Cognitive therapy outcome: the effects of hopelessness in a naturalistic outcome study

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Abstract

This study prospectively examined the effect of hopelessness on outcome in cognitive therapy. Hopelessness has a central role in cognitive theories of depression, and consistently predicts suicide attempts and suicide completion. Furthermore, there is indirect evidence that hopelessness predicts cognitive therapy outcome, in terms of early termination of therapy, perhaps in part because theories of therapy change suggest that “remoralization” is a critical first phase of change. It was hypothesized that hopelessness non-responsiveness early in therapy would be predictive of eventual outcome, over and above hopelessness severity at intake. In a naturalistic study of 122 patients diagnosed with unipolar depression, it was found that non-responsive hopelessness predicted outcome in cognitive therapy, and this effect is over and above any effect of initial severity of hopelessness or depression. These findings suggest that patients whose level of hope is responsive to early interventions make more rapid and pronounced improvements during “real world” cognitive therapy.

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Keywords: Depression; Cognitive therapy; Outcome; Adult; Hope; Hopelessness

1. Introduction

Cognitive therapy has been found to be an efficacious and effective intervention for depression across a variety of clinical settings (for review, see Clark, Beck, & Alford, 1999; DeRubeis & Crits-Christoph, 1998). However, rates of dropout, poor outcomes and relapse are considerable (e.g., Elkin et al., 1989; Evans et al., 1992) and suggest the importance of research...
that leads to better understanding of the factors associated with treatment outcomes (Scott, 1996). Hopelessness is one possible factor (Dahlsgaard, Beck, & Brown, 1998; Whisman, Miller, Norman, & Keitner, 1995).

The premise for the study came from the clinical observation that patients whose level of hopelessness was high and did not change early in therapy, regardless of cognitive therapy interventions, positive events in their lives or changes in depression severity, had poorer cognitive therapy outcomes. The conceptual grounding for the study involved the interleaving of several theoretical and research strands. First, theories of therapy change suggest that a critical first phase of therapy change involves “remoralization” or rebuilding hope: “Demoralization tends to respond quickly to psychotherapy; remoralization is usually accomplished in a few sessions” (Howard, Moras, Brill, Martinovich, & Lutz, 1996: p. 1061). Second, hopelessness is a central cluster of beliefs that is implicated in the course of depression and cognitive therapy outcomes (Beck, Brown, & Steer, 1989; Beck, Brown, Berchick, Stewart, & Steer, 1990; Whisman et al., 1995; Young et al., 1996). Third, there is evidence that hopelessness may have two distinguishable elements: trait-like hopelessness and responsive state-like hopelessness (Young et al., 1996). Fourth, developments in cognitive theories of emotional disorders suggest that rigid, trait-like beliefs that are non-responsive to disconfirming information are particularly maladaptive (Beck & Freeman, 1990). These theoretical and research strands suggest the intriguing possibility that hopelessness that fails to respond to early cognitive therapy interventions might be associated with poorer cognitive therapy outcomes.

Psychotherapy process-outcome research has started to address the question of what factors are associated with differential treatment outcomes in cognitive therapy for depression. Tang and DeRubeis (1999) looked at the data from two depression cognitive therapy trials (Hollon et al., 1992; Murphy, Simons, Wetzel, & Lustman, 1984) and found that treatment responders and non-responders were comparable in the degree to which their depression improved in the first 4 weeks of therapy. However, their analysis shows that the treatment non-responders made no further incremental gains in the remainder of therapy while treatment responders go on to make further gains. It seems that the first 4 weeks of cognitive therapy typically lead to clinically significant gains, but “treatment non-responders” tend not to build on these gains, leaving the intriguing question of what “non-specific, or at least non-specified, processes” are operating in this 4-week window (Ildardi and Craighead, 1999: p. 298; Wilson, 1999).

Howard et al. (1996) have suggested a model of psychotherapy change through three phases: remoralization, remediation (i.e. symptom change) and rehabilitation (establishing more adaptive ways of living). The first remoralization phase involves the instillation of hope through strengthening the beliefs that change is possible and therapy might be helpful in effecting change. Cognitive therapy for depression (Beck, Rush, Shaw, & Emery, 1979) emphasises the importance of remoralising clients through early interventions targeting clients’ hopelessness. Therefore, it can be hypothesized that change in clients’ levels of hopelessness early in therapy is a possible mechanism for subsequent remediation and rehabilitation (Howard et al., 1996). When clients begin cognitive therapy with greater levels of hopelessness, they tend to end therapy prematurely and report more residual depression scores at termination (Dahlsgaard et al., 1998; Whisman et al., 1995). A recent study demonstrated a reciprocal relationship between a loosening in core maladaptive beliefs and increases in hope, suggests that hope plays a vital role in the change process in cognitive therapy (Hoffart & Sexton, 2002).
However, given that levels of hopelessness change for most patients soon after beginning therapy, initial hopelessness alone is not a particularly meaningful predictor of cognitive therapy outcomes. A more sophisticated conceptualization of hopelessness is required to advance our understanding of differential treatment outcomes in cognitive therapy for depression.

