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CHRONIC ENVIRONMENTAL NOISE AS A BIOLOGICAL STRESSOR: MECHANISTIC PATHWAYS AND REGIONAL EXPOSURE CONTEXT IN THE REPUBLIC OF ARMENIA

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Environmental noise pollution is increasingly recognized as a profound biological stressor rather than a mere ecological nuisance. This review synthesizes current scientific evidence on the pathophysiological mechanisms of chronic noise exposure, emphasizing its role in activating the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system. Sustained acoustic stress induces neuroendocrine dysregulation, triggering the overproduction of reactive oxygen species endothelial dysfunction, and systemic inflammation, which are primary drivers of cardiovascular, metabolic, and cognitive disorders. By examining structural characteristics of the urban environment in Yerevan, where highly reflective stone architecture and high-density environments amplify acoustic stimuli the study highlights a sustained allostatic load and heightened physiological vulnerability among the population. The paper concludes that mitigating chronic noise exposure is critical for cellular and systemic physiological restoration, advocating for the integration of neurobiological parameters into public health strategies to prevent noise-induced systemic pathologies.

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Introduction. Noise pollution is recognized not merely as a daily nuisance, but as a severe *biological stressor* that triggers a cascade of detrimental physiological and psychological responses. According to the World Health Organization (WHO), ambient noise acts as a pervasive biological hazard that chronically overstimulates the human nervous system. Prolonged exposure to high-intensity acoustic signals disrupts normal sleep architecture, chronically elevates stress hormones, drives the

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pathogenesis of cardiovascular and metabolic disorders at the cellular level, and significantly degrades overall physiological well-being. These systemic biological consequences are strongly supported by extensive epidemiological analyses and experimental biomedical studies [1, 2].

Environmental noise is increasingly recognized as a critical biological risk factor with measurable effects on systemic homeostatic regulation and fundamental physiological processes across species. Although historically considered secondary to chemical pollutants, accumulating neurobiological and cardiovascular evidence identifies chronic acoustic exposure as a non-chemical environmental stressor capable of inducing endothelial dysfunction, sustained sympathetic activation, and stress-mediated endocrine dysregulation. At the cellular level, repeated acoustic stress triggers hypothalamic-pituitary-adrenal axis activation, enhances reactive oxygen species (ROS) generation, disrupts redox homeostasis, reduces endothelial nitric oxide bioavailability, and modulates glucocorticoid receptor-dependent transcriptional and inflammatory signaling pathways, thereby contributing to vascular, metabolic, and neurocognitive pathology [3–6].

Internationally, the biological burden of noise pollution constitutes a major challenge in urban environments, driven primarily by continuous traffic-related acoustic stimuli which act as a persistent trigger for the autonomic nervous system. According to estimates by the European Environment Agency (EEA), more than 30% of Europe's population is subjected to acoustic loads that exceed physiological tolerance thresholds, directly associating this exposure with severe biological consequences, most notably chronic sleep fragmentation and progressive cardiovascular pathologies [1, 4, 7].

Recognizing noise as a potent biological disrupter, the WHO advocates a stricter preventive approach. The WHO emphasizes that long-term exposure to acoustic stimuli-particularly from road, railway, and aircraft traffic-must remain below established biological safety thresholds in order to prevent chronic allostatic overload and subsequent cellular and systemic damage [1, 7].

The aim of this article is to synthesize and critically analyze the current scientific literature regarding the pathophysiological mechanisms of noise exposure, its neuroendocrine and cellular impacts, and the resulting systemic health consequences. By shifting the paradigm from purely environmental management to an integrated biological and physiological perspective, this review seeks to elucidate the exact pathways, through which acoustic stress translates into human disease. Furthermore, the synthesis of this diverse international evidence aims to identify key directions for future neurobiological research and health-centric interventions.

Literature Search Strategy. This review is based on literature retrieved from PubMed, Scopus, and Web of Science databases between 2010 and 2025 using keywords including "environmental noise", "HPA axis", "oxidative stress", "cardiovascular risk", and "sleep disturbance". Only peer-reviewed studies and international health agency reports were included.

Materials and Methods.

Noise Pollution: A Physical Stressor Inducing Chronic Biological and Physiological Disruption. In modern biomedical and ecophysiological frameworks, environmental noise is classified as a pervasive anthropogenic physical stressor

capable of disrupting homeostatic regulation in biological systems [8, 9]. Unlike chemical toxicants, acoustic energy does not accumulate within tissues; instead, it acts as a continuous, non-material environmental trigger that induces sustained neuroendocrine activation and physiological arousal [10–12].

