

PHARMACEUTICAL INTERVENTIONS FOR HEARING LOSS (PIHL)

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Non-Cochlear Effects of Noise

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The consequences of population growth, urbanization, and technological developments include the continued growth of “noise pollution”. Although a great deal of recent research has focused on investigation of its health effects, its “toxicity” was recognized thousands of years ago (Berglund and Lindvall, eds., 1995; Lee and Fleming, 2002). Chariots in ancient Rome were banned from the streets at night to prevent the noise of the wheels clattering on paving stones from disrupting sleep and annoying the citizens. Centuries later, cities in medieval Europe either banned horses and horse drawn carriages from the streets at night or covered the stone streets with straw to reduce noise and ensure residents’ peaceful sleep. In eighteenth century Philadelphia, the framers of the constitution had nearby cobblestone streets covered with earth to prevent noise-induced interruptions (Goines and Hagler, 2007).

Our knowledge of the cochlear effects of noise has increased dramatically over the last few decades, and is now understood at the molecular level. In contrast, the non-cochlear effects are less clear, although many are intuitively obvious. The World Health Organization (WHO) estimated that in high-income western European countries (population approximately 340 million), at least 1 million healthy life-years (disability-adjusted life-years, DALYs) are lost every year because of environmental noise (Fritschi, 2011). The most investigated endpoints are sleep disturbance, cardiovascular health, cognitive impairment (mainly in children), and perceived annoyance (Basner, et al., 2013).

The Link between Chronic Noise Exposure and Adverse Health Effects

Beyond communication, hearing informs us about the environment. We are constantly analyzing what we hear. This is a complex process and requires that pathways

distribute information across various areas of the brain and central nervous system. Noise effects depend upon the integrated meaning assigned to a host of characteristics (source, onset, duration, frequencies, intensity, whether exposure is voluntary or involuntary, whether it is regarded as useful or necessary, pleasant or unpleasant, etc.), which ultimately depends upon the instantaneous state of the person who hears it. Saunders (1956) referred to the myriad of interacting, transient, internal factors as “moderating variables”. These properties of the listener determine how the body will respond.

In other words, there is only limited theoretical understanding of non-auditory noise effects, and knowledge of possible mechanisms and modifiers is little more than suggestive (Babisch, 2002; Guski, 1999). Health effects attributed to noise exposure are mediated by physiological and/or psychological responses, which often overlap, and may not be separable, especially when physiological effects are the underlying cause of the psychological stress and vice versa.

Physiological Models

These models hypothesize a link between noise and health that is mediated by either the:

1. Sympathetic nervous system and the secretion of catecholamines, or
2. The pituitary-adrenocortical axis, based on a process called the general adaptation syndrome (Selye, 1956), more recently described terms of allostasis (Sterling and Eyer, 1988) or allostatic load (McEwen and Stellar, 1993).

Psychological Models

From the psychological perspective, four major constructs have been proposed to account for the non-cochlear effects of noise:

1. Information overload
2. Arousal
3. Coping strategies
4. Loss of control

Health Effects

Sleep Disturbance

Undisturbed sleep of sufficient length is necessary for daytime alertness and performance, quality of life, and health (Muzet, 2007; Fritschi et al., 2011). Therefore, sleep disturbance is regarded as the most deleterious non-auditory effect of environmental noise. Humans perceive, evaluate, and react to environmental sounds, even while asleep (Dang-Vu et al., 2010). Sound pressure levels as low as L_{Amax} 33dB can induce physiologic reaction during sleep, including autonomic, motor, and cortical arousals (e.g., tachycardia, body movements, and awakenings) (Muzet, 2007; Basner et al., 2006). Reaction to noise while sleeping depends not only on the number of noise events and their acoustical properties, but also on situational moderators (e.g., sleep stage; Basner, et al, 2010) and individual noise susceptibility (Dang-Vu et al., 2010). The elderly, children, shift-workers, and people with a pre-existing

sleep disorder are the at risk groups for noise induced sleep disturbance. Repeated arousals interfere with sleep structure, including delayed sleep onset and early awakenings, reduced deep (slow wave) and REM sleep, and an increase in time spent awake and in superficial sleep stages. Short-term effects of disturbed sleep include impaired mood, daytime sleepiness, and impaired cognitive performance (Basner, 2008; Elmenhorst et al., 2010).

