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EFFECT OF NOISE ON MENTAL HEALTH: A BRIEF REVIEW OF PRECLINICAL STUDIES

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ABSTRACT

Background: Noise pollution, a pervasive environmental issue, has been linked to a variety of health problems, including mental health disorders. Recent evidence suggests that chronic exposure to noise can exacerbate stress and affect cognitive functions, yet the mechanisms underlying these effects remain insufficiently understood. Preclinical studies using animal models provide crucial insights into how noise exposure impacts mental health, shedding light on potential pathways for intervention.

Objective: This review aims to synthesize findings from preclinical studies on the effects of noise-induced mental stress, focusing on the underlying mechanisms and potential therapeutic targets.

Methods: A comprehensive literature search was conducted to identify relevant preclinical studies investigating the impact of noise on mental health. Studies were selected based on their examination of noise exposure effects on behavior, stress responses, and brain function in animal models. We searched multiple electronic databases, including PubMed, Web of Science, and Scopus to identify relevant literature using keywords like, noise pollution, road traffic noise, mental health, Hippocampus, Behavioral Changes, Neurological effect, etc.

Results: Preclinical studies demonstrate that chronic noise exposure induces stress-related behaviors such as anxiety and depression in rodents. Mechanistically, noise exposure affects brain regions involved in stress regulation, including the hippocampus and amygdala. These studies also reveal alterations in neurotransmitter systems and inflammatory responses associated with noise-induced mental stress.

Conclusion: Preclinical research provides valuable insights into the impact of noise on mental health, highlighting significant behavioral and physiological changes associated with chronic exposure. Understanding these mechanisms is crucial for developing targeted interventions to mitigate the adverse effects of noise pollution on mental well-being.

Keywords: Noise, Mental Health, Preclinical Studies, Hippocampus, Behavioral Changes, Neuroprotection, Oxidative Stress

1.0 INTRODUCTION

1.1 Background

According to a World Health Organization (WHO) report, noise pollution is the third most dangerous type of pollution, following air and water pollution (**Basu B et al., 2021**). Noise is considered an environmental stressor that negatively impacts quality of life and wellbeing. Excessive noise not only hampers communication and concentration but also provokes emotional responses like annoyance (**Krittanawong C et al., 2023**). Rapid urbanization and changing lifestyles have made loud noise a common part of life, leading to significant health risks from both indoor and outdoor environmental noise pollution. The harmful effects of noise pollution are increasingly being seen across all age groups, including fetuses, infants, children, adolescents, and adults (**Gupta A et al., 2018**). Research shows that prolonged exposure to high noise levels is associated with various non-auditory effects, such as mental health problems like anxiety and depression, increased risk of hypertension, hormonal disruptions, adverse birth outcomes, sleep disorders, and cardiovascular diseases related to high blood pressure (**Zaman M et al., 2022**). A study by Bustaffa found that noise exposure can indirectly cause stress, lead to psychological symptoms and disorders, and contribute to brain and cardiovascular issues (**Bustaffa E et al., 2022**). Similarly, a recent cross-sectional study involving 1,005 state government employees in Malaysia confirmed the link between occupational noise exposure and the risk of hypertension, with a hypertension prevalence of 18.8% among noise-exposed workers (**Lee FYL et al., 2023**). This study is among the few in the ASEAN region to explore the relationship between occupational noise and hypertension, taking into account cultural and lifestyle differences. Additionally, Owolawi suggested that continuous exposure to noise levels between 85 and 90 dBA in industrial settings over a lifetime could lead to reduced hearing perception, resulting in hearing loss and an increased threshold for hearing sensitivity (**Owolawi IV, 2021**). Noise pollution, characterized by unwanted or harmful sounds, has emerged as a significant environmental health issue due to rapid urbanization and industrial growth. This type of pollution not only causes auditory disturbances but also impacts overall health, contributing to conditions such as cardiovascular diseases, sleep disorders, and cognitive impairments (**Basner et al., 2014**). Recent studies indicate that noise exposure can also influence mental health, particularly by exacerbating stress and affecting cognitive functions (**Stansfeld & Matheson, 2003**). The increasing prevalence of noise-related health issues necessitates a deeper understanding of how noise impacts mental health, which is where preclinical studies play a crucial role.

Preclinical research, particularly involving animal models, provides valuable insights into the mechanisms by which noise affects mental health. These studies help elucidate the biological pathways through which noise exposure induces stress and alters brain function (**Vasconcelos RO et al., 2023**). By understanding these mechanisms, researchers can identify potential targets for therapeutic intervention and develop strategies to mitigate the negative effects of noise. Though often overshadowed by more noticeable environmental issues, noise pollution is a widespread and increasing problem that requires our focus, innovative solutions, and collective effort to address. In the upcoming sections of this review, we will explore the causes of noise pollution, examine its harmful effects on human health, cognitive function, society, and overall well-being, and outline measures for its mitigation.

1.2 Noise Pollution Sources

A detailed understanding of the sources of noise pollution is essential for developing effective strategies to reduce its impact. The key factors contributing to noise pollution, both natural and human-made, are outlined below.

Industrial Machinery: Noise in industrial settings arises from various processes involving impact, reciprocating movements, vibrations, friction, and turbulence in air or gas streams (Maqsood N et al., 2019).

Construction Noise: Construction noise is characterized by its sudden, temporary nature, high intensity, and limited duration, making it difficult to control and significantly affecting urban residents (Mir M et al., 2023). Major sources of construction noise include knocking, hammering, piling, welding, and material transportation (Shaikh HH et al., 2023).

Rail Traffic: Railways are a significant noise source, producing high noise levels in a short time, which can be harmful to human health. Noise from railways is mainly generated by train frequency, speed, the condition of the tracks, and the intensity of train horns (Polak K et al., 2021).

Air Traffic: Aircraft noise is intermittent, with successive noise events separated by periods of silence. During take-off, the primary noise source is the aircraft engines, while aerodynamic noise from flaps, gears, and other components becomes more prominent during landing (Gely D et al., 2022).

Road Traffic: Road traffic is the leading cause of community noise, with levels increasing as traffic volumes rise. Road traffic noise depends on factors such as traffic volume, vehicle speed, and the presence of heavy vehicles and motorcycles. At low speeds, the primary noise source is the power transfer mechanism, while at speeds above 30–50 km/h, tire noise dominates, and aerodynamic noise becomes significant at speeds over 80 km/h (Grubesa S et al., 2020).

While various sources contribute to urban noise pollution, road traffic remains the most significant, leading to physiological effects that increase the overall disease burden. Globally, road traffic noise is a major environmental pollutant, with estimates suggesting an annual loss of about 1 million healthy life years in Western Europe alone (Moroe N et al., 2022).

