, conditioning or learned patterns that are not necessarily appropriate to the live moment here and now. They simultaneously indicate that the individual has temporarily lost sight of what HR asserts is his or her own role in creating experience. Pleasant or desired feelings (such as a sense of well-being, gratitude, compassion, peace, etc.) indicate, within the HR model, that the quality of one's thinking is exactly as it needs to be.

HR holds that the therapeutic "working through" of personal issues from the past to achieve wholeness is unnecessary. According to the HR model, people are already whole and healthy. The traumas of the past are only important to the extent that the individual lets them influence his or her thoughts in the present. According to HR, one's "issues" and memories are just thoughts, and the individual can react to them or not. The more the clients' experience is that they themselves are creating their own painful feelings via their own "power of Thought," HR suggests, the less these feelings bother them. Sedgeman has compared this to what happens when we make scary faces at ourselves in the mirror: because we know it is just us, it is impossible to scare ourselves that way.

Thus HR deals with personal insecurities and dysfunctional patterns almost en masse, aiming for an understanding of the "key role of thought", an understanding that ideally allows the individual to step free at once from a large number of different patterns all connected by insecure thinking. With this approach, it is rare for the practitioner to delve into specific content beyond the identification of limiting thoughts. When specific thoughts are considered to be limiting or based on insecurity or conditioning, the counselor encourages the individual to disengage from them.

**Relationships**

From the perspective of HR, relationship problems result from the partners' low awareness of their role in creating their own experience via thought and consciousness. Partners who respond to HR reportedly stop blaming and recriminating and react differently to each other. HR counselors aim to get couples to consider that each one's own feelings are not determined by one's partner and that the great majority of issues that previously snarled their interactions were based on insecure, negative, and conditioned thinking. HR counselors further suggest that every person goes through emotional ups and downs and
that one's thinking in a "down" mood is likely to be distorted. HR teaches that it is generally counterproductive to try to "talk through" relationship problems when the partners are in a bad mood. Instead HR suggests that partners wait until each has calmed down and is able to discuss things from a place of inner comfort and security.

**Chemical dependency and addiction**

HR sees chemical dependency and related behaviors as a response to a lack of a sense of self-efficacy, rather than the result of disease. That is, some people who are, in HR terms, "unaware" of their own "innate health" and their own role in creating stress via their thoughts turn to alcohol, drugs, or other compulsive behaviors in the attempt to quell their stressful feelings and regain some momentary sense of control. HR aims to offer deeper relief by showing that negative and stressful feelings are self-generated and thus can be self-quieted and it seeks to provide a pathway to well-being that does not depend on external circumstances.

**Community applications**

The Health Realization ("HR") model has been applied in a variety of challenging settings. An early project, which garnered national publicity under the leadership of Roger Mills, introduced HR to residents of a pair of low-income housing developments in Miami known as Modello and Homestead Gardens. After three years, there were major documented reductions in crime, drug dealing, teenage pregnancy, child abuse, child neglect, school absenteeism, unemployment, and families on public assistance. Jack Pransky has chronicled the transformation that unfolded there, in his book Modello, A Story of Hope for the Inner City and Beyond.

Later projects in some of the most severely violence-ridden housing developments in New York, Minnesota, and California and in other communities in California, Hawaii, and Colorado built upon the early experience in the Modello/Homestead work. The Coliseum Gardens housing complex in Oakland, California, for example, had previously had the fourth highest homicide rate of such a complex in the US, but after HR classes were launched, the homicide rate began to decline. Gang warfare and ethnic clashes between Cambodian and African-American youth ceased. In 1997, Sargeant Jerry Williams was awarded the California Wellness Foundation Peace Prize on behalf of the Health Realization Community Empowerment Project at Coliseum Gardens. By the year 2006, there had been no homicides in the Complex for nine straight years.

The HR model has also found application in police departments, prisons, mental health clinics, community health clinics and nursing, drug and alcohol rehabilitation programs, services for the homeless, schools, and a variety of state and local government programs. The County of Santa Clara, California, for example, has established a Health Realization Services Division which provides HR training to County employees and the public. The Services Division "seeks to enhance the life of the individual by teaching the understanding of the psychological principles of Mind, Thought and Consciousness, and how these principles function to create our life experience," and to "enable them to live healthier and
more productive lives so that the community becomes a model of health and wellness." The Department of Alcohol and Drug Services introduced HR in Santa Clara County in 1994. The Health Realization Services Division has an approved budget of over $800,000 (gross expenditure) for FY 2008, a 41% increase over 2007, at a time when a number of programs within the Alcohol and Drug Services Department have sustained budget cuts.

HR community projects have received grant funding from a variety of sources. For example, grant partners for the Visitacion Valley Community Resiliency Project, a five-year, multimillion-dollar community revitalization project, have included Wells Fargo Bank, Charles Schwab Corporation Foundation, Charles and Helen Schwab Foundation, Isabel Allende Foundation, Pottruck Family Foundation, McKesson Foundation, Richard and Rhoda Goldman Fund, S.H. Cowell Foundation, San Francisco Foundation, Evelyn & Walter Haas, Jr. Fund, Milagro Foundation, and Dresdner RCM Global Investors. Other projects based upon the HR approach have been funded by the National Institute of Mental Health, the U.S. Department of Justice, the National Institute on Drug Abuse, the California Wellness Foundation, and the Shinnyo-en Foundation.

Organizational applications

From the original applications, as people in the business world have been introduced to HR or the "Three Principles" (as the core understanding is known), they have started to bring these ideas into the business world they have come from. The approach has been introduced to people in medicine, law, investment and financial services, technology, marketing, manufacturing, publishing, and a variety of other commercial and financial roles. It has been reported anecdotally to have had significant impact in the areas of individual performance and development, teamwork, leadership, change and diversity. According to HR/Three Principles adherents, these results flow naturally as the individuals exposed to the ideas learn how their thoughts have been creating barriers to others and barriers to their own innate creativity, common sense, and well being. As people learn how to access their full potential more consistently, HR adherents say, they get better results with less effort and less stress in less time.

Two peer-reviewed articles on effectiveness with leadership development were published in professional journals in 2008 (ADHR) and 2009 (ODJ). See "Organizations and Business" section below (Polsfuss & Ardichvili).

