Environmental Noise and Non-Aural Health Effects – A Research Summary

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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, Berglind, 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 (OR_{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 (FBN) = 53 to 63 dB(A)(Rosenlund, Berglind, Pershagen, Järup, & Bluhm, 2001). When noise categories were combined, the effect was significant for FBN > 55 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 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 (L_{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 L_{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 indictor 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 glucocortcoid 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_{TOT}=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 sequalae (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

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Appendices

Appendix 1

Table 1 Summary of Key Evidence

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
Category A: Better study design, comprehensive statistical approach and strong evidence							
Sorensen et al., 2011[1]	Copenhagen, Denmark	Population Based Prospective Cohort	57,053 people of 50-64 years of age	Road traffic noise was calculated as the equivalent continuous A weighted sound pressure level (L_{Aeq}) at the most exposed facade of the dwelling at each address for the day (L_d; 07:00–19:00 h), evening (L_e; 19:00–22:00 h), and night (L_n; 22:00–07:00 h) and expressed as L_{den} (as an indicator of the overall noise level during the day, evening, and night) by applying a 5 dB penalty for the evening and a 10 dB penalty for the night.	Incident of Stroke.	Cox Proportional Hazards Model with age as underlying time	Incidence rate ratio (IRR) of 1.14 for stroke [95% confidence interval (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)
Hardoy et al., 2005[2]	Sardinia, Italy	Matched Case-Control	71 Cases and 284 matched controls with ages ranging between 18 and 75 years	Cases were selected from a suburban housing estate situated in the immediate vicinity of a large international airport exerting a strong degree of environmental noise (with night flying) and controls were selected from three areas of the same Italian region (Sardinia) which had not been exposed to airport noise	Specific lifetime DSM-IV diagnosis	Univariate analysis with odds ratio and 95% confidence interval	Exposed subjects showed a higher frequency of Generalized Anxiety Disorder and Anxiety Disorder Not Otherwise Specified (NOS) compared to non-exposed subjects.

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
Babisch, 2008[3]		Pooled Meta-Analysis	2 descriptive (Cross-Sectional) and 5 analytical (case-control and cohort) studies	Road traffic noise	Cardiovascular risk	odds ratio and 95% confidence interval	A pooled dose-effect curve for the association between road traffic noise levels and the risk of myocardial infarction were calculated. 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.
Beelen at al., 2009[4]	The Netharlands	Case-Cohort	120, 852 Subjects from Netharlands Study on Diet and Cancer (follow up:1987-1996)	Road Traffic noise at 1986 home address was estimated using EMPARA, a state-of-the –art model for noise mapping. Exposure were classified into categories of 5 dB(A), from ≤50 dB(A) to >65 dB(A).	Cardiovascular mortality	Cox Proportional Hazards Model	Traffic intensity was associated with cardiovascular mortality, with highest relative risk (95% confidence interval) for ischemic heart disease (IHD) mortality: 1.11 (1.03 - 1.20) (increment 10,000 mvh/24h). Relative risks for black smoke concentrations were elevated for cerebrovascular (1.39 (0.99 - 1.94)) and heart failure mortality (1.75 (1.00 - 3.05) (increment 10 µg/m3). These associations were insensitive to adjustment for traffic noise. There was an excess of cardiovascular mortality in the highest noise category (>65 dB(A)), with elevated risks for IHD (1.15 (0.86 - 1.53) and heart failure mortality (1.99 (1.05 - 3.79). After adjustment for BS and