1.3 Burden of Noise-Induced Mental Stress

The burden of noise-induced mental stress is substantial and multifaceted. Epidemiological evidence links chronic noise exposure to a range of mental health issues, including anxiety and depression (Gong X et al., 2022). Individuals living in high-noise environments, such as near busy roads or airports, are more likely to experience mental health problems compared to those in quieter areas (Lim J et al., 2018). This association is corroborated by preclinical studies, which demonstrate that noise exposure can induce stress-related behaviors and physiological changes in animal models (Hahad O et al., 2022). The economic impact of noise-induced mental stress is also significant. According to the **World Health Organization (WHO 2011)**, noise pollution contributes to increased healthcare costs and reduced quality of life. The burden extends beyond direct health effects to include impacts on productivity and overall well-being. Addressing noise-induced mental stress is therefore crucial for improving public health and reducing economic costs.

1.4 Challenges in Understanding Noise-Induced Mental Stress

Several challenges complicate the study of noise-induced mental stress. One major issue is the complexity of noise exposure itself. Noise is a multifaceted stimulus that varies in frequency, intensity, and duration, making it challenging to isolate specific effects and establish clear

causal relationships (Hahad O et al., 2019). This complexity can obscure the precise mechanisms through which noise affects mental health. Another challenge is the variability in individual responses to noise. Factors such as genetic predispositions, pre-existing mental health conditions, and environmental influences can affect how individuals respond to noise exposure (Munzel et al., 2014). This variability makes it difficult to develop universal guidelines and interventions. In preclinical research, translating findings from animal models to human conditions presents additional challenges. Differences in noise exposure patterns, brain structure, and behavioral responses between species can affect the relevance of preclinical results to human health (Goines & Hagler, 2007). Moreover, inconsistencies in study design and methodologies across research studies can lead to varying results and hinder progress (Stansfeld & Matheson, 2003). Chronic noise exposure can also lead to increased stress levels, which may further impair cognitive function (Tao Y et al., 2020). In a study involving 2,680 children aged 7–10 years in Barcelona, Spain, researchers investigated the impact of road traffic noise exposure at both school and home on cognitive development over one year, using standardized computerized cognitive assessments. The findings indicated that higher traffic noise exposure at school, especially noise fluctuations within classrooms, was associated with slower development of working memory and attention. However, no significant link was found between traffic noise exposure at home and cognitive development (Foraster M et al., 2022).

1.5 Mechanistic Approach of Noise-Induced Mental Stress

Understanding the mechanisms through which noise induces mental stress is crucial for developing effective interventions and mitigating its effects. Noise-induced mental stress involves a complex interplay of sensory processing, neural activation, and physiological responses. This section outlines the key mechanistic pathways involved in noise-induced mental stress, focusing on sensory input, brain region activation, oxidative stress, inflammation, and resultant behavioural and physiological changes.

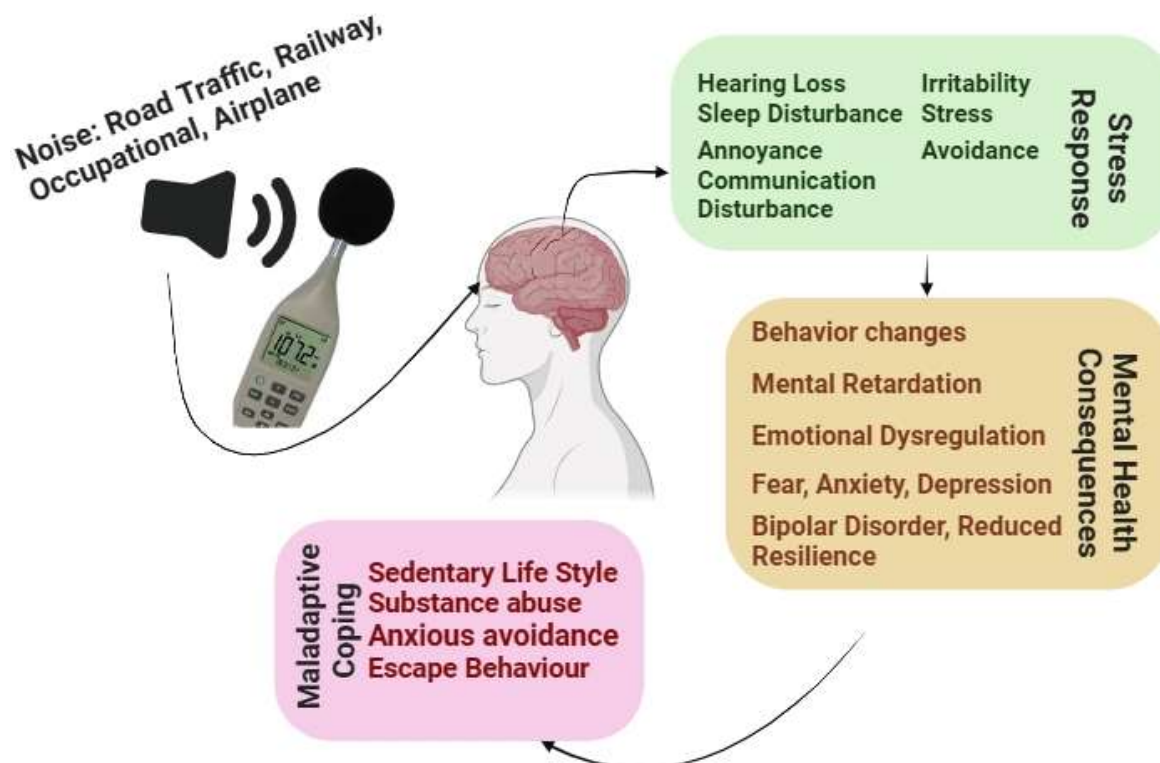


Figure 1: Consequences of Noise on Mental Health

(Various sources, including road traffic, railways, airplanes, and occupational environments, serve as chronic noise environmental stressors. Upon exposure, the auditory system triggers a stress response, leading to hearing loss, sleep disturbances, communication disruption, irritability, and elevated stress levels. Persistent stress from noise exposure can result in significant mental health consequences, such as behavioral changes, cognitive impairments, emotional dysregulation, anxiety, depression, and reduced psychological resilience. In response, individuals may adopt maladaptive coping strategies, including a sedentary lifestyle, substance abuse, and anxious avoidance, which further exacerbate mental health challenges.)

