Original Research Paper | Zoology | Volume : 5 ¦ Issue : 9 ¦ Sep 2020 ¦ e-ISSN : 2456-5040

A COMPREHENSIVE REVIEW OF THE PHYSIOLOGICAL AND BEHAVIORAL IMPACTS OF NOISE POLLUTION ON HUMANS AND WILDLIFE

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ABSTRACT

Noise pollution has emerged as a pervasive anthropogenic stressor with profound implications for biological systems. This paper synthesizes current research on the physiological and behavioral impacts of environmental noise, highlighting its role as a "silent threat." For humans, chronic noise exposure is linked to cardiovascular diseases, sleep fragmentation, and cognitive decline. For wildlife, it disrupts communication, foraging, and predator avoidance, often leading to community-wide ecological shifts. The study concludes that noise must be managed as a primary pollutant to safeguard public health and biodiversity.

KEYWORDS: Noise Pollution, Anthropogenic Noise, Environmental Health, Physiological Stress

INTRODUCTION

For the vast majority of evolutionary history, the acoustic environment was defined by geophony and biophony. However, the rapid acceleration of the Anthropocene has introduced a dominant third category: anthropophony. Noise pollution, defined by the World Health Organization (WHO, 2018) as unwanted or harmful outdoor sound, is no longer a localized urban nuisance. It is a pervasive physiological and psychological stressor that violates the biological thresholds of nearly every living system (Basner et al., 2014).

When the brain perceives noise from the source, it activates the hypothalamic-pituitary-adrenal (HPA) axis. This biological "alarm system" prepares an organism for immediate survival. While adaptive in the short term, chronic exposure leads to sustained sympathetic arousal and elevated cortisol levels. In human, this leads to vascular inflammation and endothelial dysfunction (Daiber et al., 2019). For wildlife, the acoustic environment is a vital resource. Anthropogenic noise creates "acoustic smog" that masks signals (Slabbekoorn & Peet, 2003), effectively blinding animals that rely on sound to navigate and survive (Barber et al., 2010). . This review synthesizes current literature regarding the anthropogenic acoustic transition of the 21st century.

MATERIALS AND METHODS

This review utilized a systematic search of databases including PubMed and Web of Science. Selection criteria focused on:
Large- scale longitudinal cohorts for human health (e.g., European Environment Agency [EEA], 2018).

Controlled field experiments regarding wildlife behavior (Shannon et al., 2016).

Acoustic metrics including (day-evening-night) and weighted decibels (dB(A)).

OBSERVATIONS AND RESULTS

Impacts on Human Populations

The physiological response to noise follows a "Non-Auditory" pathway. Even when hearing is not physically damaged, the sub-cortical regions of the brain trigger autonomic responses.

Cardiovascular Morbidity: Research indicates a 3.2% increase in the risk of myocardial infarction for every 10 dB (A) increase in road traffic noise (Münzel et al., 2018).

Metabolic Disruption: Nighttime noise (as low as 40 dB) increases the risk of Type 2 Diabetes by triggering cortisol-induced insulin resistance (Basner & McGuire, 2018).

Table 1: Outlines the biological and clinical effects observed at different nighttime noise levels, as defined in the WHO Night Noise Guidelines.

Noise Level (dB)	Observed Health Effects	WHO Status
Up to 30 dB	No substantial biological effects; modest increase in body movements.	**No Observed Effect Level (NOEL)**
30 to 40 dB	Sleep disturbance, awakenings, and arousals; vulnerable groups (elderly, children) are affected.	**Lowest Observed Adverse Effect Level (LOAEL)**
40 to 55 dB	Adverse health effects frequent; population must adapt lifestyle to cope; increased use of sedatives.	**Guideline Target (40 dB)**
Above 55 dB	High risk of cardiovascular disease; sizeable proportion of population highly annoyed and sleep-disturbed.	**Interim Target (IT)**

Neuro-developmental Delays: Children in schools near noisy transportation hubs show measurable deficits in reading comprehension and long-term memory (Stansfeld et al., 2005; Clark & Paunovic, 2018).

Fetal and Neonatal Development: The intrauterine environment does not fully shield the fetus from low-frequency

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noise. Maternal noise exposure is strongly linked to intrauterine growth restriction (IUGR) and low birth weight (LBW) (Smith et al., 2018). Infants born in high-noise zones (e.g., near airports) may weigh up to 69g less than those in quiet areas.

Neonatal Physiological Stress: In Neonatal Intensive Care Units (NICUs), noise levels often exceed the recommended 45 dB. This triggers immediate tachycardia, decreased oxygen saturation, and sleep fragmentation in newborns, potentially hindering neurological maturation (Wachman & Lahav, 2011).

2. Impacts on Wildlife Populations

Wildlife impacts are categorized by "Acoustic Masking" and "Sensory Overload."

Communication Alteration: Urban bird species have been observed to increase their song pitch to be heard over traffic noise (Slabbekoorn & Peet, 2003).

Foraging Efficiency: Predator species like owls show an 8% decrease in hunting success for every 1 dB increase in ambient noise (Senzaki et al., 2016).

