People typically characterize environmental issues as physical health or technological problems. But the physical environment may also have adverse psychological impacts. Suboptimal environmental conditions, such as noise and crowding (Cohen, Evans, Stokols, & Krantz, 1986; Evans & Cohen, 1987), air pollution (Evans, 1994), or disasters (Baum & Fleming, 1993), may elicit psychological coping reactions, including cardiovascular and psychoneuroendocrine activation (Baum, Grunberg, & Singer, 1982; Frankenhaeuser, 1986; Lundberg, 1984). In the present article, we provide prospective, longitudinal evidence that chronic noise elevates psychophysical stress and depresses perceived quality of life.

Although noise can cause hearing deficits (Kryter, 1994), nonauditory, stress effects of noise have not been demonstrated definitively because of methodological and conceptual limitations in prior research. Laboratory studies indicate that acute noise elevates psychophysiological stress (e.g., blood pressure, epinephrine), but these responses habituate rapidly (Evans, in press; Glass & Singer, 1972; Hygge, 1997).

Psychophysiological stress responses to chronic noise have been investigated in industrial settings and in communities proximate to highways and airports. These cross-sectional studies, unfortunately, are subject to an array of plausible alternative explanations, particularly self-selection bias. Although suggestive trends linking occupational noise exposure and hypertension exist, the designs of these studies are so weak that definitive conclusions cannot be drawn (Evans, in press; Thompson, 1993). Poor or nonexistent control groups, nonrigorous assessments of blood pressure (e.g., one reading while on the job), and insufficient estimation of noise exposure plague industrial studies of noise and cardiovascular parameters.

Community studies of aircraft noise reveal elevated psychophysiological stress among children (Evans, Hygge, & Bullinger, 1995; Evans & Lepore, 1993). All of these studies are cross-sectional and, with one exception (Evans et al., 1995), limited to blood pressure. Knipschild (1977) has also shown a dose-response function between noise exposure in the community and hypertension among adults around the Amsterdam airport. The most clear-cut evidence that noise causes elevated psychophysiological stress comes from a primate laboratory study (Peterson, Augenstein, Tanis, & Augenstein, 1981). Simulated air- and road-traffic noise produced stable, elevated arterial blood pressure over a several-week period in the laboratory.

Many noise studies have obscured not only methodological issues, but also important conceptual issues. First, with few exceptions, investigators have not screened for hearing damage. One cannot presently say with any confidence whether stress correlates of community and occupational noise exposure are mediated by hearing loss. Second, nearly all the field studies have confounded acute and chronic noise exposure. Typically, testing has occurred in situ. If individuals are not tested under quiet, carefully controlled conditions, one cannot confidently attribute stress responses to chronic versus acute noise exposure.

The present study took advantage of a natural experiment created by the opening of a new international airport located in a rural area 35 km outside of Munich, Germany. Resting blood pressure, overnight levels of neuroendocrine hormones, and quality of life were measured over a 2-year period among elementary school children residing in the flight paths of this airport before and after its inauguration. Comparison groups of sociodemographically well-matched children from nearby rural communities were also assessed over the same time period.

METHOD

Subjects

Participants were 217 third- and fourth-grade children (mean age = 9.90 years at the study’s onset) living either proximate to the new Munich International Airport or in nearby communities outside the noise impact zone of the new facility. The total of 217 participants reflects attrition of 10 and 14 children, respectively, from the noisy and quiet areas. Attrition was unrelated to the outcome variables.

The measures of noise were 24-hr dBA Leq, an unweighted mean of energy level expressed in decibels, and dBA L01, the dBA level exceeded 1% of the time over the sampling period (24 hr in the present case). The dBA scale is logarithmic, and the human observer experiences an increase in 10 dBA as approximately a doubling of loudness. Following inauguration of the new airport, dBA Leq equaled 62, with a dBA L01 of 73, in the noise-impacted communities. Among the quiet communities at the same time period, dBA Leq was 55, with a dBA L01 of 64. Prior to the opening of the new airport, dBA Leq was 53, with a dBA L01 of 63, in the airport communities, and noise levels were comparably low in the comparison areas (dBA Leq = 53, dBA L01 = 64).

