# Noise and health, cardiovascular risk and susceptible groups

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## ABSTRACT

Chronic exposure to noise in residential, work and some recreational situations can lead to a range of health effects. These are usually subdivided into well-being effects, such as annoyance and sleep disturbance, and clinical effects, such as hearing damage and cardiovascular diseases (CVD). The association between noise and CVD has been studied for several decades now and the weight of evidence clearly supports a causal link between the two. Nevertheless, many questions remain, such as the magnitude and threshold level of the adverse effects of noise, how noise and other pollutants (such as particulate matter) interact in disease causation, identifying vulnerable populations, and how epidemiologic study methodology can be improved. After a general introduction and a short description of potential mechanisms, this paper reviews the state of the art as described in literature over the past years in the area of noise and CVD with a focus on susceptible groups, and noise and air pollutants interaction and some attention for threshold levels.

## BACKGROUND

The effect of noise exposure on the cardiovascular system is one of the key focuses in current noise and health research. In the past decade sufficient evidence has been collected to conclude chronic exposure to environmental noise negatively affects the cardiovascular system. Particular effects are increased risk of hypertension (ICD-10: I10) and ischemic heart disease (ICD-10: I20-I25), including myocardial infarction (ICD-10: I21) and angina pectoris (ICD-10: I20) Most noise studies have looked into hypertension and ischemic heart disease. More recently a large cohort study in Denmark has found an association between road traffic noise and the incidence of stroke with an increased risk of 14% with an increment of 10dB (Sørensen et al., 2011)

Several reviews have suggested that noise exposure is associated with blood pressure changes and ischemic heart disease (Babisch (2006, 2008; van Kempen, et al., 2002; Babisch & van Kamp, 2009). The biological plausibility of the hypothesis of the effects of noise on the cardiovascular system is high and assumes that noise acts as a stress factor and as such has the potential of directly and indirectly causing disease (van Kempen et al., 2002). The associations are weak for blood pressure changes and hypertension and somewhat stronger for ischemic heart disease (Bodin et al, 2009). For hypertension as well as ischemic heart disease, proposed threshold values (WHO Guidelines, 2000, NHC, 2000) range between $L_{Aeq}$ levels of 65–70 dBA environmental noise exposure outdoors. Recent meta-analyses partially confirm the conclusion of the WHO that the association between road and aircraft noise exposure and cardiovascular disease is comparable. No definite conclusions can be drawn regarding the exposure–effect relations or the possible threshold values. However, preliminary exposure–response curves are available that can be used for a quantitative risk assessment (Babisch, 2008). One restriction is that cardiovascular effects have only been investigated in limited population groups (middle-aged men). Since some studies regarding hypertension were cross-sectional, no final inferences can be made about causality and long-term effects. Also it is relevant whether exposure and outcome have been assessed objectively. It is highly unlikely that ill health or noise sensitivity lead to noise exposure when reporting bias is accounted for, so in most cases the risk of underestimating the effect is larger than the risk of overestimation. Recent large meta-analyses on ischemic heart disease refer to prospective cohort and case-control studies, in which new and objectively diagnosed cases were detected over the study period and exposure was objectively assessed independently of disease incidence. This therefore enabled more definite inferences about the relation between noise and cardio-vascular disease (Babisch, 2003, 2008; van Kempen & Babisch, 2012).

### Mechanisms

Figure 1 shows the potential mechanisms by which noise can lead to health problems. The model is based on a publication of the Netherlands Health Council and is one of the prevailing approaches to noise and health based on a cognitive stimulus-response model.

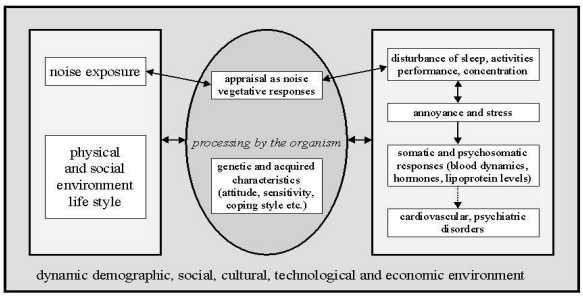


Figure 1: Blueprint of a conceptual model of noise and health. (Source: Health Council Netherlands, 1999).

