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Auditory and non-auditory effects of noise on health

[Mathias Basner](#), MD, [Wolfgang Babisch](#), PhD, [Prof. Adrian Davis](#), PhD, [Mark Brink](#), PhD, [Charlotte Clark](#), PhD, [Sabine Janssen](#), PhD, and [Prof. Stephen Stansfeld](#), PhD

Mathias Basner, Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA;

[Contributor Information](#).

Correspondence to: Dr Mathias Basner, Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA 19104–6021, USA, basner@upenn.edu

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Abstract

Noise is pervasive in everyday life and can cause both auditory and non-auditory health effects. Noise-induced hearing loss remains highly prevalent in occupational settings, and is increasingly caused by social noise exposure (eg, through personal music players). Our understanding of molecular mechanisms involved in noise-induced hair-cell and nerve damage has substantially increased, and preventive and therapeutic drugs will probably become available within 10 years. Evidence of the non-auditory effects of environmental noise exposure on public health is growing. Observational and experimental studies have shown that noise exposure leads to annoyance, disturbs sleep and causes daytime sleepiness, affects patient outcomes and staff performance in hospitals, increases the occurrence of hypertension and cardiovascular disease, and impairs cognitive performance in schoolchildren. In this Review, we stress the importance of adequate noise prevention and mitigation strategies for public health.

Introduction

Evolution has programmed human beings to be aware of sounds as possible sources of danger.¹ Noise, defined as unwanted sound, is a pollutant whose effects on health have been neglected, despite the ability to precisely measure or calculate exposure from peak levels or energy averaged over time ([panel 1](#), [figure 1](#)). Although people tend to habituate to noise exposure, degree of habituation differs for individuals and is rarely complete.² If exposure to noise is chronic and exceeds certain levels, then negative health outcomes can be seen. Health effects were first recognised in occupational settings, such as weaving mills, where high levels of noise were associated with noise-induced hearing loss.³ Occupational noise is the most frequently studied type of noise exposure. Research focus has broadened to social noise (eg, heard in bars or through personal music players) and environmental noise (eg, noise from road, rail, and air traffic, and industrial construction). These noise exposures have been linked to a range of non-auditory health effects including annoyance,⁴ sleep disturbance,⁵ cardiovascular disease,^{6,7} and impairment of cognitive performance in children.⁸ The health effects of noise from entertainment venues and from neighbours are elusive, but nevertheless, cause many complaints to local authorities. The meaning attributed to sounds might affect our response to them—eg, the response to aircraft noise might differ between an airport employee and a resident who fears long-term health consequences due to the noise exposure. Noise is pervasive in urban environments

and the availability of quiet places is decreasing. In the European Union, about 56 million people (54%) living in areas with more than 250 000 inhabitants are exposed to road traffic noise of more than average L_{DEN} 55 dB per year, which is thought to be risky to health.⁹ Thus, understanding of occupational and environmental noise is important for public health. In this Review, we summarise knowledge and research related to noise exposure and both auditory and non-auditory health effects.

Panel 1

Glossary of terms used to describe sound

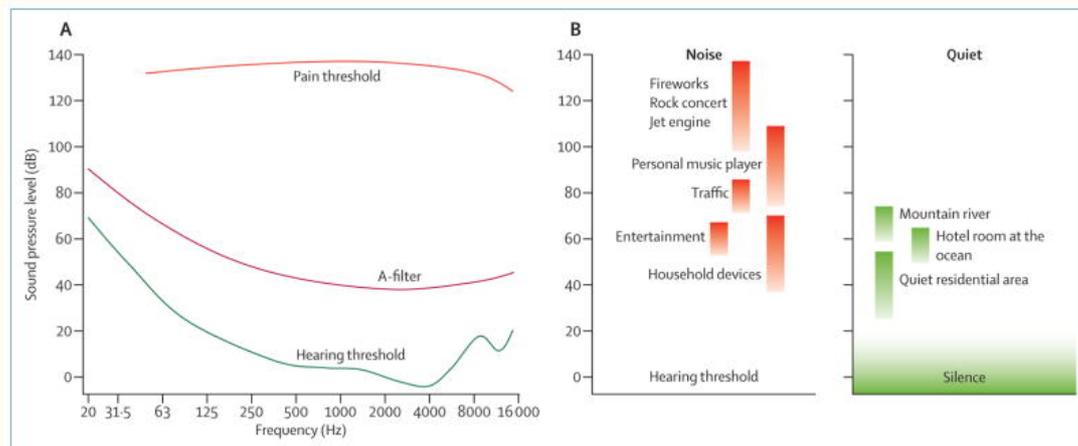
Sound pressure level Sound pressure level is a logarithmic measure of the effective pressure of a sound relative to a reference value. It is measured in decibels (dB, see below) higher than a reference level. The reference sound pressure in air is 20 μPa (2×10^{-5} Pa), which is thought to be the human hearing threshold at a sound frequency of 1000 Hz.