Philosophical context

Health Realization ("HR") rests on the non-academic philosophy of Sydney Banks, which Mr. Banks has expounded upon in several books. Mr. Banks was a day laborer with no education beyond ninth grade (age 14) in Scotland who, in 1973, reportedly had a profound insight into the nature of human experience. Mr. Banks does not particularly attempt to position his ideas within the larger traditions of philosophy or religion; he is neither academically trained nor well read. His philosophy focuses on the illusionary, thought-created nature of reality, the three principles of "Mind", "Thought", and "Consciousness", the potential relief of human suffering that can come from a fundamental
shift in personal awareness and understanding and the importance of a direct, experiential grasp of these matters, as opposed to a mere intellectual comprehension or analysis. Mr. Banks suggests that his philosophy is best understood not intellectually but by "listening for a positive feeling;" and a grasp of HR is said to come through a series of "insights," that is, shifts in experiential understanding.

Teaching of health realization

Health Realization ("HR"), like Sydney Banks's philosophy, is deliberately not taught as a set of "techniques" but as an experiential "understanding" that goes beyond a simple transfer of information. There are no steps, no uniformly-appropriate internal attitudes, and no techniques within it. The "health of the helper" is considered crucial; that is, trainers or counselors ideally will "live in the understanding that allows them to enjoy life," and thereby continuously model their understanding of HR by staying calm and relaxed, not taking things personally, assuming the potential in others, displaying common sense, and listening respectfully to all. Facilitators ideally teach in the moment, from "what they know" (e.g. their own experience), trusting that they will find the right words to say and the right approach to use in the immediate situation to stimulate the students' understanding of the "Three Principles". Rapport with students and a positive mood in the session or class are more important than the specific content of the facilitator's presentation.

Evaluations of health realization

A recent peer-reviewed article evaluating the effectiveness of HR suggests that the results of residential substance abuse treatment structured around the teaching of HR are equivalent to those of treatment structured around 12-step programs. The authors note that "these results are consistent with the general findings in the substance abuse literature, which suggests that treatment generally yields benefits, irrespective of approach."

A small peer-reviewed study in preparation for a planned larger study evaluated the teaching of HR/Innate Health via a one-and-a-half day seminar, as a stress- and anxiety-reduction intervention for HIV-positive patients. All but one of the eight volunteer participants in the study showed improved scores on the Brief Symptom Inventory after the seminar, and those participants who scored in the "psychiatric outpatient" range at the beginning of the seminar all showed improvement that was sustained upon follow-up one month later. The study's authors concluded that "The HR/IH psychoeducational approach deserves further study as a brief intervention for stress-reduction in HIV-positive patients."

A recent pilot study funded by the National Institutes of Health evaluated HR in lowering stress among Somali and Oromo refugee women who had experienced violence and torture in their homelands, but for whom Western-style psychotherapeutic treatment of trauma was not culturally appropriate. The pilot study showed that "the use of HR with refugee trauma survivors was feasible, culturally acceptable, and relevant to the participants." In a post-intervention focus group, "many women reported using new strategies to calm down, quiet their minds and make healthier decisions." Co-investigator Cheryl Robertson,
Assistant Professor in the School of Nursing at the University of Minnesota, was quoted as saying, "This is a promising intervention that doesn't involve the use of highly trained personnel. And it can be done in the community."

The Visitacion Valley Community Resiliency Project (VVCRP) was reviewed by an independent evaluator hired by the Pottruck Foundation. Her final report notes that "Early program evaluation...found that the VVCRP was successful in reducing individuals' feelings of depression and isolation, and increasing their sense of happiness and self-control. The cumulative evaluation research conducted on the VVCRP and the HR model in general concludes that HR is a powerful tool for changing individuals' beliefs and behaviors." In the Summary of Case Studies, the report goes on to state, "The VVCRP was effective over a period of five years of sustained involvement in two major neighborhood institutions...at influencing not just individuals, but also organizational policies, practices, and culture. This level of organizational influence is impressive when the relatively modest level of VVCRP staff time and resources invested into making these changes is taken into account. The pivotal levers of change at each organization were individual leaders who were moved by the HR principles to make major changes in their own beliefs, attitudes, and behaviors, and then took the initiative to inspire, enable, and mandate similar changes within their organizations. This method of reaching “critical mass” of HR awareness within these organizations appears to be both efficient and effective when the leadership conditions are right. However, this pathway to change is vulnerable to the loss of the key individual leader."

Research efforts on effectiveness

Pransky has reviewed the research on HR (through 2001) in relation to its results for prevention and education, citing about 20 manuscripts, most of which were conference papers, and none peer-reviewed journal articles, although two were unpublished doctoral dissertations. (Kelley (2003) cites two more unpublished doctoral dissertations.) Pransky concludes, "Every study of Health Realization and its various incarnations, however weak or strong the design, has shown decreases in problem behaviors and internally experienced problems. This approach appears to reduce problem behaviors and to improve mental health and well-being. At the very least, this suggests the field of prevention should further examine the efficacy of this ... approach by conducting independent, rigorous, controlled, longitudinal studies...."

Since at least 2008 peer-reviewed professional journal articles on its effectiveness have been published. See C.L.Polsfuss, A.Ardichvili articles in "Organizations and Business" section below.

Criticism

In their writings about HR, authors Roger Mills and Elsie Spittle note several kinds of objections to it.
In a criticism of the philosophy of Sydney Banks and, by implication, the HR approach, Bonelle Strickling, a psychotherapist and Professor of Philosophy, is quoted in an article in the Vancouver Sun as objecting that "it makes it appear as if people can, through straightforward positive thinking, 'choose' to transcend their troubled upbringings and begin leading a contented life." She goes on to say that "it can be depressing for people to hear it's supposed to be that easy. It hasn't been my experience that people can simply choose not to be negatively influenced by their past." Referring to Banks's own experience, she says, "Most people are not blessed with such a life-changing experience.... When most people change, it usually happens in a much more gradual way."

The West Virginia Initiative for Innate Health (at West Virginia University Health Sciences Center), which promotes HR/Innate Health and the philosophy of Sydney Banks through teaching, writing, and research, was the center of controversy soon after its inception in 2000 as the Sydney Banks Institute for Innate Health. Initiated by Robert M. D'Alessandri, the Dean of the medical school there, the institute reportedly was criticized (without attribution) as pushing "junk science," and Banks's philosophy was characterized (also without attribution) as "a kind of bastardized Buddhism" and "New Age." William Post, an orthopedic surgeon who quit the medical school because of the institute, was reported along with other unnamed professors to have accused the Sydney Banks Institute of promoting religion in a state-funded institution, and Harvey Silvergate, a civil-liberties lawyer, was quoted as agreeing that "essentially [the institute] seems like a cover for a religious-type belief system which has been prettified in order to be secular and even scientific." A Dr. Blaha, who resigned as chairman of Orthopedics at WVU, was quoted as criticizing the institute as being part of a culture at the Health Sciences Center that, in his view, places too much emphasis on agreement, consensus, and getting along. Other professors reportedly supported the institute. Anthony DiBartolomeo, chief of the rheumatology section, was quoted as calling it "a valuable addition" to the health-sciences center, saying its greatest value was in helping students, residents, and patients deal with stress.

