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Environmental noise and sleep disturbances: A threat to health?



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ABSTRACT

Environmental noise, especially that caused by transportation means, is viewed as a significant cause of sleep disturbances. Poor sleep causes endocrine and metabolic measurable perturbations and is associated with a number of cardiometabolic, psychiatric and social negative outcomes both in adults and children. Nocturnal environmental noise also provokes measurable biological changes in the form of a stress response, and clearly affects sleep architecture, as well as subjective sleep quality. These sleep perturbations are similar in their nature to those observed in endogenous sleep disorders. Apart from these measurable effects and the subjective feeling of disturbed sleep, people who struggle with nocturnal environmental noise often also suffer the next day from daytime sleepiness and tiredness, annoyance, mood changes as well as decreased well-being and cognitive performance. But there is also emerging evidence that these short-term effects of environmental noise, particularly when the exposure is nocturnal, may be followed by long-term adverse cardiometabolic outcomes. Nocturnal environmental noise may be the most worrying form of noise pollution in terms of its health consequences because of its synergistic direct and indirect (through sleep disturbances acting as a mediator) influence on biological systems. Duration and quality of sleep should thus be regarded as risk factors or markers significantly influenced by the environment and possibly amenable to modification through both education and counseling as well as through measures of public health. One of the means that should be proposed is avoidance at all costs of sleep disruptions caused by environmental noise.

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Background

The World Health Organization (WHO) has documented seven categories of adverse health and social effects of noise pollution, whether occupational, social or environmental: hearing impairment, interference with spoken communication, cardiovascular disturbances, mental health problems, impaired cognition, negative social behaviors and sleep disturbances [1]. The latter is considered the most deleterious non-auditory effect because of its impact on quality of life and daytime performance [2–4]. Environmental noise, especially that caused by transportation means, is a growing problem in our modern cities [5]. It is considered a major cause of exogenous sleep disturbances, after somatic problems and day tensions [6,7].

Sleep is an important modulator of hormonal release, glucose regulation and cardiovascular function. In particular slow-wave sleep, the most restorative sleep stage, is

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associated with decreased heart rate, blood pressure, sympathetic nervous activity and cerebral glucose utilization, compared with wakefulness. During this sleep stage, growth hormone is released while stress hormone cortisol is inhibited [8,9]. Healthy sleep plays also an important role in memory consolidation [10]. Poor sleep causes measurable changes on these systems. Experimental studies demonstrated that both sleep restriction and poor quality sleep affect glucose metabolism by reducing glucose tolerance and insulin sensitivity [11–13] and that sleep restriction dysregulates appetite (lower levels of leptin and higher levels of ghrelin) [14,15] as well as cortisol levels [8]. Sleep restriction has also been shown to increase blood pressure [16,17] and affect immune processes [18]. It has been hypothesized that these perturbations cause long-term consequences on health [19].

Indeed there is increasing evidence that quantitative and qualitative sleep disturbances may play a role in the development of cardiometabolic disease. A number of cardiovascular risk factors and cardiovascular outcomes have been associated with disturbed sleep: coronary artery calcifications, atherogenic lipid profiles, atherosclerosis, obesity, type 2 diabetes, hypertension, cardiovascular events [19,20]. Increased mortality from all causes has also been observed [21]. During the past years, the relationship between insomnia and psychiatric disorders has come to be considered synergistic, including bi-directional causation. It has become clear that insomnia is not merely a symptom of psychiatric disorders, but contributes also to the risk of future relapse or the development of new onset mood, anxiety, and substance use disorders, as well as to the severity of psychiatric symptoms [22]. Disturbed sleep has also been associated with increased frequency of violent acts as well as domestic violence, work and vehicle accidents, increased work absenteeism [23–26]. The observed associations between poor sleep and obesity, diabetes, depression, aggressive and delinquent behaviors concern children and adolescents, too [27-30]. As a result of sleep disturbances, children also suffer from impaired cognition and worsening of attention deficit hyperactivity disorder symptoms [31].

Nocturnal environmental noise also provokes measurable metabolic and endocrine perturbations (increased secretion of adrenaline, noradrenaline, cortisol), increased heart rate and arterial pressure, and increased motility. These biological responses to noise during sleep are most of the time unnoticed [32–34]. Noise also affects sleep architecture, as well as subjective sleep quality. Nocturnal air traffic causes nocturnal awakenings at levels as low as 48 dB, and physiological reactions in the form of increased vegetative hormonal secretions, cortical arousals and body movements at even lower levels, probably around 33 dB [33,35,36]. Nocturnal noise has been shown to fragment sleep, and as a consequence lead to a redistribution of time spent in the different sleep stages, typically increasing wake and stage 1 sleep and decreasing slow wave sleep and REM sleep, i.e. causing a shallower sleep [7,32,37]. Basner et al. showed that although these effects on sleep structure and continuity are relatively modest, they have a significant impact on subjective assessments on sleep quality and recuperation: Subjects experience their sleep as disturbed and with low recuperative value.

Also, despite being most of the time in an unconscious state, subjects are able to distinguish between nights with low and high degrees of traffic noise exposure. Their reaction time at next day performance test is also slightly but significantly increased [7]. These findings corroborate with previous observations that noise is indeed a widespread factor of selfreported sleep disturbances [38].

Apart from these measurable effects and the subjective feeling of disturbed sleep, people who struggle with nocturnal environmental noise often also suffer the next day from daytime sleepiness and tiredness, annoyance, mood changes as well as decreased well-being and cognitive performance [1,4,10,39-42]. Associations between exposure to aircraft noise and the following health complaints and health indicators have been demonstrated: headache, poor self-rated health status, use of medication for cardiovascular diseases and use of sleep medication [40,43]. Could these short-term effects be also followed by long-term adverse health outcomes? Data show that exposure to traffic noise, not specifically at night, is associated with increased incidence of diabetes [44], hypertension [45] and stroke among the elderly [46], as well as increased incidence and mortality from coronary heart disease [47-50]. But interestingly some epidemiological data support the hypothesis that exposure to noise at night time may be especially relevant in terms of negative cardiovascular outcomes, perhaps due to the fact that repeated autonomic arousals habituate to a much lesser degree to noise than cortical arousals [2,7]. Indeed data show that exposure to traffic noise especially at night increases the risk for hypertension [43], also in children [51], as well as the risk for heart disease and stroke [52]. These results confirm previous findings of studies looking at the association between subjective responses to community noise and cardiovascular outcomes that suggested that night-time noise may be more a determinant of noise-induced cardiovascular effects than daytime exposure [53–55].

As we previously described, poor sleep triggers biological mechanisms contributing to the deterioration of somatic health and is clearly associated with significant psychiatric morbidity, too. Whereas exposure to traffic noise around-theclock seems to be clearly associated with adverse health outcomes, the question of health consequences of noise exposure specifically at night still needs to be further explored, since the majority of evidence so far comes either from observational field studies that look at the immediate consequences of nocturnal noise exposure or from epidemiological studies that do not usually separate nocturnal from diurnal exposure. However, although sleep structure perturbations in the context of nocturnal noise seem less severe than in sleep pathology such as obstructive sleep apnea, they tend to be similar in their nature [2]. It is thus reasonable to hypothesize that poor sleep may act as a mediator between nocturnal noise pollution and increased risk of cardiovascular morbidity, through impaired endocrine and metabolic functions [11]. Also, by affecting sleep architecture, environmental noise pollution causes sleep disturbances that lead to subjective distress in the form of daytime sleepiness and tiredness, decreased well-being and cognitive performance, as well as mood changes and potentially more serious psychopathology and psychiatric morbidity, although this remains

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