International Journal of Speech and Audiology

E-ISSN: 2710-3854
P-ISSN: 2710-3846
IJSA 2022; 3(1): 08-10
© 2022 IJSA
www.rehabilitationjournals.com
Received: 22-10-2021
Accepted: 08-12-2021

Domina Petric
University Hospital Center Split, Soltanska Ulica 1, Croatia

Correspondence
Domina Petric
University Hospital Center Split, Soltanska Ulica 1, Croatia

Noise pollution and health

Domina Petric

Abstract
Noise pollution is an increasing problem in modern societies and includes environmental noise pollution, defined as all the unwanted sounds in human communities except that which originates in the workplace (such as neighbors listening television or music far too loud, night traffic, obsolete household appliances), and occupational loud noise exposure, which occurs at work. Noise pollution as such is associated with detrimental health effects, including arterial hypertension, psychological stress, tinnitus and hearing loss, sleep disturbances, and even faster cognitive decline.

Keywords: Noise, pollution, hearing, tinnitus, health

Introduction
Environmental noise pollution, that consists of all the unwanted sounds in human communities except that which originates in the workplace, is a threat to health and well-being. It is more severe and widespread than ever before, and it will continue to increase in magnitude and severity because of population growth, urbanization, and the associated growth in the use of increasingly powerful, varied, and highly mobile sources of noise, as well as because of sustained growth in highway, rail, and air traffic [1]. Noise pollution is associated with arterial hypertension, high levels of psychological stress, tinnitus and hearing loss, sleep disturbances, and faster cognitive decline [2-5]. The first category of noise exposure is ″traumatic″ noise exposure, which is common, for example, in heavy manufacturing (>105 dB for 1 hour) or due to firearm noise (>130 dB for a few seconds), and which has long been known to produce significant levels of permanent hearing loss6. The second is ″threatening″ noise levels, ranging between 90 dB and 105 dB, with exposure times of 2 hours or more, which are commonly encountered in concerts or night-clubs. People attending such events often experience a subsequent temporary hearing loss for a few hours. The third is ″safe″ levels, below 80 dB, which have been considered harmless for the auditory system for any exposure duration, but even such environmental noise may progressively degrade hearing through alterations in the way sound is represented in the adult auditory cortex [7].

Neuropsychiatric effects of environmental noise pollution
Noise pollution can have negative effects on autistic adults and children8. Autistic patients can have hyperacusis (abnormal sensitivity to sound [9]). People with autism who experience hyperacusis may have unpleasant emotions, such as fear and anxiety, and uncomfortable physical sensations in noisy environments with loud sounds [10]. Autistic patients avoid environments with noise pollution, which in turn can result in social isolation and negatively affect their quality of life [8].
Noise pollution and social isolation is a vicious circle for autistic patients. Autism itself causes these patients to be more socially isolated than the general population, and on the other hand, noise pollution causes autistic patients to become more and more socially isolated, what closes the vicious circle of noise pollution, social isolation and autism.
Prolonged exposure to loud noise alters how the brain processes speech, potentially increasing the difficulty in distinguishing speech sounds [11]. In the study conducted by Frenzilli and coworkers (2017) [12] authors discovered that loud noise exposure lasting 12 h causes immediate DNA, and long-lasting neurotransmitter and immune-histochemical alterations within specific brain areas of the rat.
Researchers concluded that loud noise exposure represents a detrimental stimulus for specific brain areas, which consists mostly on decreased catecholamine innervation involving multiple brain regions.