Reportedly in response to the controversy, the WVIIH changed its name from The Sydney Banks Institute to the West Virginia Initiative for Innate Health, although its mission remains unchanged.

Support for specific tenets of HR from other philosophies and approaches

Some of the tenets of HR are consistent with the theories of philosophers, authors and researchers independently developing other approaches to change and psychotherapy.

A large body of peer-reviewed case literature in psychotherapy by Milton Erickson, M.D., founding president of the American Society for Clinical Hypnosis, and others working in the field of Ericksonian psychotherapy, supports the notion that lasting change in psychotherapy can occur rapidly without directly addressing clients' past problematic experiences.

Many case examples and a modest body of controlled outcome research in solution focused brief therapy (SFBT), have likewise supported the notion that change in psychotherapy can
occur rapidly, without delving into the clients' past negative experiences. Proponents of SFBT suggest that such change often occurs when the therapist assists clients to step out of their usual problem-oriented thinking.

The philosophy of social constructionism, which is echoed in SFBT, asserts that reality is reproduced by people acting on their interpretations and their knowledge of it. (HR asserts that thought creates one's experience of the world.)

A major body of peer-reviewed research on "focusing," a change process developed by philosopher Eugene Gendlin, supports the theory that progress in psychotherapy is dependent on something clients do inside themselves during pauses in the therapy process, and that a particular internal activity — "focusing" — can be taught to help clients improve their progress. The first step of the six-step process used to teach "focusing" involves setting aside one's current worries and concerns to create a "cleared space" for effective inner reflection. Gendlin has called this first step by itself "a superior stress-reduction method." (HR emphasizes the importance of quieting one's insecure and negative thinking to reduce stress and gain access to "inner wisdom," "common sense," and well-being.)

Positive psychology emphasizes the human capacity for health and well-being, asserts the poor correlation between social circumstances and individual happiness, and insists on the importance of one's thinking in determining one's feelings.

Work by Herbert Benson argues that humans have an innate 'breakout principle' which provides creative solutions and peak experiences which allow the restoration of a 'new-normal' state of higher functioning. This breakout principle is activated by severing connections with current circular or repetitive thinking. This is heavily reminiscent of Health Realization discussion of the Principle of Mind and of how it is activated.

Finally, resilience research, such as that by Emmy Werner, has demonstrated that many high-risk children display resilience and develop into normal, happy adults despite problematic developmental histories.

**Positive psychology**

Positive psychology is a recent branch of psychology whose purpose was summed up in 1998 by Martin Seligman and Mihaly Csikszentmihalyi: "We believe that a psychology of positive human functioning will arise that achieves a scientific understanding and effective interventions to build thriving in individuals, families, and communities." Positive psychologists seek "to find and nurture genius and talent", and "to make normal life more fulfilling", not simply to treat mental illness. The field is intended to complement, not to replace traditional psychology. It does not seek to deny the importance of studying how things go wrong, but rather to emphasize the importance of using the scientific method to determine how things go right. Researchers in the field analyze things like states of pleasure or flow, values, virtues, talents, as well the ways that they can be promoted by social systems and institutions.
Background

Several humanistic psychologists—such as Abraham Maslow, Carl Rogers, and Erich Fromm—developed theories and practices that involved human happiness. Recently the theories of human flourishing developed by these humanistic psychologists have found empirical support from studies by positive psychologists. Positive psychology has also moved ahead in a number of new directions.

Positive psychology began as a new area of psychology in 1998 when Martin Seligman, considered the father of the modern positive psychology movement, chose it as the theme for his term as president of the American Psychological Association, though the term originates with Maslow, in his 1954 book Motivation and Personality, and there have been indications that psychologists since the 1950s have been increasingly focused on promoting mental health rather than merely treating illness. Seligman pointed out that for the half century clinical psychology "has been consumed by a single topic only - mental illness", echoing Maslow's comments. He urged psychologists to continue the earlier missions of psychology of nurturing talent and improving normal life.

The first positive psychology summit took place in 1999. The First International Conference on Positive Psychology took place in 2002. More attention was given by the general public in 2006 when, using the same framework, a course at Harvard University became particularly popular. In June 2009, the First World Congress on Positive Psychology took place.

Historical roots

Positive psychology finds its roots in the humanistic psychology of the 20th century, which focused heavily on happiness and fulfillment. Earlier influences on positive psychology came primarily from philosophical and religious sources, as scientific psychology did not take its modern form until the late 19th century. (See History of psychology)

Judaism promotes a Divine command theory of happiness: happiness and rewards follow from following the commands of the divine.

The ancient Greeks had many schools of thought. Socrates advocated self-knowledge as the path to happiness. Plato's allegory of the cave influenced western thinkers who believe that happiness is found by finding deeper meaning. Aristotle believed that happiness, or eudaimonia is constituted by rational activity in accordance with virtue over a complete life. The Epicureans believed in reaching happiness through the enjoyment of simple pleasures. The Stoics believed they could remain happy by being objective and reasonable, and they describe many "spiritual exercises" that have been compared to the psychological exercises employed in CBT and Positive Psychology.

Christianity continued to follow the Divine command theory of happiness. In the Middle Ages, Christianity taught that true happiness would not be found until the afterlife. The
seven deadly sins are about earthly self-indulgence and narcissism. On the other hand, the Four Cardinal Virtues and Three Theological Virtues were supposed to keep one from sin.

During the Renaissance and Age of Enlightenment, individualism came to be valued. Simultaneously, creative individuals gained prestige, as they were now considered to be artists, not just craftsmen. Utilitarian philosophers such as John Stuart Mill believed that moral actions are those actions that maximize happiness for the most number of people, suggesting an empirical science of happiness should be used to determine which actions are moral (a science of morality). Thomas Jefferson and other proponents of democracy believed that "Life, liberty and the pursuit of happiness" are inalienable rights, and that it justifies the overthrow of the government.

The Romantics valued individual emotional expression and sought their emotional "true selves," which were unhindered by social norms. At the same time, love and intimacy became the main motivations for people to get married.

Methods

"Happiness" encompasses many different emotional and mental phenomena (see below). One method of assessment is Ed Diener's Satisfaction with Life Scale. This 5-question survey corresponds well with impressions from friends and family, and low incidences of depression.

