Prevention

Cardiovascular effects of environmental noise exposure

Thomas Münzel1*, Tommaso Gori1, Wolfgang Babisch2, and Mathias Basner3

1 II Medizinische Klinik für Kardiologie, University Medical Center Mainz, Mainz, Germany; 2 Department of Environmental Hygiene, Federal Environment Agency, Dessau/Berlin, Germany; and 3 Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA

Received 10 November 2013; revised 7 January 2014; accepted 13 January 2014

The role of noise as an environmental pollutant and its impact on health are being increasingly recognized. Beyond its effects on the auditory system, noise causes annoyance and disturbs sleep, and it impairs cognitive performance. Furthermore, evidence from epidemiologic studies demonstrates that environmental noise is associated with an increased incidence of arterial hypertension, myocardial infarction, and stroke. Both observational and experimental studies indicate that in particular night-time noise can cause disruptions of sleep structure, vegetative arousals (e.g. increases of blood pressure and heart rate) and increases in stress hormone levels and oxidative stress, which in turn may result in endothelial dysfunction and arterial hypertension. This review focuses on the cardiovascular consequences of environmental noise exposure and stresses the importance of noise mitigation strategies for public health.

Keywords Cardiovascular disease • Noise • Pollutants • Sleep • Hypertension • Myocardial infarction • Stroke

Introduction

The Nobel Prize Winner Robert Koch predicted in 1910 that ‘One day man will have to fight noise as fiercely as cholera and pest’. Acutely, noise interferes with communication, disturbs sleep, and causes annoyance. At the same equivalent noise level, annoyance and self-reported sleep disturbance are usually highest for aircraft noise, and higher for road compared with rail traffic noise (Figure 1). Further, long-term exposure to relevant noise levels has been shown to be associated with negative health outcomes. Importantly, an impact on cardiovascular and autonomic homeostasis has been shown, even for noise levels that are quite commonly observed in urbanized regions: it is estimated that ~40% of the population in European Union countries is exposed to road traffic noise at levels exceeding 55 dB of L_{DEN} (‘day–night level’, a measure of noise which summarizes average noise exposure > 24 h, see Table 1) and that >30% is exposed to levels exceeding 55 dB at night (L_{NIGHT}, average noise level during the nighttime hours, usually between 11 p.m. and 7 a.m.).

The health burden of environmental noise has recently been quantified in a report of the World Health Organization (WHO) in terms of disability-adjusted life years (i.e. the number of years lost because of disability or death, a measure that combines both morbidity and mortality). The WHO estimates that—in western Europeans—annually 45 000 years are lost due to noise-induced cognitive impairment in children, 903 000 due to noise-induced sleep disturbance, 61 000 due to noise-induced cardiovascular disease, and 22 000 due to tinnitus. Additionally, while not being a disease per se, noise-induced annoyance decreases quality of life and thus also causes disability, quantified in 587 000 disability-adjusted life years lost in the western European population.

The present narrative review by subject matter experts is based on the current literature on the mechanisms and impact of noise on the cardiovascular system, will describe such non-auditory effects, with a particular focus on mechanisms and epidemiology of noise-induced arterial hypertension, acute myocardial infarction, and stroke.

Physiological responses to acute noise

Noise exposure modifies the function of multiple organs and systems. Acute noise exposure, in both laboratory settings where traffic noise was simulated and in real-life environments, can cause increases in blood pressure, heart rate, and cardiac output, likely mediated by the release of stress hormones such as catecholamines (for review, see Babisch). As shown by field studies, these acute

DOI: 10.1093/eurheartj/ehu030

© The Author 2014. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

Figure 1. Night-time hours, usually between 11 p.m. and 7 a.m.)