Sensory Processing and Noise Perception

The first step in the mechanistic pathway of noise-induced mental stress involves the perception of noise by the auditory system. Noise is detected by the cochlea in the inner ear, which transduces sound waves into neural signals. These signals are then transmitted to the auditory cortex via the auditory nerve and brainstem pathways. The auditory cortex processes the acoustic information and integrates it with other sensory inputs to create a coherent perception of the noise environment. Chronic exposure to high levels of noise can overwhelm the auditory system, leading to altered sensory processing and heightened sensitivity to sound stimuli. This heightened sensitivity can contribute to increased stress responses and anxiety (Jafari MJ et al., 2019).

Neural Activation and Brain Region Responses

Once noise is perceived, it activates several brain regions involved in stress response and emotional regulation. The hippocampus, prefrontal cortex, and amygdala are particularly important in mediating the effects of noise-induced stress.

Hippocampus

The hippocampus plays a key role in memory formation and stress regulation. Noise exposure has been shown to impact hippocampal structure and function, leading to cognitive and emotional changes. Chronic noise exposure can decrease neurogenesis and alter synaptic plasticity in the hippocampus, contributing to impairments in learning and memory. Noise-induced oxidative stress in the hippocampus reduces neurogenesis and disrupts hippocampal-dependent cognitive functions (McEwen BS et al., 2015).

Prefrontal Cortex

The prefrontal cortex is involved in higher-order executive functions, including decision-making and emotional regulation. Noise exposure can impair prefrontal cortex function, leading to deficits in cognitive control and emotional stability. Chronic noise exposure reduces prefrontal cortex activity, affecting working memory and attentional control. Structural changes, such as reduced dendritic spine density, further contribute to these cognitive deficits (Jafari Z et al., 2018).

Amygdala

The amygdala is crucial for processing emotions, particularly fear and anxiety. Noise exposure has been shown to increase amygdala activity, leading to heightened anxiety and stress responses. Noise-induced hyperactivity in the amygdala is associated with increased anxiety-like behaviors. This hyperactivity is often accompanied by changes in neurotransmitter systems and increased inflammatory responses (Hahad O et al., 2024).

Oxidative Stress and Inflammation

Oxidative stress and inflammation are key physiological mechanisms through which noise exposure affects brain function and mental health. Noise-induced oxidative stress results from an imbalance between reactive oxygen species (ROS) and antioxidant defenses. Elevated ROS levels can lead to neuronal damage and disrupt normal brain function. Chronic noise exposure increases oxidative stress markers in the hippocampus, contributing to neuronal damage and cognitive deficits (**Paciello F et al 2023**). Additionally, noise exposure can trigger inflammatory responses in the brain. Inflammatory cytokines, such as interleukin-1 β and tumor necrosis factor-alpha, are elevated following noise exposure, leading to further neuronal damage and altered brain function (**Shukla M et al., 2020**).

Behavioural and Physiological Changes

The activation of stress pathways and subsequent oxidative stress and inflammation lead to a range of behavioral and physiological changes. Individuals exposed to chronic noise may exhibit increased anxiety, depression, and cognitive impairments. Behavioral changes include heightened sensitivity to stressors, impaired memory, and decreased cognitive performance (**Chu B et al., 2024**).

Recent studies have explored the impact of noise on physiological markers of stress, such as cortisol levels and blood pressure. Chronic noise exposure elevates cortisol levels, indicating increased stress response. Similarly, noise exposure is associated with increased blood pressure, reflecting the physiological impact of noise-induced stress (**Sivakumaran K et al., 2022**).

2.0 NOISE-INDUCED MENTAL STRESS IN LABORATORY ANIMALS

2.1. Impact on the Hippocampus

Effects of Noise on Hippocampal Function and Structure

The hippocampus, a critical region for learning, memory, and emotional regulation, is significantly affected by chronic noise exposure. Research demonstrates that noise-induced stress leads to structural and functional changes in the hippocampus, impacting its role in cognitive and emotional processes. Noise exposure results in hippocampal atrophy, characterized by reductions in hippocampal volume and alterations in dendritic architecture. These structural changes are associated with impairments in spatial memory and learning abilities (**Zhang Y et al., 2021**).

Chronic noise exposure elevates glucocorticoid levels, which can inhibit neurogenesis in the hippocampus. Elevated cortisol levels are known to affect hippocampal neurons, leading to impaired synaptic plasticity and increased susceptibility to stress (**Lupien et al., 2009**). Moreover, noise-induced oxidative stress and inflammation further contribute to hippocampal damage. Increased oxidative stress leads to the production of reactive oxygen species (ROS), which can damage neuronal cells and exacerbate stress-induced hippocampal dysfunction (**Daiber A et al., 2020**).

Summary of Studies Examining Noise-Induced Hippocampal Changes

Several studies have explored the impact of noise on hippocampal structure and function in animal models (**Table 1**). For example, rodents exposed to chronic noise have shown significant impairments in spatial memory tasks, such as the Morris water maze, which are linked to reductions in hippocampal dendritic spine density and neuronal proliferation (**Goines & Hagler, 2007**). Another study demonstrated that noise exposure increases the expression of pro-inflammatory cytokines in the hippocampus, correlating with behavioral changes

indicative of anxiety and depression (**Hahad O et al., 2024**). These findings underscore the detrimental impact of chronic noise on hippocampal health and function.

2.2. Impact on the Prefrontal Cortex

Mechanisms of Noise-Induced Stress Affecting Cognitive Functions

The prefrontal cortex (PFC) is essential for executive functions, including decision-making, working memory, and cognitive control. Chronic noise exposure disrupts PFC function through several mechanisms. One primary mechanism involves alterations in neurotransmitter systems. Noise exposure has been associated with reduced levels of dopamine and serotonin in the PFC, which are critical for maintaining cognitive processes and emotional regulation (**Stansfeld & Matheson, 2003**).

Chronic noise also affects the structural integrity of the PFC. Noise-induced stress has been shown to decrease dendritic branching and spine density in the PFC, which can impair synaptic plasticity and cognitive functions (**Koolhaas et al., 2011**). Additionally, noise exposure can lead to alterations in brain-derived neurotrophic factor (BDNF) levels, which play a crucial role in maintaining neuronal health and cognitive function (**Pisani A et al., 2023**).

Evidence from Preclinical Models

Preclinical studies provide valuable insights into the impact of noise on the PFC. For example, rodent models exposed to chronic noise exhibit significant impairments in working memory and executive functions, which correlate with structural changes in the PFC (**Munzel et al., 2014**). Electrophysiological recordings in these models reveal altered neuronal firing patterns and reduced synaptic transmission in the PFC, highlighting the functional consequences of noise-induced stress (**Morrison & Baxter, 2012**). Additionally, studies using optogenetics and pharmacological interventions have demonstrated that noise-induced changes in neurotransmitter systems and PFC connectivity contribute to cognitive deficits (**McEwen & Gianaros, 2010**).