Cardiovascular Disease

Noise exposure causes a number of short-term physiological activation responses mediated through the autonomic nervous and endocrine systems (including increased heart rate and blood pressure, peripheral vasoconstriction), and causes the release of stress hormones (including catecholamines and glucocorticoids).

Long-term studies have provided biological mechanisms and plausibility for the hypothesis that long-term exposure to environmental noise affects the cardiovascular system in humans and animals, and causes manifest disease (including hypertension, ischemic heart disease, and stroke) in animals (Babisch, 2011). However, effects in humans and animals cannot be directly compared. The effect mechanism is thought to be the general stress model, which comprises the two pathways discussed earlier: The direct (physiological) pathway (non-conscious stress from interactions between the central auditory system and other regions of the CNS), and the indirect (psychological) pathway (emotional stress due to the cognitive reaction to noise). The latter is certainly different in humans (WHO, 2011).

The association of noise exposure and cardiovascular disease is supported by several epidemiology studies of occupationally-(van Kempen, et al., 2002; and Tomei, et al., 2010; Davies and van Kamp, 2012) and environmentally (Huss, et al., 2010; Sorensen, et al., 2011; and Gan et al., 2012) exposed populations. However, the studies are not completely convincing. The risk estimates for occupational noise at ear-damaging intensities tend to be higher than are those for environmental noise, but still relatively small ($RR < 2$) – within the range where they could be explained by incomplete control for confounding and/or various biases, e.g., reporting bias, and selection bias.

Babisch (2011) points out an additional obstacle to Interpretation. Non-auditory noise effects do not follow the toxicological principle of dosage. This means that it is not simply the accumulated sound energy that causes the adverse effects (dealing with decibels is not like summing up micrograms as we do for chemical exposures). Instead, the individual situation and disturbed activity need to be taken into account (time activity patterns). It may be very well that 80 decibels has less effect than 65 decibels when carrying out mental tasks at home or 50 decibels when trying to sleep. In this respect, the evening hours, when people come home from work for relaxation and the nighttime, when the body physically recovers from daytime load and brain restoration takes place, may be particularly important with respect to noise-induced health effects. Sleep is also an important modulator of cardiovascular function. Noise-

disturbed sleep, in this respect, must be considered as a particular potential pathway for the development of cardiovascular disorders.

Cognitive Effects

More than 20 studies have shown environmental noise exposure has a negative effect on children's learning outcomes and cognitive performance (Evans and Hygge, 2007), and that children with chronic aircraft, road traffic, or rail noise exposure at school have poorer reading ability, memory, and performance on national standardized tests than do children who are not exposed to noise at school (Hygge et al., 2002; Bronzaft, 1981; Lercher et al., 2003). 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 relationship between aircraft noise exposure at school and a child's reading comprehension and recognition memory after adjusting for a range of socioeconomic factors (Stansfeld et al., 2005; Clark et al., 2006). This linear association between exposure and effect suggests that there is no effect threshold, and any reduction in noise level at school should improve a child's cognition.

Annoyance

Annoyance is the most prevalent community response in a population exposed to environmental noise. Noise-related annoyance can result from interference with daily activities, feelings, thoughts, sleep, or rest, and may be accompanied by anger, displeasure, exhaustion, and by stress-related symptoms. In severe forms, it could be thought to affect wellbeing and health, and because of the large number of people affected, annoyance substantially contributes to the burden of disease from environmental noise.

Conclusions

The evidence for non-cochlear effect of noise on health is strongest for annoyance, sleep, and cognitive performance in adults and children. Occupational noise exposure shows some association with increased blood pressure. Dose-response relationships can be demonstrated for annoyance and, less consistently for blood pressure. The effects of noise are strongest for those outcomes that, like annoyance, can be classified under "quality of life" rather than illness. Nevertheless, what these effects lack in severity, they make up for in number of people affected.

Adaptation to long-term noise needs further study. Most people exposed to chronic environmental noise, for example from major airports, tend to tolerate it (they do not move). Yet questionnaire studies suggest that high levels of annoyance do not decline over time. One possible explanation is that adaptation to noise is achieved with a cost to health. These points ought to be taken into consideration when designing a research study involving noise exposures for heightened protection of human subjects as well as for additional data collection considerations to pursue.

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