Noise exerts its biological impact through intensity, frequency composition, duration, and repetition of exposure. In heavily urbanized environments, chronic acoustic stimulation alters circadian regulation, interferes with neuroethological communication mechanisms, and promotes maladaptive behavioral and physiological responses [13–16]. Over time, sustained exposure contributes to cumulative allostatic load and may influence cardiovascular, metabolic, and neurocognitive vulnerability [12, 17].

From an ecological perspective, anthropogenic noise disrupts species communication, predator–prey dynamics, and reproductive signaling, thereby affecting population stability and biodiversity patterns [13–16]. Although noise does not bioaccumulate, its chronic and spatially pervasive nature positions it among persistent environmental stressors [8, 14].

To quantify acoustic exposure and associated biological burden, international health authorities employ standardized metrics. The Lden (Day–Evening–Night Level) represents a 24-hour weighted exposure index incorporating physiological penalties of +5 *dB*(*A*) for evening exposure (19:00–23:00) and +10 *dB*(*A*) for nighttime exposure (23:00–07:00) [1, 7, 17]. The Lnight indicator specifically evaluates nocturnal exposure and its relationship to sleep fragmentation and cardiometabolic risk [1, 7].

These standardized exposure metrics enable cross-national comparison of environmental noise burdens and facilitate correlation between acoustic exposure and systemic physiological outcomes.

Main Sources of Environmental Noise Pollution at the Global Scale. According to recent international scientific literature and updated assessments, the main sources of environmental noise pollution are predominantly anthropogenic in origin. Transport-related noise represents the most widespread and impactful source of environmental noise, particularly in urban and peri-urban areas [3, 18, 19].

Transport noise includes emissions from road, rail, and air transport systems. In densely populated cities, road traffic remains the dominant contributor due to high traffic volumes and increasing mobility demands. According to the latest assessments published by the European Environment Agency using 2025 reporting and evaluation frameworks, road transport accounts for the largest share of the overall environmental noise burden in Europe, while railway and aircraft noise affect smaller proportions of the population but may cause significant local disturbances in specific areas [20, 21].

Industrial noise continues to pose a serious environmental challenge, particularly in industrial zones and manufacturing cities. Noise generated by heavy machinery, production lines, and energy infrastructure frequently exceeds permissible thresholds and may extend into adjacent residential areas, negatively affecting quality of life.

Additional major sources of urban noise include construction activities, leisure and entertainment facilities, and building ventilation and technical systems.

Inadequate urban planning, mixed land use patterns, and insufficient green infrastructure contribute to increased noise propagation and exposure levels.

Based on 2025 European noise exposure estimates, approximately 20–30% of the European population is exposed to environmental noise levels exceeding health-based threshold values and considered an environmental risk factor [22]. Within the framework of the Environmental Noise Directive (END), around 109 million people are exposed to long-term transport-related noise levels of 55 *dB* Lden or higher, with approximately 57 million individuals affected by harmful night-time noise exposure [22, 23].

Health-based guideline values proposed by the WHO are significantly lower than END thresholds. When WHO recommendations are applied, it is estimated that approximately 145 million people, corresponding to more than 30% of Europe's population, are exposed to transport-related noise levels associated with adverse health outcomes [1, 22].

Chronic exposure to transport noise is associated with increased risks of cardiovascular disease, metabolic disorders, sleep disturbance, and adverse mental health outcomes. While modest reductions in aircraft noise exposure were observed in recent years due to post-pandemic traffic adjustments, the number of people exposed to railway noise in urban areas continues to increase during both daytime and night-time periods [1, 22].

Despite targeted noise mitigation measures, overall exposure levels remain high due to urban population growth, increased transport demand, and intensified mobility patterns. Consequently, the European Commission has reaffirmed its commitment under the Zero Pollution Action Plan to reduce the number of people chronically disturbed by transport-related noise by 30% by 2030, relative to 2017 baseline levels, with progress monitoring continuing through 2025 and beyond [22, 23].

International Assessment of Health Effects of Noise Pollution. The health effects of environmental noise pollution have been extensively studied at the international level and are supported by a strong and growing body of scientific evidence. Environmental noise is widely recognized as a chronic stressor, and long-term exposure contributes to the development of both physiological and psychological disorders through sustained activation of stress response mechanisms [24].