Data provided by this study lend substance to clinical findings showing impaired memory, mood alterations and other behavioral alterations induced by prolonged noise exposure. The occurrence of nigrostriatal dopamine innervation further strengthens this association. In Parkinson's disease a loss of auditory function occurs, which is compatible with the loss of cochlear dopamine innervation, which in turn protects from the effects of loud noise. Thus, a vicious circle may occur, where the excitotoxic effects of loud noise may destroy dopamine nerve endings producing a loss of dopamine in their terminal fields, including the efferent synapses with cochlear hair cells, where dopamine exerts a gating control. In this way, the transmission of loud noise would no longer be hampered despite a loss in the detection of pure tones. Findings of this study provided a bridge between environmental exposure to loud noise and the onset of neuropsychiatric alterations, such as cognitive impairment, depressive symptoms, behavioral abnormalities, and movement disorders [12].

Noise-Induced Audiological Health Problems

Most standards for occupational noise exposure limits specify an 8-h occupational noise exposure limit (LEX) of 85 dBA. This limit assumes that some workers exposed at the limit will develop hearing loss. To eliminate the risk of hearing loss, a 24-h equivalent continuous level (LEQ24h) limit of 70 dBA is appropriate. When comparing the differences in the risk of hearing loss from occupational noise versus recreational sound, there is some evidence that the effects of occupational noise on hearing may be worse than energetically equivalent recreational sound. A recreational sound limit of 80 dBA LEX, equivalent to a 75 dBA LEQ24h, will virtually eliminate the risk of recreationally induced hearing loss in adults, but lower limits may be warranted for vulnerable or susceptible individuals [13]. Repeated overexposure to noise at or above 85 dBA can cause permanent hearing loss, tinnitus, and difficulty understanding speech in noise. It is also associated with cardiovascular disease, depression, balance problems, and lower income. About 22 million U.S. workers are currently exposed to hazardous occupational noise. Approximately 33% of working-age adults with a history of occupational noise exposure have audiometric evidence of noise-induced hearing damage, and 16% of noise-exposed workers have material hearing impairment. While the Mining, Construction, and Manufacturing sectors typically have the highest prevalence of noise exposure and hearing loss, there are noise-exposed workers in every sector and every sector has workers with hearing loss [14]. Turcot and coworkers conducted a study, which aim was to determine whether hearing impairment is worse in noise-exposed workers with vibration white finger (VWF) than in workers with similar noise exposure but without VWF, and found greater hearing loss at higher frequencies in workers with VWF, supporting the association between combined noise and hand-arm vibration exposure with noise-induced hearing loss [15].

Tinnitus is a phantom auditory sensation that significantly reduces the quality of life. Most cases of tinnitus are associated with hearing loss caused by ageing or noise exposure. Exposure to loud recreational sound is common among the young, and this group are at increasing risk of developing tinnitus. Head or neck injuries can also trigger the development of tinnitus, as altered somatosensory input can affect auditory pathways and lead to tinnitus or modulate its intensity.

Emotional and attentional state could be involved in the development and maintenance of tinnitus via top-down mechanisms. Thus, military personnel in combat are particularly at risk owing to combined risk factors (hearing loss, somatosensory system disturbances and emotional stress). Animal model studies have identified tinnitus-associated neural changes that commence at the cochlear nucleus and extend to the auditory cortex and other brain regions. Maladaptive neural plasticity seems to underlie these changes: it results in increased spontaneous firing rates and synchrony among neurons in central auditory structures, possibly generating the phantom percept [16]. In animal models, equivalent cochlear damage occurs in animals with and without behavioral evidence of tinnitus. But cochlear-nerve-recipient neurons in the brainstem demonstrate distinct, synchronized spontaneous firing patterns only in animals that develop tinnitus, driving activity in central brain regions and ultimately giving rise to phantom perception [17]. Jafari and coworkers found in the retrospective large-scale study that chronic noise exposure has significant contribution in auditory aging and the precipitation of both age-related hearing loss and tinnitus18. Williams and Carter found in their study that 63% of the study subjects (sample of 1435 young Australians, aged 11-35 years) experienced tinnitus in some form, but not associated with hearing threshold levels or variation in otoacustic emissions. Males experienced more permanent tinnitus at significantly grater rate than females [19].