dB scale A logarithmic scale to measure sound pressure level. A two-fold increase in sound energy (eg, two identical jackhammers instead of one) will cause the sound pressure level to increase by 3 dB. A ten-fold increase in sound energy (10 jackhammers) will cause the sound pressure level to increase by 10 dB, which is perceived as about twice as loud.

L_{max} The highest sound pressure level in a given time period.

L_{eq} Average level of sound pressure within a certain time period. If the A-filter is used for frequency-weighting (figure 1), the average level is referred to as L_{Aeq} . The filter and time period used for averaging are often indicated in subscript—eg, L_{Aeq8h} , $L_{Aeq23-7h}$, or L_{night} .

L_{DEN} L_{DEN} (Day-Evening-Night-Level), also referred to as DENL, is the A-filtered average sound pressure level, measured over a 24 h period, with a 10 dB penalty added to the night (2300–0700 h or 2200–0600 h, respectively), and a 5 dB penalty added to the evening period (1900–2300 h or 1800–2200 h, respectively), and no penalty added to the average level in the daytime (0700–1900 h or 0600–1800 h, respectively). The L_{DN} measure is similar to the L_{DEN} , but omits the 5 dB penalty during the evening period. The penalties are introduced to indicate people's extra sensitivity to noise during the night and evening. Both L_{DEN} and L_{DN} are based on A-weighted sound pressure levels, although this factor is not usually indicated in subscript.



[Figure 1](#)

Sound pressure levels

(A) The sensitivity of the auditory system depends on sound frequency and sensitivity is highest between 2000 Hz and 5000 Hz (green line). The A-filter (dark red line) is a frequency-weighting of sound pressure levels that mimics the sensitivity of the auditory system (eg, low-frequency sounds contribute little to the A-weighted dB level). (B) A-weighted sound pressure levels for several environmental sounds, emphasising that whether or not a sound is perceived as noise depends largely on the context and the individual, and is only partly determined by its sound pressure levels. For example, spectators attending a rock concert might not perceive the music as noise, whereas residents in the vicinity of the venue might call it noise, even though sound pressure levels are much lower there than for inside.

Auditory health effects

Noise-induced hearing loss

Noise is the major preventable cause of hearing loss. Noise-induced hearing loss can be caused by a one-time exposure to an intense impulse sound (such as gunfire), or by steady state long-term exposure with sound pressure levels higher than L_A 75–85 dB—eg, in industrial settings. The characteristic pathological feature of noise-induced hearing loss is the loss of auditory sensory cells in the cochlea. Because these hair cells cannot regenerate in mammals, no remission can occur; prevention of noise-induced hearing loss is the only option to preserve hearing. Hearing loss leading to the inability to understand speech in everyday situations can have a severe social effect. It can also affect cognitive performance and decrease attention to tasks. Accidents and falls are also associated with undiagnosed hearing loss, with excess mortality of 10–20% in 20 years.¹⁰

Noise-induced hearing loss is a public health problem. Global Burden of Disease 2010¹¹ estimated that 1.3 billion people are affected by hearing loss and investigators rated hearing loss as the 13th most important contributor (19.9 million years, 2.6% of total number) to the global years lived with disability (YLD). Adult-onset hearing loss unrelated to a specific disease process accounted for 79% of YLD from hearing loss. In the USA and Europe, 26% of adults have a bilateral hearing disorder that impairs their ability to hear in noisy environments, and a further 2% have substantial unilateral hearing issues. Age-adjusted prevalence is similar in Asia.¹² WHO estimates that 10% of the world population is exposed to sound pressure levels that could potentially cause noise-induced hearing loss. In about half of these people, auditory damage can be attributed to exposure to intense noise.¹³

Search strategy and selection criteria

We searched PubMed, Science Citation Index Expanded, and Social Sciences Citation Index, and references from relevant articles for English language articles from Jan 1, 1980, to Feb 1, 2013, using the search terms: “hearing loss”, “tinnitus”, “annoyance”, “cardiovascular disease”, “hypertension”, “high blood pressure”, “myocardial infarction”, “stroke”, “sleep”, “cognitive performance”, “reading ability”, and “hospital”, in combination with “noise”. Each author did their own search, and is also a subject matter expert in their field. We focused on articles published in the past 5 years; however, used older articles if they represent seminal research or are necessary to understand more recent findings. We included reports from recent meetings if we regarded them to be relevant.