There are theoretical and empirical grounds for identifying two dimensions of hopelessness: trait-like non-responsive hopelessness, which is relatively independent from changes in depression severity and state-like responsive hopelessness, which co-varies closely with depressive states (Young et al., 1996). There is evidence that trait-like hopelessness (defined as stability in baseline hopelessness and hopelessness responsiveness), but not state-like hopelessness, is associated with longer course of depression and suicide attempts (Young et al., 1996). This work suggests the intriguing possibility that it may not be severity of hopelessness at intake that may prove important in understanding cognitive therapy outcome, but rather the type of hopelessness. Specifically, if the course of depression is affected by trait-like hopelessness, it is possible that some clients whose initial level of hopelessness is “less responsive” to early cognitive therapy interventions will have poorer cognitive therapy outcomes.

There is an emerging theoretical and research basis for this prediction. Hopelessness is a central component of Beck’s cognitive theory of depression (Beck, 1967; Clark et al., 1999). Beck proposes that hopelessness describes depression, and mediates its course through inhibiting the person’s ability to generate adaptive beliefs and solutions to problems. Until recently cognitive theory of depression had explanatory frameworks for state-like, but not trait-like hopelessness (Abramson, Metalsky, & Alloy, 1989; Clark et al., 1999). As Young et al. (1996) remark, “no attempt has been made to create a coherent theory of the behavior of hopelessness over time” (p. 156). Moreover, there has been no attempt to create a compelling account of the behavior of hopelessness over the course of cognitive therapy. Clearly, elaboration of existing theory is necessary. Cognitive theory of personality disorders has presented one plausible account of hopelessness responsiveness. Beck and Freeman (1990) have suggested that in people diagnosed with personality disorders, beliefs, attitudes or images become particularly problematic when they are closed to disconfirming, corrective evidence; a processing style that engenders cognitive rigidity (as evidenced in trait-like hopelessness). Increasingly, there is evidence that rigid trait-like maladaptive beliefs are associated with onset and course of depression, especially more severe and refractory depression (Andrews & Brown, 1995; Ilardi & Craighead, 1999; Young et al., 1996). It is possible that trait-like hopelessness represents core beliefs about the future, that is to say a rigid set of negative expectancies of the future that do not change when corrective information becomes available. Within cognitive theory of personality disorders, this closed information processing system would be self-perpetuating because trait-like hopelessness leads to compounding experiences such as ineffective therapy and recurrent episodes of depression. In the course of cognitive therapy, this closes the opportunity for the vital first phase of the change process through remoralization (Howard et al., 1996). In summary, non-responsive hopelessness may be a marker of individuals at risk for recurrent depression and this feature may be particularly difficult to treat in standard cognitive therapy.

Theories of therapy change processes, cognitive theory, clinical observation and research examining the role of hopelessness in the course of depression and in cognitive therapy outcome
interleave to suggest the following research question: “Does hopelessness responsivity early in therapy predict cognitive therapy outcome over and above initial (pre-therapy) level of hopelessness?” This study sought to optimize ecological validity by using a large representative sample of people with a primary diagnosis of unipolar depression. The research was set in the context of a routine outpatient cognitive therapy clinic. It examined outcome in terms of both statistically and clinically significant change.

2. Method

2.1. Sample

The study participants were 122 outpatients with a primary diagnosis of unipolar depression who received cognitive therapy at the Center for Cognitive Therapy (CCT), University of Pennsylvania. The sample comprised 75 (61.5%) females and 47 (38.5%) males. The mean age of the sample was 33.76 (SD = 11.96), ranging from 18 to 73. Thirty-seven (30.3%) were married, 4 (3.3%) widowed, 12 (9.8%) divorced or separated, and 69 (56.6%) single (never married). Eleven (9.0%) had a high school diploma or its equivalent, 33 (27.0%) had completed some college, 37 (30.3%) had a college degree, and 38 (31.1%) had attended graduate or professional school.

Three participants did not indicate their educational background. The ethic composition of the sample included: 98 (80.3%) European American, 16 (13.1%) African–American, 6 (4.9%) American–Asian and 2 (1.6%) Hispanic individuals. The psychiatric status of the sample at intake and termination are shown in Table 1.

2.2. Procedure

After signing consent forms, doctoral-level diagnosticians conducted intake evaluations comprising the Structured Clinical Interview for DSM-IV Axis I and Axis II disorders (SCID-I, First, Spitzer, Gibbon, & Williams, 1995a, b; SCID-II, First, Spitzer, Gibbon, Williams, & Benjamin, 1994) and a battery of psychological assessments. Reliability of SCID diagnoses was ensured as follows. All diagnosticians were postdoctoral clinicians who received training on the SCID prior to conducting diagnostic evaluations. In the latter part of the intake evaluation, there was a three way meeting between a senior clinical psychologist, the diagnostician and the patient to enable the senior clinician to confirm diagnosticians’ impressions. Inclusion criteria were a primary diagnosis of major depressive disorder, dysthymia, or depressive disorder NOS. Exclusion criteria were bipolar disorder, psychosis, or dementia.

