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THE ADVERSE EFFECTS OF ENVIRONMENTAL NOISE EXPOSURE ON HEALTH

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Abstract
Noise has always been very common environmental problem for mankind. Noise leading to hearing loss is one of the most common neurological disorders. In defence scenario, armed forces are exposed to the following types of noise: tanks, aircrafts, bombardment, missile launching, ammunition trials etc. Noise incites both direct (hearing loss, tinnitus) as well as indirect effects (anxiety, depression, annoyance, etc.). Environmental noise has multiple effects on human health. Some of these effects such as raised blood pressure; hearing loss and cognitive impairments may have implications for adult health as well.

In this review, we consigned that noise affects memory storage, cognitive decision making, executive functions that are the foremost requirements for human being. Psychological behaviour like argumentativeness, mood fluctuations, incapability to face stressful situations hamper the performance and lead to a poor quality of life. Noise exposures disturb the balance of awake and sleep modes of brain activity leading to neurological change

Keywords
Noise; Cognitive; Oxidative Stress; Hearing Loss.

INTRODUCTION

Noise is defined as unwanted sound or a combination of sounds that has adverse effects on health. The effects can manifest in the form of physiologic damage or psychological harm through a variety of mechanisms (Bahng and Lee, 2020). WHO reported that around one million healthy life years are lost every year due to environmental noise. Noise Induced Hearing Loss (NIHL) is thought to be one of the major causes of preventable hearing loss. The psychological effects of noise are usually not well characterized and often ignored. However, their effect can be equally devastating and may include hypertension, tachycardia, elevated cortisol levels and increased physiologic stress. Collectively, these effects can have severe adverse consequences on daily living and globally on economic production (Seidman and Standring, 2010). Currently the armed forces are facing severe disabilities secondary to noise. It has been shown that the top two disabilities now facing the American Military are hearing loss and tinnitus (Bahng and Lee, 2020). It is reported that the physical and emotional effects of persistent noise lead to irritability, anger, nausea, headache, sleep disturbances and higher sense of helplessness, lack of control, tension, stress, unhappiness, anxiety and depression (Fooladi, 2012). In a self-reported study on the uncontrolled noise exposures to honking and shouting, the participants indicated their responses as being rage and frustration (Fooladi, 2012). Noise may cause cognitive impairment by a variety of mechanisms. Earlier studies have demonstrated that children

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in noisy environments have decreased attention on task and have lower performance on cognitive assignments compared to children in quiet environment (Hygge et al., 2003); Shield et al, 2015). A more recent study by Ljung et al., found that Traffic noise significantly impaired reading ability and comprehension as well as basic mathematics performance in children (Ljung et al, 2009). These psychological and physiological non-auditory effects of noise result in detrimental health consequence and a decreased quality of life(Seidman and Standring, 2010).

i. NOISE AND HEALTH IMPACT

Increased arousal, learned helplessness, frustration, annoyance and consequences of sleep disturbance on performance are reported to contribute to cognitive changes (Stansfeld et al., 2000;(Evans, 2006). Noise exposure may slow rehearsal in memory, influence process of selectivity in memory and choice of strategies for carrying out a task (Smith et al, 1997). It may reduce helping behaviour, increase aggression and reduce the processing of social cues during task performance (Jones et al., 1981). Other physiological effects of noise exposure include symptoms such as nausea, headache, argumentativeness and change in mood and anxiety (Stansfeld et al., 2000). Noise induced disruption was also found for non- auditory tasks like serial recall of visually presented lists and reading (Klatte et al., 2013). Studies undertaken in rats by Rabat have shown that noise exposure was correlated to an incapability to face stressful situations (Rabat, 2007). 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, and exhaustion and by stress related symptoms(Tiesler et al., 2013).