Tinnitus—ie, change in sound perception, such as ringing, that cannot be attributed to an external source—often follows acute and chronic noise exposure, and persists in a high proportion of affected individuals for extended periods.¹⁴ Tinnitus can affect quality of life in several ways, including through sleep disturbance, depression, or the inability to sustain attention.¹⁵ The fact that hearing loss and tinnitus are reported in combination suggests that both symptoms share common pathophysiological pathways.

Occupational noise-induced hearing loss

Despite the introduction of standards for hearing protection, reduction in occupational noise exposure in developed countries, and extensive public health efforts, hearing loss induced by exposure to occupational noise remains a dilemma and is the focus of extensive research. Noise-induced hearing is the most common occupational disease in the USA: about 22 million US workers are exposed to hazardous noise levels at work, and, annually, an estimated US\$242 million is spent on compensation for hearing loss disability.¹⁶

Many countries enforce general health and safety legislation that specifies maximum exposure levels and requirements for action, including noise assessments, regular audiometric testing, protective equipment, and monitoring, which are intended to protect both workers and the public from excessive noise exposure. However, the available evidence for associations between occupational noise exposure and hearing loss is complex and its quality varies. Many studies have a lack of appropriate non-exposed controls, and longitudinal studies are scarce. Contributors to a Cochrane collaboration review¹⁷ concluded that “higher quality prevention programs, better quality of studies especially in the field of engineering controls and better implementation of legislation are needed to better prevent noise-induced hearing loss”. This Review also indicated that current efforts for hearing loss prevention focus on hearing protection rather than on noise control.

The exact level of noise exposure in industrial settings that carries risk of hearing damage is debated internationally. For example, in the UK, the Control of Noise at Work Regulations (2005)¹⁸ set levels for action at L_{Aeq8h} 80 dB (protection made available) and 85 dB (protection mandatory). A 3 year follow-up investigation of 19 UK companies that had varying degrees of compliance reported that these values were safe.¹⁹ However, studies with a longer follow-up are needed to lend support to these findings. The US Occupational Safety and Health Administration (OSHA) promotes less strict standards than do others and sets the permissible exposure limit at L_{Aeq8h} 90 dB. However, according to OSHA regulations, employers have to implement a hearing conservation programme if workers are exposed to levels higher than L_{Aeq8h} 85 dB. Although noise-induced hearing loss is well recognised in industrial settings, individuals with other occupations such as musicians^{20,21} or those working for the military,^{22,23} also contribute substantially to the overall burden of noise-induced hearing loss.

Social noise exposure

Excessive noise is often accepted as part of the recreational environment. Although occupational noise has decreased since the early 1980s, the number of young people with relevant degrees of social noise exposure has tripled in the same period.²⁴ A growing body of work is assessing the risk of hearing loss in adolescents due to personal music player use.²⁵ In one study, 66% of young adults attending

nightclubs or rock concerts in the Nottingham area of England reported temporary auditory effects or tinnitus.²⁴ Prospective cohort studies like OHRKAN²⁶ are needed to conclude whether widespread exposure to loud music in adolescence increases the prevalence of hearing loss and tinnitus in older ages. Both safer products and public health campaigns are needed to reduce the risk of hearing loss from personal music player use. Noise-cancelling headphones are effective preventive measures for reducing hazards for users of personal music players.²⁷

Noise-induced hearing loss and age

Noise-induced hearing loss is determined by noise exposure and life-course events, all age groups can be affected. Exposure to different types of noise from early childhood might have cumulative effects on hearing impairment in adulthood. Evidence is increasing that early social and biological factors might affect hearing in middle age (eg, a study of patients assessed at age 45 years²⁸). Prevalence of hearing loss is highly related to age.²⁹ How noise and age interact is a major gap in the specialty's knowledge. Data suggest that pathological but sublethal changes from early noise exposure substantially increase risk of inner ear ageing and related hearing loss.^{30,31} In addition to noise, factors such as alcohol and tobacco use and hyperglycaemia are associated with age-related hearing loss. Thus, public health initiatives need to address both general health and auditory health.