Table 1. Further, long-term exposure to relevant noise levels has been shown to be associated with negative health outcomes. Importantly, an impact on cardiovascular and autonomic homeostasis has been shown, even for noise levels that are quite commonly observed in urbanized regions: it is estimated that ~40% of the population in European Union countries is exposed to road traffic noise at levels exceeding 55 dB of L_{DEN} (‘day–night level’, a measure of noise which summarizes average noise exposure > 24 h, see Table 1) and that >30% is exposed to levels exceeding 55 dB at night (L_{NIGHT}, average noise level during the nighttime hours, usually between 11 p.m. and 7 a.m.).

The health burden of environmental noise has recently been quantified in a report of the World Health Organization (WHO) in terms of disability-adjusted life years (i.e. the number of years lost because of disability or death, a measure that combines both morbidity and mortality). The WHO estimates that—in western Europeans—annually 45 000 years are lost due to noise-induced cognitive impairment in children, 903 000 due to noise-induced sleep disturbance, 61 000 due to noise-induced cardiovascular disease, and 22 000 due to tinnitus. Additionally, while not being a disease per se, noise-induced annoyance decreases quality of life and thus also causes disability, quantified in 587 000 disability-adjusted life years lost in the western European population.

The present narrative review by subject matter experts is based on the current literature on the mechanisms and impact of noise on the cardiovascular system, will describe such non-auditory effects, with a particular focus on mechanisms and epidemiology of noise-induced arterial hypertension, acute myocardial infarction, and stroke.

Physiological responses to acute noise

Noise exposure modifies the function of multiple organs and systems. Acute noise exposure, in both laboratory settings where traffic noise was simulated and in real-life environments, can cause increases in blood pressure, heart rate, and cardiac output, likely mediated by the release of stress hormones such as catecholamines (for review, see Babisch). As shown by field studies, these acute

* Corresponding author. Tel: +49 6131 17 7250, Fax: +49 6131 17 6615, Email: tmuenzel@uni-mainz.de

© The Author 2014. Published by Oxford University Press on behalf of the European Society of Cardiology.
effects occur not only at high sound levels in occupational settings but also at relatively low environmental noise levels when concentration, relaxation, or sleep is disturbed.6

The model describing the generalized psychophysiological reactions to stress formulated in 1977 by Henry and Stephens can be applied to noise.5,7,8 In this model, any form of stress (or any stimulus that is felt as such) provokes the activation of two different neuro-hormonal systems which ultimately help to cope with the stressor or at least to limit its damages.6,28 These reactions include the activation of sympathetic responses (fight–flight reactions) as well as the release of corticosteroids (defeat reaction). Particularly intense noise stimuli (for instance, when healthy volunteers are exposed to noise simulating a military low-altitude flight or a racing car), especially when their content appears aggressive or frightening, trigger the fight–flight reaction, with the secretion of adrenalin and noradrenalin from the adrenal medulla.9 The effects of this sympathetic arousal would help the organism to remove the stressor by actively confronting the problem or fleeing from it. Further, high-level noise events beyond the pain threshold and frightening sounds at lower levels also increase plasma cortisol levels,9 a so-called defeat reaction aimed at mitigating the damages expected from the stressor. Notably, such changes do not require the involvement of cortical structures, i.e. the cognitive perception of noise does not appear to be necessary for its effects on cardiovascular homeostasis to become manifest. Indeed, the activation of fight–flight and defeat reactions is thought to involve subcortical regions of the brain like the hypothalamus, which has inputs to the autonomic nervous system, the endocrine system, and the limbic system.10 Such stress responses, in turn, result in changes in a number of physiological functions and in the homeostasis of several organs, including blood pressure, cardiac output, blood lipids (cholesterol, triglycerides), free fatty acids, plasma proteins and hormones, and others.11 Such neuro-hormonal systems which ultimately help to cope with the stressor or at least to limit its damages.6

