Editorial

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Noise Stress

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Editorial

Noise is frequently described as an intermittent or chronic unsolicited sound, impairing communication and health. Noise pollution, especially in municipal environs, has escalated in the recent century ranking among the environmental stressors with the highest public health impact [1]. Although different individuals have varying spectrum of audible sensitivity to sound frequencies, most humans are sensitive to noise in the range of 500 Hz to 4 kHz, which includes the range of conventional speech.

In the central nervous system, sound proceeds in two directions; one to the auditory center, where it is perceived and interpreted; and the other to the deep parts of brain, where it activates the autonomic nervous system and is liable for a wide range of monaural effects. The effect of noise on the central nervous system is reliant on the state of the brain. In an exhausted individual the compensatory mechanisms are more susceptible than in a rested individual. Genetics has been identified to play a key role in the mechanisms of noise-induced auditory and non-auditory perturbations. It has been proven that certain patients are more vulnerable to permanent threshold shifts, based on auditory brainstem response (ABR) threshold measurements, when exposed to a similar noise experience. Loci of vulnerability to noise have not been identified in humans, but several mouse models have been shown to uphold genetic composition in noise induced impairments [2].

Noise stress has been confirmed to aggravate mechanical mutilation of the cochlea inducing permanent hearing loss [2]. The detrimental effects of noise however transcend its auditory effects owing to the stimulated increase in generation of reactive oxygen and nitrogen species (ROS and RNS), which trigger apoptosis, damage DNA and disrupt lipid and protein molecules, triggering loss of function and cell death. It activates the hypothalamo-pituitary-adrenocortical (HPA) axis, resulting in behavioral, autonomic, neuroendocrine and immunological reactions; often linked with increased generation of free radicals which subsequently induce oxidative stress and associated health impairments [3].

Studies affirm that noise exposure is allied with increased incidence of diabetes mellitus [4], mortality from cardiovascular diseases [5], poor sleep quality [6], annoyance and pain [7], anxiety, impaired memory and cognitive development [3]. In addition, noise exposure delays healing of surgical wounds [8], suppress immune function [9] and impairs fertility, reproductive physiology and energy consumption in animals [10]. Reports of psychoneurotic and psychosomatic complaints have also been documented following noise exposures [11].

According to WHO [1], noise causes daily health damage estimated at 4 million dollars. Recent studies have focused on the efficacy of exogenous antioxidant agents to mitigate noise-induced stress. This potential has been attributed to their imperative ability to enhance the expression of endogenous antioxidant enzymes and scavenge generated free radicals.

There is however a need to enact and strengthen policies advocating for implementation of noise exposure limits in residential, educational, commercial and industrial metropolis. Governments and civil societies should enlighten, re-orientate, and emphasize intentional, maximum increase in distance from noise sources and decrease in noise exposure time, for occupationally exposed population as this is more appropriate for preventing noise induced anomalies especially in regions where potent antioxidants are inaccessible. In conclusion, future research on human subjects, should focus on the identification of the loci of vulnerability to noise, thus providing genetic basis for subsequent selection and modification.

References

1. World Health Organization (2016) Environmental Noise Guidelines for European Region. Geneva, Switzerland.
2. Akefe IO, Ayo JO, Sinkalu VO (2017) Mitigative effects of antioxidants in noise stress. J Clin Nutr Diet 3: 21.
3. Wang S, Yu Y, Feng Y, Zou F, Zhang X, et al. (2016) Protective effect of the orientin on noise induced cognitive impairments in mice. Behav Brain Res 296: 290-300.
4. Sorensen M, Andersen ZJ, Nordsborg RB, Becker T, Tjonneland A, et al. (2013) Long-term exposure to road traffic noise and incident diabetes: A cohort study. Env Health Perspect 121: 217-222.
5. Baliatsa C, Van Kemp I, Van Poll R, Yzermans, J (2016) Health effects from low frequency noise and infrasound in the general population: Is it time to listen? A systematic review of observational studies. Sci Total Env 10: 557-558.

6. Van Kamp I, Davies H (2013) Noise and health in vulnerable groups: a review. Noise Health 15: 153-158.
7. Michaud SD, Feder K, Keith ES, Voiscescu SA, Mam L, et al. (2016) Personal and situational variables associated with wind turbine noise annoyance. J Acoust Soc Am 139: 1455–1466.
8. Zheng KC, Ariizumi MJ (2007) Modulations of immune functions and oxidative status induced by noise stress. Occup Health Saf 49: 32–38.
9. Cheng L, Wang SH, Chen OC, Liao XM (2011) Moderate noise induced cognition impairment of mice and its underlying mechanisms. Physiol Behav 104: 981-988.
10. Brouček J (2014) Effect of noise on performance, stress, and behaviour of animals. Slovak J Animal Sci 47: 111-123.
11. Martinho PAJ, Castelo BMS, Castelo BNA (1999) The palmomental reflex in vibro acoustic disease. Aviat Space Env Med 70: 100-117.