Scientific advances and therapeutic strategies

In the past 5 years, several studies and advances have improved understanding of the causes and factors affecting susceptibility to noise-induced hearing loss. Noise-induced hearing loss is widely accepted to be a symptom of a complex disease that results from the interaction of genetic and environmental factors. Heritability might explain up to 50% of hearing loss variability in individuals after exposure to noise, but definitive studies are needed. Identification of susceptibility genes might help to identify the population at high risk and improve targeted hearing protection in predisposed individuals.³² Much progress has been made in the understanding of molecular mechanisms involved in hair-cell and nerve damage. Recent research from investigators using stem cells to recover the damaged sensory circuitry in the cochlea is at a very early stage, but could lead to potential therapeutic strategies.³³ Attention is increasing on the risks of combined exposure to high-level noise and ototoxic drugs, which can affect the structures of the inner ear and the auditory nerves.^{34,35} A small but substantial number of people have hearing loss as a complication of cancer treatments such as cisplatin, which might be further exacerbated by high levels of noise (eg, in MRI scanners).

Several therapeutic avenues have been recently explored, and oral drugs to protect against noise-induced hearing loss are expected to become available in the next 10 years.¹³ Investigators have reported that oxidative stress could contribute to cochlear cell damage; antioxidant compounds, such as glutathione, have improved noise-induced hearing loss in animals and might prevent noise-induced hearing loss.^{36,37} An oral otoprotective drug, D-methionine, prevents noise-induced hearing loss in animals even when first given within hours after a noise exposure; however, only formal clinical trials will provide the data needed to assess safety and efficacy in human beings.³⁸ Clinical trials of D-methionine in the US Army, funded by the US Department of Defense, are scheduled to begin soon (NCT01345474).³⁸

Diagnosis of noise-induced hearing loss

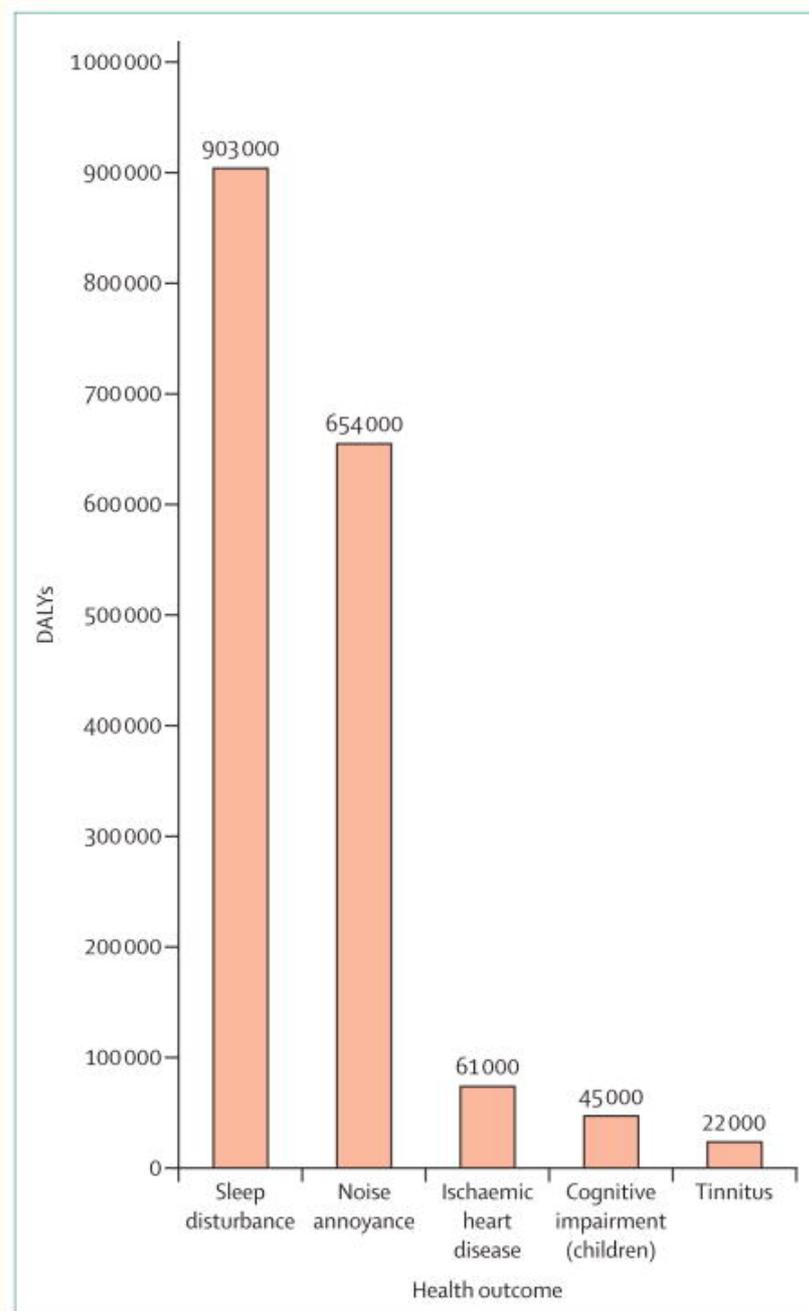
The development of otoacoustic emission testing has been an important technological advance in audiological assessment. Otoacoustic emissions are a release of acoustic energy from the cochlea that can be recorded in the ear canal. Otoacoustic emission testing is used to identify hearing defects in newborn babies and young children. Hall and Lutman³⁹ reported that otoacoustic emission testing was twice as sensitive as was audiometry to detect a change in hearing threshold level and suggested that it could improve monitoring for noise-induced hearing loss in the workplace. A longitudinal study⁴⁰ also suggested that otoacoustic emissions could indicate noise-induced changes in the inner ear undetected by audiometric tests. Otoacoustic emissions might therefore be a superior diagnostic predictor for