Table 1 Noise metrics and their definition

<table>
<thead>
<tr>
<th>Metric</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SPL</strong></td>
<td>The sound pressure level (SPL) is a logarithmic measure of the effective pressure of a sound relative to a reference value. It is measured in decibels (dB, see below) higher than a reference level. The reference sound pressure in air is 20 μPa (2 × 10⁻⁶ Pa), which is equivalent to the human hearing threshold at a sound frequency of 1000 Hz.</td>
</tr>
<tr>
<td><strong>dB</strong></td>
<td>A logarithmic scale to measure sound pressure levels.</td>
</tr>
<tr>
<td><strong>L₁₆ₐₗ₎</strong></td>
<td>The energy-equivalent average A-weighted sound pressure level (L₁₆ₐₗ₎) expressed in decibels is the most commonly used noise exposure metric reflecting (energetically) averaged noise exposure over a certain time period. The A-weighting (represented by the A in L₁₆ₐₗ₎) accounts for the different sensitivity of the human ear at different sound frequencies. The duration of the averaging period within the 24 h day is often amended (e.g., L₁₆ₐₗ₎ 16 h, usually reflecting the period from 7 a.m. to 11 p.m.). The L₁₆ₐₗ₎ is often calculated for long periods (e.g., over one year, the busiest 6 months of the year, etc.).</td>
</tr>
<tr>
<td><strong>Lₙιₙτₐ</strong></td>
<td>Weighted L₁₆ₐₗ₎ for the night period (usually 11 p.m. and 7 a.m.).</td>
</tr>
<tr>
<td><strong>L₉₅ₐₗ₅</strong></td>
<td>Weighted L₁₆ₐₗ₎ over a 24 h period with a 10 dB penalty for nocturnal noise exposure (usually 11 p.m. to 7 a.m.).</td>
</tr>
<tr>
<td><strong>L₉₅ₐₗ₅ₙ</strong></td>
<td>The 24 h L₁₆ₐₗ₎ with a 5 dB penalty for the evening (usually 6 p.m. to 10 p.m. or 7 p.m. to 11 p.m.) and a 10 dB penalty for the night (usually 10 p.m. to 6 a.m. or 11 p.m. to 7 a.m.). The penalties are introduced to indicate people’s extra sensitivity to noise during the evening and the night. With respect to long-term health effects, these metrics are usually calculated as average annual exposure indicators.</td>
</tr>
<tr>
<td><strong>L₉₅ₐₗ₅ₚₖₚₖ</strong></td>
<td>Maximum noise level in a given time period (during the passing of a train). L₉₅ₐₗ₅ₚₖₚₖ is often better at predicting acute effects of single noise events than average noise levels. With respect to long-term health effects, however, equivalent sound levels seem more appropriate exposure metrics.</td>
</tr>
</tbody>
</table>

The energy-equivalent average A-weighted SPL (L₁₆ₐₗ₎) as expressed in decibels is the most commonly used indicator of the noise exposure that people perceive outside and inside their homes. The A-weighting accounts for the different sensitivity of the human ear at different sound frequencies.

Experimental laboratory studies, observational field studies, and epidemiological studies all play important roles in elucidating the relationship between noise exposure and adverse health effects.3,4,29,50 Such neuro-hormonal systems which ultimately help to cope with the stressor or at least to limit its damages.6

Figure 1 Percentage of persons highly annoyed by aircraft, road, and rail traffic noises. The curves were derived for adults on the basis of surveys (26 for aircraft noise, 19 for road noise, and 8 for rail noise) distributed over 11 countries. Adapted from Miedema and Oudshoorn.1
effects of environmental noise on cardiovascular health. Figure 2 shows a proposed reaction scheme for the effects of noise on the organism (from Sørensen et al. and Schmidt72,95). As described above, noise may exert its effects either directly, through synaptic interactions, or indirectly, through the emotional and the cognitive perception of sound. In other words, both the objective noise exposure (sound level) and its subjective perception determine the impact of noise on neuroendocrine homeostasis.