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Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
							traffic intensity noise risks became unity for IHD mortality and slightly reduced for heart failure mortality.
Selander et al., 2009[5]	Stockholm, Sweden	Population based case-control	Total 3666 study subjects: 1571 cases and 2095 controls	Multiple sources of noise exposure: road traffic noise for all addresses of each subject; railway noise if lived within 200 m of a railway for at least 1 year; aircraft noise for the year 1997 at the Bromma and Arlanda airport; occupational noise exposure and self assessed noise annoyance	Risk for Myocardial Infarction (MI)	Unconditional logistic regression and multinomial regression to calculate odds ratio and 95% confidence intervals	The adjusted odds ratio for MI associated with long-term road traffic noise exposure of 50 dBA or higher was 1.12 (95% confidence interval = 0.95-1.33). In a subsample, defined by excluding persons with hearing loss or exposure to noise from other sources, the corresponding odds ratio was 1.38 (1.11-1.71), with a positive exposure-response trend. No strong effect modification was apparent by sex or cardiovascular risk factors, including air pollution from road traffic.
Sbihi et al., 2008[6]	British Columbia	Cohort	10 872 sawmill workers in British Columbia from 1991 to 1998	Used four exposure metrics: cumulative exposure, and duration of exposure above thresholds of 85 dB(A), 90 dB(A) and 95 dB(A).	Mortality or hospital admission for hypertension and doctor diagnosed hypertension	Poisson regression calculating relative risks	The results showed a monotonic increase in hypertension incidence with cumulative exposure. The risk in the highest exposed population was 32% higher than baseline. Similar results were found using duration of exposure metrics. The highest relative risk was 1.5 in workers exposed for more than 30 years at 85 dB(A). Exposure-response trends were statistically significant.
Babisch et	Berlin, Germany	Matched Case-Control	1881 cases (age 20-69	Combined scale of noise exposure: subjective annoyance	Incident of Myocardial	Conditional logistic	The adjusted odds ratio for men exposed to sound levels

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
al., 2005[7]			years) with confirmed diagnosis with myocardial infarction during 1998-2001 and 2234 sex, age and hospital matched controls	and objective measure of noise level from environment and work place. Environmental exposure was assessed using Berlin traffic noise map for living address of each subject and work sound level was determined according to ISO norm 9921/1.	Infarction (MI)	regression calculating odds ratio and 95% confidence intervals	of more than 70 dB(A) during the day was 1.3 (95% confidence interval=0.88-1.8) compared with those where the sound level did not exceed 60 dBA. In the subsample of men who lived for at least 10 years at their present address, the odds ratio was 1.8 (1.0-3.2). Noise-exposed women were not at higher risk
Category B: Better study design, appropriate statistical approach and sufficient evidence							
Willich et al., 2006[8]	Berlin, German	Matched case-control	4115 admitted cases from 32 major hospitals in Berlin with diagnosis of myocardial infarction and controls matched on gender, age and hospital	Combined scale of noise exposure: subjective annoyance and objective measure of noise level from environment and work place. Environmental exposure was assessed using Berlin traffic noise map for living address of each subject and work sound level was determined according to ISO norm 9921/1.	Incident of Myocardial Infarction (MI)	Conditional logistic regression calculating odds ratio and 95% confidence intervals	There was a marginally increased risk of myocardial infarction associated with annoyance by environmental noise in women (adjusted odds ratio 1.47, 95% confidence interval 0.95-2.25, P=0.081) but not in men, and not associated with annoyance by work noise. Environmental sound levels were associated with increased risk in men and women (odds ratios 1.46, 1.02-2.09, P=0.040 and 3.36, 1.40-8.06, P=0.007) and work sound levels in men only (1.31, 1.01-1.70, P=0.045).
Belojevic et al., 2008[9]	Belgrade, Croatia	Cross-sectional	328 preschool children (174 boys and 154 girls) aged 3–7 years, who attended	Noise exposures were measured in two night intervals in front of children's residences: between 10 p.m. and midnight and between midnight and 1.30 a.m. In front of each kindergarten	Children's blood pressure and heart rate	Multiple linear regression calculating regression coefficients	The prevalence of children with hypertensive values of blood pressure was 3.96% (13 children, including 8 boys and 5 girls) with higher prevalence in children from

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
			10 public kindergartens	noise measurements were performed in two daily periods (9 a.m.–10.30 a.m. and 1.30 p.m.–3 p.m.). Time interval of each measurement was 15 min; the speed of sampling was 10 per second, with 9000 samples collected per measurement. A composite night time Leq was calculated for each street and a composite daytime Leq for each kindergarten.		and 95% confidence intervals	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 pressure (B=1.056; p=0.009).
Jarup et al, 2008[10]	London, Berlin, Amsterdam, Stockholm, Milan and Athens	Cross Sectional	4,861 persons 45–70 years of age at the time of interview, with a minimum length of residence of 5 years living near one of six major European airports	Noise levels for separate periods of the day were modeled linking to each participant's home address using geographic information systems technique. For both aircraft and road traffic noise the levels had a 1-dB resolution, except for the United Kingdom, where only 5-dB classes for road traffic noise could be procured.	Automated blood pressure instruments measured hypertension	Multivariable logistic regression calculating odds ratio and 95% confidence intervals	Significant exposure–response relationships between night-time aircraft as well as average daily road traffic noise exposure and risk of hypertension after adjustment for major confounders. For night-time aircraft noise, a 10-dB increase in exposure was associated with an odds ratio (OR) of 1.14 [95% confidence interval (CI),