The "Remembering self" may not be the best source of information for pleasing the "Experiencing self"

Rather than long-term, big picture appraisals, some methods attempt to identify the amount of positive affect from one activity to the next. Some scientists use beepers to remind volunteers to write down the details of their current situation. Alternatively, volunteers complete detailed diary entries each morning about the day before. An
interesting discrepancy arises when researchers compare the results of these short-term "experience sampling" methods, with long-term appraisals. Namely, the latter may not be very accurate; people may not know what makes their life pleasant from one moment to the next. For instance, parents' appraisals mention their children as sources of pleasure, and yet 'experience sampling' indicates that they were not enjoying caring for their children compared to other activities.

Psychologist Daniel Kahneman explains this discrepancy by differentiating between happiness according to the 'Experiencing Self' compared to the 'Remembering Self'. Kahneman explains that, when we are asked to reflect on experiences, memory biases like the Peak-End effect (e.g. we mostly remember the dramatic parts of a vacation, and how it was at the end) play a large role. One of his more striking findings was in a study of colonoscopy patients. By adding 60 seconds to this invasive procedure, Kahneman actually got participants to report the colonoscopy as more pleasant. He accomplished this by making sure that, for the extra 60 seconds, the colonoscopy instrument was not moved, since movement is the source of the most discomfort. Thus, Kahneman was appealing to the Remembering Self's tendency to focus on the end of the experience. Such findings help explain human error in Affective forecasting - people's ability to predict their future emotional states.

Michael Argyle developed the Oxford Happiness Questionnaire as a broad measure of psychological well-being. This has been criticized as an aggregate of self-esteem, sense of purpose, social interest and kindness, sense of humor and aesthetic appreciation.

**Neuroscientific approach**

Neuroscience and brain imaging has shown increasing potential for helping science understand happiness and sadness. Though it may be impossible to achieve any comprehensive measure of happiness objectively, some physiological correlates to happiness can be measured. Stefan Klein, in his book The Science of Happiness, links the dynamics of neurobiological systems (i.e., dopaminergic, opiate) to the concepts and findings of positive psychology and social psychology.

Nobel prize winner Eric Kandel and researcher Cynthia Fu describe their findings that depression can be diagnosed very accurately just by looking at fMRI brain scans. The idea is that, by identifying neural correlates for emotions, scientists may be able to use methods like brain scans to tell us more about all the different ways of being "happy".

**Evolutionary approach**

The evolutionary perspective offers an alternative approach to understand what happiness or quality of life is about. Briefly, it focuses on the questions: What features are included in the brain that allow humans to distinguish between positive and negative states of mind, and how do these features improve humans' ability to survive and reproduce? It claims that answering these questions points towards an understanding of what happiness is about and how to best exploit the capacities of the brain with which humans are endowed.
perspective is presented formally and in detail by the evolutionary biologist Bjørn Grinde in his book Darwinian Happiness.

**General findings by topic**

Money, once one reaches middle class, may be best spent ensuring one's job and social ties are enjoyable.

Happiness has become a very popular discussion topic in popular culture, especially in the Western world. There are many studies being done to demystify the factors that play into happiness. Although "happiness" can be used to refer to many things, the following describes research that is generally related.

One website, Subjective Well-Being Across Cultures, takes an inside look on the differences of happiness on an international level. Eunkook M. Suh, a professor at University of California, and Shigehiro Oishi, a professor at University of Minnesota, discuss key components to different cultures' views on what creates well-being and happiness. An example of how much well-being can vary is illustrated by a study of over 6,000 students in 43 nations to identify their "mean of Subjective Well-Being" on a scale of 1-7. The rate varied wildly, with China coming in at 3.3, and Brazilians at 6.2. Other studies suggested that two of the main factors for this variation are wealth of the country, and whether the country is individualistic or collectivist. They authors go into more detail about how these matter.

**Age**

The Midlife crisis may mark the first reliable drop in happiness during the average human's life. Evidence suggests that, with the exception of the years 40 - 50, most people generally get happier as they get older. Researchers specify that people in both their 20s and 70s tend to be happier than during midlife, although the measures of happiness change at different rates (e.g. feelings of stress and anger tend to decline after age 20, worrying drops after age 50, enjoyment had been very slowly declining but finally starts to rise after 50, etc.). These findings are based on decades of data, and controls for cohort groups; the data avoids the risk that the drops in happiness during midlife are due to populations’ unique midlife experiences, like a war. The studies have also controlled for income, job status and
parenting (as opposed to childfreedom) to try and isolate the effects of age. Researchers found support for the notion that there are changes inside of the individual with age that affect happiness.

This could be for any number of things. Psychological factors could include a greater awareness of one's self and preferences; an ability to control desires and hold more realistic expectations; getting closer to death may motivate people to pursue more goals; improved social skills, like forgiveness, may take years to develop; or happier people may live longer and are slightly overrepresented in the elderly population. Chemical changes that come with age may also be playing a role.

Other studies have found that older individuals report more health problems, but fewer problems overall. Young adults reported more anger, anxiety, depression, financial problems, troubled relationships and career stress. Researchers also suggest that depression in the elderly is often due largely to passivity and inaction - they recommend that people continue to do the things that bring happiness, even in old age.

Buying happiness

In his book Stumbling on Happiness, psychologist Dan Gilbert describes research suggesting that money makes a big difference to the poor (where basic needs are not yet met) but has greatly diminished effects once one reaches middle class (i.e. the Easterlin paradox). Professor of Economics Richard Easterlin notes that job satisfaction does not depend on salary. In other words, having extra money for luxuries does not increase happiness as much as enjoying one's job or social network. Gilbert is thus adamant that people should go to great lengths in order to (a) figure out which jobs they would enjoy and (b) find a way to do one of those jobs for a living (that is, provided one is also attentive to social ties).

Studies have routinely showed that nations are happier when people's needs are met. Some studies suggest, however, that people are happier after spending money on experiences, rather than physical things.

Education and intelligence

English poet Thomas Grey said "Where ignorance is bliss, Tis folly to be wise." Research suggests that neither a good education nor a high IQ reliably increase happiness. Anders Ericsson argues that an IQ above 120 has a decreasing influence on success. Presumably, IQs above 120 do not go much further to cause other happiness indicators like success (with the exception of careers like Theoretical physics, where high IQs are more predictive of success). Above that IQ level, other factors start to matter more like social skills or a good mentor. One of the main benefits of intelligence and education may simply be that it allows one to reach the middle-class level of need satisfaction (as mentioned above, being richer than this seems to do little for happiness).
Martin Seligman has said that "As a professor, I don't like this, but the cerebral virtues — curiosity, love of learning — are less strongly tied to happiness than interpersonal virtues like kindness, gratitude and capacity for love."