According to the EEA, more than 110 million people in Europe are exposed to environmental noise levels considered harmful to health, primarily from road, rail, and aircraft traffic [25]. Chronic exposure to transport-related noise is estimated to result in approximately 66 000 premature deaths annually across Europe. In addition, environmental noise contributes to around 48 000 new cases of ischemic heart disease per year and is associated with approximately 22 000 new cases of Type 2 diabetes annually (Tab. 1) [26].

The WHO identifies environmental noise as one of the most significant environmental health risks in the WHO European Region, ranking second only to air pollution in terms of public health impact. The WHO estimates that environmental noise exposure leads to the loss of more than 1–1.6 million disability-adjusted life years (DALYs) each year in Western Europe, reflecting both premature mortality and years lived with disability [26].

The health impacts of environmental noise primarily affect three major systems:

- *Cardiovascular system* – long-term exposure to environmental noise increases the risk of hypertension, ischemic heart disease, and stroke through stress-induced changes in blood pressure regulation and autonomic nervous system function [27].
- *Nervous system and mental health* – noise exposure is associated with elevated levels of annoyance, anxiety, depressive symptoms, cognitive impairment, and reduced academic performance in children, largely mediated by chronic stress and sleep disturbance [17].
- *Sleep disturbances* – environmental noise is a major cause of chronic sleep disruption. An estimated millions Europeans suffer from severe noise-related sleep disturbance, which in turn contributes to the development of cardiovascular, metabolic, and mental health disorders [26].

Overall, these findings indicate that the health burden of noise pollution is comparable to, and in some cases exceeds, that of other well-known environmental risk factors, such as exposure to second-hand tobacco smoke or certain chemical pollutants [26, 27].

Table 1

Health burden of environmental noise in Europe

Indicator	Value
Population exposed to harmful noise levels	>110 million people
Share of population exposed	~20–30%
Premature deaths per year	~66 000
New ischemic heart disease cases	~48 000/year
New type 2 diabetes cases	~22 000/year
Population with severe sleep disturbance	6–7 million
Healthy life years lost (DALYs)	>1.6 million/year

Mechanisms of Noise-Induced Health and Ecological Impact. Environmental noise is no longer viewed merely as a local nuisance but as a complex environmental stressor that triggers a cascade of biological responses. Unlike many other pollutants, noise exerts its effects through a "dose-response" relationship, where the harm is determined by the intensity (decibels), frequency, and duration of exposure. Modern research distinguishes between the direct pathway (immediate damage to the auditory system) and the indirect pathway (long-term physiological and psychological stress responses) [26]. To better contextualize these mechanisms, Tab. 2 summarizes typical environmental noise levels and their associated biological and health effects.

Physiological Pathways: The Stress Response and Cardiovascular Damage. The primary mechanism of noise-induced harm is the activation of the body's non-specific stress response. Even at relatively low levels (as low as 40–50 *dB*), noise can be perceived by the brain as a biological threat, especially during sleep. These physiological responses follow a dose–response relationship, where increasing noise intensity and exposure duration amplify stress-mediated damage.

Table 2

Environmental noise levels and associated biological and health effects

Noise Level, *dB*	Biological and Health Impact	Typical Context / Source
30–40	subtle alterations in sleep architecture, particularly light sleep and REM stages. Hormonal effects are minimal and indirect	quiet residential area at night
40–55	clinically relevant sleep disturbance; increased annoyance and activation of psychological stress responses, especially during nighttime exposure	evening traffic in suburban areas
55–65	chronic stress threshold: elevation of blood pressure, activation of stress hormones, and interference with verbal communication	busy offices; heavy urban street traffic
65–75	clear increase in long-term cardiovascular risk (hypertension, ischemic heart disease) with sustained exposure, particularly at night	near highways or railway lines
75–85	persistent psychological stress and anxiety; possible metabolic and immune dysregulation with prolonged exposure (moderate evidence)	industrial environments; loud music venues
85–110	auditory damage zone: high risk of permanent hearing loss and tinnitus with repeated or prolonged exposure	construction sites; concerts; emergency sirens
≥120	threshold of pain: immediate risk of acoustic trauma, including damage to the eardrum	jet engine takeoff; explosions

Neuroendocrine Activation. Acoustic signals are processed by the auditory cortex and relayed to the amygdala and hypothalamus leading to corticotropin-releasing hormone (CRH) secretion and subsequent ACTH release from the pituitary gland. This triggers the Hypothalamic-Pituitary-Adrenal (HPA) axis and the sympathetic nervous system. The resulting secretion of stress hormones-cortisol, adrenaline, and noradrenaline-prepares the body for a "fight-or-flight" response, even when the person is stationary or asleep [28, 29]. The primary mechanism of noise-induced harm is the activation of the body's non-specific stress response (Fig. 1).