2.3. Impact on Other Brain Regions

Overview of Findings from Studies on the Amygdala, Hypothalamus, and Other Relevant Areas

In addition to the hippocampus and PFC, other brain regions are also affected by chronic noise exposure. The amygdala, a key structure involved in emotional processing, exhibits heightened activity in response to chronic noise. Noise exposure has been shown to increase the expression of stress-related biomarkers, such as corticotropin-releasing hormone (CRH) and glucocorticoid receptors, in the amygdala (**Kim & Yoon, 1998**). This heightened activity is associated with increased anxiety-like behaviors and altered emotional responses in noise-exposed animals (**Peng X et al., 2023**).

The hypothalamus, which regulates the hypothalamic-pituitary-adrenal (HPA) axis, is also impacted by chronic noise exposure. Noise exposure has been linked to dysregulation of the HPA axis, including increased release of CRH and other stress hormones (**McEwen & Gianaros, 2010**). This dysregulation can lead to alterations in stress responses and emotional regulation.

Other brain regions, such as the striatum and periaqueductal gray, also show changes in response to chronic noise exposure. The striatum, involved in reward processing, exhibits altered dopaminergic activity, which can affect motivation and reward-related behaviors (**Newsom RJ et al., 2012**). The periaqueductal gray, involved in pain modulation, shows

changes in neuronal activity and neurotransmitter levels following noise exposure (**Goines & Hagler, 2007**). Table 1 represents the outcomes of preclinical studies involving different noise level using various animal model.

Table 1: Few Evidences Based On Preclinical Studies

Ref.	Noise Induction	Animal Model	Intervention	Outcomes
Naqvi F et al., 2012	100 dB, 4 h daily for 15 days	Adult male albino rats of Wistar strain	-	Decreased tendency to move in open field, decrease in time spent in open arm and number of entries in open arm in rats, and increase in immobility time in tail suspension test
Ha S et al., 2021	115 dB, 4 hours per day for 3 days	Sprague-Dawley rats	-	miR-758-5p, miR210-5p, miR370-5p, miR-652-5p, miR-3544, miR-128-1-5p, miR-665, miR-188-5p, and miR-874-5p up regulated, whereas, miR-448-3p, miR-204-5p, miR-204-3p, and miR-204-3p were down regulated in rat hippocampi with noise-induced hearing loss
Guthrie OW et al., 2016	6 h per day, five days/week for four weeks	female Fischer344 rats	20 inhalation exposures to 1000mg/m ³ of jet propulsion fuel-8 (Organic Solvent)	Fuel plus noise exposure alter neural responses
Zhuang H et al., 2020	Sound pressure level of 123 dB for 2 h	CBA mice	-	age-related decline in hippocampal neurogenesis accelerated in mice with noise induced hearing loss
Cui B et al., 2012	100 dB sound pressure level, 4 h/d×14 d	male Wistar rats	-	Significant increased levels of tau phosphorylation, and generation of Long-lasting pathological neurofibrillary tangle
Cunha AOS et al., 2018	1 minute of 110 dB sound, applied two times per day for 10 days	Male Wistar rats	-	Decreased resting membrane potential, increased membrane input resistance and time constant, and decreased action potential threshold. High-intensity sound affects intrinsic membrane properties of hippocampal pyramidal neurons, mainly by decreasing the amplitude of I _h
Molina SJ et al., 2021	2 Hr to white noise at 95–97 dB SPL for 1 or 5 days	Male and female albino Wistar rats (pups used at 7 or 15 PND)	Enriched environment	Exposure to noise induced changes in hippocampal oxidative state in the Short T, as well as variations in ROS levels, antioxidant defences and aminoacidergic

				neurotransmission markers during early/mid adolescence at Long T.
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3. CHALLENGES AND SOLUTIONS

3.1. Challenges

Methodological Limitations in Preclinical Studies

Preclinical studies provide valuable insights into the effects of noise on mental health, yet several methodological limitations impact their reliability and generalizability. One major limitation is the variability in experimental designs, including differences in noise exposure parameters such as intensity, duration, and frequency. These variations can lead to inconsistent findings across studies, complicating efforts to draw definitive conclusions (**Lim J et al., 2018**).

Another challenge is the use of different animal models, which may not fully replicate human responses to noise. Rodent models, commonly used in preclinical research, have different auditory processing mechanisms and stress responses compared to humans, which can limit the applicability of findings (**Reul & de Kloet, 1985**). Additionally, the lack of standardization in behavioural testing methods across studies can introduce variability in results and hinder the comparison of findings (**Miller & O'Callaghan, 2002**).

Variability in Noise Exposure Protocols

The variability in noise exposure protocols is a significant challenge in preclinical research. Studies often differ in terms of noise sources, exposure levels, and duration, which can affect the reproducibility and interpretation of results. For example, some studies use continuous white noise, while others employ intermittent noise or specific frequencies, leading to inconsistent effects on mental health outcomes (**Basner et al., 2014**). Moreover, the intensity of noise exposure used in studies can vary widely, from relatively low levels to those that are uncomfortably loud. This variability makes it difficult to determine dose-response relationships and to establish clear thresholds for noise-induced mental stress (**Goines & Hagler, 2007**). Standardizing noise exposure protocols and clearly defining exposure parameters are essential for improving the reliability and comparability of preclinical studies.

Translation of Animal Models to Human Contexts

Translating findings from animal models to human contexts presents another significant challenge. While preclinical studies offer insights into potential mechanisms of noise-induced mental stress, the direct applicability of these findings to human populations is not always straightforward. Differences in auditory perception, stress response, and brain structure between animals and humans can limit the relevance of animal model findings (**McEwen BS, 2007**). Additionally, human exposure to noise is often complex and multifactorial, involving not only the physical characteristics of the noise but also psychological and social factors that may not be fully captured in animal models (**Basner et al., 2014**). Bridging the gap between animal research and human health requires careful consideration of these differences and the development of translational strategies that account for the complexities of human noise exposure.