**Parenting**

While parenting is sometimes held as the necessary path of adulthood, studies are actually mixed as to whether parents are more likely to report being happier than non-parents. When quantitatively measured by self-report, researchers have found that parents prefer doing almost anything else to looking after their children. On the other hand, parents in self-reports are happier than non-parents. This may be due to already happy people tending to have more children than already non-happy people or that more long-term having children gives more meaning to life. One study found having up to three children increased happiness among married couples, but not among other groups with children. Proponents of Childfreedom maintain that this is because one can enjoy a happy, productive life without ever being a parent.

**Weather**

There is some evidence that suggests that sunnier climates do not predict happiness. In one study, both Californians and Midwesterners expected the former’s happiness ratings to be higher due to a sunnier environment. In fact, the Californian and Midwestern happiness ratings did not show a significant difference. Other researchers say the minimum daily-dose of sunlight is as little as 30min.

That is not to say that the weather is never a factor for happiness. Some psychologists suggest changing norms of sunlight in particular can cause Seasonal affective disorder.

**Religion**

Various studies have examined the relationship between Religion and happiness.

**Changes in happiness levels**

The human ability of emotional Hedonic Adaptation explains why beauty, fame and money do not generally have lasting effects on happiness (this effect has also been called the Hedonic treadmill). The tendency to adapt is clearly illustrated by studies showing that lottery winners are no happier years later. Other studies have shown that, after equally few years, paraplegics are almost as happy as control groups that are not paralyzed (p. 48). Daniel Kahneman explains that "they are not paraplegic full time...It has to do with allocation of attention". Contrary to our impact biases, lotteries and paraplegia do not change experiences in the ways we think, or even to as great a degree. After adaptation, the paraplegics almost returned to their baseline happiness. Adaptation is much slower for other distracting life changes, like the death of a spouse, or losing one's job. These events can show measurable changes in happiness levels for several years. Thus, adaptation does mitigate the emotional effects of many life events, but not entirely.
Factors Influencing Chronic Happiness Levels

Some research suggests that large portions of happiness are within a human's control.

Research indicates that genetics play a very significant role in determining one's baseline happiness levels, according to David Lykken. In her book The How of Happiness, Sonja Lyubomirsky similarly argues that people's happiness varies around a genetic set point. Importantly, an individual's base-line happiness is not entirely determined by their genetics, and not even by the early life influences on those genetics. Whether or not a person manages to elevate their base-line to the heights of their genetic possibilities depends partly on factors including actions and habits. Some happiness-boosting habits seem to include gratitude, appreciation, and even altruistic behaviour. Other habits and techniques of increasing happiness have emerged from research and are discussed on this page.

Besides training new habits and antidepressants, getting better exercise and a healthier diet have proven to have strong effects on mood. In fact, exercise is sometimes called the "miracle" or "wonder" drug - alluding to the wide variety of proven benefits that it provides.

**Gender**

Gender effects on wellbeing are paradoxical in that while men report feeling less happy than women, women are more susceptible to depression. Possible explanations include that women may experience more variance (more extremes) in emotion, although women are generally happier.

**Marriage**

Martin Seligman writes that "Unlike money, which has at most a small effect, marriage is robustly related to happiness... In my opinion, the jury is still out on what causes the proven fact that married people are happier than unmarried people." (pp. 55–56) There is
also little data on alternatives like Polyamory. On the other hand, at least one large study in Germany found no difference in happiness between married and unmarried people.

**Personality**

An emotionally stable (the opposite of Neurotic) personality correlates well with happiness. Not only does emotional stability make one less prone to negative emotions, it also predicts higher social intelligence - which helps to manage relationships with others (an important part of being happy, discussed below).

Cultivating an extroverted temperament may correlate with happiness for the same reason: it builds relationships and support groups. Some people may be lucky, then, that many personality theories leave room for the idea that individuals have some control over their long term behaviours and cognitions. Genetic studies indicate that it is genes for personality (specifically extraversion, neuroticism and conscientiousness) and a general factor linking all 6 traits that account for the heritability of subjective well-being.

**Social ties**

In the article “Finding Happiness after Harvard” George Vaillant concludes a study on what aspects of life are important for “successful living”. Back in the 1940s Arlie Bock, who was in charge of the Harvard Health Services, started a study by selecting 268 Harvard students from graduating classes of 1942, 43', and 44'. He wanted to find out what aspects of life create “successful living”. In 1967 psychiatrist, George Vaillant helped continue the study, he did follow up interviews with many of the students, now in their 50's, to see how their lives were going. Then in 2000 Vaillant again interviewed these students on the progress of their lives. The characteristics Vaillant was observing were health, close relationships, and how one dealt with their troubles. George Vaillant found that a key aspect to successful living is healthy and strong relationships.

A widely publicized study from 2008 in the British Medical Journal reported that happiness in social networks may spread from person to person. Researchers followed nearly 5000 individuals for 20 years in the long-standing Framingham Heart Study and found clusters of happiness and unhappiness that spread up to 3 degrees of separation on average. Happiness tended to spread through close relationships like friends, siblings, spouses, and next-door neighbors, and the researchers reported that happiness spread more consistently than unhappiness through the network. Moreover, the structure of the social network appeared to have an impact on happiness, as people who were very central (with many friends, and friends of friends) were significantly more likely to be happy than those on the periphery of the network. Overall, the results suggest that happiness can spread through a population like a virus.

**Culture**

One website, Subjective Well-Being Across Cultures, takes an inside look on the differences of happiness on an international level. Eunkook M. Suh, a professor at University of
California, and Shigehiro Oishi, a professor at University of Minnesota, discuss key components to different cultures’ views on what creates well-being and happiness. An example of how much well-being can vary is illustrated by a study of over 6,000 students in 43 nations to identify their "mean of Subjective Well-Being" on a scale of 1-7. The rate varied wildly, with China coming in at 3.3, and Brazilians at 6.2. Other studies suggested that two of the main factors for this variation are wealth of the country, and whether the country is individualistic or collectivist. They authors go into more detail about how these matter.

**Political views**

Conservatives are happier than liberals. One explanation being greater acceptance of income inequalities in society.

**Theory**

**Broad theories**

Some researchers in this field posit that positive psychology can be delineated into three overlapping areas of research:

- Research into the Pleasant Life, or the "life of enjoyment", examines how people optimally experience, forecast, and savor the positive feelings and emotions that are part of normal and healthy living (e.g. relationships, hobbies, interests, entertainment, etc.). Martin Seligman says that this most transient element of happiness may be the least important, despite the attention it is given.
- The study of the Good Life, or the "life of engagement", investigates the beneficial effects of immersion, absorption, and flow that individuals feel when optimally engaged with their primary activities. These states are experienced when there is a positive match between a person’s strength and the task they are doing, i.e. when they feel confident that they can accomplish the tasks they face. (See related concepts, Self-efficacy and play)
- Inquiry into the Meaningful Life, or "life of affiliation", questions how individuals derive a positive sense of well-being, belonging, meaning, and purpose from being part of and contributing back to something larger and more permanent than themselves (e.g. nature, social groups, organizations, movements, traditions, belief systems).

