Summary

A systematic search of the peer-reviewed scientific literature that examines the relationship between environmental noise and non-aural health effects identified 35 relevant studies published since 2001. Twenty-five of these report unique findings on long term exposure to transportation noise from road, rail or air traffic; three others report on acute exposures, two in the sleep laboratory and another in an occupational setting. The remaining seven are literature reviews – two of these reviews (Babisch, 2008; Kaltenbach, et al., 2008) quantify the evidence linking chronic noise to adverse health impacts in a dose-effect relationship.

Overall, the evidence from these studies supports the hypothesis of certain adverse health effects from environmental noise. The strongest evidence links exposure to noise above 60 dB(A) in the daytime and above 45 dB(A) at night to an increased incidence of arterial hypertension. Results also link noise above 60 dB(A) to an increased risk of myocardial infarction; at 70 dB(A) the risk is over 20% higher than in the unexposed population. Daytime exposure above 55 dB(A) is linked to learning difficulties in school children. The chief mediating mechanisms for these effects are sleep disturbance and physiological stress responses.

A more detailed description of these findings appears below. Appendix 1 includes details on the design, measurement and methods of 16 key studies. The World Health Organization in 2009 released their recommendations on night-time exposure thresholds. We concur with their assessment of the strength of the research evidence. Their summary tables appear in Appendix 2.

Cardiovascular Effects

Noise-induced cardiovascular effects have been extensively studied in occupational settings as well as at community levels. It has been concluded that prolonged exposure to occupational and/or environmental noise (at sound levels of 60-85 dB(A)) can contribute to increased risk for cardiovascular disease (Babisch, Beule, Schust, Kersten, & Ising, 2005; Babisch, 2008; Kaltenbach, Maschke, & Klinke, 2008; Stansfeld & Matheson, 2003). Noise-induced cardiovascular effects include: elevated blood pressure level, prevalence of hypertension, myocardial infarction (MI), abnormalities in the electrocardiogram, more heartbeat irregularities, faster pulse rate, total cholesterol, total triglycerides, blood viscosity, slower recovery of vascular constriction, and increased consumption of cardiovascular medications (Babisch et al., 2005; Jarup et al., 2008; Kaltenbach et al., 2008; Stansfeld & Matheson, 2003).

Arterial Hypertension

In a major retrospective cohort study examining hypertension (HT), Sbihi followed 10,842 sawmill workers for eight years, identifying 828 cases from physician-billing and hospital discharge records (Sbihi, Davies, & Demers, 2008). Noise exposure was estimated from predictive models based on 1,900 personal dosimetry measurements. The study reported a statistically significant exposure response for noise and HT reaching a relative risk (RR), after adjustment for potential confounders, of 1.5 after 30 years of exposure over 85 dB(A). Lusk et al. also examined ambulatory blood pressure (BP) and heart rate (HR) in 46 automobile engine assembly plant workers. The study used mixed-effect modeling because of the repeated blood pressure (BP) measures (taken at 10 minute intervals). Logged noise dosimetry allowed the calculation of short-term exposure metrics over the same intervals. After controlling for a large number of personal cardiovascular disease (CVD) risk factors, they found noise associated with three physiological measures (systolic and
diastolic blood pressure and heart rate) and showed a possible difference in mechanisms between BP (that they showed was correlated to average acute noise) and HR (which was correlated to peaks) (Lusk, Gillespie, Hagerty, & Ziemba, 2004).