Effects of nocturnal noise exposure on sleep and the cardiovascular system

Sleep is a complex and very active process, incorporating many vital physiological processes (e.g. protein biosynthesis, excretion of specific hormones, memory consolidation) that, in a broad sense, serve recuperation and preparation for the next wake period. Acute and chronic sleep restriction or fragmentation has been shown to be associated with inadequate pancreatic insulin secretion,13 decreased insulin sensitivity,14 changes in appetite regulating hormones,15 and increased sympathetic tone and venous endothelial dysfunction.16 At the same time, epidemiologic studies have shown that habitual short sleep (<6 h per night) is associated with obesity,17,18 diabetes,18,19 hypertension,20 cardiovascular disease,21 and all-cause mortality,22,23 stressing the importance of undisturbed sleep of sufficient length for health in general and cardiovascular health specifically. For these reasons, sleep disturbance is usually considered the most severe non-auditory effect of environmental noise exposure.24,25

The Ascending Reticular Activating System is part of the body’s arousal system. It receives input from several sensory systems (including the auditory) and relays this information, for instance, to cardio-respiratory brainstem networks and through the Thalamus to the Cortex. Thus, we recognize, evaluate, and react to environmental sounds even while asleep.26 The Thalamus has a gating function, i.e. based on the sensory information and the current central nervous system state, information may be relayed to or withheld from the Cortex.27 If the information is passed on to the Cortex, it may lead to a Cortical arousal, that, if the subject is sleeping,
Cardiovascular effects of nocturnal noise

In a field study, nocturnal aircraft noise exposure played back with loudspeakers in the subjects’ bedrooms was shown to dose dependently affect parameters of vascular function (Figure 3), including endothelial function as measured by flow-dependent dilation of the brachial artery. Although this study was limited to single-night exposures to different levels of noise, a priming effect of noise was detected, i.e. the effect of noise was more pronounced if the subject had previously been exposed to noise. As expected, the changes in arterial function were paralleled by increased catecholamine production and impaired sleep quality, but morning plasma levels of cortisol and inflammatory markers IL-6 and C-reactive protein were unaffected by noise exposure.

These findings provide biologic plausibility for an association between nocturnal noise exposure and cardiovascular health. Increasingly, epidemiologic studies indicate that nocturnal noise exposure may be more relevant for cardiovascular health than day-time noise exposure (for a detailed discussion of epidemiologic studies and both day- and night-time noise exposure, see below). For aircraft noise, the HYENA study (‘Hypertension and Exposure to Noise near Airports’) found no significant association for day-time noise, but a significant increase in blood pressure with increases in night noise.23,43 Compatible with this evidence, it has been demonstrated that road traffic noise exposure has a larger impact in those who sleep with open windows or whose bedroom faces the road.44 A sustained decrease in blood pressure during the night (so-called dipping) seems to be important for resetting the cardiovascular system and for long-term cardiovascular health.45 Repeated nocturnal autonomic arousals may prevent blood pressure dipping and contribute to the risk for developing hypertension in those exposed to relevant levels of environmental noise for prolonged periods of time.46,47 In line with this, it was found that the risk to develop hypertension was higher in those sleeping with open windows during the night, but it was lower in those with sound insulation or where the bedroom was not facing the main road.48 A recent Swiss study showed an adverse effect of railway noise on blood pressure, that was more strongly associated with night-time exposure.49

The Night Noise Guidelines for Europe were published by the WHO in 2009 and constitute an expert consensus correlating four noise exposure ranges to negative health outcomes ranging from ‘no substantial biological effects’ to ‘increased risk of cardiovascular disease’.50 The WHO considers average nocturnal noise levels of \( L_{Aeq, outside} \geq 55 \text{ dB} \) as an interim goal when the recommended guideline value of 40 dB is not feasible in the short term for the prevention of noise-induced health effects.51

In sum, nocturnal noise has been shown to affect both autonomic regulation (via increases in heart rate mediated by sympathetic activation and/or parasympathetic withdrawal51–53 and with increases in blood pressure54) and, directly, vascular function through the induction of endothelial dysfunction. Importantly, both endothelial dysfunction and reduced heart rate variability have been demonstrated to have prognostic value in patients with peripheral artery disease, arterial hypertension, and patients with an acute coronary syndrome or chronic stable coronary artery disease.55–57
together, these observations provide a biological rationale for the increased incidence of arterial hypertension, myocardial infarction, and stroke in subjects with long-term exposure to relevant noise levels as described in the following paragraphs.