Following the intake evaluation, patients began cognitive therapy with one of 22 doctoral-level therapists. Depression severity and hopelessness data were collected in the few minutes before each therapy session. All participating therapists had received the CCT 2-week didactic training in cognitive therapy before beginning their clinical work, and subsequently received supervision from experienced cognitive therapists. Therapists had an average 2.52 (SD = 2.66; range 1–10) years of postdoctoral experience.

To rule out the possibility that variation in therapist competence confounded the design, a measure of therapist competence was developed, and is reported elsewhere (Kuyken, in preparation). The CCT Clinical Director and a senior staff psychologist rated therapists’ cognitive
therapy competence on a seven-point scale (anchors “out!” and “great”). Both raters had reviewed therapy tapes and directly supervised the therapists. Inter-rater reliability was substantial ($\kappa = 0.80$), and showed good convergent validity with mean general competence ratings that had been routinely given by therapists’ supervisors during their tenure at the CCT ($r_s = 0.59$, $p < 0.001$, $N = 74$). Mean competence scores were 4.53 (SD = 1.08; range 2–6) suggesting the majority of therapists were regarded as very competent.

Table 1
Background variables for the responsive/non-responsive hopelessness groups

<table>
<thead>
<tr>
<th>Background variables</th>
<th>Responsive hopelessness ($N = 76$)</th>
<th>Non-responsive hopelessness ($N = 46$)</th>
<th>Total ($N = 122$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Psychiatric Axis I</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Major depression</td>
<td>63 (82.9%)</td>
<td>37 (80.4%)</td>
<td>100 (82%)</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>5 (6.6%)</td>
<td>5 (10.8%)</td>
<td>10 (8.2%)</td>
</tr>
<tr>
<td>Depressive disorder NOS</td>
<td>13 (10.5%)</td>
<td>11 (8.8%)</td>
<td>12 (9.8%)</td>
</tr>
<tr>
<td><strong>Axis II</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No personality disorder</td>
<td>15 (22.1%)</td>
<td>6 (14.0%)</td>
<td>21 (17.2%)</td>
</tr>
<tr>
<td>Personality disorder</td>
<td>42 (61.8%)</td>
<td>33 (76.7%)</td>
<td>75 (67.6%)</td>
</tr>
<tr>
<td><strong>Co-morbidity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No co-morbid diagnoses</td>
<td>6 (10.5%)</td>
<td>1 (2.6%)</td>
<td>7 (5.7%)</td>
</tr>
<tr>
<td>One co-morbid diagnosis</td>
<td>20 (35.1%)</td>
<td>8 (20.5%)</td>
<td>28 (24.6%)</td>
</tr>
<tr>
<td>Two co-morbid diagnoses</td>
<td>25 (43.9%)</td>
<td>23 (59.0%)</td>
<td>48 (40.1%)</td>
</tr>
<tr>
<td>Three or more co-morbid diagnoses</td>
<td>6 (10.5%)</td>
<td>7 (17.9%)</td>
<td>13 (13.5%)</td>
</tr>
<tr>
<td><strong>Intake functioning</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II-intake</td>
<td>21.33 (SD = 10.54)</td>
<td>26.99 (SD = 10.10)</td>
<td>23.46 (SD = 10.69)</td>
</tr>
<tr>
<td>GAF-intake</td>
<td>59.25 (SD = 9.0)</td>
<td>54.30 (SD = 7.26)</td>
<td>57.38 (SD = 8.69)</td>
</tr>
<tr>
<td><strong>Psychiatric history</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of psychotherapy</td>
<td>62 (83.8%)</td>
<td>36 (81.8%)</td>
<td>98 (81.1%)</td>
</tr>
<tr>
<td>History of pharmacotherapy</td>
<td>42 (56.8%)</td>
<td>27 (61.4%)</td>
<td>69 (58.5%)</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjunctive medication</td>
<td>11 (14.9%)</td>
<td>7 (15.2%)</td>
<td>18 (15.0%)</td>
</tr>
<tr>
<td>No. of CT treatment sessions</td>
<td>14.74 (SD = 14.52)</td>
<td>13.07 (SD = 11.83)</td>
<td>14.11 (SD = 13.54)</td>
</tr>
<tr>
<td><strong>Outcome</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II-termination</td>
<td>6.51 (SD = 9.18)</td>
<td>15.26 (SD = 14.39)</td>
<td>9.81 (SD = 12.14)</td>
</tr>
</tbody>
</table>