The model assumes that most effects are a consequence of the appraisal of sound as noise. It is generally assumed that stress responses play an important role in the process by

which environmental noise leads to health effects. However, sound can also directly lead to physiological responses due to interactions of the acoustic nerve with other parts of the central nervous system. This is particularly relevant during sleep. Noise exposure is associated with annoyance, sleep and activity disturbance, and stress responses. These effects are at the base of so-called instantaneous effects such as blood pressure increases and increased secretion of cortisol, responses considered to be risk factors for cardiovascular diseases and mental pathology. Responses are partly dependent on the noise characteristics of frequency, intensity, duration, and meaning, and partly on non-acoustical aspects such as context, attitude, expectations, fear, noise sensitivity, and coping strategies.

### Knowledge and knowledge gaps

In 2008 the ICBEN (International Commission on the Biological Effects of Noise) team 3 review (Davies & van Kamp, 2008) concluded that future attention should aim at the effects of combined exposures (noise and air), the inconsistency found in cardiovascular effects in children and effects in vulnerable groups as well as gender differences. It was also concluded that exposure assessment should be improved, endpoints measured more consistently, and that more systematic adjustment for confounding factors was advised. The subsequent ICBEN team 3 review over the period 2008-2011 (Davies & van Kamp, 2012 in press) identified 50 peer-reviewed English-language papers. Over this period an emphasis on road traffic exposure was observed, as well as a focus on co-exposures to noise and traffic-related air pollution and their health effects. Correlations between traffic-related pollutants were not as high as was expected. The findings of studies on the joint effect on health were not entirely consistent, but suggest independent cardiovascular effects of noise and air pollution. Aircraft noise exposure also received considerable attention with multiple components of the Hypertension and Exposure to Noise near Airports (HYENA) project and one very large cohort study finding independent effects for aircraft noise on risk of myocardial infarction, after adjustment for air pollution. Occupational studies were still less common during this period, but new evidence showed a fairly consistent positive association between workplace noise and both hypertension and ischemic heart disease. Finally, a handful of studies were identified dealing with other issues such as policy, methodology and disease mechanisms. A separate search was performed for noise and health in vulnerable groups. In most recent reviews (WHO JRC, 2011; Clark & Stansfeld, 2007; Berry et al., 2009; Davies & van Kamp, 2008; WHO, 2000) on noise and health, this topic has been touched upon, but evidence is still scarce or scattered. There are conceptual problems and the mechanisms for these vulnerabilities have not been clearly described, nor are the mechanisms necessarily the same for different groups at risk. For an extensive description we refer to Davies & Van Kamp (2012 in press) and Van Kamp & Davies (2012 in press).

In this paper the most recent findings on noise and cardiovascular effects are summarized. A separate paragraph is devoted to the issue of vulnerable groups and describes what we know about physiological and cardiovascular effects in susceptible groups. Papers are presented by disease categories within exposure categories, followed by a section on effects in vulnerable groups.

## METHOD

This paper is based on reviews prepared for the International Commission on the Biological Effects of Noise (ICBEN) 2011 conference (Van Kamp, & Davies, 2012, in press) and (Davies & Van Kamp, 2012, in press) plus a chapter in the Praeger Handbook of Environmental health (Van Kamp, Babisch and Brown, 2012). For the reviews two literature searches were performed pertaining to the theme of the physiological effects of noise exposure and environmental quality in relation to noise and susceptible groups respectively. The search for physiological effects was limited to English language, peer reviewed and the period 2008-11, while the search on susceptible groups was limited to English, French and German language, peer reviewed and covered the period between 2006 and 2011. For the review on physiological effects we searched PubMed using keywords pertaining to noise, transportation, traffic, aircraft, railway, proximity to road; and cardiovascular disease, coronary heart disease, ischemic heart disease, myocardial infarction, hypertension, and stroke. Literature was also hand-searched for additional studies. To detect relevant peer reviewed studies on noise and health in susceptible groups Medline and Scopus were searched. A wide range of keywords was used, related to noise exposure, vulnerable groups and health outcomes. In addition, the reference sections of previous systematic reviews, key papers, conference proceedings and international reports on vulnerable groups as well databases of websites dealing with the issue of noise and vulnerability (WHO, PINCHE, ENNAH) were checked for potentially relevant references. For details on the method we would like to refer to Davies & van Kamp (2012, in press) and Van Kamp & Davies (2012, in press)