### Epidemiological studies

Studies on chronic exposure to road traffic and/or railway or aircraft noise have reported a relationship with elevated blood pressure, hypertension or the use of antihypertensive medication, ischaemic heart disease including fatal myocardial infarction, stroke, dementia, and diabetes mellitus. Different study designs and methods for the assessment of the exposure, the disease, and potentially confounding factors were used. Based on these differences, the strength of the associations varies significantly across studies. Overall, however, studies demonstrate that environmental noise carries a health burden that has medical and economic implications: in the UK, day-time noise levels of ≥55 dB have been estimated to cause an additional 542 cases of hypertension-related myocardial infarction, 788 cases of stroke, and 1169 cases of dementia, with a cost valued at around £1.09 billion annually.58

### Road traffic noise, blood pressure, and hypertension

A meta-analysis of 24 cross-sectional studies on the relationship between road traffic noise and the prevalence of hypertension reported an odds ratio (OR) of 1.07 (95% confidence interval (CI) = 1.02–1.12, P < 0.05) per 10 dB increase of the 16-h day-time average road traffic noise level ($L_{Aeq16h}$) in the range of <50 to >75 dB.59 A certain degree of heterogeneity among

---

**Figure 3** Effects of simulated aircraft noise (noise 30 and 60 reflecting 30 or 60 playback aircraft noise events) on endothelial function (as measured by flow-mediated dilation) and (lower right) stress hormone levels of healthy volunteers (adapted from Schmidt et al.95). The administration of the antioxidant vitamin C (upper right) was associated with improved endothelial function, demonstrating a role of oxidative stress.
Aircraft noise and arterial hypertension

In 2001, an increased prevalence of arterial hypertension in the vicinity of Stockholm airport was reported. Similarly, a dose–response relationship has been shown in the HYENA study with respect to night-time noise. A 14% increase in OR (CI = 1.01–1.29; P < 0.05) for arterial hypertension was in this study associated with every 10 dB increase in L_{night}; in contrast, no effect was found for day-time aircraft noise exposure (L_{day}: OR = 0.93; CI = 0.83–1.04; P = n.s.). Data from the European Union-funded RANCH (Road Traffic and Aircraft Noise Exposure and Children’s Cognition and Health) study reported an association between both day-time and nocturnal noise exposure at home and blood pressure values in 9- to 10-year-old children living near Schiphol (Amsterdam) or Heathrow (London). A meta-analysis of four cross-sectional and one cohort study on the relationship between air traffic noise and the prevalence of hypertension reported an OR of 1.13 (CI = 1.00–1.28; P < 0.05) per 10 dB increase of the day–night-weighted noise level L_{DEN} in the range of <55 to >65 dB. Studies carried out repeatedly in the area neighbouring Amsterdam’s Schiphol airport reported a higher prevalence of prescriptions for cardiovascular medications (OR ranging between 1.2 and 1.4 between high and low noise groups). Likewise, a cross-sectional study from the Cologne airport region in Germany demonstrated higher individual rates of cardiovascular medicine prescriptions in residents exposed to high aircraft noise levels, particularly during the night and the early morning hours (3–5 h). Higher risks were found for subjects for whom the average noise level during the late night period exceeded 40 dB. Results from the HYENA study also suggest an effect of aircraft noise on the use of antihypertensive medication, but this effect did not hold for all participating study centers. Results were more consistent across centres for the increased use of anxiolytics in relation to aircraft noise.