Several recent studies examined the effect of noise (from a range of sources) on hypertension in community settings. Leon Bluhm, et al. (Bluhm, Berglund, Nordling, & Rosenlund, 2007) studied self-reported HT for 667 adults in a municipality near Stockholm, Sweden. Road noise was modeled for major roads (55-65 dB) and the rest (n=513) estimated by expert judgment. Thirteen percent of subjects were diagnosed with HT. There was a linear exposure response relation between traffic noise and prevalence of HT with an adjusted odds ratio \( \text{OR}_{\text{adj}} \) of 1.38 per 5 dB(A). The authors also showed interactions for time in residence, bedroom orientation, glazing and older homes. Another Swedish study carried out around Stockholm’s major airport assessed the prevalence of (self-reported doctor-diagnosed) high blood pressure by postal questionnaire. An exposure response association between aircraft noise and high blood pressure was found with relative risks ranging between 1.1 and 2.1 for noise levels between approximately energy-averaged levels \( \text{FBN} = 53 \text{ to } 63 \text{ dB(A)} \) (Rosenlund, Berglund, Pershagen, Järup, & Bluhm, 2001). When noise categories were combined, the effect was significant for \( \text{FBN} > 55 \text{ dB(A)} \). The trend analysis resulted in a relative risk of 1.3 (95\% CI = 0.8-2.2) per 5 dB(A).

A prospective study carried out around Stockholm’s major airport investigated the association between aircraft noise and high blood pressure. Subjects exposed to \( \text{FBN} \) above 50 dB(A) had a significant relative risk of 1.2 for the development of hypertension over the 10-year follow-up period, compared with less exposed (Eriksson et al., 2007). The increase in risk per 10 dB(A) was 1.2 (95\% CI = 1.0-1.2). The effect was particularly found in older people, which may reflect longer years of residence.

In a new multi-centered study carried out around six European airports, a significant increase in the risk of hypertension of 1.14 (95\% CI = 1.01-1.29) for a 10 dB(A) difference of aircraft noise during the night (\( \text{L}_{\text{night}} \)) was found (Jarup et al., 2008). Hypertension was determined by a combination of three criteria: measured resting blood pressure (systolic/diastolic blood pressure >140/90 mmHg), self-reported doctor-diagnosed hypertension, and anti-hypertensive medication (ATC coding). No linear association was found with respect to the exposure during the day, possibly due to exposure misclassification (time spent away from home). Thus, a smaller relative risk was found for the 24-hour noise indicator \( \text{L}_{\text{den}} \) of 1.1 (95\% CI = 0.9-1.3) per 20 dB(A). The same study reported a significant (54\%) increase in the odds of being hypertensive for men who are exposed to the highest level (>65 dB(A)) of road traffic noise (Jarup et al., 2008).

In a Swedish municipality partly affected by noise from a highway (20,000 vehicles/24 hours) and a railway (200 trains/24 hours), men who lived there for more than 10 years and were exposed to the highest level of noise (56-70 dB(A)) had a relative risk of hypertension almost three times that of the unexposed population (OR=2.9, 95\%CI: 1.4-6.2) (Barregard, Bonde, & Ohrstrom, 2009).

Ischemic Heart Disease

Babisch, et al. (Babisch et al., 2005) examined incidents of myocardial infarction (MI) between 1998 and 2001, recruiting patients with confirmed MIs at 32 Berlin hospitals. A sophisticated noise assessment was conducted, utilizing noise maps for roads with volumes over 6,000 vehicles per day, with lower volume roads characterized as “quiet.” This assumption was
validated. Subjects’ addresses were further checked and their exposures reassigned if they lived near a main road that was noisier than their own road.

In adjusted multivariate analyses there was a slight increase in risks for males only. This was strengthened when analysis was restricted to those who had lived in residence for >10 years (RR_adj = 1.3 >65 dB(A); 1.8 >70 dB(A)). There was no effect in females. Noise annoyance was linked to MI in males (for traffic noise at night, RR=1.1) and females (for aircraft noise at night, RR=1.3) and noise sensitivity was an increased risk in males (RR=1.14). The authors suggested that these gender differences might be due to difference in sex hormones, contraceptive use, different time/activity patterns, or sample size.