3.2. Solutions

Recommendations for Improving Study Design

To address the methodological limitations in preclinical studies, several recommendations can be made. Standardizing noise exposure protocols is crucial for ensuring consistency and reproducibility. Researchers should establish clear guidelines for noise parameters, including intensity, frequency, and duration, to facilitate comparison across studies (**Basner et al., 2014**). Improving the selection of animal models is also important. Using a range of models that better reflect human auditory and stress responses can enhance the translational relevance of findings. For example, incorporating models with varying genetic backgrounds and pre-existing conditions may provide a more comprehensive understanding of how noise affects different populations (**Koolhaas et al., 2011**). Furthermore, adopting standardized behavioral testing methods and reporting practices can improve the comparability of results. Implementing rigorous experimental protocols and statistical analyses will help mitigate variability and enhance the reliability of findings (**Miller & O'Callaghan, 2002**).

Advances in Noise Exposure Technologies and Analytical Methods

Recent advances in noise exposure technologies and analytical methods offer promising solutions to the challenges faced in preclinical research. Newer technologies, such as programmable sound generators and automated exposure systems, allow for precise control of noise parameters and better mimic real-world noise environments (**Estrella I et al., 2024**). These advancements enable researchers to create more consistent and realistic noise exposure conditions, improving the relevance of findings. In terms of analytical methods, advancements in neuroimaging and electrophysiological techniques provide deeper insights into the effects of noise on brain function. Techniques such as functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) can be used to assess changes in brain activity and connectivity associated with noise exposure (**Munzel et al., 2014**). Additionally, novel biomarkers and molecular assays can help identify underlying mechanisms and pathways affected by noise (**Tang S et al., 2022**).

Strategies for Better Integration of Preclinical Findings into Human Health Contexts

To improve the integration of preclinical findings into human health contexts, it is essential to develop translational strategies that bridge the gap between animal research and human applications. One approach is to use computational modelling and simulation to predict human responses based on preclinical data. These models can incorporate various factors, such as individual differences and environmental conditions, to provide more accurate predictions of human outcomes (**McEwen & Gianaros, 2010**). Collaborative efforts between researchers, clinicians, and policymakers can also facilitate the translation of findings into practical applications. By aligning preclinical research with public health priorities and clinical needs, researchers can ensure that their findings address relevant issues and contribute to the development of effective interventions. Furthermore, incorporating human data into preclinical studies, such as through the use of human-derived cell cultures or brain organoids, can enhance the relevance of findings and provide insights into human-specific effects of noise exposure. This integrative approach helps ensure that preclinical research translates effectively into human health contexts.

4. FUTURE PERSPECTIVES

Emerging Research Directions and Technologies

As we advance in our understanding of noise-induced mental stress, several emerging research directions and technologies hold promise for further elucidating the mechanisms involved and developing effective interventions. One key area of interest is the use of advanced neuroimaging techniques to investigate the neural correlates of noise-induced stress in greater detail. Functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) are increasingly employed to visualize brain activity and neurotransmitter dynamics in response to noise exposure (**Salzman & Fusi, 2010**). These technologies allow for the identification of specific brain regions and pathways affected by noise, providing insights into the neural mechanisms underlying mental stress. Another promising direction is the integration of genetic and epigenetic approaches to understand individual susceptibility to noise-induced stress. Advances in genomics have enabled researchers to investigate how genetic variations influence the response to noise exposure and contribute to mental health outcomes (**Sullivan et al., 2012**). Epigenetic modifications, such as DNA methylation and histone modifications, may also play a role in how noise exposure affects gene expression and stress responses (**Leso V et al., 2020**). Understanding these genetic and epigenetic factors could lead to more targeted interventions and personalized approaches to managing noise-induced mental stress. Recent technological advancements in noise exposure systems, such as virtual reality (VR) and programmable sound environments, are also paving the way for more controlled and realistic noise exposure studies. VR systems allow for the simulation of complex and dynamic noise environments, providing a more immersive and accurate representation of real-world noise exposure (**Geronazzo M et al., 2023**). Programmable sound systems enable precise control over noise parameters, such as intensity, frequency, and duration, facilitating more standardized and reproducible research (**Kang H et al., 2021**).

Potential for Personalized Interventions and Treatments

The potential for personalized interventions and treatments is a significant area of development in addressing noise-induced mental stress. Personalized medicine involves tailoring interventions based on individual characteristics, such as genetic predisposition, environmental factors, and personal preferences. In the context of noise-induced stress, personalized approaches could include:

Genetic Profiling: By identifying genetic markers associated with susceptibility to noise-induced stress, personalized interventions can be developed to target specific vulnerabilities. For example, individuals with certain genetic variants may benefit from specific stress-reducing strategies or pharmacological treatments (**Sullivan et al., 2012**).

Customized Noise Reduction Strategies: Personalized noise reduction strategies could be developed based on individual noise sensitivity and preferences. This could include the use of noise-canceling devices, sound masking technologies, or tailored acoustic environments designed to mitigate the impact of noise on mental health (**Jaschke AC et al., 2023**).

Behavioral and Cognitive Interventions: Personalized behavioral and cognitive interventions, such as mindfulness-based stress reduction (MBSR) or cognitive-behavioral therapy (CBT), can be tailored to address the specific stressors and coping mechanisms of individuals affected by noise (**Kabat-Zinn, 1990**). These interventions can be adapted based on individual needs and preferences to enhance their effectiveness.

Pharmacological Treatments: Advances in pharmacology may lead to the development of personalized medications that target specific neurobiological pathways affected by noise

exposure. For example, drugs that modulate neurotransmitter systems or reduce inflammation may be tailored to individuals based on their specific neurobiological profiles (**Miller & O'Callaghan, 2002**).

Role of Policy and Public Health Initiatives in Mitigating Noise Pollution

Addressing noise-induced mental stress also requires effective policy and public health initiatives. Policymakers and public health agencies play a crucial role in mitigating noise pollution and protecting mental health. Key strategies include:

Noise Regulation and Standards: Implementing and enforcing noise regulation and standards is essential for reducing noise pollution in urban environments. This includes setting limits on noise levels from transportation, construction, and industrial sources, as well as promoting the use of quieter technologies and practices (**Basner et al., 2014**).

Urban Planning and Design: Urban planning and design can help mitigate noise pollution by incorporating noise-reducing features, such as green spaces, sound barriers, and zoning regulations. Designing cities to minimize noise exposure and create quiet zones can improve overall quality of life and reduce noise-related mental stress (**Babisch, 2005**).

Public Awareness and Education: Raising public awareness about the effects of noise on mental health and promoting noise-reduction strategies can empower individuals to take proactive measures. Public education campaigns can provide information on the impact of noise pollution and encourage practices such as using ear protection and reducing noise in residential areas (**Abulude FO et al., 2024**).

Research Funding and Support: Supporting research on noise-induced mental stress and its health impacts is critical for developing evidence-based policies and interventions. Funding for research initiatives can help advance our understanding of noise-related health effects and inform the development of effective public health strategies (**Munzel et al., 2014**).