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
							1.01–1.29]. The exposure–response relationships were similar for road traffic noise and stronger for men with an OR of 1.54 (95% CI, 0.99–2.40) in the highest exposure category (> 65 dB; *p* trend = 0.008).
van Kempen et al, 2006[11]	London and Amsterdam	Cross-sectional	1283 children (age 9–11 years) attending 62 primary schools around two European airports: Heathrow and Schiphol.	Noise exposure was assessed for each child by linking home and/or school addresses to modelled equivalent aircraft and road traffic noise levels. These predict the average outdoor noise exposure during a specified time interval.	Blood pressure measurements were taken in the afternoon in a quiet room in the school building using automatic blood pressure meters	Multilevel modeling was used with pooled date.	Aircraft noise exposure at school was related to a statistically non-significant increase in blood pressure and heart rate. Aircraft noise exposure at home was related to a statistically significant increase in blood pressure. Aircraft noise exposure during the night at home was positively and significantly associated with blood pressure. The findings differed between the Dutch and British samples. Negative associations were found between road traffic noise exposure and blood pressure
Category C: Observational study design, appropriate statistical approach and weaker evidence							
Stansfeld et al., 2009[12]	North Wales, UK	Cross-sectional with follow up	The sample were adults over 16 years living in 387 households exposed to high levels of road traffic along the main street in three	Noise exposure was measured outdoors in both the high and low road traffic noise exposed streets at baseline in the first two weeks of December 1997; repeat measurements were carried out 12 months later in the first week of December 1998. Noise measurements were made between 10 am and	Noise annoyance was measured with three standard questions that assessed the level of annoyance for three sources of environmental	Univariate Analysis of Variance (ANCOVA) for cross-sectional main noise effect and within-subject	No differences were found between the noise exposed and quiet areas at baseline in annoyance, or common mental disorder and no change in annoyance, health functioning or common mental disorder related to the introduction of the bypass at the follow up.

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
			towns in Clwyd, North Wales and adults living in 'control' households exposed to lower levels of road traffic in the same area.	5 pm on weekdays in both noise exposed and quiet areas with matching of time of day between baseline and follow-up.	noise at home: (Neighbors, road traffic noise and train noise). The Revised Clinical Interview Schedule assessed the prevalence of common mental disorder in a subsample. Respondents scoring above a threshold of 12 or more were considered to be potential psychiatric cases.	follow up analysis with adjustment for baseline health status	
Bodin et al., 2009[13]	Scania region of Southern Sweden	Population based cross-sectional	Randomly selected 24,328 adults (18-81 years) from a public health survey	Average road noise (LAeq 24 hr) at the current residential address using geographic information system	Self-reported hypertension	Multivariable logistic regression calculating odds ratio and 95% confidence intervals	Modest exposure effects (OR ≈ 1.1) were generally noted in intermediate exposure categories (45 -64 dB(A)), and with no obvious trend. The effect was more pronounced at > 64 dB(A) (OR 1.45, 95% CI 1.04 - 2.02). Age modified the relative effect (p = 0.018). An effect was seen among middle-aged (40 - 59 years old) at noise levels 60 - 64 dB(A) (OR = 1.27, 95% CI 1.02 - 1.58)) and at > 64 dB(A) (OR = 1.91, 95% CI 1.19 - 3.06)). An effect was also indicated among younger adults but not among