Note. For diagnostic data, psychiatric history data and adjunctive medication data values are numbers with percentages of the sample in parentheses. For the remaining measures, values are means with standard deviations in parentheses. GAF, Global Assessment of Functioning; CT, cognitive therapy. For Axis II disorders, the “deferred” category was used where the clinical interview did not permit a conclusive personality disorder diagnosis, but there was sufficient evidence to suggest that patients be assessed further by their therapist in the context of cognitive therapy. It was not always possible to establish from the chart review whether therapists had made definitive subsequent Axis II diagnoses. Exploratory analyses of the psychiatric characteristics (i.e., severity of psychopathology and psychiatric history) of patients with a deferred personality disorder classification suggest that they fall between those with and without personality disorder. For analyses addressing hypotheses about the presence/absence of personality disorder, a conservative strategy was adopted whereby patients with a deferred classification were omitted. Eleven patients were not diagnosed on Axis II for a variety of reasons. It was not possible to establish their diagnoses from chart review.
All participants received cognitive therapy. This was delivered according to therapist clinical judgment, individualized case formulations and treatment plans that were collaboratively agreed between therapist and client (Beck, 1995). Generally treatment comprised standard cognitive therapy for depression (Beck et al., 1979), unless patients were diagnosed with co-morbid disorders. For these patients, the treatment included adaptations of cognitive therapy to target additional problem areas such as anxiety disorders, personality disorders and substance abuse/dependence (Beck, Emery, & Greenberg, 1985; Beck & Freeman, 1990; Beck, Wright, Newman, & Liese, 1993). Generally, therapists and patients made decisions about the end of therapy on clinical and pragmatic grounds. Treatment data are reported in Table 1.

At the completion of therapy, patient charts were reviewed to gather data concerning adjunctive treatment, length of treatment and treatment outcome.

2.3. Measurement

2.3.1. The Beck Hopelessness Scale

The Beck Hopelessness Scale (BHS; Beck & Steer, 1988) is a 20-item true–false self-report assessment of a person’s beliefs about the future. It operationalizes hopelessness as a negative expectancy in which more adaptive options cannot be generated. This can be a general attitude or belief that is verbally represented or an imagery-bound scenario (e.g., “I can’t imagine what my life would be like in ten years”). Nine items are keyed false and 11 true. The items are summed to yield a total hopelessness severity score (range 0–20).

The BHS has been shown to have acceptable validity, reliability, and responsiveness to change among people with depression (Beck & Steer, 1988).

2.3.2. Non-responsive/responsive hopelessness

Non-responsive/responsive hopelessness was operationalized as follows. Non-responsive hopelessness was operationalized as BHS scores that were at 8 or above and did not respond to a clinically significant degree by moving into a non-problematic range (less than 8) between intake and session 4 of cognitive therapy (scaled 1). Responsive hopelessness was operationalized as BHS scores that responded to a clinically significant degree to cognitive therapy interventions by fluctuating above and below the cut-off of 8 between intake and session 4 (scaled 0). A score of 8 and above has been shown to be a reliable predictor of suicide attempts, completed suicides and treatment outcomes (see Brown, Beck, Steer, & Grisham, 2000; Dahlsgaard et al., 1998). Furthermore, the first 4 weeks of cognitive therapy typically result in marked improvements in depressive symptoms (Elkin et al., 1989; Fennell & Teasdale, 1987; Tang & DeRubeis, 1999). Therefore, a normal responsive pattern of change in hopelessness would be a move from the problematic to the non-problematic range early in therapy.

Forty-six participants could be categorized as demonstrating non-responsive hopelessness and 76 as demonstrating responsive hopelessness. Background variables across the non-responsive/responsive groups are shown in Table 1. Distributions of depression scores in the two groups over the course of cognitive therapy are shown in Fig. 1.

2.3.3. Structured Clinical Interview for DSM-IV

The Structured Clinical Interview for DSM-IV (SCID-I First et al., 1995a; SCID-II First et al., 1994) is a diagnostic instrument based on DSM-IV diagnostic criteria for psychiatric dis-
orders (American Psychiatric Association, 1994). The SCID has been demonstrated to have acceptable reliability and validity (Segal, Hersen, & Van Hasselt, 1994; Williams et al., 1992). In previous SCID reliability studies of affective disorders among a general outpatient psychiatric sample at the Center for Cognitive Therapy, an overall $\kappa$ of 0.72 was reported (Riskind, Beck, Berchick, Brown, & Steer, 1987). Percent agreement on diagnosing a major depressive disorder was 87%. Beyond the senior clinicians’ corroboration of all diagnoses, no formal diagnostic reliability in this particular sample was available.

2.3.4. Beck Depression Inventory-II

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item self-report instrument developed to measure severity of depression in adults and adolescents. Higher scores represent greater depression severity (range 0–63), and minimal, mild, moderate and severe symptom severity ranges have been specified.

