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Negative Impact of Noise Exposure on the Mean Number of Neurons in the Frontal Lobe of Adult Wistar Rats

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ABSTRACT

Introduction: Noise exposure can induce oxidative stress and alter neurotransmitter dynamics within the nervous system. This oxidative stress is also associated with structural changes in the layers of the cerebrum. However, the effects of noise exposure on neuronal populations remain inadequately understood. This study aims to quantify the mean number of neurons in the frontal lobe of Wistar rats subjected to noise exposure.
Method: This study utilized an experimental design characterized by a randomized post-test only control group framework. A total of 30 male Wistar rats were selected through simple random sampling and subsequently divided into two groups: a control group without treatment (C1, n=15) and an experimental group exposed to 95 dB noise (E1, n=15) for four hours daily. Following a two-week exposure period, their brain tissue was excised and preserved in 10% neutral buffered formalin. Histological assessment was conducted using Hematoxylin and Eosin staining to evaluate the mean number of neurons in the frontal lobe. Statistical analysis was performed using the independent T-test.
Results: The mean number of neurons was 1053.33 ± 55.529 in the E1 group and 720.167 ± 61.135 in the C1 group. The mean neurons of the frontal lobe of Wistar rats in the E1 group ws lower than the C1 group significantly ($P < 0.001$).
Conclusion: Neuronal death in the frontal lobe resulting from noise-induced stress can induce structural alterations within this region, leading to detrimental effects on cognitive function and behavior.

Keywords: Frontal lobe, neuron, noise, stress

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Introduction

Sound influences the transmission of impulses within the central nervous system, particularly in the auditory cortex, encompassing Brodmann areas 41 and 42 located in the temporal lobe.[1] Exposure to an acoustically enriched environment affects the auditory system, resulting in alterations to neuronal structure and function.[2] Auditory stimulation can impact memory, autonomic functions, consciousness, cognitive abilities, and decision-making processes in the frontal lobe via the reticular formation.[3–5] Additionally, auditory stimulation has implications for cognition during rapid eye movement (REM) sleep.[6]

In contemporary society, exposure to sound can have detrimental effects, particularly in the form of noise, defined as unwanted sound and considered a significant environmental stressor. Noise pollution poses serious health risks, contributing to issues such as hearing loss, cognitive impairment, sleep disturbances, and negative emotional responses like stress, anger, and dissatisfaction.[7–10] Chronic exposure to environmental stressors, such as transportation noise, has been associated with increased rates of deafness and hindered cognitive development, including reading skills. A study examining the impact of ambient noise on the cognitive performance of schoolchildren found significant impairments at elevated noise levels, especially among students aged 11 to 13, underscoring the necessity for effective noise mitigation strategies in educational settings.[11] Conversely, other research indicated that white noise could have beneficial effects as a sleep therapy for patients with acute myocardial infacrtion.[12]

A study involving adult male rats demonstrated that exposure to noise at an intensity of 80 dB for two hours daily over six weeks resulted in deficits in spatial learning and memory, as assessed by the Morris water maze, alongside elevated serum corticosterone levels and increased malondialdehyde (MDA) and superoxide dismutase (SOD) levels in hippocampal tissues.[13]

The effects of noise, including varying intensities, on cognitive function have been extensively investigated. One notable study reported a reduction in neurogenesis within the motor and somatosensory cortices of rat pups subjected to noise exposure.[14] However, the effects on the average number of neurons in the frontal lobe remain less understood. The quantity of neurons in this region plays a critical role in decision-making, reasoning, personality, self-regulation, and planning. Oxidative stress resulting from noise exposure adversely affects memory, sensory processing, autonomic function (e.g., elevated heart rate), and cognitive capabilities.[13] Exposure to noise levels exceeding 80 dB for durations of up to two hours over 1.5 months can induce oxidative stress within the central nervous system,

comparable to conditions experienced on public roadways. Bali, as a prominent tourist destination, has seen a rise in transportation density. In 2018, noise levels in Lumintang City Park (Denpasar, Bali) reached 92 dB on the roadway, surpassing the quality standard for green spaces set at 50 dB.[15] This indicates a concerning increase in sound intensity that can act as an environmental stressor. This study aims to investigate the effects of noise on the average number of neurons in the frontal lobe of adult Wistar rats.