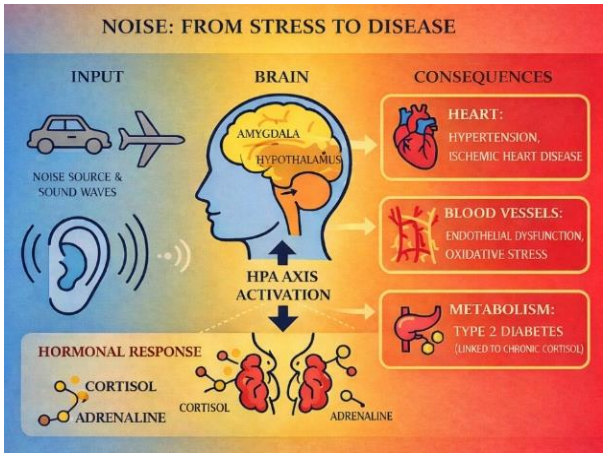

Fig. 1. Pathophysiological mechanisms of noise-induced health impacts. The diagram illustrates the systemic pathway from acoustic input to the activation of the HPA axis, resulting in hormonal responses and long-term consequences for the cardiovascular system and metabolism.

Vascular and Oxidative Stress. Chronic elevation of these hormones leads to significant changes in the cardiovascular system. Recent studies have highlighted endothelial dysfunction as a key consequence. Noise exposure increases the production of ROS in the blood vessels, leading to oxidative stress and inflammation. This process damages the inner lining of the arteries, promoting atherosclerosis, hypertension, and increasing the risk of myocardial infarction (heart attack) and stroke [30].

Metabolic Disruptions. Prolonged cortisol elevation interferes with glucose metabolism and insulin sensitivity. This biochemical shift explains the established link between chronic transport noise and the increased incidence of Type 2 diabetes [30].

Sleep Architecture and Cognitive Impairment. Sleep is essential for biological restoration and the consolidation of memory. Noise is one of the most significant external disruptors of sleep quality.

Fragmentation of Sleep. Noise causes "micro-arousals" that the individual may not consciously remember. These interruptions shift the sleeper from deep, restorative stages to lighter stages of sleep, or reduce the duration of Rapid Eye Movement (REM) sleep. This fragmentation prevents the brain from performing critical maintenance, leading to daytime fatigue, mood disorders, and impaired immune function [28, 30].

Cognitive Development in Children. Children are particularly vulnerable to noise. Scientific evidence (such as the RANCH study) shows that children attending schools near high-traffic areas or airports exhibit significant delays in reading comprehension and long-term memory. Because their brains are still developing, the chronic stress of noise interferes with their ability to filter out irrelevant stimuli, leading to diminished academic performance and increased irritability [28, 30].

Ecological Implications and Wildlife Survival. In the natural world, sound is a vital "information channel". Anthropogenic (human-made) noise creates an "acoustic masking" effect that threatens the survival of various species.

Acoustic Masking and Predator-prey Dynamics. When human-made noise overlaps with the frequencies used by animals, its "masks" important sounds. For many species, this means they can no longer hear the approach of a predator or the calls of their offspring. In marine environments, low-frequency shipping noise and high-intensity sonar can travel hundreds of kilometers, devastating the echolocation and navigation systems of whales and dolphins, often leading to mass strandings [28, 30].

The Lombard Effect and Energy Expenditure. Many birds and mammals have adapted by vocalizing louder or at higher pitches to be heard over urban noise-a phenomenon known as the lombard effect. While this allows for communication, it comes at a high metabolic cost, draining energy reserves needed for migration or reproduction, and often making the animals more conspicuous to predators [30].

Chronic Environmental Noise Exposure in Armenia: Biological Context and Health Implications. The urban acoustic environment of the Republic of Armenia, particularly in Yerevan, represents a setting of sustained environmental noise exposure shaped by specific architectural, traffic, and density-related

characteristics. While the pathophysiological mechanisms of noise-induced stress are biologically universal, local structural conditions may influence cumulative exposure intensity and duration.