Children in the quiet comparison communities were matched to the children in the noise-impacted communities according to socioeconomic status. Households did not differ in the type of occupation, \( \chi^2(4, N = 197) = 8.91; \) parental education, \( r(214) < 1.0; \) or family size, \( r(214) < 1.0. \) All of the children in the study were screened for normal hearing with an audiometric examination.
Procedure

Testing occurred in a sound-attenuated, climate-controlled mobile laboratory parked outside the child’s elementary school. A microphone (6 m above ground) interfaced with a B&K Model 4426 Community Noise Level Analyzer monitored 24-hr outdoor noise levels at the trailer. Data were collected 6 months prior to the opening of the new airport (Wave 1), 6 months after the opening (Wave 2), and again 18 months after the opening (Wave 3), for a total of three assessment phases per participant.

Resting blood pressure was assessed with an automated monitor (A & D Digital, UA 751) while the child sat comfortably with his or her right arm supported at heart height on a table. Baseline readings were calculated by taking the average of six resting indices taken on 2 consecutive days. On each day, four readings were taken after an orientation to the automated monitor, and the first reading was discarded. Reliability estimates for the six readings exceeded an alpha of .85 for both diastolic and systolic blood pressure.

Twelve-hour overnight urine samples were collected between 20:00 on the evening of the initial testing day and 8:00 the following morning. The container was kept refrigerated and contained a preservative. Parents returned the urine specimen container the next day to the trailer. Total volume was measured, and small replicate samples of urine were extracted and deep frozen at −70 °C until assayed. Half of the replicates were also pH adjusted to reduce oxidation further for the catecholamine assays. Epinephrine and norepinephrine were assayed by high-performance liquid chromatography with electrochemical detection (Riggin & Kissinger, 1977). Cortisol was determined by a radioimmune assay (Baxter Travenol Diagnostics, Cambridge, Mass.). (For further information about procedures for the collection and assay of urinary neuroendocrine samples, see Baum & Grunberg, 1995, and Grunberg & Singer, 1990.)

Quality of life was assessed by the KINDL, a valid and reliable index of the principal domains of quality of life (physical, psychological, social, functional daily life; Bullinger, von Mackensen, & Kirchberger, 1994). These domains were combined for the present analysis (α = .92).

RESULTS

The data analytic strategy was to conduct a 2 × 3 repeated measures multivariate analysis of variance (MANOVA). The principal statistic of interest is the exact F test for the interaction of group (noise impacted vs. quiet) and time (Wave 1 vs. Wave 2 vs. Wave 3).

As can be seen in Table 1, blood pressure increased in the noise-impacted communities after Wave 1, with the opening of the new airport; much smaller changes occurred among the quiet, comparison communities. The interaction of group and time was significant for systolic blood pressure, MANOVA exact F(2, 214) = 4.50, p < .01, and marginal for diastolic blood pressure, F(2, 214) = 2.83, p < .06.

The overnight urine neuroendocrine results are shown in Table 1 as well. Consistent with the elevations in blood pressure, both epinephrine, F(2, 200) = 36.86, p < .001, and norepinephrine, F(2, 200) = 22.31, p < .001, increased sharply among children living in the flight paths of the new airport after it opened; smaller increases were seen over the same period among the children residing in quiet communities. Changes in urinary cortisol over time were not systematically related to noise conditions, F(2, 200) = 1.73, n.s. Degrees of freedom vary because of missing data.

As indicated in Table 2, quality of life declined significantly in the noise-impacted communities 18 months after the opening of the new airport, but remained relatively stable in the quiet, comparison communities, F(2, 202) = 3.07, p < .05.