Methods

The Ethics Commission of the Faculty of Medicine at Udayana University had approved this study, under approval number 487/UN14.2.2.VII.14/LT/2023. The research employed an analytical experimental design with a randomized post-test control group approach. Male Wistar rats, aged three months and weighing between 240 and 260 grams, were utilized for the study. The subjects were divided into two equal groups: the treatment group (E1), which was subjected to noise exposure, and the control group (C1), which received no exposure. According to Federer's formula, a total sample size of 30 male Wistar rats was determined, and the subjects were assigned to groups through simple random sampling.

The rats in the treatment group were exposed to white noise generated by Real Time Analyzer software (version 5.2.0; Yoshimasa Electronic Inc., Japan) at an intensity of 95 dB for a duration of four hours daily over two weeks. The noise source, a loudspeaker (Sony SRS XB30, Japan), was positioned in a soundproof enclosure at a height of 30 cm above the rats. The control group (C1) was similarly housed in a soundproof box for the same duration without exposure to white noise.

On day 15, the Wistar rats were euthanized via intraperitoneal injection of ketamine at a dosage of 300 mg/kg body weight. The brains were subsequently fixed in 10% neutral buffered formalin. Histological preparations commenced with slicing paraffin-embedded blocks into 6 μm sections, followed by staining with hematoxylin-eosin.

Neuronal counts were conducted in the frontal lobe using a CX 41 microscope (Olympus, Japan) at 400x magnification, with independent assessments performed twice across four visual fields per animal. Data processing was carried out using Jamovi, and results are presented as mean values, with comparisons made between groups. An independent T-test was employed for statistical analysis to determine significance (p-value < 0.05), and findings were presented in both tabular and graphical formats.

Result

This study utilized 30 Wistar rats, which were divided into two equally sized groups maintained throughout the duration of the experiment. Both sets of numerical data were

subjected to Levene's test, yielding a *P*-value of 0.608, and the Shapiro-Wilk test, yielding a *P*-value of 0.132, indicating that the data were normally distributed and homogeneous. Subsequently, in Table 1, the C1 and E1 groups were compared using an independent samples T-test to assess for statistically significant differences in neuronal counts between the groups. The results indicated that the mean neuronal count in the group exposed to 95 dB noise for four hours daily was significantly lower than that of the control group that was not exposed to noise ($P < 0.05$), as shown in Figure 1.

Table 1. Results of the mean neurons of the frontal lobe of wistar rats.

Groups	Number (n)	Mean ± SD	*P*-value
C1	15	1054 ± 56.6	< 0.001
E1	15	720 ± 62.0	

Data were analyzed with independent T-test
C1 = control group; E1 = experimental group