In this context, environmental noise should be considered a continuous physical stimulus capable of promoting repeated neuroendocrine activation and sustained oxidative vascular stress within exposed populations.

Unique Urban Soundscapes and Source Profiles. Unlike many European cities where noise sources are spatially segregated, Yerevan's urban environment is characterized by high mixed-use density and overlapping residential, commercial, and transport zones.

Acoustic Amplification. The predominant use of volcanic tuff and other reflective stone materials in Yerevan's architecture, combined with relatively narrow street canyons, facilitates sound reflection and reverberation. These structural conditions may prolong acoustic persistence and amplify transport-related emissions, particularly from the aging vehicle fleet, which remains a primary source of environmental noise [30].

Construction and Service-related Exposure. Recent urban redevelopment and expansion of hospitality and service sectors have increased localized exposure patterns. The proximity of construction sites and high-activity nightlife venues to residential areas, frequently without sufficient acoustic buffering, may elevate chronic exposure levels and intermittently exceed internationally recognized comfort thresholds [31].

Such environmental conditions may contribute to cumulative allostatic load and reinforce mechanisms of endothelial dysfunction and oxidative stress described in previous sections [30].

Gaps in Strategic Monitoring and Data Infrastructure. A primary divergence between Armenia and established European frameworks lies in the systematic collection of longitudinal exposure data.

Mapping Challenges. While the Environmental Noise Directive mandates periodic strategic noise mapping within the European Union, Armenia currently lacks a fully integrated and continuously updated national noise registry. Initial mapping efforts in Yerevan have relied primarily on point-source or episodic measurements rather than comprehensive city-wide surveillance [32].

The absence of longitudinal exposure datasets limits precise estimation of dose–response relationships and population-level health burden.

Regulatory Divergence. Although Armenian sanitary norms provide a regulatory basis for noise control, a measurable gap remains between local enforcement thresholds and the more stringent preventive level of ≤ 53 *dB* Lden recommended by the WHO [4, 31]. This discrepancy may permit persistent low-grade exposure capable of sustaining chronic physiological stress responses even when formal regulatory limits are not exceeded.

Projected Health and Ecological Implications. In Armenia, elevated baseline urban noise is increasingly regarded as a potential contributor to public health trends, although direct epidemiological data remain limited.

Cardiometabolic Considerations. Given the high density of traffic in central Yerevan, a substantial segment of the population is likely exposed to environmental

noise levels that international studies associate with increased risk of hypertension, ischemic heart disease, and metabolic dysregulation [30]. Limited availability of acoustically protected green corridors may further restrict opportunities for physiological recovery from repeated sympathetic activation [30, 32].

Vulnerability of Sensitive Zones. Many educational and healthcare institutions are in high-traffic districts without advanced acoustic insulation. International evidence indicates that chronic environmental noise exposure in such settings is associated with sleep disturbance, stress-mediated endocrine activation, and cognitive interference in children, consistent with patterns observed in RANCH and related sleep-disruption studies [30].

Although Armenia-specific biomarker-based investigations remain limited, extrapolation from established mechanistic evidence suggests that chronic acoustic exposure may represent a modifiable contributor to vascular aging and metabolic vulnerability in urban populations.

Challenges and Prospects for Mitigation. Reduction of environmental noise exposure in Armenia may be approached as a preventive strategy targeting stress-mediated systemic pathology.

Key structural directions include:

1. *Urban planning integration*. Incorporation of acoustic considerations into early stages of urban design and construction permitting may reduce long-term exposure intensity.

2. *Technological transition*. Modernization of the transport sector and adoption of low-noise road surface technologies may decrease baseline environmental noise levels.

3. *Institutional coordination*. Development of coordinated multi-agency monitoring systems may strengthen evidence-based decision-making and align environmental health standards with broader sustainability objectives [33, 34].

Results and Discussion.

Integrating Mechanistic Evidence with Regional Exposure Context. The comprehensive evidence synthesized in this review indicates that environmental noise should be regarded not as a secondary ecological nuisance, but as a biologically active determinant of systemic health. When the global pathophysiological mechanisms outlined in Section “Mechanisms of Noise-Induced Health and Ecological Impact” are considered alongside the regional exposure characteristics described in Section “Chronic Environmental Noise Exposure in Armenia: Biological Context and Health Implications”, several integrative observations emerge [35].