Aircraft noise and coronary heart disease

The relationship between aircraft noise and coronary heart disease is less well investigated so far. Earlier studies carried out around Amsterdam’s Schiphol airport gave some hints of a higher risk of cardiovascular disorders in subjects that lived closer to the airport. More recent studies, however, provided evidence of a higher cardiovascular risk for subjects who reside near airports. In an ecological study carried out around Heathrow airport, London, an increased risk of stroke and coronary heart disease was reported in relation to day- and night-time exposure to aircraft noise in people that were more exposed to aircraft noise than others. This exposure-dependent relationship was adjusted for aggregated data regarding ethnicity, social deprivation, incidence of lung cancer as a proxy for smoking, road traffic noise exposure, and air pollution. In another new census-block-based ecological study carried out around 89 North American airports using hospital admission registry data, it was
found that persons living in areas at the 90th percentile of noise exposure among census blocks within zip codes had a higher risk of hospitalization for ischaemic heart disease and cerebrovascular disease. The results were adjusted for age, sex, and race as well as area level of socioeconomic status and ethnicity. A large cohort study in Switzerland reported an increased mortality due to myocardial infarction with increasing exposure levels and duration of aircraft noise, with a non-significant hazard ratio of 1.3 when persons exposed to noise levels ($L_{DEN}$) ≥ 60 dB compared with those exposed to < 45 dB after adjustment for several individual and geographical variables, including air pollution. As mentioned above, this hazard ratio increased to 1.5 (CI = 1.0–2.2; $P < 0.10$) and was significant when only residents exposed to noise for at least 15 years were included. In contrast, none of the other endpoints, including all-cause mortality, cerebrovascular disease, and stroke, was associated with aircraft noise. Furthermore, with increasing exposure to noise, the proportion of persons with tertiary education declined, whereas the proportion unemployed, the proportion of foreign nationals, and the proportion of people living in old and unrenovated buildings increased. Finally, in the cross-sectional HYENA study an association between (self-reported) heart disease or stroke (combined endpoint) and nocturnal aircraft noise was described. The association was particularly strong in subjects that had lived in the same place for ≥ 20 years. An OR of 1.25 (CI = 1.03–1.51; $P < 0.05$) per 10 dB increase of the night-noise level ($L_{night}$) was found.

Limitations of research in the area of environmental noise

A number of factors may modify the impact of noise on health. These include exposure-modifying factors such as the location of rooms and the quality of sound insulation, the habit to sleep with open or closed windows and length of residence and other behavioural risk factors. Most of the epidemiological studies controlled their results for a basic set of potentially confounding factors such as age, gender, socioeconomic status, smoking, body mass index, physical activity, alcohol intake, and/or ethnicity. Particularly, those studies where the subjects are actually seen provide a lot of additional information based on clinical interviews. However, response rates are sometimes low, which implies a possible selection bias. Ecological and other studies using health registries, on the other hand, do not suffer from the problem of low response rates, but individual data regarding personal risk factors are often missing. Since many behavioural risk factors that are related with worse health outcomes are correlated with socioeconomic status, they can partly be controlled. Aggregated indices of areal deprivation and socioeconomic status may serve as surrogates if individual data are not available. The fact that noise effects were seen when different methods and study types were applied supports the reasoning for an association. For example, cohort and case–control studies have a high analytic power, but observational cross-sectional studies may also provide information on relationships. Differences in study type (prospective, case–control, cross-sectional, ecologic) and the degree and quality of adjustment for confounding (i.e. residual confounding) may explain some of the discrepancies found between individual epidemiological studies.