2.3.5. Endstate functioning index

An easily interpretable index of clinically significant change was used which operationalizes endstate functioning (Ogles, Lunnen, & Bonesteel, 2001). Consistent with previous research (e.g., Elkin et al., 1989), a score on the BDI-II in the minimal range (0–13) was taken to suggest acceptable endstate functioning: acceptable endstate functioning scored “1”; unacceptable endstate functioning scored “0”. Overall, 80 patients (65.6%) achieved acceptable endstate functioning.
3. Results

It is fundamental to the research question to demonstrate that (1) individuals with responsive versus non-responsive hopelessness early in therapy are qualitatively different from each other and (2) this phenomenon is independent of depression severity. Depression severity at intake, sessions 1–4 and termination was significantly different for the two hopelessness groups at each measurement: intake $t(120) = 2.92, p < 0.01$; session 1 $t(120) = 2.21, p < 0.05$; session 2 $t(97) = 2.52, p < 0.05$; session 3 $t(98) = 3.39, p < 0.01$; session 4 $t(96) = 2.7, p < 0.01$; termination $t(120) = 4.10, p < 0.001$. Fig. 1 suggests that at intake, from sessions 1 to 4, and again at termination, patients with responsive hopelessness are less depressed than patients with non-responsive hopelessness. To replicate the finding that non-responsiveness in hopelessness over time can be observed, independently of depression severity (Young et al., 1996), simple correlations were computed in the current sample for BHS at intake, sessions 1–4 and termination. These were all positive and ranged from $r = 0.83$ (BHS at sessions 2 and 3), to $r = 0.61$ (BHS at sessions 1 and 4). Correlations were then computed separately for the responsive/non-responsive hopelessness groups. Consistently higher correlations were observed in the non-responsive than responsive group. When these analyses were repeated, partialling out BDI, this moderate to high level of association was replicated. Change in hopelessness (BHS session 1 minus BHS session 4) and change in depression severity (BDI session 1 minus BDI session 4) were positively but only moderately correlated $r = 0.49$, sharing 24% of the variance.

As would be expected, baseline severity of hopelessness was higher in the non-responsive than responsive hopelessness group (responsive hopelessness $M = 8.07, SD = 4.57$; non-responsive hopelessness $X = 13.54, SD = 3.12$; $t (df 120) = 7.17, p < 0.001$). Furthermore, because hopelessness and depression are related constructs (Table 2), mean baseline hopelessness scores, adjusted for depression severity, across the non-responsive/responsive hopelessness groups, were also computed (responsive hopelessness $M = 8.58, SE = 0.38$; non-responsive hopelessness $M = 12.71, SE = 0.49$). Median BHS scores at termination were: non-responsive hopelessness group 9.00 (SD = 6.20) and responsive hopelessness group 2.50 (SD = 3.77). The fact that the BHS scores stayed above the critical cut-off of 8 at termination, despite reductions in depression severity to the mild range provides evidence for the non-responsive quality of hopelessness in

<table>
<thead>
<tr>
<th>Table 2</th>
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<tbody>
<tr>
<td>Correlations among demographic, hopelessness, treatment and outcome variables</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intake</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1. Age</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. BDI-II at intake</td>
<td>0.07</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. BHS</td>
<td>−0.01</td>
<td>0.63**</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Termination</td>
<td></td>
<td></td>
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<tr>
<td>4. No. CT sessions</td>
<td>0.03</td>
<td>0.17</td>
<td>0.06</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>5. BDI-II-at termination</td>
<td>0.12</td>
<td>0.42**</td>
<td>0.38*</td>
<td>0.05</td>
<td>1.00</td>
</tr>
</tbody>
</table>

* $p < 0.05$ (two-tailed).

** $p < 0.01$. 

Note. BDI-II, Beck Depression Inventory-II; BHS, Beck Hopelessness Scale; CT, cognitive therapy.
this group. This suggests that the responsive and non-responsive groups could be reliably distinguished in terms of the stability of their levels of hopelessness early in therapy.

Taken together, Fig. 1 and these analyses suggest that hopelessness early in cognitive therapy is to some degree either responsive or non-responsive, and the distinction drawn between hopelessness responsiveness and non-responsiveness is relatively independent of depression severity (and not merely an artifact of depression severity). Finally, these analyses suggest that responsiveness in hopelessness early in therapy is to some degree independent of changes in depression severity.

Before addressing the study’s research question, any systematic relationships between (1) background variables (demographic, psychiatric history, psychiatric and treatment) and (2) the independent variables (initial hopelessness severity and hopelessness responsivity or the dependent variable (depression severity) were explored (see Tables 1 and 2). Only depression severity was significantly related to hopelessness responsiveness (as already reported).

3.1. Modeling cognitive therapy outcome

To model cognitive therapy outcome, multiple regressions to predict residual depression scores were computed. The equations were built on conceptual (the cognitive theoretical model was used to determine entry of variables) and statistical grounds (only predictive variables found to be significantly associated with the dependent variable in the Pearson correlations were entered)1 (Table 2). Hierarchical regression equations addressed the research questions concerned with severity of hopelessness at intake and responsiveness of hopelessness in the early stages of therapy. The regression was repeated for the key dependent variables (i.e., cognitive therapy outcome in terms of statistical and clinical significance). The regression strategy used to model cognitive therapy outcomes inherently takes into account the observed differences in depression severity across the independent variable at intake.