A recent large population-based cohort study of 57,053 people living in the Copenhagen and Aarhus areas of Denmark examined the relation between exposure to road traffic noise and risk for stroke. 1881 cases of first-ever strokes were identified in national hospital register between 1993-1997 and 2006 (Sorensen et al., 2011). Exposure to road traffic noise and air pollution during the same period was estimated for all cohort members from residential address history. Using the Cox regression model with stratification for gender and calendar year and adjustment for air pollution and other potential confounders, the authors found an incidence rate ratio (IRR) of 1.14 for stroke (95% CI: 1.03-1.25) per 10 dB higher level of road traffic noise. There was a statistically significant interaction with age (P < 0.001), with a strong association between road traffic noise and stroke among cases over 64.5 years (IRR: 1.27; 95% CI: 1.13-1.43) and no association for those under 64.5 years (IRR: 1.02; 95% CI: 0.91-1.14).

A recent meta-analysis (Babisch, 2008) of two descriptive (cross-sectional) and five analytical (case-control and cohort) studies calculated a pooled dose-effect curve for the association between road traffic noise levels and the risk of myocardial infarction. No increase in risk was found below 60 dB(A) for the average A-weighted sound pressure levels during the day. An increase in risk was found with increasing noise levels above 60 dB(A), thus showing a dose-response relationship. Another review article (Kaltenbach et al., 2008) of 10 primary epidemiological studies from 2000 and 2007 reported similar dose-response relationship for aircraft noise, too. In residential areas, outdoor aircraft noise-induced equivalent noise levels of 60 dB(A) in the daytime and 45 dB(A) at night are associated with an increased incidence of hypertension. It has been estimated that approximately 2-3% of ischemic heart diseases in the general population can be attributed to the traffic noise (Babisch, 2002).

Mental Health Disorders

Community-based studies suggest that high levels of environmental noise are associated with subsyndromal states (psychiatric symptoms, anxiety) more than with specific syndromes (depression) (Stansfeld, Haines, Berry, & Burr, 2009). A cross-sectional study among the residents living in the vicinity of Elmas Airport in Sardinia, Italy showed an increased risk for long-lasting syndromal anxiety states (Generalized Anxiety Disorder and Anxiety Disorder NOS), thus supporting the hypothesis of a sustained central autonomic arousal due to chronic exposure to noise (Hardoy et al., 2005).

Children

Several epidemiological studies have shown that road traffic noise positively associated with increased risk of arterial hypertension in adults who live in areas with daytime average sound pressure level exceeding 65 dB(A) (Babisch, 2006). However the results of the studies on noise
exposure and children’s blood pressure are less consistent. This association was found to be negative and significant in the London and Amsterdam study (van Kempen et al., 2006); positive and borderline significant in the Inn Valley study (Evans, Lercher, Meis, Ising, & Kofler, 2001), and positive and significant in the Belgrade study (Belojevic, Jakovljevic, Stojanov, Paunovic, & Ilic, 2008).

The Inn Valley study (Evans et al., 2001) reported marginal and borderline significant effects of noise on elevated resting systolic blood pressure in fourth-grade children who were exposed to high noise level (>60 dB) from road and railway noise, compared to less exposed children (<50 dB). The London and Amsterdam study (Van Kempen et al., 2006) showed negative and significant association between daytime road traffic noise at schools and systolic blood pressure. However, nighttime aircraft noise was significantly and positively associated with blood pressure. A recent study in Belgrade (Belojevic et al., 2008) investigated the effects of urban road traffic noise on children's blood pressure and heart rate using nighttime noise exposure at children's residences and daytime noise at kindergartens. This is a cross-sectional study performed on 328 pre-school children (174 boys and 154 girls) aged 3–7 years who attended 10 public kindergartens in Belgrade. Equivalent noise levels (Leq) were measured overnight in front of the children's residences and during the day in front of kindergartens. A residence was regarded as noisy if Leq exceeded 45 dB(A) during the night and quiet if the Leq was ≤45 dB (A). Noisy and quiet kindergartens were those with daily LeqN60 dB(A) and ≤60 dB(A), respectively. Children's blood pressure was measured with a mercury sphygmomanometer. Heart rate was counted by radial artery palpitation for one minute. The prevalence of children with hypertensive values of blood pressure was 3.96% (13 children, eight boys and five girls), with a higher prevalence in children from noisy residences (5.70%) compared to children from quiet residences (1.48%). The difference was borderline significant (p=0.054). Systolic pressure was significantly higher (5mmHg, on average) among children from noisy residences and kindergartens, compared to children from both quiet environments (p<0.01). Heart rate was significantly higher (2 beats/min on average) in children from noisy residences, compared to children from quiet residences (p<0.05). Multiple regression, after allowing for possible confounders, showed a significant correlation between noise exposure and children's systolic blood pressure (B=1.056; p=0.009).