## RESULTS

### Road traffic Noise

One of the cardiovascular effects often studied in relation to road traffic noise is hypertension. Findings on hypertension (HT) have been called “extremely heterogeneous” (Babisch, 2006; Barregard et al., 2009), possibly due to limitations in study design. In the more recent study of Barregard et al. (2009) physician-diagnosed HT was examined in a cohort of 1,953 adults. Information on potential confounders was collected by questionnaire and traffic noise exposures were modelled. Prevalence and Incidence of HT and use of antihypertensives in relation to levels of noise exposure were found primarily in men, especially when considering length of residency (>10 yr). In order to reduce heterogeneity Belojevic et al. (2008) used measured night-time noise data from 70 downtown streets in Belgrade to assess exposure in a cohort of 2,503 adults, who had lived in the same residence for > 10 years, and who slept on the “street side” of the house. HT prevalence was 19.2%; and again it was found that there was a positive association in men but not women after taking confounders into account. Bodin et al. (2009) studied the effect of age in the relation between road traffic noise and Hypertension. They also used modelled exposure levels, and self-reported HT. Risk of HT was “modestly” increased at levels < 60 dBA ($L_{eq,24hr}$), and increased above 60 dBA. An age effect was seen with greatest risks in the “middle aged” (40-59 years) compared to younger (18-39), or old-aged respondents (60-80). In contrast to findings reported by Belojevic no gender differences were noted. Chang et al. (2009) examined road traffic noise and HT in Taiwan and extremely high exposure levels were reported (90% above 75 dBA $L_{eq,\ 8hr}$ at residence). A dose-response in prevalence of

hypertension was observed in males only. The EU HYENA study provided interesting data on the effects of road traffic noise in a study around 6 major European airports. (Floud et al, 2010; Haralabidis et al. 2008; Jarup et al, 2008; Selander et al, 2009). A significant increase in (adjusted) risk of HT per 10 dB increase for road traffic noise was found with again a more pronounced dose-response relation for men. The same pattern showed in hypertensive medication use, but was not significant. A study on a sub-sample of HYENA data (N=149) showed a "non-dipping effect" of diastolic blood pressure at night, which has been previously identified as an independent risk factor for cardiovascular disease. Griefahn et al. (2008) showed increased heart rate evoked by night-time noise exposure levels between 45-77 dBA, with and without awakening as well as incomplete habituation to noise during sleep. These findings were supported in a rail-noise study by Tassi et al, (2010a) showing larger cardiovascular effects of night time freight train exposure and among younger subjects (Tassi et al, 2010b). In general it can be concluded that studies into the physiological effects of night time noise exposures show inconclusive results (Graham, et al., 2009; Selander et al., 2009). Finally, increased cardiovascular risk was confirmed in three studies using proximity of residence to major roads as exposure estimate (Gan et al, 2011a; Hoffman et al, 2009; van Hee et al. 2009). All of these studies acknowledged the difficulty in attributing cause to specific agents and identify noise as one such potential agent, along with air pollutants.

Combined exposure to noise and air pollutants is a key issue in the interpretation of studies of associations between road traffic and cardiovascular disease (Brook, 2010), especially in studies that use proximity to roadway as a surrogate for exposure, i.e. Gan et al. (2011a). Air pollution can be considered as a confounder or effect modifier. Several recent studies have examined the relationship between noise and air pollution. Table 1 shows the correlations found in some recent studies.

**Table 1: Correlation between traffic noise and air pollutants (pearson correlation coefficient unless otherwise stated).**

| | $NO_X$ | NO | $NO_2$ | Black smoke | UFP* |
|---|---|---|---|---|---|
| Noise[+] | 0.62[a]<br>0.64[b]<br>0.50[c] | 0.41-0.60[d]<br>0.39[e] | 0.53[b]<br>0.62[f]<br>0.16-0.62[d]<br>0.33[e] | 0.24[f]<br>0.44**[e] | 0.21-0.60[g]<br>0.22-0.41[d]<br>0.41-0.81[h] |

[+] different indicators of LAeq atmost exposed façade * Ultrafine particulate; ** Black carbon; [a] (Sorensen *et al.* 2011); [b] (Davies, *et al*. 2009); [c] (Persson *et al.* 2007); [d] (Allen *et al*. 2009); [e] (Gan *et al*. 2011a -)

Table 1: Correlation between traffic noise and air pollutants (Pearson correlation coefficient unless otherwise stated).