noise-induced hearing loss, but further longitudinal studies are needed to show whether otoacoustic emission testing can replace standard audiometry or whether the two techniques have complementary roles.¹⁹

Non-auditory health effects

Introduction

The most investigated non-auditory health endpoints for noise exposure are perceived disturbance and annoyance, cognitive impairment (mainly in children), sleep disturbance, and cardiovascular health. WHO estimated that in high-income western European countries (population about 340 million people), at least 1 million healthy life-years (disability-adjusted life-years) are lost every year because of environmental noise ([figure 2](#)).¹⁴



[Open in a separate window](#)

Figure 2

DALYs attributed to environmental noise exposure in Europe

According to WHO,¹⁴ more than 1 million healthy life years (DALYs) are lost annually because of environmental noise exposure in European A-member states alone. Most of these DALYs can be attributed to noise-induced sleep disturbance and annoyance. DALYs=Disability-adjusted life years.

Annoyance

Annoyance is the most prevalent community response in a population exposed to environmental noise. Noise annoyance can result from noise interfering with daily activities, feelings, thoughts, sleep, or rest, and might be accompanied by negative responses, such as anger, displeasure, exhaustion, and by stress-related symptoms.⁴¹ In severe forms, it could be thought to affect wellbeing and health, and because of the high number of people affected, annoyance substantially contributes to the burden of disease from environmental noise (figure 2).¹⁴ Investigators have proposed standardised questions

about residents' long-term annoyance in their home for use in surveys.⁴² Additionally, investigators have gathered substantial data for community annoyance in residents exposed to noise in their home, based on which exposure–response relationships were derived (eg, for wind turbines).^{4,43,44} These relations can be used in strategic or health impact assessments for estimating long-term annoyance in fairly stable situations. Although the overall community response depends on societal values and is most relevant to the guidance of policy, several personal (eg, age and noise sensitivity) and situational characteristics (eg, dwelling insulation) might affect the individual degree of annoyance.^{41,44}

Cardiovascular disease

Both short-term laboratory studies of human beings and long-term studies of animals have provided biological mechanisms and plausibility for the theory that long-term exposure to environmental noise affects the cardiovascular system and causes manifest diseases (including hypertension, ischaemic heart diseases, and stroke).⁴⁵ Acute exposure to different kinds of noise is associated with arousals of the autonomic nervous system and endocrine system.⁴⁶ Investigators have repeatedly noted that noise exposure increases systolic and diastolic blood pressure, changes heart rate, and causes the release of stress hormones (including catecholamines and glucocorticoids).⁴⁵ The general stress model is the rationale behind these reactions. Potential mechanisms are emotional stress reactions due to perceived discomfort (indirect pathway), and non-conscious physiological stress from interactions between the central auditory system and other regions of the CNS (direct pathway). The direct pathway might be the predominant mechanism in sleeping individuals, even at low noise levels.

