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Review Article

Advancements in Elucidating the Mechanisms of Central Nervous System Damage Induced by Infrasound Exposure

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Abstract

Infrasound, defined as sound waves with frequencies below 20 Hz, is gaining attention for its potential effects on human health, particularly the central nervous system (CNS). It can impact the CNS through direct and indirect mechanisms, such as damaging neuronal membranes, interfering with neurotransmitter release, altering intracellular signaling, and compromising the blood-brain barrier. These effects may cause neuronal dysfunction, neurotransmitter imbalances, increased oxidative stress, and inflammation, affecting mood, cognition, and memory. This review summarizes the current understanding of infrasound's impact on the CNS and its underlying mechanisms, aiming to provide a foundation for future research and applications.

Keywords

Infrasound, Biophysical Effects, Central Nervous System (CNS), Neurotransmitters, Oxidative Stress, Inflammatory Response

1. Introduction

Sound, as a core element of human communication and perception of the world, encompasses a spectrum that includes a category of low-frequency sound waves known as "infrasound," with frequencies below 20 Hz. Infrasound is widespread in both nature and human activities due to its ability to travel long distances and penetrate barriers [1]. Its sources can be traced to phenomena such as earthquakes, ocean waves, volcanic eruptions, and the operation of heavy machinery, creating a hidden dimension within our environment. With advancements in technology, the applications of infrasound have gradually expanded, particularly in the military domain, where infrasound weapons have attracted attention due to their potential impact on the central nervous system (CNS) [2, 3]. Recent in-depth studies suggest that infrasound can induce changes in nerve cells and interfere with the transmission of neural signals, leading to significant physiological and behavioral changes [4-6]. This paper aims to systematically review and analyze the existing literature on the effects of infrasound on the central nervous system, particularly focusing on the mechanisms of damage at the cellular and molecular levels.

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2. Biophysical Effects of Infrasound on the Central Nervous System

2.1. Direct Biophysical Damage to the CNS

Infrasound can interfere with the normal function of the CNS, including the brain, by triggering biological resonance, as the natural resonant frequencies of these organs (e.g., the alpha rhythm of the brain, which ranges from 7 to 12 Hz) fall within the infrasound frequency range. First, high-energy infrasound (above 120 dB) can cause micro-vibrations of cell membranes, potentially altering their physical properties, including fluidity and permeability, directly affecting neuronal excitability. For instance, Liu Bin et al. observed that after L929 cells were exposed to infrasound, normal protrusions on the membrane surface significantly shortened, indentations became shallower, and the membrane surface became smoother, resembling a cobblestone-like appearance [7]. Based on cellular biomechanics principles, the micro-vibrations induced by infrasound can affect the electrophysiological properties and signal transmission capabilities of nerve cells. This impact leads to the disruption of neuronal excitability and synaptic transmission, which may be one of the fundamental mechanisms underlying CNS dysfunction [8]. Further studies have shown that exposure to 8 Hz, 120-140 dB infrasound causes significant structural changes in key brain regions, particularly the cerebral cortex and hippocampus, in rats. These changes include neuronal damage, nuclear condensation, mitochondrial swelling, and deformation of nerve fibers [9]. Such structural damage may result in cellular dysfunction, affecting the overall efficiency of the CNS. It is noteworthy that infrasound not only acts directly on the structure of nerve cells but also activates microglia and astrocytes in the brain [10, 11]. The activation of these cells exacerbates neuronal damage through paracrine mechanisms, further demonstrating the broad impact of infrasound on the CNS. Infrasound can also cause damage to the blood-brain barrier (BBB). Studies have shown that exposure to 8 Hz, 120 dB infrasound increases the permeability of the BBB, potentially leading to vasogenic brain edema [12, 13]. When the sound intensity reaches 140 dB, regardless of the exposure duration, congestion of the pia mater, subarachnoid hemorrhage, and cortical petechial hemorrhage can be observed in brain tissue [14], further proving the severe damage effects of infrasound on brain tissue. In summary, the direct biophysical damage effects of infrasound on the CNS involve multiple levels, including the induction of mechanical stress responses in cell membranes, structural damage to cells, and the compromise of BBB integrity.