Cardiovascular and metabolic effects of noise pollution

Studies have shown that noise pollution has effects on systolic, but not diastolic blood pressure, total cholesterol, total triglycerides, blood viscosity, platelet count and glucose level, and might be considered as minor risk factor for coronary artery disease. A sudden intense exposure to noise may stimulate catecholamine secretion and precipitate cardiac dysrhythmias, although available studies in coronary care units and studies of noise from low altitude military flights failed to detect changes in cardiac rhythm attributable to noise [20]. A study, aimed to investigate associations between traffic-related nitrogen oxides (NOx) or noise pollution and risk of incident metabolic syndrome and its components in an elderly Mexican-American population, showed that each per unit increase of traffic-related NOx (2.29 parts per billion (ppb)) was associated with a 15% (HR = 1.15, 95% CI: 1.04-1.28) lower level of high-density lipoprotein cholesterol (HDL-cholesterol), and each 11.6 decibels (dB) increase in noise increased the risk of developing metabolic syndrome by 17% (HR = 1.17, 95% CI: 1.01-1.35). Authors concluded that policies aiming to reduce traffic-related air pollution and noise might mitigate the risk of metabolic syndrome and its components in vulnerable populations [21]. According to recent estimates of the World Health Organization, exposure to traffic noise is responsible for a loss of more than 1.5 million healthy life years per year in Western Europe alone, a major part being related to annoyance, cognitive impairment, and sleep disturbance. Underlying mechanisms of noise-induced mental stress are centered on increased stress hormone levels, blood pressure, and heart rate, which in turn favor the development of cerebrocardiovascular disease such as

stroke, arterial hypertension, ischemic heart disease, and myocardial infarction. Traffic noise exposure is associated with mental health symptoms and psychological disorders such as depression and anxiety, which further increase maladaptive coping mechanisms (alcohol and tobacco use). From a molecular point of view, experimental studies suggest that traffic noise exposure can increase stress hormone levels, thereby triggering inflammatory and oxidative stress pathways by activation of the nicotinamide adenine dinucleotide phosphate oxidase, uncoupling of endothelial/neuronal nitric oxide synthase inducing endothelial and neuronal dysfunction [22].

Conclusion

Both environmental and occupational noise exposure are associated with detrimental audiological (hearing loss, tinnitus) and non-audiological (neuropsychiatric, psychological, cardiovascular metabolic) health effects. Vulnerable groups of individuals, especially patients that suffer from autism, might be more affected. Autism itself causes these patients to be more socially isolated than the general population, and on the other hand, noise pollution causes autistic patients to become more and more socially isolated, what closes the vicious circle of noise pollution, social isolation and autism. With increasing population, traffic and noise-producing technology (speakers, amplifiers, television, home theater) there is an increasing need for public health measures and legal interventions in order to better regulate both environmental and occupational noise exposure. Excessive noise pollution, especially leisure loud noise exposure is unfortunately very often perceived as desirable, especially among younger population (disco music, electronic music concerts) and there is a need to raise awareness, especially among teenagers and adolescents, about the detrimental health effects of loud noise exposure. Hearing loss in that age group, associated with leisure exposure to loud noise, is usually temporary, and therefore, the special attention should be on the prevention of permanent hearing loss and chronic tinnitus-associated with noise-exposure during the early stages of noise-induced hearing damage, when it is not yet irreversible. There is also a need to raise awareness and implementing legal measures related to individual, societal and corporate liability for the preservation of healthy environment, which is one of the basic human rights, and this should include effective measures of prevention and mitigation of noise pollution.