Chronic exposure can cause an imbalance in an organism's homeostasis (allostatic load), which affects metabolism and the cardiovascular system, with increases in established cardiovascular disease risk factors such as blood pressure, blood lipid concentrations, blood viscosity, and blood glucose concentrations.^{45,47} These changes increase the risk of hypertension, arteriosclerosis, and are related to severe events, such as myocardial infarction and stroke. Studies of occupational^{48–50} and environmental^{7,51–53} epidemiology have shown a higher prevalence and incidence of cardiovascular diseases and mortality in highly noise-exposed groups. The risk estimates for occupational noise at ear-damaging intensities tend to be higher than are those for environmental noise (at lower noise levels). Because of different acoustic characteristics for different noise sources (sound level, frequency spectrum, time course, sound level rise time, and psycho-acoustic measures) noise levels from different noise sources cannot be merged into one indicator of decibels. Different exposure–response curves are needed for different noise sources. Meta-analyses were done to quantitatively assess the exposure–response link for transportation noise (exposure to road traffic and aircraft noise) and health effects (hypertension and ischaemic heart diseases, including myocardial infarction).^{6,54,55} The investigators derived increases in risk of between 7% and 17% per 10 dB increase in equivalent noise level L_{Aeq} (figure 3). Their results have been adjusted for known risk factors such as age, sex, socioeconomic status, smoking, body-mass index, and others. The researchers identified sex and age as effect modifiers. Studies of the combined effects of noise and air pollution showed largely independent effects,^{7,51–53} which can be explained by different mechanisms of how both exposures can affect health (cognitive and autonomic stress response vs inflammatory processes).

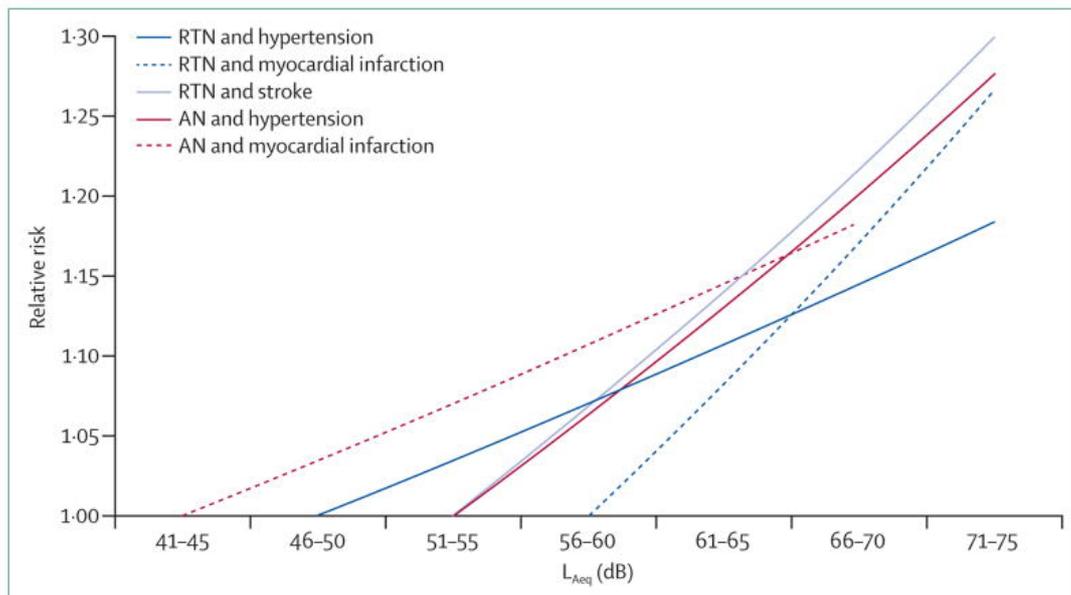


Figure 3

Exposure–response curves of road and aircraft noise and cardiovascular endpoints

RTN and hypertension (24 studies, noise indicator L_{Aeq16h}); RTN and myocardial infarction (five studies, noise indicator L_{Aeq16h}); RTN and stroke (one study, noise indicator L_{DEN}); AN and hypertension (five studies, noise indicator L_{DN}); and AN and MI (one study, noise indicator L_{DN}). RTN=road traffic noise. AN=aircraft noise.

Cognitive performance

WHO estimate that about 45 000 disability-adjusted life-years are lost every year in high-income western European countries for children aged 7–19 years because of environmental noise exposure ([figure 2](#)).¹⁴ Postulated mechanisms for noise effects on children’s cognition include communication difficulties, impaired attention, increased arousal, learned helplessness, frustration, noise annoyance, and consequences of sleep disturbance on performance.^{3,56} Investigators have also suggested psychological stress responses as a mechanism because children are poor at appraising threats from stressors and have less well developed coping strategies than do adults.³ Areas with high levels of environmental noise are often socially deprived, and children from areas with high social deprivation do worse on tests of cognition than do children not exposed to social deprivation. Therefore, measures of socioeconomic position should be taken into account in the assessment of associations between noise exposure and health and cognition.