2.2. Indirect Biophysical Effects of Infrasound on the CNS

Although infrasound is generally not directly perceived by the human auditory system, it can indirectly affect the auditory [15] and vestibular systems [16]. Research has shown that infrasound exposure may cause symptoms such as tinnitus, a feeling of fullness in the ears, and balance dysfunction [17, 18]. These symptoms are thought to result from interactions between low-frequency sound waves and the inner ear and vestibular nerves, potentially altering the function of cochlear and vestibular hair cells [19]. Additionally, infrasound can indirectly affect the CNS by influencing cognitive function and behavior. Prolonged exposure to infrasound may lead to decreased attention, memory decline, and reduced work efficiency, changes that are usually associated with alterations in neurotransmitter levels in specific brain regions such as the hippocampus [20]. The stress response induced by infrasound may alter hippocampal function (especially the activation of Connexin 43 hemichannels in hippocampal astrocytes [20]) and neural plasticity (downregulation of the BDNF-TrkB signaling pathway related to infrasound-induced neuronal plasticity [21]), thereby affecting cognitive function. At the same time, infrasound exposure may also trigger psychological stress and affect emotional states [22], manifesting as symptoms such as anxiety, fatigue, and irritability. These psychological and emotional changes may be related to the activation of the hypothalamic-pituitary-adrenal (HPA) axis and subsequent changes in cortisol levels [22], thereby affecting overall mood and stress levels. Infrasound, including low-frequency noise, can disrupt normal sleep patterns, potentially reducing sleep quality by causing micro-arousals or slight disturbances to the sleep cycle, even if the disturbance is not sufficient to wake the individual [23, 24]. This may lead to decreased cognitive function and increased stress, exacerbating the indirect effects of infrasound on the CNS. Although the immediate effects of infrasound are significant, its potential long-term consequences for CNS health should not be overlooked. Chronic exposure may lead to sustained structural and functional changes in the brain, particularly in areas sensitive to stress and acoustic trauma, such as the hippocampus and cortex, possibly increasing the risk of developing neurological disorders or exacerbating existing conditions. However, epidemiological studies on the health impacts of low-frequency noise are still scarce [25]. In summary, the indirect biophysical effects of infrasound on the CNS encompass a broad spectrum of disturbances related to sensation, cognition, psychology, and sleep. The diversity of these effects demonstrates the ability of infrasound to impact the CNS through indirect pathways and complex interactions of physiological responses.

•**Direct Pathway**: Infrasound exposure → Micro-vibrations → Membrane permeability changes → Structural damage (e.g., mitochondrial swelling, neuronal damage) → CNS dysfunction.
•**Indirect Pathway**: Infrasound exposure → Auditory/Vestibular System Disruption → Balance/Cognitive Impairments → CNS Dysfunction. Infrasound exposure → HPA Axis Activation → Neurotransmitter Changes (e.g., reduced BDNF, increased glutamate) → Cognitive and Emotional Dysfunctions.

Figure 1. *A schematic diagram used to visually distinguish between direct and indirect biophysical damage.*

3. Biochemical and Molecular-Level Effects of Infrasound on the CNS

The biochemical and molecular-level effects of infrasound on the CNS are particularly critical in terms of neurotransmitter release, cellular signaling pathways, and neural cell metabolism.

3.1. Effects on Neurotransmitter Release

Infrasound can induce functional changes in calcium channels on neuronal cell membranes by altering their physical properties, particularly membrane fluidity. These physical changes partially open calcium channels, especially TRPV4 [8], increasing calcium influx and triggering the release of neurotransmitters [26]. Since neurotransmitter release is a calcium-dependent process, the increased calcium ions facilitate the fusion of neurotransmitter vesicles with the cell membrane, leading to the release of large amounts of neurotransmitters into the synaptic cleft, thereby affecting neural signal transmission. Prolonged or high-intensity exposure to infrasound can disrupt neurotransmitter homeostasis. For instance, exposure to an environment of 8 or 16 Hz at 120 dB for two hours significantly increased glutamate levels [20, 27], while other studies found reduced levels of norepinephrine [28] and dopamine [29]. These neurotransmitters play essential roles in regulating emotions, cognitive functions, and memory formation. The disruption of neurotransmitter balance caused by infrasound may lead to abnormal emotional regulation, cognitive decline, and impaired memory formation, revealing the biological basis for the extensive effects of infrasound on the nervous system.

3.2. Effects on Apoptotic Signaling Pathways

Infrasound exposure can cause abnormal increases in intracellular calcium levels, activating apoptotic signaling pathways. Through the aforementioned signal transduction mechanisms, infrasound alters the expression of specific genes, thereby affecting cell cycle regulation, DNA repair mechanisms, and antioxidant defense systems, promoting cell survival and proliferation. For example, the increased intracellular calcium can activate protein kinase C (PKC), MAP kinase pathways, and other signaling molecules involved in regulating cell growth, differentiation, death, and many other physiological processes.