References

1. Goines L, Hagler L. Noise pollution: a modem plague. South Med J. 2007;100(3):287-294.
2. Münzel T, Schmidt FP, Steven S, Herzog J, Daiber A, Sørensen M. Environmental Noise and the Cardiovascular System. J Am Coll Cardiol. 2018;71(6):688-697.
3. Rosen S, Olin P. Hearing Loss and Coronary Heart Disease. Archives of Otolaryngology. 1965;82:236.
4. Kerns E, Masterson EA, Themann CL, Calvert GM. Cardiovascular conditions, hearing difficulty, and occupational noise exposure within US industries and occupations. Am J Ind Med. 2018;61(6):477-491.
5. Paul KC, Haan M, Mayeda ER, Ritz BR. Ambient Air Pollution, Noise, and Late-Life Cognitive Decline and Dementia Risk. Annu Rev Public Health. 2019;40:203-220.
6. Beliefs SOF. Position Statement on Preventing Noise-Induced Hearing Loss. AAA. 2004;6(4):4-7.
7. Gourévitch B, Edeline JM, Occelli F, Eggermont JJ. Is the din really harmless? Long term effects of non-traumatic noise on the adult auditory system. Nat Rev Neurosci. 2014;15(7):483-91.
8. Reaven J. Autism & Anxiety: Parents seek help for extreme reaction to loud noise. https://www.autismspeaks.org/expert-opinion/autism-anxiety-parents-seek-help-extreme- reaction-loud-noise-0 september 5, 2018.
9. ASHA. Tinnitus and Hyperacusis. https://www.asha.org/practice-portal/clinical-topics/tinnitus-and-hyperacusis/ january 24, 2022.
10. Stiegler LN, Davis R. Understanding Sound Sensitivity in Individuals with Autism Spectrum Disorders. FOCUS. 2010;25(2):67-75.
11. Reed AC, Centanni TM, Borland MS, Matney CJ, Engineer CT, Kilgard MP. Behavioral and neural discrimination of speech sounds after moderate or intense noise exposure in rats. Ear Hear. 2014;35(6):e248-e261.
12. Frenzilli G, Ryskalin L, Ferrucci M, *et al*. Loud Noise Exposure Produces DNA, Neurotransmitter and Morphological Damage within Specific Brain Areas. Front Neuroanat. 2017;11:49.
13. Neitzel RL, Fligor BJ. Risk of noise-induced hearing loss due to recreational sound: Review and recommendations. J Acoust Soc Am. 2019;146(5):3911.
14. Themann CL, Masterson EA. Occupational noise exposure: A review of its effects, epidemiology, and impact with recommendations for reducing its burden. J Acoust Soc Am. 2019;146(5):3879.
15. Turcot A, Girard SA, Courteau M, Baril J, Larocque R. Noise-induced hearing loss and combined noise and vibration exposure. Occup Med (Lond). 2015;65(3):238-244.
16. Shore SE, Roberts LE, Langguth B. Maladaptive plasticity in tinnitus--triggers, mechanisms and treatment. Nat Rev Neurol. 2016;12(3):150-160
17. Shore SE, Wu C. Mechanisms of Noise-Induced Tinnitus: Insights from Cellular Studies. Neuron. 2019;103(1):8-20.
18. Jafari Z, Copps T, Hole G, Kolb BE, Mohajerani MH. Noise Damage Accelerates Auditory Aging and Tinnitus: A Canadian Population-Based Study. Otol Neurotol. 2020;41(10):1316- 1326.
19. Williams W, Carter L. Tinnitus and leisure noise. Int J Audiol. 2017;56(4):219-225.
20. Stansfeld SA, Matheson MP. Noise pollution: non-auditory effects on health. Br Med Bull. 2003;68:243-257.
21. Yu Y, Paul K, Arah OA, Mayeda ER, Wu J, Lee E, *et al*. Air pollution, noise exposure, and metabolic syndrome - A cohort study in elderly Mexican-Americans in Sacramento area. Environ Int. 2020;134:105269.
22. Hahad O, Prochaska JH, Daiber A, Muenzel T. Environmental Noise-Induced Effects on Stress Hormones, Oxidative Stress, and Vascular Dysfunction: Key Factors in the Relationship between Cerebrocardiovascular and Psychological Disorders. Oxid Med Cell Longev. 2019;2019:4623109.