Somewhat unexpectedly, correlations were consistently low to moderate. The lower than expected correlations are likely due to differences in actual source and propagation paths, traffic density, and meteorological conditions (Davies et al., 2009; Foraster et al., 2011; Allen et al., 2011). Correlations of modelled estimates (Sørenson, 2011, Gan et al, 2011b) were similarly low - even though both noise and transport related air pollution (TrAP) models used similar inputs and might have been expected to generate higher correlations.

Three recent large cohort studies looked specifically at road traffic noise and heart disease, examining also the joint effect of noise and air pollution (Beelen et al., 2009; Gan et al, 2011b; Selander et al., 2009). Gan et al. (2011a) found a 9% (95%CI 1%-18%) increase in CHD mortality associated with a 10 dB(A) increase in residential noise (modelled, all transport sources, $L_{DEN}$). Beelen et al. (2009) investigated several CVD mortality endpoints in the Netherlands Cohort Study on Diet and Cancer, using modelled noise levels. For the highest noise exposure category (>65 dBA $L_{DEN}$) they found an increased relative risk (adjusted) for all CVD of 1.25 (95% Confidence Intervals between 1.01-1.53); risk of heart failure, ischemic heart disease and dysrhythmia were also elevated. None of the three latter effects was statistically significant. Selander et al. (2009) confirmed an increased risk for acute myocardial infarction (MI) morbidity in the Stockholm Heart Epidemiology Program. With respect to the joint effect of traffic-related air and noise pollution on CVD, the findings of these three studies were similar. The most important finding is that each pollutant seemed to have independent effects on CVD outcomes. As one of the first, Sørensen et al. examined the association between road traffic exposure and incidence of stroke in a Danish cohort of 57,053 (Sørenson et al., 2011). Road traffic noise ($L_{DEN}$) was positively associated with incidence of stroke with a relative risk of 1.14 (95%CI 1.03-1.25) overall. In people over 64.5 years an elevated risk showed at levels of 55 and 60 dBA.

Finally, Sobotova et al. (2010) showed that exposure to road traffic noise was associated with elevated risk factor scores such as the Framingham SCORE60 and the European Society of Cardiology's relative risk SCORE. Both metrics are used to predict a person's chance of having a heart attack in the next 10 years.

### Aircraft Noise

Babisch & van Kamp (2009) summarized the literature on aircraft noise and hypertension. They concluded that there was sufficient evidence of association between aircraft noise and hypertension, but that only a "best guess" quantitative effect estimate could be made, i.e. an increased relative risk of 1.13 (95% CI 1.00-1.28). In the period between 2008-2011 a series of papers was published describing the HYENA study, investigating noise near airports and hypertension. (Jarup et al, 2008; Haralabidis et al, 2008, 2011; Eriksson et al, 2010). It involved six major European airports and 4,861 subjects, used modelled exposure data for aircraft noise, measured blood pressure levels and collected data on potential confounders and effect modifiers. A dose-response pattern for hypertension was found for $L_{night}$ but not $L_{Aeq,16hr}$. Hypertensive medication use was positively associated with $L_{night}$ and $L_{Aeq,16hr}$, but strength of association varied by country and was only significant in UK (both metrics) and the Netherlands ($L_{night}$ only). No apparent gender effect was found. The non-dipping effect seen for night-time road traffic was not confirmed for air traffic but blood pressure was increased – systolic blood pressure by 6.2 mmHg, and diastolic blood pressure by 7.4 mmHg during 15-minute measurement intervals in which there was an aircraft event (Barregard et al., 2009) Finally, Selander et al. (2009) found elevated morning cortisol levels in a subset of 439 HYENA subjects, in relation to aircraft noise at night, but only in women, and especially those who were currently working. Chronically elevated levels of cortisol are considered as an indicator of chronic stress and as such a risk factor for cardiovascular disease.

Exposure to military aircraft noise was studied in Korea (Rhee et al, 2008). Exposed subjects lived within 5km of either a helicopter base ($L_{Aeq,8hr}$ 71-72 dBA) or a fighter jet base (68-82 dBA), for a minimum of 10 years and were compared to non-exposed control group. Risk for HT (blood pressure of >140/90 or use of antihypertensive medication) was elevated in both exposed groups, but was statistically significant only for helicopter exposure.

Eriksson et al. (2010) examined cumulative incident HT (physician diagnosed or blood pressure >140/90) in 4,721 subjects exposed to noise from Stockholm airport, and who

were followed for 8-10 years. Elevated risk for HT was apparent in males only and thus comparable to findings in relation to road traffic noise. Estimated risk increased when the analysis was restricted to non-smokers.