An efficient evaluation of noise effect should include an analysis of its frequency spectrum (Mahendra Prashanth and Venugopalachar, 2011). (Shukla, 2012) showed that traffic noise produces both a high frequency component around 1 kHz and a low frequency component around63 Hz, responsible for the health relevant hazards. Some of the problems of high frequency noise include hearing impairment, hypertension, high blood pressure, speech interface, annoyance and disturbance to daily activities whereas low frequency noise could lead to annoyance, sleep deprivation, physiological disorders, etc. (Davies and Kamp, 2012; Shukla, 2012). In another study, Prashanth et al. (2008) reported that repeated noise exposures in the low (> 22- 500 Hz) and mid frequency (>500 Hz to 2 kHz) octave bands lead to ear vibration, chronic fatigue, headache, awakening from sleep, neck pain, backache, eye ball pressure, and ear pulsation.

Animal Studies in Wister rat show that these extra-auditory effects of noise exposure were due to modified levels of oxidative markers in two areas of the brain, mainly the hippocampus and cerebellum (Shukla et al., 2019; Uran et al., 2010). Significant decrease in the volume of granule layer and cells in cerebellum, decrease in somal volume of Purkinje cells and increased plasma corticosterone concentration was found in noise exposed animals (Hosseini-Sharifabad and Sabahi, 2014).

ii. NOISE AND OXIDATIVE STRESS

Certain environmental challenges can increase the production of reactive oxygen species (ROS) in different structures, which may override the cellular antioxidant defences and can lead to oxidative stress (Vicente et al., 2004). Interestingly, the abundance of polyunsaturated fatty acids and the low-level of defensive mechanisms, together with the high oxygen consumption, make the brain more susceptible to oxidative damage than other organs. In particular, after experimental noise exposure in laboratory animals, superoxide anion radicals emerge in the stria vascularis (Yamane et al., 1995), hydroxyl radicals significantly increase in the cochlea (Henderson et al., 2008), hydrogen peroxide-induced cell damage to the inner ear occurs in vitro (Dehne et al., 2000), glutathione increases in the lateral wall (Yamasoba et al., 1998) and glutathione peroxidase and malondialdehyde activities increase progressively with noise intensity in hair cells (Yamashita et al., 2004). Although experimental data show that loud noise can increase ROS in the auditory pathway (Yamashita et al., 2004; Pouyatos et al., 2005; Le Prell et al., 2007), reports concerning the influence of noise stress on oxidative status in extra-auditory CNS structures are scarce (Manikandan et al., 2006; Rabat et al., 2006; Turner et al., 2005).

In addition, experimental data in developing animals are lacking. In consequence, since the tonotopic organization of the auditory cortex is much more susceptible to perturbations of acoustic inputs in infancy than in older animals, as suggested by Wang studies (2004), the concept of a 'critical' or sensitive period of development after which plasticity is more restricted could be tested by exposing immature animals(Wang, 2004). The adverse effects of environmental noise on human mental health have been reported by several authors. In particular, it has been suggested that loud noise can induce a variety of symptoms, including changes in anxiety, emotional stress, increase in social conflicts, as well as general psychiatric disorders (Rabat et al., 2006). Similarly, animal experiments have demonstrated that acute and chronic noise exposures can also induce temporary or permanent changes related to the central nervous system (CNS)(Goble et al., 2009; Manikandan et al., 2006).

iii. NOISE AND HEARING LOSS

Noise-induced hearing loss (NIHL) acquired in leisure or occupational settings is a common cause of hearing impairment in industrialized countries, with a prevalence second only to age-related hearing loss (ARHL) (Stanbury 2008). Hearing loss associated with mild acoustic overexposure is reversible, and hearing recovers within 2–3 weeks [Dengerink 1985]. This temporary loss is known as a temporary threshold shift (TTS), and is probably due to reversible damage to the stereocilia of hair cells [Gao 1992] and/or swelling, followed by recovery, of cochlear nerve terminals [Pujol 1999]. Noise intensity and duration of exposure determine the level of noise damage to an organism.