These categories appear to be neither widely disputed nor adopted by researchers across the 12 years that this academic area has been in existence. Martin Seligman originally proposed these 3 categories, but has since suggested that the last category, "meaningful life", be considered as 3 different categories. The resulting acronym is PERMA (Positive Emotions, Engagement, Relationships, Meaning and purpose, and Accomplishments).
Professor Philip Zimbardo suggests we might also analyze happiness from a "Time Perspective". Zimbardo suggests sorting people's focus in life by valence (positive or negative) but also by their time perspective (past, present, or future orientation). Doing so may reveal some conflicts between individuals to be conflicts, not over whether an activity is enjoyed, but whether one prefers to risk delaying gratification further. Zimbardo also believes that research reveals an optimal balance of perspectives for a happy life; he says our focus on reliving positive aspects of our past should be high, followed by time spent believing in a positive future, and finally spending a moderate (but not excessive) amount of time enjoying the present.

The broaden-and-build theory of positive emotions suggests that positive emotions (e.g. happiness, interest, anticipation) broaden one's awareness and encourage novel, varied, and exploratory thoughts and actions. Over time, this broadened behavioral repertoire builds skills and resources. For example, curiosity about a landscape becomes valuable navigational knowledge; pleasant interactions with a stranger become a supportive friendship; aimless physical play becomes exercise and physical excellence.

This is in contrast to negative emotions, which prompt narrow survival-oriented behaviors. For example, the negative emotion of anxiety leads to the specific fight-or-flight response for immediate survival.

Below, research is sorted according to which of Seligman's categories it may be most (but not strictly) related (i.e. the "pleasant", "good", or "meaningful" life). Since these are still fuzzy classifications, research mentioned in one section may be quite relevant in another.

**The pleasant life**
Simple exercise, such as running, is cited as key to feeling happy.

Abraham Maslow proposed a hierarchy of needs in which more primitive desires must be met (basic physiological, sense of safety) before social needs can be met (e.g. intimacy), and certainly before one can effectively pursue more conceptual needs (e.g. morality).

There is evidence suggesting that negative emotions can be damaging. In an article titled "The undoing effect of positive emotions", Barbara Fredrickson et al. hypothesize that positive emotions undo the cardiovascular effects of negative emotions. When people experience stress, they show increased heart rate, higher blood sugar, immune suppression, and other adaptations optimized for immediate action. If individuals do not regulate these changes once the stress is past, they can lead to illness, coronary heart disease, and heightened mortality. Both lab research and survey research indicate that positive emotions help people who were previously under stress relax back to their physiological baseline. Other research shows that improved mood is one of the various benefits of physical exercise.

The good life

Ideas of well-being as a good life trace their origins to Aristotelian ideas of eudaimonia. A range of concepts have grown out of this model including self-efficacy, personal effectiveness, flow, mindfulness etc.

Self-efficacy is one's belief in one's ability to accomplish a task by one's own efforts. Low self-efficacy is associated with depression; high self-efficacy can help one overcome abuse, overcome eating disorders, and maintain a healthy lifestyle. High self-efficacy also improves the immune system, aids in stress management, and decreases pain. A related but somewhat differing concept is Personal effectiveness which is primarily concerned with the methodologies of planning and implementation of accomplishment.
Flow

Flow, or a state of absorption in one's work, is characterized by intense concentration, loss of self-awareness, a feeling of being perfectly challenged (neither bored nor overwhelmed), and a sense that "time is flying." Flow is an intrinsically rewarding experience, and it can also help one achieve a goal (e.g. winning a game) or improve skills (e.g. becoming a better chess player). Anyone can experience flow in many different regards, such as play, creativity, and work. To experience flow, one needs to have the correct ratio of challenge for their particular skill set. Therefore, if one is very skilled in a certain regard, they need a lot of challenge or if they are unskilled they need a small amount of challenge. If one is too challenged it results in a state of anxiety and if one is not challenged enough the result is boredom. Being challenged means flow is, of course, temporarily exciting and stressful, but this Eustress is not harmful because it is not chronic stress.

Czikszentmihalyi identifies nine elements of flow: 1. There are clear goals every step of the way, 2. There is immediate feedback to one's action, 3. There is a balance between challenges and skills, 4. Action and awareness are merged, 5. Distractions are excluded from consciousness, 6. There is no worry of failure, 7. Self-consciousness disappears, 8. The sense of time becomes distorted, 9. The activity becomes "autotelic" (an end in itself, done for its own sake).

Mindfulness

Mindfulness, may be defined as the intentionally-focused awareness of one's immediate experience. The experience is one of a moment-by-moment attention to thoughts, emotions, physical sensations, and surroundings. To practice mindfulness is to become grounded in the present moment; one's role is simply as observer of the arising and passing away of experience. One does not judge the experiences and thoughts, nor do they try to 'figure things out' and draw conclusions, or change anything - the challenge during mindfulness is to simply observe. Benefits of mindfulness practice include reduction of stress, anxiety, depression, and chronic pain. See also Reverence (emotion).

Advocates of focusing on present experiences also mention research by Psychologist Daniel Gilbert, who suggests that daydreaming, instead of focusing on the present, may impede happiness. Other psychologists (see Zimbardo's "Time Perspectives" above) say that it is still important to spend time recalling past positive experiences, and building positive expectations for the future.

The meaningful life

After several years of researching disgust, University of Virginia professor Jonathan Haidt and others studied its opposite, and the term "elevation" was coined. Elevation is a moral emotion and is pleasant. It involves a desire to act morally and do "good"; as an emotion it has a basis in biology, and can sometimes be characterized by a feeling of expansion in the chest or a tingling feeling on the skin.
Optimism and helplessness

There is reason to focus on one's immediate locus of control, and to recognize that this behaviour - from everyone - would solve massive world issues.

Learned optimism is the idea that a talent for joy, like any other, can be cultivated. It is contrasted with learned helplessness, which is when one believes that they have no control over what occurs, rather it is something external that dictates their ability to accomplish a task, succeed, etc. Learning optimism is done by consciously challenging self talk if it describes a negative event as a personal failure that permanently affects all areas of the person's life. Reports of happiness have also been correlated with the general ability to "rationalize or explain" social and economic inequalities.

Hope is a learned style of goal-directed thinking in which the person utilizes both pathways thinking (the perceived capacity to find routes to desired goals) and agency thinking (the requisite motivations to use those routes).