Finally Huss et al. (2010) found a 50% increase in acute myocardial infarction (AMI) mortality in those exposed $L_{DN}$ > 60 dBA in six million subjects who were followed for 5 years. $PM_{10}$ was not linked to increased risk of AMI, but residing within 100 metres of a major road was associated with an increased risk of approximately 18%. No link between aircraft noise exposure and stroke mortality was confirmed in this study.

### Occupational Noise

Occupational noise studies are not very common but for several reasons are extremely valuable. There is larger control over exposure levels and these studies are less sensitive to confounding.

Two cohort studies of occupational exposure and hypertension were reported in the period between 2008 and 2011 (Lee et al., 2009; Sbihi et al., 2008). In a cohort of 10, 872 sawmill workers followed for 8-yrs Sbihi et al found that cumulative occupational noise exposure was a strong predictor of risk of HT, with a relative risk of 1.32 in the highest exposed group (>115 dBA*year) compared to controls (<95 dBA*year). There was a significant dose-response trend. Inconsistent trends over different "threshold" levels were investigated by Sbihi et al. by adjusting measured noise exposures for hearing protection devices. Lee et al. followed 530 male metal manufacturing workers for nine years, obtaining annual blood pressure measurements and found that systolic, but not diastolic, blood pressure increased over time in all three exposed groups, in a dose-response fashion; 1.7, 2.0 and 3.8 mmHg for groups I, II, and III respectively.

Chang et al. (2009, 2010) examined the role of workplace co-exposure of noise exposure and organic solvents in a pair of studies. In the first, cross-sectional, study 59 subjects exposed to either noise or solvents showed greatly increased risk of hypertension, 7-8 fold compared to a control group, but co-exposure to both pollutants did not significantly increase this risk. In the second study, twenty subjects were divided into 4 similar exposure groups (none, noise-only, solvent only and combined) and undertook 24-hour ambulatory blood pressure monitoring. Only combined-exposure showed significantly elevated blood pressure. Both studies were challenged by small numbers, cross-sectional study design and poorly controlled confounding.

In a meta-analysis of 15 studies into noise and hypertension (18,658 subjects), heart rate and ECG abnormalities, positive results were found for all hypothesized associations (systolic and diastolic blood pressure, heart rate, and electro cardiogram [ECG] abnormality) (Tomei et al., 2010).

A secondary data analysis of the US National Health and Nutrition Examination Survey (NHANES) by Gan et al. (2011b) observed excess risk for angina pectoris, myocardial infarction and coronary heart disease across subjects employed in a broad range of industries. Dose-response trends were statistically significant for angina and coronary heart disease (CHD). Risks for CHD were stronger in younger age groups, current smokers, and in men. Noise was also associated with isolated diastolic blood pressure >90 mmHg, and a systolic blood pressure below 140 mmHg), but not with any other blood pressure measures or any biomarkers.

### Vulnerable groups

Several recent reviews have emphasised the importance of studying health effects of noise in specific susceptible groups (WHO & JRC, 2011; Clark & Stansfeld, 2007; Berry et al., 2009; Davies & van Kamp, 2008; WHO, 2000). In the framework of this paper, which addresses cardiovascular effects, only age and gender specific studies are considered relevant.

Gender differences have shown up in most studies on cardiovascular effects. In general men are at greater risk than women for noise-related cardiovascular disease irrespective of noise source (road vs. aircraft) or outcome (hypertension or heart disease), with some exceptions. The results regarding gender differences in cardiovascular effects of noise are labelled as inconclusive (WHO & JRC, 2011; Babisch, 2006e). Babisch (2006) showed that people with prevalent chronic diseases run a slightly higher risk of heart diseases as a result of traffic noise than those without.