High-intensity noise exposure damages inner hair cells (IHCs), primarily through two pathways: direct mechanical damage, in which noise can destroy the static cilia of hair cells, resulting in hair cell loss and damage to supporting cells and spiral ganglia (Shukla et al., 2019)

iv. NOISE AND COGNITIVE FUNCTION

The damaging effect of noise, however, is not limited in the auditory system, but extend to many other systems (Basner et al.,2014). Recent studies have warned of noise-related impairment of learning ability and cognitive performance(Cheng et al., 2011; Cui et al., 2009; Wright et al., 2006). Studies suggest that the evidence of the effects of noise on children's cognition has grown stronger over recent years, with over 20 studies showing detrimental effects of noise on children's memory and reading outcomes [Belojevic 2012]. children exposed to chronic aircraft or road traffic noise at school have poorer reading comprehension and memory than children who are not exposed [Haines 2001].

v. HYPOTHESIZED RELATIONSHIPS BETWEEN HEARING LOSS AND COGNITIVE DECLINE

Several possible relationships have been postulated (see Figure 1). Cognitive decline may reduce the cognitive resources that are available for auditory perception, manifesting as hearing loss. In contrast, Lin and colleagues (Lin et al., 2013) have suggested that hearing loss causes cognitive decline that is either permanent (the "sensory-deprivation"), or potentially remediable (the "information-degradation"). Another possibility is that a third factor causes both declines (the "common cause").