Marine Trauma: Low-frequency sonar and seismic airguns reduce the effective communication range of blue whales by up to 90% (Hatch et al., 2012).

Behavioral Interference: The Foraging-Vigilance Trade-off The 8% Rule: Experimental field data reveals that for every **1 dB increase** in ambient noise, the hunting success of specialized predators (such as the Short-eared Owl) **decreases by 8%** [Senzaki et al., 2016].

Acoustic Masking and Space Reduction: For every **10 dB increase** in anthropogenic noise, the "active space"—the area over which a biological signal can be perceived—is reduced by **50%.** A **20 dB increase** results in a **90% reduction** in communication range [Barber et al., 2010].

Pollination Disruption: Noise levels typical of industrial sites can reduce the visitation rate of avian and insect pollinators by **up to 70%,** leading to a significant decline in the seed set and reproductive success of native plant species [Franci et al., 2015].

DISCUSSION

The evidence suggests that noise acts as a "molecular stressor." In humans, the primary mechanism is oxidative stress. Noise-induced arousal activates NADPH oxidase in blood vessels, leading to endothelial damage (Daiber et al., 2019). In wildlife, noise creates a "Landscape of Fear" (Francis & Barber, 2013). This "acoustic fragmentation" is as damaging as physical deforestation because it isolates populations that avoid noisy corridors, reducing genetic diversity (Barber et al., 2010; Shannon et al., 2016). Furthermore, the data suggests a disturbing evolutionary trade-off. Species that adapt to noise—such as urban birds singing at higher pitches (Slabbekoorn & Peet, 2003)—may suffer from reduced signal quality or increased metabolic costs. This "forced adaptation" can lead to a decrease in the overall fitness of the population. In the marine environment, the impact is even more severe due to the efficiency of sound travel in water. The reduction of the "communication space" for cetaceans from hundreds of kilometers to less than ten (Hatch et al., 2012) suggests that we are effectively isolating marine populations in an "acoustic fog," hindering their ability to coordinate migrations or find mates across vast oceanic distances.

For the most vulnerable human demographics pregnant women and newborns—the discussion must pivot to epigenetic programming. The elevation of maternal catecholamines and glucocorticoids (cortisol) due to environmental noise can traverse the placental barrier. This exposure during critical windows of gestation may "program" the fetal HPA axis, potentially predisposing the offspring to metabolic syndromes and heightened stress reactivity in adulthood (Dzhambov et al., 2019). In the neonatal phase, especially within the high-decibel environments of many NICUs, the lack of "acoustic protection" can disrupt the pruning of neural pathways. Because newborns lack the cognitive ability to habituate to loud, unpredictable sounds, they remain in a state of constant autonomic arousal, which can manifest as delayed weight gain and impaired neurodevelopment (Wachman & Lahav, 2011).

Table 2: WHO Recommended Noise Exposure Limits (2018)

Noise Source	Average Exposure (dB)	Nighttime Exposure (dB)	Strength of Recommendation
Road Traffic	< 53 dB	< 45 dB	Strong
Railway	< 54 dB	< 44 dB	Strong
Aircraft	< 45 dB	< 40 dB	Strong
Wind Turbines	< 45 dB	No specific recommendation*	Conditional
Leisure Noise	≤ 70 dB (Yearly average)	N/A	Conditional

In the ecological sphere, the discussion must move beyond individual behavioral shifts to community-wide restructuring. Wildlife relies on "acoustic space" as a finite resource. When anthropogenic noise occupies the low-frequency bands used by large mammals or the high-frequency bands used by songbirds and insects, it creates an "acoustic squeeze." The phenomenon of acoustic masking does more than just hinder mating; it creates a "sensory void" where the subtle cues of moving prey or an approaching predator are lost (Senzaki et al., 2016). This creates what ecologists term a "Landscape of Fear" (Francis & Barber, 2013). Animals may perceive a noisy forest as "high-risk" even if it is abundant in resources, leading to habitat abandonment. This "acoustic fragmentation" is particularly dangerous because it is invisible; a habitat may appear geographically intact on a map while being functionally fragmented and biologically impoverished (Barber et al., 2010).

Furthermore, the data suggests a disturbing evolutionary trade-off. Species that adapt to noise—such as urban birds singing at higher pitches (Slabbekoorn & Peet, 2003)—may suffer from reduced signal quality or increased metabolic costs. This "forced adaptation" can lead to a decrease in the overall fitness

of the population. In the marine environment, the impact is even more severe due to the efficiency of sound travel in water. The reduction of the "communication space" for cetaceans from hundreds of kilometers to less than ten (Hatch et al., 2012) suggests that we are effectively isolating marine populations in an "acoustic fog," hindering their ability to coordinate migrations or find mates across vast oceanic distances.

CONCLUSION

Noise pollution is an invisible threat requiring immediate intervention. Mitigation must include source control, such as "quiet tires," and urban design involving "Acoustic Greenbelts". Protecting acoustic integrity is a fundamental biological necessity. The biological evidence demands that noise be reclassified. It is not an inevitable byproduct of progress but a primary environmental toxin. Whether through the molecular degradation of human arteries or the acoustic displacement of apex predators, noise pollution is systematically eroding the biological integrity of our planet.

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