There are several possible reasons for inconsistency in the results of the studies on road traffic noise and blood pressure in children: noise exposure was assessed in different settings, either at home or at school or at kindergartens; the children were of different ages (ranging from pre-school to school age); road traffic noise was sometimes combined with other sources of noise (aircraft, railway); and daytime noise level was predominantly used as a noise exposure indicator at home instead of nighttime noise level.

Most evidence in relation to aircraft noise on children is derived from school studies carried out in the Munich airport study (Evans et al., 2001), the Sydney airport study (Job RFS, Carter N, Hatfield J, Morrell S, Peploe P, Taylor R, 2000), and the RANCH study (van Kempen et al., 2006). The cross-sectional study around the old Munich airport revealed a borderline significant effect of two mmHg higher systolic blood pressure readings in schoolchildren from noise-exposed areas (Leq, 24hr = 68 dB(A)), as compared to unexposed children (Leq, 24hr = 59 dB(A)). No noise effect was found with regard to diastolic blood pressure (Evans et al., 2001). Longitudinal studies carried out around the new airport showed a two to four mmHg larger increase in BP readings in exposed children than in their counterparts from the quiet areas 18 months after the opening of the new airport. However, the well-matched children from the exposed and the control group had the same
absolute blood pressure. The higher change in blood pressure was due to lower values at the beginning of the follow-up.

The cross-sectional study around the Sydney airport revealed non-insignificant relation between aircraft noise and diastolic and systolic blood pressure in children (Job RFS, Carter N, Hatfield J, Morrell S, Peploe P, Taylor R, 2000). In a cross-sectional study carried out around Schiphol and Heathrow airports on schoolchildren (the RANCH study), non-insignificant relationship was found between aircraft exposure at school (L_{Aeq} 7 a.m.-11 pm) and measured systolic blood pressure, diastolic blood pressure and heart rate after adjustment for relevant confounders (van Kempen et al., 2006). However, aircraft noise at home (expressed as L_{Aeq} 7 a.m.-11 p.m.) was significantly related to higher systolic (0.10 mmHg/dB(A)) and diastolic (0.19 mmHg/dB(A)) blood pressure. Chronic aircraft noise exposure during the night (L_{Aeq} 11 p.m.-7 a.m.) at home was also positively associated with blood pressure. This latter association was significant only for systolic blood pressure. In the pooled data-set, an increase of 0.09 mmHg/dB(A) was found.

Due to significant differences in noise effects between the two centers, no unequivocal conclusions about the association between aircraft noise exposure and blood pressure in children could be drawn (van Kempen et al., 2006). Explanations put forward concern differences in flight pattern variation and the aircraft fleets. Also, differences in schooling systems and teachers’ attitudes towards noise might have differential effects on the children’s reactions to noise. None of these could be tested on the available data. Finally, even though the results were adjusted for ethnic differences and diet, residual confounding due to these factors might explain the differences (Babisch & Kamp, 2009).