The fact that studies often only find noise associations with some but not all of their cardiovascular endpoints, and that not everybody will be affected (e.g. only men, only those within a certain age range) stresses the need to further investigate the exact mechanisms of how noise affects the cardiovascular system and why some are more susceptible than others. Of note, the question of what comes first (temporality) may be easier to address with regards to noise epidemiology as compared with other associations between suspected risk factors and disease prevalence as it is unlikely that the decision to relocate to noisier areas might be determined by the presence of a disease.

There is evidence that ambient particle concentrations are also associated with the incidence of cardiovascular diseases. The potential confounding effect of air pollution was therefore considered in more recent studies, and, given the stronger association with air pollution, particularly in road traffic noise studies. Complicating these considerations, however, adjustment for air pollution indicators in multivariable statistical models is complex because modelled indicators of both exposures refer to the same input data (e.g. traffic volume and composition, traffic flow and speed, proximity to the road). It is an advantage of noise assessment that meteorology plays less of a role compared with air pollution within urban distances where houses are close to the streets. The fact that noise effects are also seen for noise sources other than road traffic (e.g. aircraft noise, railway noise, occupational noise that do not contribute as much to the air pollution exposure of the dwellings were the people live) supports the concept of an independent effect of noise. This is addressed in the present issue of the European Heart Journal. Using data from the German Heinz Nixdorf Recall Study, a population-based cohort study, the authors have demonstrated independent associations of long-term exposure to both fine particulate matter (PM) and road traffic noise with thoracic aortic calcification, a reliable measure of subclinical atherosclerosis. In this study, PM$_{2.5}$ (PM with an aerodynamic diameter ≤ 2.5 μm) and $L_{night}$ (average level of nocturnal noise), but not $L_{DEN}$, were associated with an increasing coronary calcification-burden, and the association remained valid after mutual adjustment. The authors concluded that long-term exposure to fine PM and night-time traffic noise are both independently associated with subclinical atherosclerosis and may both contribute to the association between road traffic and coronary artery disease. In a recent systematic review, it was concluded that confounding of cardiovascular effects by noise or air pollutants was low, even though this may have to do with the quality of the assessment methods of air pollutants.

Regarding effect modification, the studies showed a tendency towards stronger effects in middle-aged subjects and a tendency towards stronger effects in males. However, this was not consistent across studies. Furthermore, pre-existing co-morbidity was also found to be an effect modifying factor. Finally, a cumulative effect of noise from multiple sources of noise (e.g. work and traffic noise) has been shown.

Conclusions

Taken together, the present review provides evidence that noise not only causes annoyance, sleep disturbance, or reductions in quality of life, but also contributes to a higher prevalence of the most important cardiovascular risk factor arterial hypertension and the incidence of
cardiovascular diseases. The evidence supporting such contention is based on an established rationale supported by experimental laboratory and observational field studies, and a number of epidemiological studies. Meta-analyses have been carried out to derive exposure–response relationships that can be used for quantitative health impact assessments. Noise-induced sleep disturbance constitutes an important mechanism on the pathway from chronic noise exposure to the development of adverse health effects. The results call for more initiatives aimed at reducing environmental noise exposure levels to promote cardiovascular and public health. Recent studies indicate that people's attitude and awareness in particular towards aircraft noise has changed over the years. Noise mitigation policies have to consider the medical implications of environmental noise exposure. Noise mitigation strategies to improve public health include noise reduction at the source, active noise control (e.g., noise-optimized take-off and approach procedures), optimized traffic operations (including traffic curfews), better infrastructural planning, better sound insulation in situations where other options are not feasible, and adequate limit values.

Funding
M.B. was funded through NIH (grant R01 NR004281). T.G. and T.M. received from the Federal Ministry of Education and Research (BMBF01OE1003). T.G. also received from the Robert Müller Foundation, Foundation Heart of Mainz. The authors are responsible for the contents of this publication. Funding to pay the Open Access publication charges for this article was provided by the University Medical Center of Mainz.

Conflict of interest: none declared.

References
Cardiovascular effects of environmental noise exposure