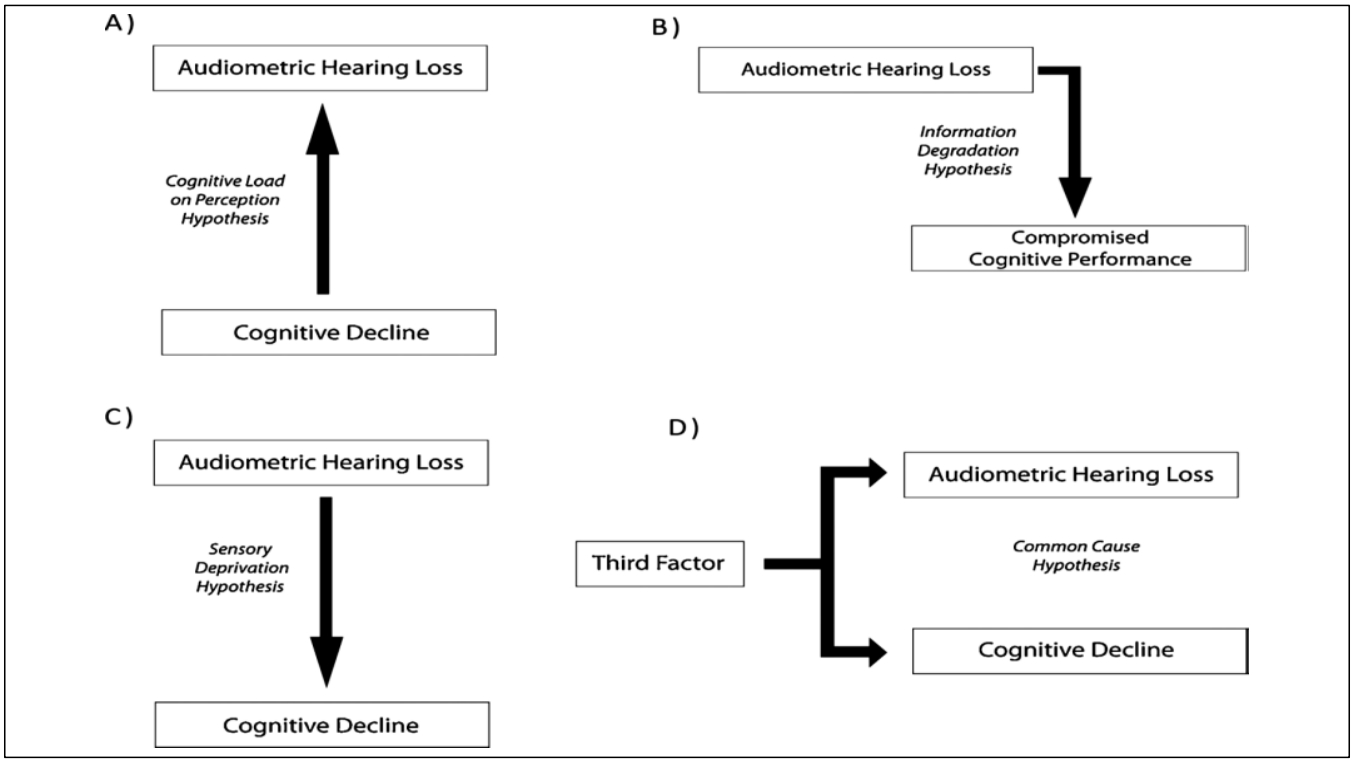

Figure 1: A summary of the four hypotheses for the causal relationship between hearing loss and cognitive decline. (A) Cognitive load on perception hypothesis: cognitive decline leads to audiometric hearing loss; (B) Information degradation hypothesis: audiometric hearing leads to diminished (but reversible) cognitive performance; (C) Sensory deprivation hypothesis: audiometric hearing loss causes cognitive decline; (D): Common cause hypothesis: both audiometric hearing loss and cognitive decline are caused by a third, common factor.

Soldiers who were exposed to unnecessary noise levels including explosions and blast waves shown severe NIHL and tinnitus (Kapoor 2019), as well as cognitive discrepancies and memory impairment (Belanger et al., 2009). The mechanisms underlying cognitive function decline after noise exposure are not entirely clear. Noise exposure impair cognitive functions through different pathway. One is related to the oxidative reaction initiated by noise exposure. Increased oxidative stress has been reported in many studies as the cause of neuronal degeneration seen in many auditory nuclei as well as in the brain regions critical for cognitive functions (Shukla 2019, 2020). The other way is due to change of auditory input to the cognitive brain after hearing loss induced by noise. This latter approach has not been considered intensively in the past, but the possibility has been supported by the connection between the auditory brain and cognitive brain (Kraus and Canlon, 2012)and established by the hippocampal deterioration and deteriorated spatial memory in C57BL/6J mice with age related hearing loss (Yu et al., 2011) and the destruction of hippocampal neurogenesis in the rat after noise-induced unilateral hearing loss (Kraus et al., 2010) (Shukla 2019; 2020).

In the auditory system, environmental noise induces cell death (Basta, et al., 2005) and a threshold shift (Syka and Rybalko, 2000), and causes abnormal neural coding

(Pienkowski and Eggermont 2012) in the auditory cortex (AC) and other cortical regions responsible for acoustic processing (Cheng 2016). In contrast, environmental noise also affects non-auditory brain regions such as the hippocampus, a major limbic region that receives direct or indirect neural input from the central auditory system (Kraus and Canlon, 2012). Accumulating evidence illustrates a significant effect of noise on hippocampal-related cognition (Manikandan, et al., 2006). A number of studies including our own have highlighted that exposure to moderate noise can affect the physiological structure and subsequently the function of both the AC and hippocampus (Cheng, et al., 2011). However, does the AC, the primary brain region to process acoustic information, have high susceptibility to environmental noise than the hippocampus in non-auditory system? It has not been determined. Previous studies have shown that as a stressor, noise may cause extensive oxidative stress along the lemniscal ascending pathway including the AC and the hippocampus (Manikandan, et al., 2006; Cheng, et al., 2011). A moderate level of noise was used to avoid direct physical damage. The central nervous system undergoes progressive structural and functional maturation during postnatal development, and noise exposure, as a stressor, may therefore affect brain function of mice to a greater extent at this stage than in adults (Chang and Merzenich, 2003).

vi. NOISE AND CARDIOVASCULAR RISK

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) (Basner 2014). Acute exposure to different kinds of noise is associated with arousals of the autonomic nervous system and endocrine system (Lusk 2004). 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) Basner 2014. 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 homoeostasis (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 (Basner 2014; Babisch 2011). These changes increase the risk of hypertension, arteriosclerosis, and are related to severe events, such as myocardial infarction and stroke. Studies of occupational (Davies 2012) and environmental epidemiology have shown a higher prevalence and incidence of cardiovascular diseases and mortality in highly noise-exposed groups (Sørensen 2012) .

CONCLUSION

In summary, there is sufficient evidence for the effects of environmental noise in human being on hormonal changes, annoyance, well-being and cognitive effects such as reading comprehension, long-term memory and performance. The review finding that aircraft/environmental noise is related to hyperactivity symptoms requires some systematic investigation and undoubtedly the prolonged use of personal listening devices on hearing needs to be assessed in longitudinal studies, not least because of the public health implications of almost universal use in young people.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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