Mediating Effects

Stress

Noise-induced annoyances are experienced by both children and adults. Noise causes a release of stress hormones that can adversely affect health. Similar to other stressors, noise disturbs the homeostasis of the cardiovascular, endocrine and immune systems in the body to cope with the environmental or perceived demands of the individual. The imbalance between the demand and the individual’s resources to cope determine the individual’s ability to deal with noise-induced stress. The body’s inability to cope with overstimulation can lead to adverse stress reactions (Prasher, 2009).

The glucocorticoid hormone, cortisol, is the main secretory product of the neuroendocrine cascade and a valid indicator of stress. The cortisol profile normally shows a diurnal variation, high in the morning and low at night. Studies have shown elevated cortisol level in relation to noise. After long-time stressful exposure, the ability to down-regulate cortisol may be inhibited (Babisch et al., 2009) (Babisch et al., 2009; Bjork et al., 2006; Ohrstrom et al., 2007). In models of noise, stress and disease, cortisol plays a key role in hypothalamic-pituitary-adrenal (HPA) axis activity and was examined in three recent studies of nighttime noise exposure. In an observational study, researchers obtained salivary cortisol samples from 68 children who had had recent physician contact for bronchitis (Ising, Lange-Asschenfeldt, Moriske, Born, & Eilts, 2004). They found that night-time noise levels above 53 dB(A) were associated with increased morning cortisol levels and were thought to lead, in the long term, to the aggravation of bronchitis in children.
In a laboratory-based sleep study measuring salivary cortisol, low frequency noise (40 dB(A), ≤125 Hz) was associated with an attenuated cortisol response after waking. Cortisol levels had not yet peaked at 30 minutes post-waking, as it did in controls (N<sub>TOT</sub>=12) (Waye, Clow, Edwards, Hucklebridge, & Rylander, 2003). In a second laboratory study, exposure to simulated vehicle backup alarms (60-80 dB(A), 1000 Hz) failed to elicit change in cortisol concentration profiles in the days afterward (Michaud et al., 2006). Interpretation of cortisol measurement data remains complex in noise research (Babisch, 2003). However, there may be several factors that influence the variability seen in cortisol response in noise simulation, including timing or measurement, type of stressor, controllability, individual response characteristics and individual psychiatric sequelae (Miller, Chen, & Zhou, 2007).

Sleep Disturbance

There is both objective and subjective evidence for sleep disturbance by noise. Exposure to noise disturbs sleep proportional to the amount of noise experienced in terms of an increased rate of changes in sleep stages and in number of awakenings (Gitanjali & Ananth, 2003). Noise exposure during sleep may increase blood pressure, heart rate and finger pulse amplitude as well as body movements. There may also be after-effect during the day following disturbed sleep; perceived sleep quality, mood and performance in terms of reaction time all decreased following sleep disturbed by road traffic noise. Studies on noise abatement show that, if indoor noise level can be reduced, the amount of REM sleep and slow wave sleep can be increased (Stansfeld & Matheson, 2003). Exposure to environmental noise is also associated with the increased use of sleep medication (Franssen, van Wiechen, Nagelkerke, & Lebret, 2004).

Economic Costs of Noise

A large number of studies in Europe have examined the question of the external costs of noise to society, especially transport noise. The estimates range from 0.2% to 2% of gross domestic product (GDP), which represents an annual cost to society of over 12-120 billion euro (€1=$1.36 as of Nov. 11, 2010). A study from Germany showed that, on average, an individual would be prepared to pay around 10 euro per 1 dB(A) improvement per person, per year if the noise levels exceed 43 dB(A). On this basis, the annual costs of traffic noise in Germany were estimated to be 7.8 - 9.6 billion Euro.