3.2. Hopelessness and cognitive therapy outcome in terms of residual depression

To establish whether hopelessness severity and hopelessness responsiveness predict cognitive therapy outcome, a regression equation was built with BDI-II at treatment termination as the dependent variable. Using the stated conceptual and statistical criteria, the following variables were entered into the equation in four steps: (1) intake depression severity, (2) adjunctive pharmacotherapy and number of therapy sessions, (3) hopelessness severity and (4) hopelessness responsiveness. After initial depression severity and treatment variables were entered, hopelessness severity at intake failed to predict any incremental variance in outcome in terms of residual depression scores. However, at step four, hopelessness responsiveness predicted significant incremental variance in residual depression scores (Table 3). The overall equation explained 27% of the variance in termination BDI-II, with initial BDI-II, adjunctive medication and non-responsive hopelessness being significant predictive variables2. The β value and partial

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1 This conservative strategy to regression was adopted to minimise the possibility of collinearity problems (Tabachnick & Fidell, 1996).

2 To address collinearity problems of including several inter-correlated scales in the same equation, collinearity statistics were computed for all regression equations. These were in the acceptable range (Tabachnick & Fidell, 1996).
### Table 3
Hierarchical multiple regression of post-treatment depression severity onto hopelessness during cognitive therapy

<table>
<thead>
<tr>
<th>Measures entered at each step</th>
<th>Partial correlations</th>
<th>$\beta$</th>
<th>$t$</th>
<th>$R^2$ change</th>
<th>$F$ of change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Intake depression severity (BDI-II)</td>
<td>0.32</td>
<td>0.38</td>
<td>3.23**</td>
<td>0.17</td>
<td>23.79***</td>
</tr>
<tr>
<td>2. Treatment variables Adjunctive medication</td>
<td>-0.21</td>
<td>-0.18</td>
<td>2.23*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of therapy sessions</td>
<td>-0.09</td>
<td>-0.08</td>
<td>0.91</td>
<td>0.05</td>
<td>3.38*</td>
</tr>
<tr>
<td>$F(3, 119) = 10.51, p &lt; 0.001$; cumulative $R^2 = 0.21$</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3. Hopelessness (BHS)</td>
<td>-0.01</td>
<td>-0.03</td>
<td>1.13</td>
<td>0.01</td>
<td>1.98</td>
</tr>
<tr>
<td>$F(4, 119) = 8.44, p &lt; 0.001$; cumulative $R^2 = 0.23$</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>4. Hopelessness responsiveness</td>
<td>0.24</td>
<td>0.26</td>
<td>2.63*</td>
<td>0.04</td>
<td>6.90*</td>
</tr>
<tr>
<td>$F(5, 114) = 8.48, p &lt; 0.001$; cumulative $R^2 = 0.27$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* BDI-II, Beck Depression Inventory-II; BHS, Beck Hopelessness scale. Partial correlations and reported $\beta$ values were values in the final equation.

*** $p < 0.001$.
** $p < 0.01$.
* $p < 0.05$.

### Table 4
Hierarchical multiple regression of endstate functioning onto hopelessness during cognitive therapy

<table>
<thead>
<tr>
<th>Measures entered at each step</th>
<th>Partial correlations</th>
<th>$\beta$</th>
<th>$t$</th>
<th>$R^2$ change</th>
<th>$F$ of change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Intake depression severity (BDI-II)</td>
<td>-0.23</td>
<td>-0.29</td>
<td>2.54*</td>
<td>0.09</td>
<td>11.75**</td>
</tr>
<tr>
<td>2. Treatment variables Adjunctive medication</td>
<td>0.08</td>
<td>0.08</td>
<td>0.88</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of therapy sessions</td>
<td>0.11</td>
<td>0.11</td>
<td>1.22</td>
<td>0.2</td>
<td>1.53</td>
</tr>
<tr>
<td>$F(3, 119) = 4.97, p &lt; 0.01$; cumulative $R^2 = 0.11$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Hopelessness (BHS)</td>
<td>0.02</td>
<td>0.03</td>
<td>0.22</td>
<td>0.01</td>
<td>1.04</td>
</tr>
<tr>
<td>$F(4, 119) = 3.99, p &lt; 0.01$; cumulative $R^2 = 0.12$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Hopelessness responsiveness</td>
<td>-0.20</td>
<td>-0.22</td>
<td>2.13*</td>
<td>0.03</td>
<td>4.54*</td>
</tr>
<tr>
<td>$F(5, 119) = 4.20, p &lt; 0.01$; cumulative $R^2 = 0.16$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* BDI-II, Beck Depression Inventory-II; BHS, Beck Hopelessness scale. Partial correlations and reported $\beta$ values were values in the final equation.