More than 20 studies have shown environmental noise exposure has a negative effect on children’s learning outcomes and cognitive performance,⁵⁷ and that children with chronic aircraft, road traffic, or rail noise exposure at school have poorer reading ability, memory, and performance on national standardised tests than do children who are not exposed to noise at school.^{58–60} Investigators have examined exposure–effect links between noise exposure and cognition to identify the exposure level at which noise effects begin.^{61,62} The RANCH study of 2844 children aged 9–10 years attending 89 schools around Heathrow (London, UK), Schiphol (Amsterdam, the Netherlands), and Madrid-Barajas (Spain) airports showed a linear exposure–effect relation between aircraft noise exposure at school and a child’s reading comprehension and recognition memory after adjusting for a range of socioeconomic factors.^{61,62} A L_{Aeq} 5 dB increase in aircraft noise exposure was associated with a 2 month delay in reading age in children in the UK and a 1 month delay in those in the Netherlands. These linear associations suggest that there is no threshold for effects and any reduction in noise level at school should improve a child’s cognition.

WHO Community Noise Guidelines⁶³ suggest that the background sound pressure level should not exceed L_{Aeq} 35 dB during teaching sessions. Intervention studies and natural experiments have shown that reductions in noise exposure from insulation or the closure of airports are associated with improvements in cognition, suggesting that noise reduction can eliminate noise effects on cognition.^{58,59}

Sleep disturbance

Sleep disturbance is thought to be the most deleterious non-auditory effect of environmental noise exposure (figure 2), because undisturbed sleep of a sufficient length is needed for daytime alertness and performance, quality of life, and health.^{5,14} Human beings perceive, evaluate, and react to environmental sounds, even while asleep.⁶⁴ Maximum sound pressure levels as low as L_{Amax} 33 dB can induce physiological reactions during sleep including autonomic, motor, and cortical arousals (eg, tachycardia, body movements, and awakenings).^{5,65} Whether noise will induce arousals depends not only on the number of noise events and their acoustical properties,² but also on situational moderators (such as momentary sleep stage⁶⁶) and individual noise susceptibility.⁶⁴ Elderly people, children, shift-workers, and people with a pre-existing (sleep) disorder are thought of as at-risk groups for noise-induced sleep disturbance.⁵ Repeated noise-induced arousals interfere with sleep quality through changes in sleep structure, which include delayed sleep onset and early awakenings, reduced deep (slow-wave) and rapid eye movement sleep, and an increase in time spent awake and in superficial sleep stages.^{2,66} However, these effects are not specific for noise,⁶⁷ and generally less severe than those in clinical sleep disorders such as obstructive sleep apnoea.⁶⁸ Short-term effects of noise-induced sleep disturbance include impaired mood, subjectively and objectively increased daytime sleepiness, and impaired cognitive performance.^{69,70} Results of epidemiological studies indicate that nocturnal noise exposure might be more relevant for the creation of long-term health outcomes such as cardiovascular disease than is daytime noise exposure,⁷¹ probably because of repeated autonomic arousals that have been shown to habituate to a much lesser degree to noise than other—eg, cortical—arousals.² In 2009, WHO published the Night Noise Guidelines for Europe, an expert consensus mapping four noise exposure groups to negative health outcomes ranging from no substantial biological effects to increased risk of cardiovascular disease (panel 2).⁷² WHO regards average nocturnal noise levels of less than $L_{Aeq, outside}$ 55 dB to be an interim goal and 40 dB a long-term goal for the prevention of noise-induced health effects.

Panel 2

WHO definitions of health effects of different average night noise levels⁷²

Below 30 dB $L_{Aeq, night, outside}$ Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{A, eq, night, outside}$ of 30 dB is equivalent to the no observed effect level (NOEL) for night noise.

30–40 dB $L_{Aeq, night, outside}$ A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill, and elderly people) are more susceptible. However, even in the worst cases the effects seem modest. $L_{A, eq, night, outside}$ of 40 dB is equivalent to the lowest observed adverse effect level (LOAEL) for night noise.

40–55 dB $L_{Aeq, night, outside}$ Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.