Implications for Mental Health Research and Public Policy

The implications of these findings for mental health research and public policy are substantial. Research into noise-induced mental stress provides valuable insights into the neurobiological mechanisms of stress and its impact on mental health. This knowledge can inform the development of targeted interventions and treatments, as well as guide policy and public health initiatives aimed at reducing noise pollution and its adverse effects. Effective policies and public health strategies are crucial for mitigating noise pollution and protecting mental health. Implementing noise regulations, promoting urban planning strategies, and raising public awareness are key components of a comprehensive approach to addressing noise-induced stress.

Final Thoughts on Future Research Needs and Directions

Future research in the field of noise-induced mental stress should focus on several key areas. These include exploring emerging technologies and research directions, such as advanced neuroimaging, genetic and epigenetic approaches, and personalized interventions. Additionally, efforts to improve study design, standardize noise exposure protocols, and bridge the gap between animal models and human health are essential for advancing our understanding of noise-related health effects. Collaboration between researchers, policymakers, and public health professionals will be critical in translating research findings into effective interventions and policies. By addressing the challenges and leveraging emerging opportunities, we can work towards reducing the impact of noise on mental health and enhancing overall well-being.

5. CONCLUSION

The review of noise-induced mental stress in laboratory animals highlights several key findings. Chronic noise exposure leads to significant alterations in brain function and structure, particularly in regions such as the hippocampus, prefrontal cortex, amygdala, and hypothalamus. These changes are associated with impairments in cognitive functions, emotional regulation, and overall mental health. The impact of noise on these brain regions underscores the importance of understanding the mechanisms underlying noise-induced stress and developing effective interventions. Methodological limitations in preclinical studies, variability in noise exposure protocols, and challenges in translating animal models to human contexts are significant obstacles in this field. Addressing these challenges through standardized protocols, advanced technologies, and personalized approaches is essential for improving the reliability and applicability of research findings.

REFERENCE

1. Abulude FO, Ademilua SO, Gbotoso AO. Noise Pollution in Libraries: Impacts, Strategies, and Recommendations. *Indonesian Journal of Multidisciplinary Research*. 2024;4(1):127-44.
2. Babisch W. Noise and health. *Environ Health Perspect*. 2005 Jan;113(1):A14-5. <https://doi.org/10.1289/ehp.113-a14> PMID: 15631951.
3. Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, Stansfeld S. Auditory and non-auditory effects of noise on health. *Lancet*. 2014 Apr 12;383(9925):1325-1332. [https://doi.org/10.1016/S0140-6736\(13\)61613-X](https://doi.org/10.1016/S0140-6736(13)61613-X) Epub 2013 Oct 30. PMID: 24183105.
4. Basu B, Murphy E, Molter A, et al. Investigating changes in noise pollution due to the COVID-19 lockdown: the case of Dublin, Ireland. *Sustain Cities Soc* 2021;65:102597.
5. Bustaffa E, Curzio O, Donzelli G, et al. Risk associations between vehicular traffic noise exposure and cardiovascular diseases: a residential retrospective cohort study. *Int J Environ Res Public Health* 2022;19:10034.
6. Chu B, Marwaha K, Sanvictores T, et al., Physiology, Stress Reaction. [Updated 2024 May 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK541120/>
7. Corona-Strauss FI, Schick B, Delb W, Strauss DJ. Notched-noise embedded frequency specific chirps for objective audiometry using auditory brainstem responses. *Audiol Res*. 2012 Feb 27;2(1):e7. <https://doi.org/10.4081/audiores.2011.e7> PMID: 26557336; PMCID: PMC4630940.
8. Cui B, Zhu L, She X, Wu M, Ma Q, Wang T, Zhang N, Xu C, Chen X, An G, Liu H. Chronic noise exposure causes persistence of tau hyperphosphorylation and formation of NFT tau in the rat hippocampus and prefrontal cortex. *Exp Neurol*. 2012 Dec;238(2):122-9. <https://doi.org/10.1016/j.expneurol.2012.08.028> Epub 2012 Sep 4. PMID: 22971273.
9. Cunha AOS, Ceballos CC, de Deus JL, Leão RM. Long-term high-intensity sound stimulation inhibits h current (I_h) in CA1 pyramidal neurons. *Eur J Neurosci*. 2018 Jun;47(11):1401-1413. <https://doi.org/10.1111/ejn.13954> Epub 2018 Jun 4. PMID: 29779233.