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
							elderly. No apparent effect modification by gender, country of origin, disturbed sleep or strained economy was noted.
Barregard et al., 2009[14]	Lerum, Sweden	Population based cross-sectional	1953 residents between 18-75 years of age and had resided at their present address for at least 6 months	A weighted 24 hr average sound levels (LAeq 24 hr) from a highway (20,000 vehicles/24 hr) and railway (200 train/24 hr) were calculated at the current residential address using geographic information system	Self-reported hypertension and hypertensive medication use	Logistic regression, Poisson regression and Cox Hazard model.	When road traffic noise, age, sex, heredity and body mass index were included in logistic regression models, and allowing for >10 years of latency, the OR for hypertension was 1.9 (95% CI 1.1 to 3.5) in the highest noise category (56-70 dBA) and 3.8 (95% CI 1.6 to 9.0) in men. The incidence rate ratio was increased in this group of men, and the relative risk of hypertension in a Poisson regression model was 2.9 (95% CI 1.4 to 6.2). There were no clear associations in women or for railway noise.
Leon Bluhm et al., 2007[15]	Stockholm, Sweden	Population based cross-sectional	Stratified random sample of 667 residents aged 19-80 years	The outdoor equivalent traffic noise level (Leq 24 h) at the residence of each individual was determined using noise-dispersion models and manual noise assessments. The individual noise exposure was classified in units of 5 dB(A), from <45 dB(A) to >65 dB(A).	Self-reported hypertension	Multivariable logistic regression calculating odds ratio and 95% confidence intervals	The odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% confidence interval (CI) 1.06 to 1.80) per 5 dB(A) increase in noise exposure. The association seemed stronger among women (OR 1.71; 95% CI 1.17 to 2.50) and among those who had lived at the address for >10 years (OR 1.93; 95% CI 1.29 to 2.83).

Author and pub year	Location	Study design	Sample	Definition of Exposure	Primary Outcome	Statistics	Findings
							Analyses of categorical exposure variables suggested an exposure–response relationship. The strongest association between exposure to traffic noise and hypertension was found among those with the least expected misclassification of true individual exposure, as indicated by not having triple-glazed windows, living in an old house and having the bedroom window facing a street (OR 2.47; 95% CI 1.38 to 4.43).
Selander et al., 2009[16]	UK, Germany, Sweden, The Netherlands, Italy and Greece	Cross-sectional	A subsample of 439 participants of HYENA study	Aircraft noise exposure was assessed for each participant's home address using geographic information system mapping.	Saliva Cortisol level for each subject at three times (morning, lunch and evening) during 1 day	Linear regression calculating regression coefficients and 95% confidence intervals	An elevation of 6.07 nmol/L [95% confidence interval (CI), 2.32-9.81 nmol/L] in morning saliva cortisol level in women exposed to aircraft noise at an average 24-hr sound level (L(Aeq,24h)) > 60 dB, compared with women exposed to L(Aeq,24h) < or = 50 dB, corresponding to an increase of 34%. Employment status appeared to modify the response. No association was found between noise exposure and saliva cortisol levels in men.

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Appendix 2

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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."

Effect		Indicator	Threshold, dB
Biological effects	Change in cardiovascular activity	*	*
	EEG awakening	$L_{Amax,Inside}$	35
	Motility, onset of motility	$L_{Amax,Inside}$	32
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	$L_{Amax,Inside}$	35
Sleep quality	Waking up in the night and/or too early in the morning	$L_{Amax,Inside}$	42
	Prolongation of the sleep inception period, difficulty getting to sleep	*	*
	Sleep fragmentation, reduced sleeping time	*	*
	Increased average motility when sleeping	$L_{night,outside}$	42
Well-being	Self-reported sleep disturbance	$L_{night,outside}$	42
	Use of somnifacient drugs and sedatives	$L_{night,outside}$	40
Medical conditions	Environmental insomnia**	$L_{night,outside}$	42

Table 1 Summary of effects and threshold levels for effects where *sufficient* evidence is available

* *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.*

Table 2
Summary of effects and threshold levels for effects where *limited* evidence is available**

Effect		Indicator	Estimated threshold, dB
Biological effects	Changes in (stress) hormone levels	*	*
Well-being	Drowsiness/tiredness during the day and evening	*	*
	Increased daytime irritability	*	*
	Impaired social contacts	*	*
	Complaints	$L_{night,outside}$	35
	Impaired cognitive performance	*	*
Medical conditions	Insomnia	*	*
	Hypertension	$L_{night,outside}$	50
	Obesity	*	*
	Depression (in women)	*	*
	Myocardial infarction	$L_{night,outside}$	50
	Reduction in life expectancy (premature mortality)	*	*
	Psychic disorders	$L_{night,outside}$	60
	(Occupational) accidents	*	*

** 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 as the evidence for the effects in this table is limited, the threshold levels also have a limited weight. In general they are based on expert judgement of the evidence.*

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