With respect to age, noise effects in (school) children are the the best documented and include some cardiovascular indicators. Analysis on the pooled data set (Heathrow, Schiphol) of the RANCH study (van Kempen et al, 2006) indicated that aircraft noise exposure at school was related to a non-significant increase in blood pressure and heart rate in children. Road traffic noise showed an unexplained "protective" effect. Babisch & van Kamp (2009) found an inconsistent association between aircraft noise and children's blood pressure. Likewise recent reviews concluded a tendency towards positive associations, but large methodological differences between studies and inconsistent associations of aircraft noise with systolic blood pressure in children (Paunovic et al, 2011; Stansfeld & Crombie, 2011). Based on a study among children (Babisch et al. 2009) it was concluded that road traffic noise at home could affect children's blood pressure. There is some evidence that short-term cardiovascular reactions during sleep are more pronounced in children (Stansfeld & Crombie, 2011). Lepore et al. (2010) found that in comparison with quiet-school children, noisy-school children had significantly lower blood pressure increases, when exposed to either acute noise or non-noise stressors, indicative of a generalized habituation effect. Studies in Serbia (Belojevic et al., 2008, 2011) among schoolchildren and preschool children indicated a raised blood pressure among children from noisy schools and quiet residences, compared to children from both-quiet environments. There is no consistent evidence that the effect of traffic noise on cardiovascular diseases is greater in older than younger people. (Belojevic et al, 1011). Bodin et al. (2009) found strong evidence for an age effect in the noise blood pressure association, with a stronger relation in the middle aged; age group-specific models could account for differences in prevalence in future studies.

Taking physiological changes as endpoint, a study in France (Mir, 2008) among 10 year-old schoolchildren showed that school noise exposure was associated with higher cortisol levels indicative of a stress reaction. These finding are supported by a Swedish study (Wålinder et al., 2007) that found increased prevalence of reduced diurnal cortisol variability in relation with classroom Leq during school day levels between 59 to 87 dBA.

## CONCLUSION

Recent studies into the effect of noise on the cardiovascular system support and refine previous findings of a positive association between them. Several of the earlier identified gaps have been addressed in recent studies. Especially important progress was made on "disentangling" the cardiovascular effects of traffic-related coexposures, noise, and traffic related air pollution (TrAP). The correlations between these exposures were not as high as many researchers feared, and epidemiological studies could therefore be successfully pursued. Four large health studies examining joint effects, were consistent in suggesting that both air pollution and noise are likely independent risk factors for cardiovascular disease. This is consistent with evidence derived from animal and occupational studies that are less susceptible to confounding and in line with the fact that plausible biological mechanisms exist for exposure to air pollution as well as noise (Babisch, 2006).

With respect to effects of gender on health associations, the majority of the studies found men to be at greater risk than women for noise-related cardiovascular disease irrespective of noise source (road vs. aircraft) or outcome (hypertension or heart disease). However there are some exceptions regarding cortisol response, self-reported hypertension, and stroke (Selander et al., 2010; Bodin et a., 2009; Sørensen et al., 2011). Analysis on the HYENA data set showed that these gender effects are quite complicated. While men showed to be more at risk in general, only women were at risk in the 65 and over sample (Jarup et al., 2008).

Several reviews concluded on an inconsistent association between aircraft noise and children's blood pressure. Effects of road traffic are understudied in children. There is some evidence that cardiovascular response to nighttime exposure is stronger in children than adults and inconsistent evidence of a generalized habituation effect of children frequenting high-noise schools. In general studies into effects of noise on the cardiovascular system in specific and potentially susceptible groups are scarce. As a consequence, clear effects are few and this is partly due to the lack of targeted and well designed studies making clear comparisons between the general population and potentially susceptible groups and quantifying these differences in terms of noise levels. Setting specific limit values for susceptible groups is not possible yet based on the available evidence.

Evidence does not indicate that the elderly are more vulnerable to effects of noise in terms of annoyance and sleep disturbance, but possibly the elderly are more vulnerable regarding cardiovascular effects and this may be a combined effect of air pollution and noise (Gan et al., 2012).

Generally speaking the methodological quality of research has increased compared to earlier efforts in this field. The problem of misclassification of exposure has been addressed by the work on TrAP and noise correlations and in the occupational arena by the work of Sbihi et al. (2008, 2010a, 2010b).

With respect to future research, reviewers are consistent in their call for more prospective studies to help elucidate underlying mechanisms of disease and the study of children, where results have been inconsistent. The distribution of noise over groups based on socioeconomic status (SES) deserves more attention, as well as the accumulation of exposures (noise & air), the accumulation of residential and work-related exposures and places with less opportunity for recovery from daily stressors (lack of restoration). It may also be fruitful to study differential effects of noise from a more contextual viewpoint and take life course and life phase related aspects into account. This includes looking at health effects of noise in groups based on e.g. socio-economic status, working situations and places as well as looking at specific susceptibility for noise during the life stages and an accumulation of risk during the life course.

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