10. Daiber A, Kröller-Schön S, Oelze M, Hahad O, Li H, Schulz R, Steven S, Münzel T. Oxidative stress and inflammation contribute to traffic noise-induced vascular and cerebral dysfunction via uncoupling of nitric oxide synthases. *Redox Biol.* 2020 Jul;34:101506. <https://doi.org/10.1016/j.redox.2020.101506> Epub 2020 Apr 20. PMID: 32371009.
11. Estrella I, Pacheco A, Marin W, Pacheco-Pumaleque L, Cuba-Carbajal N, Pucuhuayla-Revatta F, Felix-Poicon E, Añaños-Bedriñana M. Tackling Noise: Technology Integration for Improved Noise Pollution Monitoring. *Environ Health Insights.* 2024 Mar 13;18:11786302241235013. <https://doi.org/10.1177/11786302241235013> PMID: 38482524.
12. Foraster M, Esnaola M, López-Vicente M, et al. Exposure to road traffic noise and cognitive development in school children in Barcelona, Spain: a population-based cohort study. *PLoS Med* 2022;19:1004001.
13. Gely D, Marki F. Understanding the basics of aviation noise. In: Leylekian L, Covrig A, Maximova A, eds. *Aviation Noise Impact Management*. Cham: Springer 2022:1–9.
14. Geronazzo M, Barumerli R, Cesari P. Shaping the auditory peripersonal space with motor planning in immersive virtual reality. *Virtual Reality.* 2023 Dec;27(4):3067-87.
15. Goines L, Hagler L. Noise pollution: a modern plague. *South Med J.* 2007 Mar;100(3):287-94. <https://doi.org/10.1097/smj.0b013e3180318be5> PMID: 17396733.
16. Gong X, Fenech B, Blackmore C, Chen Y, Rodgers G, Gulliver J, Hansell AL. Association between Noise Annoyance and Mental Health Outcomes: A Systematic Review and Meta-Analysis. *Int J Environ Res Public Health.* 2022 Feb 25;19(5):2696. <https://doi.org/10.3390/ijerph19052696> PMID: 35270388.
17. Grubesa S, Suhanek M. Traffic noise. In: Siano D, Gonzales AE (eds). *Noise and Environment*. IntechOpen. 2020;1–21.
18. Gupta A, Gupta A, Jain K, Gupta S. Noise pollution and impact on children health. *Indian J Pediatr* 2018;85:300–6.
19. Guthrie OW, Wong BA, McInturf SM, Reboulet JE, Ortiz PA, Mattie DR. Background Noise Contributes to Organic Solvent Induced Brain Dysfunction. *Neural Plast.* 2016;2016:8742725. <https://doi.org/10.1155/2016/8742725> Epub 2016 Jan 18. PMID: 26885406.
20. Ha S, Kim KW, Lee SM, Lee CH, Kim SY. MicroRNA Dysregulation in the Hippocampus of Rats with Noise-Induced Hearing Loss. *Oxid Med Cell Longev.* 2021 Sep 6;2021:1377195. <https://doi.org/10.1155/2021/1377195> PMID: 34527169.
21. Hahad O, Jimenez MT, Kuntic M, Frenis K, Steven S, Daiber A, Muenzel T. Cerebral consequences of environmental noise exposure. *Environment international.* 2022 Jul 1;165:107306.
22. Hahad O, Kuntic M, Al-Kindi S, Kuntic I, Gilan D, Petrowski K, Daiber A, Münzel T. Noise and mental health: evidence, mechanisms, and consequences. *Journal of Exposure Science & Environmental Epidemiology.* 2024 Jan 26:1-8.
23. 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 Nov 11;2019:4623109. <https://doi.org/10.1155/2019/4623109> PMID: 31814877.
24. Jafari MJ, Khosrowabadi R, Khodakarim S, Mohammadian F. The Effect of Noise Exposure on Cognitive Performance and Brain Activity Patterns. Open Access Maced J Med Sci. 2019 Aug 30;7(17):2924-2931. <https://doi.org/10.3889/oamjms.2019.742> PMID: 31844459.
 25. Jafari Z, Kolb BE, Mohajerani MH. Chronic traffic noise stress accelerates brain impairment and cognitive decline in mice. Exp Neurol. 2018 Oct;308:1-12. <https://doi.org/10.1016/j.expneurol.2018.06.011> Epub 2018 Jun 22. PMID: 29936225.
 26. Jaschke AC, Bos AF. Concept and considerations of a medical device: the active noise cancelling incubator. Front Pediatr. 2023 Jul 3;11:1187815. <https://doi.org/10.3389/fped.2023.1187815> PMID: 37465419; PMCID: PMC10350684.
 27. Kabat-Zinn, J. *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain, and illness* (15th anniversary ed.). Delta Trade Paperback/Bantam Dell. 2005.
 28. Kang H, Sung S, Hong J, Jung S, Hong T, Park HS, Lee DE. Development of a real-time automated monitoring system for managing the hazardous environmental pollutants at the construction site. Journal of Hazardous Materials. 2021 Jan 15;402:123483.
 29. Kim JJ, Yoon KS. Stress: metaplastic effects in the hippocampus. Trends Neurosci. 1998 Dec;21(12):505-9. [https://doi.org/10.1016/s0166-2236\(98\)01322-8](https://doi.org/10.1016/s0166-2236(98)01322-8) PMID: 9881846.
 30. Koolhaas JM, Bartolomucci A, Buwalda B, de Boer SF, Flügge G, Korte SM, Meerlo P, Murison R, Olivier B, Palanza P, Richter-Levin G, Sgoifo A, Steimer T, Stiedl O, van Dijk G, Wöhr M, Fuchs E. Stress revisited: a critical evaluation of the stress concept. Neurosci Biobehav Rev. 2011 Apr;35(5):1291-301. <https://doi.org/10.1016/j.neubiorev.2011.02.003> Epub 2011 Feb 21. PMID: 21316391.
 31. Krittanawong C, Qadeer YK, Hayes RB, et al. Noise exposure and cardiovascular health. Curr Probl Cardiol 2023;48:101938.
 32. Lee FYL, Ismail NH, Liew PM, Lim SH. A cross-sectional study of occupational noise exposure and hypertension in Malaysia. Cureus 2023;15:e48758.
 33. Leso V, Fontana L, Finiello F, De Cicco L, Luigia Ercolano M, Iavicoli I. Noise induced epigenetic effects: A systematic review. Noise Health. 2020 Oct-Dec;22(107):77-89. https://doi.org/10.4103/nah.NAH_17_20 PMID: 33402608.
 34. Lim J, Kweon K, Kim HW, Cho SW, Park J, Sim CS. Negative impact of noise and noise sensitivity on mental health in childhood. Noise Health. 2018 Sep-Oct;20(96):199-211. https://doi.org/10.4103/nah.NAH_9_18 PMID: 30516173.
 35. Lim J, Kweon K, Kim HW, Cho SW, Park J, Sim CS. Negative impact of noise and noise sensitivity on mental health in childhood. Noise Health. 2018 Sep-Oct;20(96):199-211. https://doi.org/10.4103/nah.NAH_9_18 PMID: 30516173.
 36. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci. 2009 Jun;10(6):434-45. <https://doi.org/10.1038/nrn2639> Epub 2009 Apr 29. PMID: 19401723.