Above 55 dB $L_{Aeq, night, outside}$ The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

Hospital noise

Although most environmental noise guidelines list hospitals as noise-sensitive facilities, studies of external (eg, traffic) noise effects on hospital environments are very rare. However, research on the understanding and prevention of indoor hospital noise effects on patients and staff has been increasing. An extensive meta-analysis of hospital sound levels indicated that hospital noise has increased by about L_{Aeq} 10 dB since the 1960s.⁷³ Noise levels in hospitals are now typically more than L_{Aeq} 15–20 dB higher than those recommended by WHO.⁶³ Hospital noise could therefore be an increasing threat to patient rehabilitation and staff performance.

The sound environment in hospitals, especially in intensive care units, can be characterised by irregularly occurring noises from sources such as medical devices (eg, alarms), telephones or pagers, conversations, door sounds, and nursing activities. Such noise worsens patient health outcomes through factors such as increased cardiovascular stress, longer healing times, increases in doses of pain-relief drugs, and increased patient readmission rates.⁷⁴ Neonates, long-term patients, and elderly people are thought to be particularly at-risk to the effects of noise. Sleep disruption is the most common noise-related patient complaint.^{75,76} Researchers of a sleep laboratory study developed arousal probability curves for 14 noises typically encountered in hospitals.⁷⁷ The most disturbing noises were intravenous pump alarms and telephone rings, which are intentionally designed to alert staff members.

Evidence of negative effects of noise on hospital staff is increasing, particularly for nurses, with noise-induced stress linked to burnout, diminished wellbeing, and reduced work performance.⁷⁸ Substantial proportions of staff report annoyance, irritation, fatigue, and tension headaches, which they assign to the noisy workplace environment.⁷⁹ Noise also affects speech intelligibility and could therefore lead to misunderstandings that result in medical errors.^{73,78}

Improved acoustics such as sound-absorbing ceilings are relevant factors for staff performance and reduced work strain,⁸⁰ and have been associated with a decrease in rates of patients being readmitted to hospital.⁷⁴ Reduction of background sound levels and ringtone volume of telephones is recommended to improve patient recovery at night.⁷⁷ Researchers noted promise in reductions of rates of false alarms of medical devices and modification of staff behaviour to avoid unnecessary noise.⁸¹

Conclusions

Hearing loss caused by occupational or recreational noise exposure is highly prevalent and constitutes a public health threat needing preventive and therapeutic strategies. In this Review, we emphasise that non-auditory health effects of environmental noise are manifold, serious and, because of the widespread exposure, very prevalent. These factors stress the need to regulate and reduce environmental noise exposure (ideally at the source) and to enforce exposure limits to mitigate negative health consequences of chronic exposure to environmental noise. Educational campaigns for children and adults can promote both noise-avoiding and noise-reducing behaviours, and thus, mitigate negative health consequences. Efforts to reduce noise exposure will eventually be rewarded by lower amounts of annoyance, improved learning environments for children, improved sleep, lower prevalence of cardiovascular disease, and, in the case of noise exposure in hospitals, improved patient outcomes and shorter hospital stays.

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Footnotes

Contributors

MBA wrote the abstract, the section about noise effects on sleep, and the conclusion; did a literature search for the section about noise effects on sleep; contributed [panel 2](#); and helped to design [figure 1](#). WB wrote the introduction to the section about non-auditory health effects; did the literature search for and wrote the section on cardiovascular noise effects; and contributed [figures 2](#) and [3](#). AD did the literature search for and wrote the chapter on auditory effects of noise on health. MBR did the literature search for and wrote the section on the effects of hospital noise. CC did the literature search for and wrote the section on the effects of noise on cognitive performance. SJ did the literature search for and wrote the section on community effects of noise. SS wrote the introduction. All authors read and revised the Review.

Conflicts of interest

We declare that we have no conflicts of interest.

Contributor Information

Mathias Basner, Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA.

Wolfgang Babisch, Department of Environmental Hygiene, Federal Environment Agency, Berlin, Germany.

Prof. Adrian Davis, Public Health England, Wellington House, Waterloo Road, London, UK. Ear Institute, University College, London, UK.

Mark Brink, D-MTEC Public and Organizational Health, ETH Zurich, Zurich, Switzerland.

Charlotte Clark, Centre for Psychiatry, Wolfson Institute of Preventive Medicine, Barts and London School of Medicine and Dentistry, Queen Mary University of London, UK.

Sabine Janssen, Department of Urban Environment and Safety, TNO (Netherlands Organization for Applied Scientific Research), Delft, Netherlands.

Prof. Stephen Stansfeld, Centre for Psychiatry, Wolfson Institute of Preventive Medicine, Barts and London School of Medicine and Dentistry, Queen Mary University of London, UK.

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