Table 1. Measures of psychophysiological stress in the noise-impacted and quiet communities

<table>
<thead>
<tr>
<th>Measure and community</th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic blood pressure (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noise</td>
<td>97.2 (11.6)</td>
<td>101.6 (9.9)</td>
<td>102.4 (10)</td>
</tr>
<tr>
<td>Quiet</td>
<td>100.8 (8.9)</td>
<td>102.2 (8.9)</td>
<td>102.6 (12)</td>
</tr>
<tr>
<td><strong>Diastolic blood pressure (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noise</td>
<td>60.5 (7)</td>
<td>63.2 (6.1)</td>
<td>64.4 (6.2)</td>
</tr>
<tr>
<td>Quiet</td>
<td>62.6 (7.1)</td>
<td>63.6 (6.4)</td>
<td>64.8 (6.8)</td>
</tr>
<tr>
<td><strong>Epinephrine (ng/hr)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noise</td>
<td>229.2 (153.4)</td>
<td>328.1 (130.4)</td>
<td>341.9 (168.1)</td>
</tr>
<tr>
<td>Quiet</td>
<td>251.8 (57)</td>
<td>280.9 (64.6)</td>
<td>246.2 (83.7)</td>
</tr>
<tr>
<td><strong>Norepinephrine (ng/hr)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noise</td>
<td>610.7 (338.6)</td>
<td>1,228.5 (659.7)</td>
<td>1,556.3 (703.6)</td>
</tr>
<tr>
<td>Quiet</td>
<td>660.0 (506.9)</td>
<td>879.7 (457.7)</td>
<td>950.7 (525.5)</td>
</tr>
<tr>
<td><strong>Cortisol (µg/hr)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noise</td>
<td>355.8 (189.3)</td>
<td>435.9 (538.9)</td>
<td>514.4 (689.3)</td>
</tr>
<tr>
<td>Quiet</td>
<td>330.5 (189.3)</td>
<td>237.3 (614.7)</td>
<td>377.7 (288.9)</td>
</tr>
</tbody>
</table>

*Note.* The data shown are means, with standard deviations in parentheses.
DISCUSSION

Chronic exposure to ambient aircraft noise elevates psychological stress in human beings. Children living proximate to the new Munich International Airport experienced significant elevations in resting blood pressure after the airport opened. During this same time period, well-matched children in nearby, similar communities experienced stable levels of resting blood pressure. The catecholamine data, which are consistent with the blood pressure effects, underscore the value of conceptualizing noise and other suboptimal environmental conditions as stressors. Elevated urinary catecholamines have consistently been shown to reliably and sensitively mark chronic exposure to stressors (Baum et al., 1982; Frankenhaeuser, 1986; Lundberg, 1984). Urinary cortisol has proven less reliable as an index of chronic stress exposure (Frankenhaeuser, 1986; Lundberg, 1984).

The self-report data show the same pattern, but with a delayed time course. Childrens’ perceived quality of life dropped more markedly in the noise-impacted communities than in the quiet communities, but this drop did not occur until 18 months after the new airport opened.

As in any field study, some questions remain. We cannot disentangle the apparent effects of chronic noise from the uncontrollability of that exposure. Although the primary environmental change near the new airport was noise, other changes also were witnessed, including increased land development, more road traffic, and the like.

Our prospective data add evidence to previous cross-sectional results that have shown elevated stress among adults and children working and residing, respectively, in chronically noisy environments. In young children, chronic noise exposure appears to cause increased psychological stress, as measured by cardiovascular, neuroendocrine, and affective indicators. These effects occur among children who suffer no detectable hearing damage while living in the immediate vicinity of an airport.

<table>
<thead>
<tr>
<th>Community</th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noise</td>
<td>110.3 (13.8)</td>
<td>112.4 (13.9)</td>
<td>104.8 (16.0)</td>
</tr>
<tr>
<td>Quiet</td>
<td>112.5 (16.4)</td>
<td>112.1 (17.0)</td>
<td>109.6 (15.5)</td>
</tr>
</tbody>
</table>

Note: The scale for the quality-of-life measure ranges from 40 to 200, with greater values indicating higher perceived quality of life. The data shown are means, with standard deviations in parentheses.

REFERENCES


Acknowledgments—We are grateful to the families who cooperated with this research. We thank Sylvia von Mackensen, Markus Meis, Gerhard Helm, Gunnar Söderqvist, Christian Wigren, Nils Antoni, and Pat O’Keeffe for their dedication to this project. This research was partially supported by the Society for the Psychological Study of Social Issues, the National Institutes of Health (ROI HL 4732 01A), the Nordic Scientific Group for Noise Effects, the Swedish Environmental Protection Agency, and the German Research Foundation. The second author was also supported by the DFG Heisenberg Fellowship.