37. Maqsood N, Younes I, Minallah MN. Industrial noise pollution and its impact on the hearing capacity of workers: a case study of Gujranwala city, Pakistan. *Int J Econ Environ Geol* 2019;10:45–9.
38. McEwen BS, Nasca C, Gray JD. Stress Effects on Neuronal Structure: Hippocampus, Amygdala, and Prefrontal Cortex. *Neuropsychopharmacology*. 2016 Jan;41(1):3-23. <https://doi.org/10.1038/npp.2015.171> Epub 2015 Jun 16. PMID: 26076834.
39. McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol Rev*. 2007 Jul;87(3):873-904. <https://doi.org/10.1152/physrev.00041.2006> PMID: 17615391.
40. Miller DB, O'Callaghan JP. Neuroendocrine aspects of the response to stress. *Metabolism*. 2002 Jun;51(6 Suppl 1):5-10. <https://doi.org/10.1053/meta.2002.33184> PMID: 12040534.
41. Mir M, Nasirzadeh F, Bereznicki H, Enticott P, Lee SH, Mills A. Construction noise effects on human health: evidence from physiological measures. *Sustain Cities Soc* 2023;91:104470.
42. Molina SJ, Buján GE, Guelman LR. Noise-induced hippocampal oxidative imbalance and aminoacidergic neurotransmitters alterations in developing male rats: Influence of enriched environment during adolescence. *Dev Neurobiol*. 2021 Mar;81(2):164-188. <https://doi.org/10.1002/dneu.22806> Epub 2021 Jan 27. PMID: 33386696.
43. Moroe N, Mabaso P. Quantifying traffic noise pollution levels: a cross-sectional survey in South Africa. *Sci Rep* 2022;12:3454.
44. Morrison JH, Baxter MG. The ageing cortical synapse: hallmarks and implications for cognitive decline. *Nat Rev Neurosci*. 2012 Mar 7;13(4):240-50. <https://doi.org/10.1038/nrn3200> PMID: 22395804.
45. Munzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. *Eur Heart J*. 2014 Apr;35(13):829-36. <https://doi.org/10.1093/eurheartj/ehu030> Epub 2014 Mar 9. PMID: 24616334.
46. Naqvi F, Haider S, Batool Z, Perveen T, Haleem DJ. Sub-chronic exposure to noise affects locomotor activity and produces anxiogenic and depressive like behavior in rats. *Pharmacol Rep*. 2012;64(1):64-9. [https://doi.org/10.1016/s1734-1140\(12\)70731-4](https://doi.org/10.1016/s1734-1140(12)70731-4) PMID: 22580521.
47. Newsom RJ, Osterlund C, Masini CV, Day HE, Spencer RL, Campeau S. Cannabinoid receptor type 1 antagonism significantly modulates basal and loud noise induced neural and hypothalamic-pituitary-adrenal axis responses in male Sprague–Dawley rats. *Neuroscience*. 2012 Mar 1;204:64-73.
48. Owolawi IV. Noise exposure and its auditory effect on industrial workers. *Int J Otolaryngol Head Neck Surg* 2021;10:365–75.
49. Paciello F, Pisani A, Rinaudo M, Cocco S, Paludetti G, Fetoni AR, Grassi C. Noise-induced auditory damage affects hippocampus causing memory deficits in a model of early age-related hearing loss. *Neurobiology of Disease*. 2023 Mar 1;178:106024.
50. Peng X, Mao Y, Tai Y, Luo B, Dai Q, Wang X, Wang H, Liang Y, Guan R, Liu C, Guo Y. Characterization of anxiety-like behaviors and neural circuitry following chronic moderate noise exposure in mice. *Environmental Health Perspectives*. 2023 Oct 5;131(10):107004.

51. Pisani A, Paciello F, Del Vecchio V, Malesci R, De Corso E, Cantone E, Fetoni AR. The Role of BDNF as a Biomarker in Cognitive and Sensory Neurodegeneration. *J Pers Med.* 2023 Apr 10;13(4):652 <https://doi.org/10.3390/jpm13040652> PMID: 37109038.
52. Polak K, Korzeb J. Identification of the major noise energy sources in rail vehicles moving at a speed of 200km/h. *Energies* 2021;14:3957.
53. Reul JM, de Kloet ER. Two receptor systems for corticosterone in rat brain: microdistribution and differential occupation. *Endocrinology.* 1985 Dec;117(6):2505-11. <https://doi.org/10.1210/endo-117-6-2505> PMID: 2998738.
54. Salzman CD, Fusi S. Emotion, cognition, and mental state representation in amygdala and prefrontal cortex. *Annu Rev Neurosci.* 2010;33:173-202. <https://doi.org/10.1146/annurev.neuro.051508.135256> PMID: 20331363.
55. Shaikh HH, Zainun NY, Khahro SH. Impact of noise pollution at construction sites of Sindh Pakistan. *E3S Web Conf* 2023;437:02003.
56. Shukla M, Mani KV, Shukla S, Kapoor N. Moderate noise associated oxidative stress with concomitant memory impairment, neuro-inflammation and neurodegeneration. *Brain, Behavior, & Immunity-Health.* 2020 May 1;5:100089.
57. Sivakumaran K, Ritonja JA, Waseem H, AlShenaiber L, Morgan E, Ahmadi SA, Denning A, Michaud D, Morgan RL. Impact of Noise Exposure on Risk of Developing Stress-Related Metabolic Effects: A Systematic Review and Meta-Analysis. *Noise Health.* 2022 Oct-Dec;24(115):215-230. https://doi.org/10.4103/nah.nah_21_22 PMID: 36537446.
58. Stansfeld SA, Matheson MP. Noise pollution: non-auditory effects on health. *Br Med Bull.* 2003;68:243-57. <https://doi.org/10.1093/bmb/ldg033> PMID: 14757721.
59. Sullivan PF, Neale MC, Kendler KS. Genetic epidemiology of major depression: review and meta-analysis. *Am J Psychiatry.* 2000 Oct;157(10):1552-62. <https://doi.org/10.1176/appi.ajp.157.10.1552> PMID: 11007705.
60. Tang S, Yuan K, Chen L. Molecular biomarkers, network biomarkers, and dynamic network biomarkers for diagnosis and prediction of rare diseases. *Fundamental Research.* 2022 Nov 1;2(6):894-902.
61. Tao Y, Chai Y, Kou L, Kwan M. Understanding noise exposure, noise annoyance, and psychological stress: incorporating individual mobility and the temporality of the exposure-effect relationship. *Appl Geogr* 2020;125:102283.
62. Vasconcelos RO, Gordillo-Martinez F, Ramos A, Lau IH. Effects of Noise Exposure and Ageing on Anxiety and Social Behaviour in Zebrafish. *Biology.* 2023 Aug 24;12(9):1165.
63. World Health Organization. Burden of disease from environmental noise: Quantification of healthy life years lost in Europe. 2011. Available from: https://www.euro.who.int/__data/assets/pdf_file/0008/136466/e94888.pdf
64. Zaman M, Muslim M, Jehangir A. Environmental noise-induced cardiovascular, metabolic and mental health disorders: a brief review. *Environ Sci Pollut Res Int* 2022;29:76485–500.
65. Zhang Y, Zhu M, Sun Y, Tang B, Zhang G, An P, Cheng Y, Shan Y, Merzenich MM, Zhou X. Environmental noise degrades hippocampus-related learning and memory. *Proceedings of the National Academy of Sciences.* 2021 Jan 7;118(1):e2017841117.

66. Zhuang H, Yang J, Huang Z, Liu H, Li X, Zhang H, Wang J, Yu S, Liu K, Liu R, Bi M, Wang J, Salvi RJ, Hu B, Teng G, Liu L. Accelerated age-related decline in hippocampal neurogenesis in mice with noise-induced hearing loss is associated with hippocampal microglial degeneration. *Aging (Albany NY)*. 2020 Oct 11;12(19):19493-19519. <https://doi.org/10.18632/aging.103898> Epub 2020 Oct 11. PMID: 33041264.