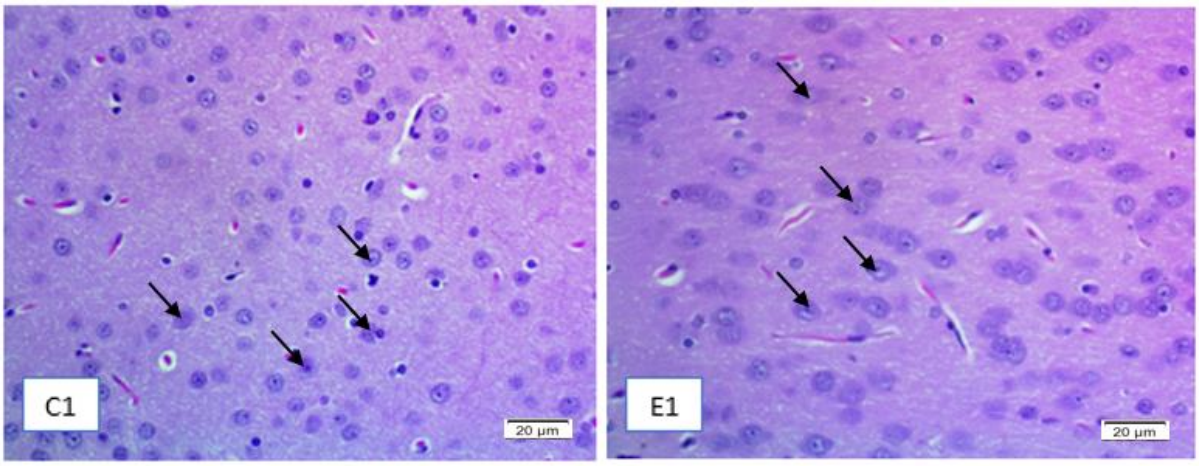

Figure 1. Histology of the frontal lobe of Wistar rats with HE staining at 400x magnification. In the C1 group, the neuron mean was higher than the E1 group. The black arrows indicate neurons.

Discussion

The present finding indicates that Exposure to 95 dB noise in the E1 group significantly reduced the mean number of frontal lobe neurons in Wistar rats compared to the C1 group. Auditory stimuli are initially transmitted to the primary auditory cortex (Brodmann areas 41 and 42) within the temporal lobe and subsequently processed in the auditory association cortex. Beyond perceptual processing, auditory input exerts significant influence on emotional regulation through projections from the medial and inferior temporal gyri to the amygdala. The amygdala provides efferent outputs to the hypothalamus via the bed nucleus of the stria

terminalis and the amygdalofugal pathways, thereby modulating autonomic and neuroendocrine activity. In parallel, auditory signals engage the hippocampus, contributing to memory formation and consolidation, with reciprocal hippocampal–hypothalamic connections further integrating auditory input into neuroendocrine regulation. Auditory information also reaches the reticular activating system, facilitating cortical arousal and consciousness. Within this network, the reticular formation—particularly the locus coeruleus—plays a central role in mediating motor and behavioral responses during heightened arousal states.[1] Chronic or acute exposure to noise levels above 85 dB is associated with adverse effects not only on the auditory system but also on extra-auditory physiological functions as showed on Figure 2. Such stress-related outcomes are mediated through activation of the hypothalamic–pituitary–adrenal (HPA) axis, the sympatho-adrenomedullary (SAM) system, and the inflammatory response. Activation of the HPA axis results in increased levels of glucocorticoids and corticotropin-releasing hormone (CRH), both of which induce dysfunction of cortisol regulation.[16,17] This dysfunction is associated with elevated extracellular glutamate and enhanced glutamate receptor activity. Notably, nearly 50% of brain neurotransmission is mediated by glutamatergic pathways, with 90% of cortical neurons utilizing glutamate as their neurotransmitter. Elevated extracellular glutamate levels facilitate an influx of Ca^{2+} ions, which can trigger neuronal death.[18]

Chronic exposure to noise also activates the NLRP3 inflammasome, which is associated with neuroinflammation and cognitive decline. While neuroinflammation is known to play a critical role in noise-induced cognitive deficits, the underlying mechanisms responsible for this neuroinflammation remain poorly understood.[19] Reactive oxidative stress (ROS) is pivotal in inhibiting N-methyl-D-aspartate receptors (NMDARs), leading to glutamate excitotoxicity and increased intracellular Ca^{2+} concentrations. This cascade of events results in mitochondrial dysfunction and neuronal death, contributing to a reduction in neuronal cell count. The excessive oxidative conditions induced by ROS can also lead to lipid peroxidation, DNA oxidation, and protein modifications across various cellular compartments.[19–22] Furthermore, another study found that noise exposure decreases levels of brain-derived neurotrophic factor (BDNF) and its receptor TrkB, increases amyloid levels, and impairs cognitive functions in aged rats.[23]