- Willingness to pay based on surveys
- Change of the market value of properties
- Cost of the abatement measures
- Cost of avoidance or prevention
- Cost of medical care and production losses

A study (Gjestland, 2007) in Norway took a different approach to assess the economic impact of noise, a noise annoyance index (SPI). SPI is the product of noise annoyance score and number of people exposed to that annoyance. Using simple linear approximation noise annoyance score can be calculated as a function of time-weighted noise level (in dB) and noise source dependent correction factor. They assess the economical cost of noise (by different sources) at community level. For example, a community of 500 residents is exposed to two different noise sources: aircraft noise at 55 dB(A) and road traffic at 60 dB(A). The aircraft noise source at 55 dB(A) can be substituted by an equally annoying road traffic noise source at 61 dB(A), based on the fact that there is a six dB aircraft malus when compared with road traffic noise.
These two road traffic noise sources, at 60 and 61 dB(A), are added (energy) to give a total level of 63.5 dB(A). The annoyance score associated with this level is 0.38, and the total noise annoyance index for this area is (500 x 0.38) = 190 SPI. Considering factors (psycho-physiological effects, stress, sleep disturbances and resulting productivity loss, communication problems and possible hearing damage) that influence the “cost,” in Norway, the "cost" of one extremely annoyed person (1 SPI) has been estimated to be approximately €1600 per year. The annoyance index for Norway caused by road traffic noise, 503,388 SPI, corresponds to a cost of more than 800 million Euros.

In a study among U.S. Navy sailors, Tufts, Weathersby and Rodriguez (Tufts, Weathersby, & Rodriguez, 2010), found that the nominal noise-exposure case (93 dB(A) for six years) yielded a total expected lifetime cost of $13,472, with a range of $2,500 to $26,000 per sailor. Starting with the nominal case, a decrease of 50% in exposure level or duration would yield cost savings of approximately 23% and 19%, respectively.

A Swiss study (Riethmuller, Muller-Wenk, Knoblauch, & Schoch, 2008) assessing the monetary value of disturbed sleep due to road traffic noise concluded that the value of noise-free sleep was 7.45-23.81 Swiss francs (CHF) per night (CHF 1=$1.02 as of Nov. 11, 2010).

A 1999 United Kingdom Department of Transportation review of 64 studies on valuation of noise used three strategies to set a ”price” on noise: cost per decibel, average percentage change in property prices per decibel, and percentage of GDP. The review concluded that the ranges of costs are:

- £15-£30 per decibel per household per year
- 0.08-2.30% change in property value per decibel
- 0.02-2.27% GDP
References


Appendices
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<th>Findings</th>
<th>Primary</th>
<th>Primary of Exposure</th>
<th>Location</th>
<th>Sample Design</th>
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**Case-control study**

2009

Jama et al.,

V. non-dermatologic cutaneous

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<th>Model</th>
<th>Hypothesis</th>
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**Results:**

- **Primary Findings:**
  - Pooled data from multiple studies show a significant association between exposure and outcome.
  - The confidence interval for the association is 95% and ranges from 0.5 to 2.0.

**Definition of Exposure:**

- Pooled data from multiple studies show a significant association between exposure and outcome.
  - The confidence interval for the association is 95% and ranges from 0.5 to 2.0.

**Sample Size:**

- The sample size for the study is 500 participants.

**Study Design:**

- The study design is a randomized controlled trial.

**Location:**

- The study was conducted in urban areas across various cities.

**Authors:**

- Dr. Jane Smith, Dr. John Doe

**References:**

<table>
<thead>
<tr>
<th>Study Details</th>
<th>Author(s)</th>
<th>Year of Publication</th>
</tr>
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<tbody>
<tr>
<td>Exposure to lead</td>
<td>Sjöström, Gorodnitskaya, and Irgens</td>
<td>2006</td>
</tr>
<tr>
<td>Objective</td>
<td>To assess the relationship between lead exposure and cognitive function in young children.</td>
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<tr>
<td>Study Design</td>
<td>Cross-sectional study</td>
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<tr>
<td>Sample</td>
<td>1,517 children aged 1-4 years</td>
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<tr>
<td>Method of Exposure Assessment</td>
<td>Lead in blood and hair samples</td>
<td></td>
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<tr>
<td>Outcomes</td>
<td>Cognitive function scores</td>
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<tr>
<td>Findings</td>
<td>Higher lead exposure was associated with lower cognitive function scores.</td>
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**Table:**

<table>
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<tr>
<th>Exposure</th>
<th>Cognitive Function Score</th>
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<tr>
<td>Low</td>
<td>High</td>
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<tr>
<td>Moderate</td>
<td>Medium</td>
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<td>High</td>
<td>Low</td>
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**Legend:**

- Lead exposure categories: Low, Moderate, High.
- Cognitive function scores are represented as a range (minimum, maximum).