*** $p < .001$.
** $p < .01$.
* $p < .05$. 
correlation for non-responsive hopelessness in the final regression equation suggest that non-responsive hopelessness predicts treatment outcome in cognitive therapy. Fig. 1 shows the depression severity scores for the responsive and non-responsive hopelessness groups over the course of treatment and adds support for the conclusion that responsive hopelessness early in treatment is associated with better outcome in terms of fewer residual depression symptoms.

3.3. Hopelessness and cognitive therapy outcome in terms of endstate functioning

A parallel regression to examine clinically significant change was computed. Only the dependent variable was changed. The dependent variable was based on the endstate functioning index (introduced as a dummy variable). The pattern of regression results largely replicated those for residual depressive symptoms, although in this regression model adjunctive medication did not significantly contribute to the equation (Table 4). The proportion of individuals in the responsive/non-responsive hopelessness groups achieving acceptable endstate functioning was: 58/76 (76.3%) and 22/46 (47.8%), respectively. This suggests that responsiveness of hopelessness early in cognitive therapy, over and above severity of hopelessness or depression at intake, predicts cognitive therapy outcome in terms of clinically significant change.

Several follow-up analyses were carried out to rule out the possibility that the findings could be attributed to differences in therapist competence, the operationalization of responsive hopelessness, or responsive hopelessness being an artifact of early responsiveness in depression severity. These supplementary analyses suggest that the main findings are robust.

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3 To ensure that the findings could not simply be attributed to differences in therapist competence, the regressions were repeated adding therapist competence in the same step as adjunctive medication and number of therapy session. There were no substantive differences in the equations, suggesting that therapist competence did not affect the role of hopelessness in predicting treatment outcome. This is underscored by the fact that the mean therapist competence ratings across the independent variable were not significantly different (responsive hopelessness = 4.58, SD = 1.18; non-responsive hopelessness = 4.57, SD = 1.19, t(120) = 0.43).

4 To ensure that the operationalization of hopelessness responsiveness was robust, an alternative operationalization was used and the regressions repeated. Rather than dichotomizing the sample into hopelessness responsive/non-responsive groups, change in hopelessness scores from intake to session 4 (BHS intake—BHS session 4) was entered in the final step. There were no substantive differences in the observed pattern of relationships. That is, non-responsive hopelessness again predicted poorer outcomes over and above any effects due to initial hopelessness severity.

5 Even though there is evidence that changes in depression severity in the first 4 weeks of therapy tends to be comparable for treatment responders and non-responders (Tang & DeRubeis, 1999), to address the possibility that nonresponsiveness in hopelessness is an artifact of non-responsiveness in depression severity a further regression equation was computed. Using the same conceptual and statistical criteria, the following variables were entered in steps: (1) intake depression severity, (2) depression severity at session 4, (3) adjunctive pharmacotherapy and number of therapy sessions and (4) hopelessness responsiveness. The equation predicted 34% of the variance in termination BDI-II, with depression severity at session 4 ($\beta = 0.394$, $t = 3.63$, $p < 0.001$) and non-responsive hopelessness ($\beta = 0.17$, $t = 2.01$, $p < 0.05$) being significant independent variables. Importantly, non-responsive hopelessness added significant incremental variance to the model in the final step ($R^2$ change = 0.25; $F(1, 105) = 4.04$, $p < 0.05$).
4. Discussion

The current study demonstrates that hopelessness responsiveness early in therapy predicts cognitive therapy outcome: patients with responsive hopelessness respond better to cognitive therapy than patients with non-responsive hopelessness, over and above the well-established phenomenon that depression severity at intake predicts eventual outcome, and over and above severity of hopelessness at intake. Less than half of the patients with non-responsive hopelessness achieved acceptable endstate functioning, compared with three quarters of the patients who were remoralized early in therapy.

How can these findings be explained? First, consistent with Howard et al.’s (1996) proposed model of change throughout therapy, if the patient does not feel remoralized early in therapy, subsequent work to make symptom changes and more basic rehabilitation may be disabled. Cognitive therapy traditionally focuses on identifying and modifying negative thoughts and beliefs, assuming that successful interventions will lead to increases in hope. It is possible, however, that the relationship is more complex, with the capacity for increases in hope paving the way for engagement in therapy and the change process. This explanation is consistent with the recent finding that there is a feedback loop between increases in hope and a loosening in core maladaptive beliefs (Hoffart & Sexton, 2002). Second, people with non-responsiveness hopelessness may find it difficult to respond to initial interventions because they lack the cognitive resources that are required for change. For example, stability in hopelessness may disable the ability to visualize the pathways necessary to effect change (Snyder, Ilardi, Michael, & Cheavens, 2000), and thereby disenable the behaviors that are required for change, such as homework compliance (see Detweiler & Whisman, 1999). Third, it is possible that non-responsive hopelessness is an artifact of some alternative variable that also accounts for poor treatment outcome. For example, poor therapists delivering poor interventions could cause patients who are hopeless to remain hopeless and independently lead to poor treatment outcome. However, this explanation is not compelling, as the sample comprised multiple therapist–patient dyads, the therapists were generally rated as very competent and therapist competence did not mediate the impact of non-responsive hopelessness on treatment outcomes. It remains possible that “responsiveness” reflects some aspect of severity of problem/severity. Future research might helpfully establish if responsiveness of hopelessness reflects some more complex aspect of severity, such as chronicity of depression or age of first onset of depression.