Author and journalist J.B. MacKinnon provides a cognitive tool for avoiding helplessness (e.g. paralysis in the face of earth's many problems) in the form of what he calls "Vertical Agitation". The concept comes from research on denial by sociologist Stanley Cohen. Cohen explains that, in the face of massive problems, people tend towards learned helplessness rather than confronting the dissonant facts of the matter. Vertical Agitation, according to MacKinnon, means focusing on only one portion of the problem at a time, and holding oneself accountable for the solving of that problem - all the way to the highest level of government, business and society (e.g. advocating strongly for one thing: eco-friendly lightbulbs). This allows each individual in society to make the vital "trivial" (read:small) changes, without being intimidated by the work that needs to be done as a whole. MacKinnon adds that this will also keep individuals from getting too 'holier than thou' (harassing friends and family about every possible improvement).

Good work
As mentioned above, having more money does not reliably cause more happiness. Psychologist Howard Gardner has done more extensive research on what it takes to do good work at one’s job. He says young generations (particularly in the United States) have been taught to focus on selfish pursuit of money for its own sake. Gardner’s alternatives loosely follow the pleasant/good/meaningful life classifications; he believes young people should be trained to pursue excellence in their field, as well as engagement (see flow, above) in accordance with their moral belief systems.

**Strengths and virtues**

The Buddhist saying that "Life is Suffering", according to Jordan Peterson, refers to the fact that nature can be harsh and indifferent, which highlights the importance of cultivating virtues.

Research and clinical psychologist Jordan Peterson argues that it is natural (but of course not good) for human beings to suffer a great deal. He says this is because living beings are limited and restrained in many important ways. For this reason, Peterson adopts a Buddhist saying that "Life is suffering". He does not think this view is pessimistic, and asserts that accepting the universe’s harsh indifference towards living things can free one from the expectation that they should always be happy. This realization can ultimately help one overcome suffering, which is no longer unexpected. This also means that individuals should be all the more delighted when they flourish, when others flourish, or when they build a society where flourishing is anywhere near the norm at all. To Peterson, virtues are important because they give people the tools to escape suffering (e.g. the strength to admit dissonant truths to themselves). Peterson thus believes that suffering is sometimes caused by a false philosophy (one that denies how natural suffering is), and sometimes by a lack of strong virtues - on our part or on the part of people that affect us.

The development of the Character Strengths and Virtues (CSV) handbook represents the first attempt on the part of the research community to identify and classify the positive psychological traits of human beings. Much like the Diagnostic and Statistical Manual of Mental Disorders (DSM) of general psychology, the CSV provides a theoretical framework to assist in understanding strengths and virtues and for developing practical applications for positive psychology. This manual identifies six classes of virtue (i.e., "core virtues"), made up of twenty-four measurable character strengths.

The introduction of CSV suggests that these six virtues are considered good by the vast majority of cultures and throughout history and that these traits lead to increased happiness when practiced. Notwithstanding numerous cautions and caveats, this
suggestion of universality hints that in addition to trying to broaden the scope of psychological research to include mental wellness, the leaders of the positive psychology movement are challenging moral relativism and suggesting that we are "evolutionarily predisposed" toward certain virtues, that virtue has a biological basis.

The organization of these virtues and strengths is as follows:

- Wisdom and Knowledge: creativity, curiosity, open-mindedness, love of learning, perspective, innovation
- Courage: bravery, persistence, integrity, vitality
- Humanity: love, kindness, social intelligence
- Justice: citizenship, fairness, leadership
- Temperance: forgiveness and mercy, humility, prudence, self control
- Transcendence: appreciation of beauty and excellence, gratitude, hope, humor, spirituality

The organization of these virtues into 6 groups is contested. It has been suggested that the 24 strengths identified are more accurately grouped into just 3 or 4 categories: Intellectual Strengths, Interpersonal Strengths, and Temperance Strengths or alternatively Interpersonal Strengths, Fortitude, Vitality, and Cautiousness These general traits, and even their classifications, have emerged independently elsewhere in literature on values. Some examples have been described by Paul Thagard, including Jeff Shrager's workshops that attempt to discover the habits of highly creative people.

Application

Practical applications of positive psychology include helping individuals and organizations identify their strengths and use them to increase and sustain their respective levels of well-being. Therapists, counselors, coaches, and various psychological professionals, as well as HR departments, business strategists, and others are using these new methods and
techniques to broaden and build upon the strengths of individuals who are not necessarily suffering from mental illness or disorder.

Researcher Dianne Hales described a person as emotionally healthy as someone who exhibited flexibility and adaptability to different circumstances, had a sense of meaning and affirmation in life as well as an "understanding that the self is not the center of the universe", had compassion and the ability to be unselfish, along with increased depth and satisfaction in intimate relationships, and who had a sense of control over the mind and body.

Proponents of replacing Gross domestic product with Gross national happiness as the predominant measure of a nation's success often cite positive psychology research.

**Life coaching**

In his paper *The Primacy of Positivity*, Dr Timothy Sharp argues that happiness is an important first step in achieving success and productivity in one's life. Dr Sharp's 'The Happiness Institute' promotes the achievement of happiness and positive emotions. Sharp believes that positive psychology better equips coaches because it helps individual clients to achieve their goals and to progress effectively. For instance, Dr Sharp's uses positive psychology as a method of weight loss, known as The Happiness Diet. Positive Psychology is used to promote happiness which in turn can increase one's commitment to traditional methods such as diet and exercise.

In Norman Vincent Peale's 1952 book "The Power of Positive Thinking" the positive psychology concept of optimism is introduced. Optimistic thought has led to a significant market for self-help books as well as feel-good religious movements such as the prosperity gospels of Robert H. Schuller.

**In education**

Positive psychology is beneficial to schools and students as it encourages individuals to strive to do the best they can whereas scolding has the opposite effect. Clifton and Rath discuss the research conducted by Dr. Elizabeth Hurlock in 1925, who designed a study where fourth, fifth and sixth graders were either praised, criticized or ignored based on their work on math problems. The study found that students who had been praised improved by 71%, those who were criticized improved only by 19%, and those who had been given no feedback improved only by 5%. This early study illustrates that praise is the most effective method of fostering improvement.

According to Clifton and Rath, ninety-nine out of one hundred people would prefer to be around positive people. People believe that they work more productively when they are around positive people. Positive emotions are contagious so having a teacher or student who is positive can help the other students to be positive and work to the best of their abilities. If there is one negative person, it can ruin the entire positive vibe in an
environment. Clifton and Rath believe that ‘positive emotions are an essential daily requirement for survival’.

In 2008 a whole-of-school implementation of Positive Psychology was undertaken by Geelong Grammar School (Victoria, Australia) in conjunction with the Positive Psychology Center at the University of Pennsylvania. This involved initial training of teaching staff in the principles and skills of positive psychology. Ongoing support was provided by The Positive Psychology Center staff remaining in-residence for the entire year (Seligman et al. 2008).