3.3. Effects on Neural Cell Metabolism

Infrasound may also affect the energy metabolism of neural cells, including ATP production and consumption. By influencing cellular signaling pathways, infrasound can indirectly impact cellular metabolic activity. For example, the stress response induced by infrasound may increase energy consumption to maintain cellular defense mechanisms such as protein repair and antioxidant defense systems. Additionally, infrasound may affect mitochondrial function, thereby impacting ATP production. Mitochondria, being the energy factories of cells, play a direct role in the energy balance of cells, and their dysfunction can lead to increased cellular stress or even cell death. Under high-intensity infrasound, cells may experience increased oxidative stress, DNA damage, and imbalance in the intracellular environment, triggering programmed cell death. The gene expression changes induced by infrasound, such as the upregulation of pro-apoptotic genes and downregulation of anti-apoptotic genes, constitute key molecular mechanisms leading to infrasound-induced apoptosis. Zhang Meng Yao and colleagues exposed SD rats to 140 dB (8 Hz) infrasound for three days, two hours per day, and observed significant damage to the hippocampal ultrastructure through transmission electron microscopy (TEM), with recovery beginning one week after exposure. TUNEL data showed that neuronal apoptosis at 24 and 48 hours post-exposure was significantly higher than in the control group, while apoptotic cells decreased one week post-exposure [30]. Jiang Shan and colleagues found that infrasound exposure triggered apoptosis by upregulating P53 protein and downregulating B-cell lymphoma (Bcl-2) protein levels [31]. P53 is a key factor in apoptosis and cell cycle regulation, while Bcl-2 is a major anti-apoptotic protein that regulates cellular response to apoptotic signals [32].

3.3.1. Oxidative Stress

Long-term exposure to infrasound increases oxidative stress in the brain, promoting excessive free radical production and lipid peroxidation [33] accumulation, damaging neuronal cell mem-

branes, DNA, and proteins, affecting normal cell function and survival, and potentially inducing apoptosis. Oxidative stress refers to the imbalance between free radicals and antioxidants in the body, leading to damage to cellular structures and functions. The study by Zhang Xinwei and colleagues showed that infrasound could increase the production of reactive oxygen species (ROS), reduce the activity of antioxidant enzymes such as superoxide dismutase (SOD) and glutathione (GSH) [34], causing lipid peroxidation, DNA damage, and protein oxidation, which further disrupts intracellular ion balance, particularly calcium ions (Ca^{2+}). Abnormal calcium ion flow can trigger various intracellular responses, including the activation of signaling pathways leading to cell death. Cui Fang and colleagues found that after prolonged exposure to 8 Hz infrasound of different intensities, the activity of GSH-PX in the brain, liver, and lung tissues of rats was significantly reduced compared to the control group [35].

3.3.2. Neuroinflammatory Response

Infrasound can elevate the levels of pro-inflammatory cytokines in the hippocampus, such as interleukin (IL)-1β and tumor necrosis factor α (TNFα). These cytokines interfere with neural signal transmission and neuroplasticity by affecting the function of neurotransmitters and receptors [31]. Infrasound exposure can activate the fibroblast growth factor (FGF) 2/FGF receptor (FGFR) 1 pathway, and after three days of infrasound exposure, A1 astrocytes were induced in the hippocampal CA1 region, with an increase in the number of microglia, indicating the occurrence of neuroinflammation [10, 11]. Infrasound can cause rapid overactivation of glial cells and lead to sustained inflammation. The research by Shi, Ming, and colleagues indicates that the calmodulin and protein kinase C signaling pathways in glial cells are involved in NF-kappaB activation triggered by TRPV4, a calcium-permeable mechanosensitive channel, which is a potential key factor in infrasound-induced neuronal damage [8]. Long-term or repeated exposure to infrasound may lead to chronic inflammatory responses, which further increase oxidative stress, creating a vicious cycle.

Table 1. *Infrasound effects of cellular mechanisms and signaling pathways.*

Cell Type	Affected Pathways	Molecular Mechanisms	Implications
Astrocytes	Connexin 43 hemichannels, BDNF signaling	Altered ion homeostasis Reduced BDNF levels	Impaired synaptic plasticity and signaling
Microglia	NF-kappaB signaling	Increased pro-inflammatory cytokines (TNF-α, IL-1β) Chronic neuroinflammation	Neuronal damage and cognitive decline
Neurons	TRPV4 calcium channels, MAPK pathways	Calcium overload -Activation of apoptotic pathways (P53, Bcl-2 downregulation)	Apoptosis and neurotransmitter imbalance
Endothelial Cells	Oxidative stress pathways (ROS, GSH, SOD)	Lipid peroxidation Increased BBB permeability	Blood-brain barrier disruption
Mitochondria	Oxidative phosphorylation, antioxidant mechanisms	Reduced ATP production Increased oxidative stress	Energy depletion and cell death

3.4. Frequency and Intensity-Dependent Effects of Infrasound on the CNS

Infrasound can vary in frequency and intensity, both of which play crucial roles in determining the magnitude and nature of its effects on the CNS. Different frequencies may interact with CNS structures in different ways, and variations in sound intensity (measured in dB) at the same frequency can cause varying degrees of cellular and molecular damage.