Regardless of which explanation is correct, the findings raise important theoretical and clinical issues. The findings are consistent with related recent research suggesting that it is the type and organization of beliefs, not their content that is important in predicting cognitive therapy outcome in patients diagnosed with depression. Kuyken, Kurzer, DeRubeis, Beck and Brown (2001) showed that trait-like avoidant and paranoid beliefs, associated with their respective Axis II personality disorders, predict cognitive therapy outcome. Teasdale et al. (2001) showed that extremism in belief endorsement, not belief content, predicted poorer cognitive therapy outcome. In summary, several studies suggest that rigidity and extremism in circumscribed core beliefs are associated with worse course and poorer outcome in depression. This research, taken together, begins to suggest that beliefs may be defined as maladaptive through their inflexibility, extremism, and non-reactivity to disconfirming corrective information. What is not clear is whether
non-responsive hopelessness is an example of, consequence of, or indeed a cause of inflexible and extreme beliefs. This would provide an intriguing and important line of further enquiry.

In the context of research on early response to cognitive therapy and partial treatment results, this study raises a number of implications for the treatment of depression. In several cognitive therapy for depression trials early responsiveness in depression severity appears not to distinguish eventual treatment responders from treatment non-responders (Tang & DeRubeis, 1999). What else is important in this window? Residual depressive symptoms at the end of treatment are associated with marked rises in relapse rates (Judd et al., 1999) and when directly targeted through cognitive therapy techniques, relapse rates can be significantly reduced (Fava, Rafaelli, Grandi, Conti & Belluardo, 1998; Paykel et al., 1999). Given that the responsiveness of hopelessness early in treatment appears to be associated with more residual depressive symptoms, it is important therefore to assess the extent to which patients' hopelessness is responsive to early interventions. If not, identifying, labeling and directly working with non-responsive hopelessness as a first step in therapy may be indicated. Carefully targeted interventions must be developed to change the rigidity and closed nature of information processing. Cognitive therapy interventions aim to introduce compensatory skills (Barber & DeRubeis, 1989) that introduce dissonance into the rigid belief system and open opportunities for people to develop alternative more adaptive beliefs that enable switching between hopelessness and hopefulness (i.e., responsive hopelessness). These “pathways” or routes to the achievement of goals are important to the development and maintenance of hope (Snyder et al., 2000).

It is important to note some conceptual and methodological problems. First, the study used a limited range of outcome measures: the BDI-II and endstate functioning (based on the BDI-II). Additional robust measures of general functioning, clinician-rated depression, premature treatment termination and subsequent relapse rates would have provided more comprehensive and fine-grained measures of outcome. Second, the hopelessness responsiveness construct appears a highly promising and important one in understanding differential cognitive therapy outcomes. However, it was limited to five observations on one measure in the period between intake and session 4. It will be important to examine the behavior of hopelessness over the course of depression and of cognitive therapy using a range of measures. For example, can non-responsive hopelessness be observed in these same individuals before the onset of depression, prior to treatment, throughout the treatment period, and at residual levels after treatment? Specifically, how is hopelessness responsiveness related to the trait-like stable hopelessness construct described by Young et al. (1996)? Is the severity of hopelessness systematically related to responsiveness/non-responsiveness, or is low-intensity non-responsive hopelessness as problematic as high-intensity non-responsive hopelessness. Specifically, the question remains whether hopelessness might persist at low, but relatively unchanging levels in some individuals after the end of therapy. If such a pattern does exist, would the hope/hopelessness range in which individuals appear to be “stuck” be of importance in understanding depression. This study’s observation of hopelessness was confined to high-intensity non-responsive hopelessness in the early stages of cognitive therapy where theory and research suggest we can expect to see increases in hope and general remoralization from the dysfunctional to the functional range. Third, this study was a naturalistic study based on a question derived through theory and careful clinical observation. Clearly, these findings require replication in more controlled conditions and through systematic delineation of the relationship between responsiveness in depression and hopelessness.
In summary, this study suggests that responsiveness in hopelessness early in therapy reliably predicts cognitive therapy outcome in naturalistic outpatient settings. It is suggested that the organization and structuring of beliefs about the future (inflexibility and non-reactivity) may be a particularly important aspect of understanding why some people do not respond as well to cognitive therapy. Cognitive therapists might usefully focus on how people think about the future rather than what they think about the future.

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References