Staats, Hupp and Hagley (2008) have used positive psychology to explore academic honesty, by identifying positive traits that were displayed by heroes and then determining if the presence of these traits in students could be used to predict their future intent to cheat. Their research has resulted in ‘an effective working model of heroism in the context of the academic environment’ (Staats, Hupp & Hagley, 2008).

**Applications to Clinical Psychology**

A strengths-based approach aims to change clinical psychology to have an equally weighted focus on both positive and negative functioning when attempting to understand and treat distress. The rationale is based on several empirical findings. Positive characteristics interact with negative life events to predict disorder (so studying only negative life events would produce misleading results). Interventions that focus on strengths and positive emotions can be as effective in treating disorder as other more commonly used approaches such as cognitive behavioral therapy. The terms positive clinical psychology and other terms are a bit odd as this field has always had scientists and clinicians that address quality of life outcomes. While positive psychology can inform clinical psychology, it is not helpful to stretch beyond this point. This is about changing priorities to address the breadth and depth of the human experience in clinical settings.

**In the workplace**

Positive psychology has also been implemented in business management practice, but Wong & Davey (2007) acknowledges that although managers can introduce this concept to a workplace, they don't always have the ability to apply it to employees in a positive way. Furthermore, if positive psychology must be applied to an organisation with transparency if it is to be welcomed and committed to by employees. Managers must also understand that the sheer implementation of positive psychology will not combat any commitment challenges they may face. However, it may help employees to be more optimistic to new concepts or management practices.

In their article The Benefits of Frequent Positive Affect: Does Happiness Lead to Success?, S. Lyubomirsky et al. say: "The cross-sectional evidence reveals that happy workers enjoy multiple advantages over their less happy peers. Individuals high in subjective well-being are more likely to secure job interviews, to be evaluated more positively by supervisors once they obtain a job, to show superior performance and productivity, and to handle
managerial jobs better. They are also less likely to show counter-productive workplace behavior and job burnout."

Positive psychology, when applied correctly can provide employees with a greater opportunity to use skills and vary work duties. However, it is important to remember that changing work conditions and roles can lead to stress amongst employees if they are not properly supported by management in their venture. This particularly holds true for employees who must meet the expectations of organisations with unrealistic goals and targets.

So how does an organization implement change? Lewis et al. (2007) have developed Appreciative inquiry (AI) which is an integrated, organizational-level methodology for approaching organizational development. Appreciative inquiry is based on an understanding of how organizational resourcefulness is generated through accessing many human psychological processes, such as positive emotional states, imagination, social cohesion and the social construction of reality.

**In offender rehabilitation**

Traditionally, working with offenders has focused on their deficits (with respect to socialization, schooling etc.) and other "criminogenic" risk-factors. Rehabilitation more often than not takes the form of forced treatment or training for the good of the community. The experience with this approach has not been very rewarding. Positive psychology has made some inroads recently with the advent of the "Good Lives Model", developed by Tony Ward, Shadd Maruna and others with respect to rehabilitation: "Individuals take part ... because they think that such activities might either improve the quality of their life (an intrinsic goal) or at least look good to judges, parole boards and family members (an extrinsic goal)."

**Other future research**

Positive psychology research and practice is also currently being conducted and developed in various countries throughout the world. In Canada, for example, Charles Hackney of Briercrest College applies positive psychology to the topic of personal growth through martial arts training, and Paul Wong, president of the International Network on Personal Meaning, is developing an existential approach to positive psychology.

An ‘intense affect’ can certainly be considered with cognitive and behavioral change, which is more slight and complex and is becoming a legitimate area of study, specifically with the links in cognition and motivational responses. For researchers to make further progress there is a need for past theories and methods to be overcome and to encourage the more contemporary research, says Isen (2009). Chang (2008) believes emotional intelligence is not definitive to positive affect and researchers have a number of paths that allow the enhancement of emotional intelligence; however more study is required to track the gradient of positive affect in psychology.
Academic Programs that Teach Positive Psychology

The University of Pennsylvania's Positive Psychology Center has developed a masters degree program in positive psychology (MAPP) which is described which they designate "the world's first degree program in positive psychology". This relatively new degree program is under the direction of The Center for Applied Psychology's "senior scholar," James Pawelski, Ph.D. In addition to his work as administrator of the program, Pawelski also teaches courses on "positive intervention" in the program. In a lecture presented at the John C. Dalton Institute of College Student values, Pawelski describes the degree as consisting of monthly intensives coupled with online course work which can be "pursued without interrupting your career." The degree "explores the history, theory, and basic research methods of positive psychology", "focuses on such issues as the empirical study of positive emotions, strengths-based character, and healthy institutions", and mentors the student in using "these aspects of positive psychology in" their "particular professional setting."

Another academic program that focuses on training students in Positive Psychology and features both an M.A. and Ph.D. tracts, is offered at the Claremont Graduate University's School of Behavioral and Organizational Sciences and is affiliated with the school's Quality of Life Research Center. This program "aim[s] to provide excellent graduate education and to facilitate the production of practical knowledge" and emphasizes "sampling methods as well as more traditional experimental and quasi-experimental designs, surveys, and interviews, our faculty and students focus their research on life-long processes and outcomes of behavior as they affect the quality of life".

Criticism

Sample (2003) notes that it is argued by Steven Wolin, a clinical psychiatrist at George Washington University in Washington DC, that the study of positive psychology is a reiteration of older ways of thinking in positive psychology.

The uptake of positive psychology by the popular press, primarily promoting among other claims the health benefits of positive psychology. Snyder and Lopez (cited in Held 2004, p. 17) warn of possible damage to the field of positive psychology through the scientific community becoming caught up in the media's claims of positive psychology. Warning researchers of the field, Snyder and Lopez suggest that they remain within the parameters of scientific professionalism and utilise any research or studies appropriately.

Some negative attributes of positive psychology as described by Held (2004) include the movement’s lack of consistency towards the aspect of negativity. She raised issues with the simplistic approach taken by some psychologists in the application of positive psychology. A ‘one size fits all’ approach is not seen by Held to be beneficial to the advancement of the field of positive psychology, and she suggested a need for individual differences to be incorporated into its application.
Held (2004) argued that while positive psychology makes contributions to the field of psychology, that it is not without its faults. Her 2004 article in the Journal of Humanistic Psychology, vol.44, no.1. offered insight into topics including the negative side effects of positive psychology, the negativity that can be found within the positive psychology movement and the current division inside the field of psychology caused by the differing opinions held by psychologists on positive psychology.