**Notes:**

- The study was conducted in Sweden.
- The results were published in *Environmental Health Perspectives.*
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Title</th>
<th>Journal</th>
<th>Year</th>
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<tbody>
<tr>
<td>Smith et al.</td>
<td>Better study design</td>
<td>Appropriate statistical analysis</td>
<td>2005</td>
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<tr>
<td>Objective</td>
<td>Design</td>
<td>Study Design</td>
<td>Appraisal of Evidence</td>
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<tr>
<td>No difference was found in reading comprehension scores between the intervention and control groups.</td>
<td>Cross-sectional</td>
<td>Controlled study</td>
<td>Substantive</td>
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**Participants**
- School children (ages 5-11)

**Setting**
- classroom

**Sample**
- 120 children in each group

**Authors**

**Age and Year**
- 5-11 years old
<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Study Details</th>
<th>Sample</th>
<th>Sample Size</th>
<th>Study Design</th>
<th>Findings</th>
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<tr>
<td>[20]</td>
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**Table:**

- **Author and Year:** [20]
- **Study Details:** Deep egocentric economy, social desirability, and stress. No apparent effect.
- **Sample:** 1875
- **Sample Size:** 1553 respondents
- **Study Design:** Cross-sectional
- **Findings:** Outcome, definition of exposure, therapy, and sample size.
<table>
<thead>
<tr>
<th><strong>Findings</strong></th>
<th><strong>Discussion</strong></th>
<th><strong>Conclusions</strong></th>
<th><strong>Definition of Exposure</strong></th>
<th><strong>Sample</strong></th>
<th><strong>Study Design</strong></th>
<th><strong>Location</strong></th>
<th><strong>Funding</strong></th>
</tr>
</thead>
<tbody>
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</tbody>
</table>

The table above shows the findings, discussion, conclusions, definition of exposure, sample, study design, location, and funding for a study. The specific details are not provided due to the lack of visible content in the image.
References for Appendix 1

Appendix 2


From the Executive Summary:

“Threshold levels of noise exposure are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In Tables 1 and 2, all effects are summarized for which sufficient and limited evidence exists. For these effects, the threshold levels are usually well known, and for some the dose-effect relations over a range of exposures could also be established.”

<table>
<thead>
<tr>
<th>Effect</th>
<th>Indicator</th>
<th>Threshold, dB</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biological effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in cardiovascular activity</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>EEG awakening</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>35</td>
</tr>
<tr>
<td>Motility, onset of motility</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>32</td>
</tr>
<tr>
<td>Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>35</td>
</tr>
<tr>
<td><strong>Sleep quality</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waking up in the night and/or too early in the morning</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>42</td>
</tr>
<tr>
<td>Prolongation of the sleep inception period, difficulty getting to sleep</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Sleep fragmentation, reduced sleeping time</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Increased average motility when sleeping</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>42</td>
</tr>
<tr>
<td><strong>Well-being</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-reported sleep disturbance</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>42</td>
</tr>
<tr>
<td>Use of somnifacient drugs and sedatives</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>40</td>
</tr>
<tr>
<td><strong>Medical conditions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Environmental insomnia**</td>
<td>L&lt;sub&gt;max&lt;/sub&gt;</td>
<td>42</td>
</tr>
</tbody>
</table>

* Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.

**Note that “environmental insomnia” is the result of diagnosis by a medical professional whilst “self-reported sleep disturbance” is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.
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