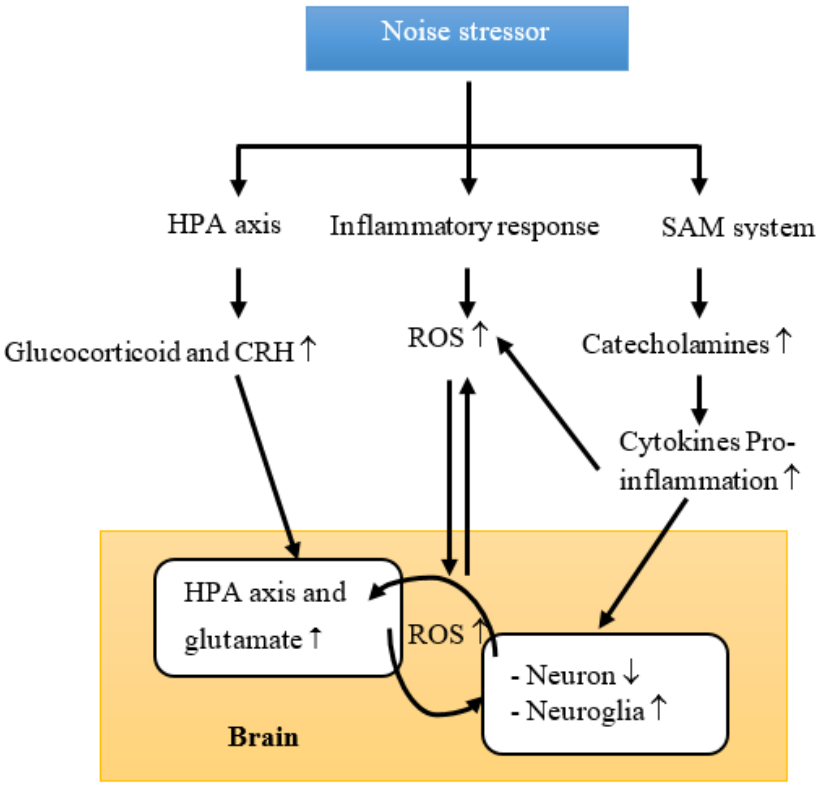

Figure 2. Noise can cause stress and activate the HPA axis, SAM system, and inflammatory cytokine interactions, ultimately leading to increased ROS in the brain.[17,20,21,24]

A study indicated that Wistar rats exposed to 95 dB noise for four hours daily exhibited a significant decrease in the number of neurons in the temporal lobes compared to control groups ($P < 0.005$).[25] In addition to affecting the temporal lobes, noise exposure can also alter the structural integrity of the amygdala, hippocampus and prefrontal cortex.[26,27] Neuronal loss in the frontal lobe due to oxidative stress can result in structural modifications that yield negative consequences. The frontal lobe is essential for executive functions, working memory, attention, decision-making, and emotional regulation. Damage to this region can lead to diminished cognitive abilities, including impairments in planning, organization, problem-solving, attention deficits, decision-making difficulties, and challenges in emotional regulation.[24,28–30] Furthermore, a study reported that 54 young participants exposed to 95 dB noise experienced significant reductions in mental workload and visual/auditory attention.[31] Oxidative stress is closely associated with the development and progression of Alzheimer's disease. Prolonged exposure to noise may contribute to the onset of Alzheimer's disease and related forms of dementia, which represent significant public health concerns.[32]

This study provides important insights into the impact of noise exposure on the central nervous system, highlighting mechanisms that remain relatively underexplored. However, the present work did not address the role of oxidative stress in noise-induced neurotoxicity. In addition, this study has limitations in the histological representation of other regions of the central nervous system, such as the temporal lobe, hippocampus, thalamus, and brainstem. Therefore, future investigations are warranted to evaluate oxidative stress in the frontal lobe and histological appearance in other regions as a potential pathway contributing to neuronal degeneration. Elucidating the molecular mechanisms underlying noise-related neuronal cell death may facilitate the identification of preventive strategies and the development of targeted therapeutic interventions.

Conclusion

This study concludes that noise exposure contributes to neuronal loss in the frontal lobe of Wistar rats, as evidenced by a lower mean neuronal count relative to the control group.

Conflicts of Interest

We have no conflicts of interest to report.

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