3.4.1. Frequency Effects

Infrasound frequencies between 8 Hz and 16 Hz are commonly observed to affect brain regions involved in cognitive functions and emotional regulation [20, 27]. For example, exposure to 8 Hz infrasound has been shown to primarily affect hippocampal and cerebral cortex neurons, leading to significant neuronal damage and synaptic dysfunction. In contrast, higher frequencies such as 16 Hz tend to affect auditory and vestibular systems, often leading to balance dysfunction, tinnitus, and other auditory disturbances. The frequency range (typically under 20 Hz) also intersects with the natural resonant frequencies of some brain regions, which could explain some of the observed neural excitability and functional changes.

3.4.2. Intensity Effects (dB)

The intensity of infrasound at the same frequency plays a crucial role in its effects on the CNS. For instance, exposure to 8 Hz at 120 dB causes substantial mechanical stress on cell membranes, leading to structural damage in neurons and glial cells [9]. However, at higher intensities, such as 140 dB, the damage becomes more severe, including damage to the blood-brain barrier (BBB) and the onset of vasogenic edema [9, 12, 13]. Interestingly, exposure to the same frequency (8 Hz) at varying intensities results in different levels of oxidative stress, inflammation, and neurotransmitter imbalance, which can contribute to long-term cognitive and behavioral changes.

Table 2. *Frequency and Intensity Effects of Infrasound on the CNS.*

Frequency (Hz)	Intensity (dB)	Cellular Effects	Molecular Mechanisms	CNS Impacts
8 Hz	120 dB	Micro-vibrations in neuronal membranes & Mitochondrial swelling	Activation of TRPV4 calcium channels & Increased oxidative stress	Neuronal damage in hippocampus & Disrupted synaptic function
8 Hz	140 dB	Blood-brain barrier permeability increase & Cell apoptosis	Higher calcium influx & Elevated ROS levels & DNA damage	Severe brain tissue damage & Vasogenic edema
16 Hz	120 dB	Disruption of auditory system function & Vestibular system effects	Activation of Connexin 43 hemichannels & Increased pro-inflammatory cytokines	Tinnitus & Balance dysfunction
16 Hz	140 dB	Auditory cell damage & Neuronal apoptosis in brain regions involved in sensory processing	Overactivation of MAPK pathways & Apoptotic signaling	Loss of hearing & Severe balance impairments

In summary, current research reveals that infrasound causes damage to the central nervous system by affecting various aspects, including cellular signal transduction, neurotransmitter balance, oxidative stress, and inflammation. Future research should focus on identifying more molecular markers, developing protective strategies, and validating the results of animal models and cell experiments in humans, thereby providing a solid scientific basis for assessing and mitigating the health risks of infrasound.

4. Conclusion and Outlook

With the advancement of research on the effects of infrasound, its molecular mechanisms of CNS damage have become increasingly clear. These damages, depending on the intensity of infrasound exposure, can be classified into reversible functional damage and irreversible organic damage, with certain temporal characteristics and adaptive phenomena of the body. Despite the progress made, the complete mechanisms of infrasound effects and their long-term impacts require further investigation, especially regarding the long-term effects and effective protective measures against infrasound-induced CNS damage. Future research needs to focus on identifying more molecular markers, developing new protective strategies, and validating the results of animal models and cell experiments in humans to provide a more solid scientific foundation for the assessment and prevention of infrasound health risks.

Abbreviations

CNS	Central Nervous System
dB	Decibels
TRPV4	Transient Receptor Potential Vanilloid 4
ROS	Reactive Oxygen Species
SOD	Superoxide Dismutase
GSH	Glutathione
GSH-PX	Glutathione Peroxidase
IL-1β	Interleukin 1 Beta
TNFα	Tumor Necrosis Factor Alpha
FGF	Fibroblast Growth Factor
FGFR	Fibroblast Growth Factor Receptor
NF-kappaB	Nuclear Factor Kappa B
HPA	Hypothalamic-Pituitary-Adrenal
BDNF	Brain-Derived Neurotrophic Factor
TrkB	Tropomyosin Receptor Kinase B
Bcl-2	B-Cell Lymphoma 2
P53	Tumor Protein 53
TEM	Transmission Electron Microscopy
TUNEL	Terminal Deoxynucleotidyl Transferase dUTP Nick-End Labeling

Conflicts of Interest

The authors declare no conflicts of interest.

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