Activation of the HPA axis, sympathetic adrenergic signaling, and oxidative stress pathways represent conserved biological responses to chronic acoustic stimulation. However, the magnitude and persistence of these responses are shaped by exposure intensity, duration, and environmental modulation of acoustic propagation [36]. Architectural morphology, traffic density, and limited vegetative buffering may therefore influence the cumulative biological stress burden in specific urban settings.

In Yerevan, the predominance of stone-based construction materials and high-density urban canyons may enhance sound reflection and prolong acoustic

persistence. Such structural characteristics may increase the continuity of sympathetic activation and oxidative vascular stress compared to urban layouts characterized by dispersed infrastructure or extensive green corridors. However, direct biomarker-based data in Armenia remain limited [37]. Consequently, although hypertension, endothelial dysfunction, and metabolic disruption are mechanistically universal outcomes of chronic noise exposure, accurate risk stratification requires localized environmental exposure data [38].

An important translational consideration concerns the relationship between internationally established exposure thresholds and national monitoring capacity. The WHO and the EEA recommend preventive benchmarks, including the ≤53 *dB* Lden threshold for cardiovascular protection [1]. In the Armenian context, limitations in longitudinal strategic noise mapping constrain precise estimation of exposure distribution and health burden. The absence of a comprehensive Strategic Noise Map represents a significant limitation for quantitative dose–response assessment and for estimating DALYs attributable to environmental noise [39].

Although model-based platforms such as noise-map.com provide indicative visualizations, they do not replace systematic measurement-based monitoring capable of supporting epidemiological correlation with cardiovascular and metabolic outcomes [40]. Without longitudinal exposure datasets comparable to those available in EU member states, accurate quantification of population-level biological burden remains challenging [41].

Nevertheless, extrapolation from established mechanistic evidence suggests that chronic acoustic exposure may contribute to cumulative allostatic load within susceptible urban populations. Interventions aimed at reducing baseline exposure intensity, particularly in high-density traffic zones and sensitive institutional areas-may therefore function as preventive strategies targeting stress-mediated systemic pathology.

The transition toward integrated noise management should thus be interpreted not solely within a regulatory framework but as a component of cardiovascular and metabolic disease prevention. Nature-based buffering strategies, such as vegetative corridors and green infrastructure, may simultaneously attenuate acoustic intensity and support physiological recovery processes, including autonomic recalibration and stress-hormone normalization. Such approaches offer dual environmental benefits, potentially influencing both air quality and noise exposure dynamics.

In summary, although Armenia remains in a developmental phase regarding systematic environmental acoustic monitoring, alignment of national public health priorities with mechanistic cardiovascular and neuroendocrine evidence provides a scientifically grounded pathway for risk reduction and health-centered urban development [42].

Translational Implications and Preventive Strategies. The mitigation of environmental noise exposure should be interpreted within a preventive biomedical framework targeting stress-mediated systemic pathology. Reduction of chronic acoustic stimulation may contribute to lowering cumulative sympathetic activation, oxidative vascular stress, and long-term cardiometabolic risk.

As illustrated in Fig. 2, integrated noise management requires coordinated engineering, technological, and regulatory approaches.

Fig. 2. Integrated noise management strategy for urban environments.

The framework summarizes structural and monitoring-based interventions aimed at reducing chronic acoustic exposure and associated biological stress burden.

In the Armenian urban context, priority measures include the implementation of nature-based buffering systems and targeted infrastructure modernization. Expansion of vegetative corridors and urban canopy coverage may reduce acoustic intensity, while simultaneously supporting thermoregulation and urban resilience [42]. Such interventions may facilitate autonomic recalibration and improve opportunities for physiological recovery from repeated stress activation.

Modernization of the transport sector, including electric mobility transition and low-noise surface technologies, represents a direct method for reducing baseline environmental noise levels. From a biological perspective, lowering chronic exposure intensity may attenuate sustained HPA axis activation and endothelial oxidative stress.

These measures should be complemented by improved exposure monitoring systems capable of linking environmental metrics with health-relevant indicators [41]. Integration of systematic surveillance enables more accurate dose–response assessment and strengthens preventive cardiovascular strategies.

Public engagement and educational initiatives may further enhance awareness of the health implications of chronic acoustic exposure. Citizen-based monitoring platforms can supplement professional measurement systems and increase transparency of exposure distribution [42].

Collectively, these approaches support alignment between environmental management and evidence-based public health protection, reinforcing preventive strategies against stress-mediated systemic disease [23].

Conclusion. Environmental noise should be recognized as a biologically active, non-chemical environmental stressor with measurable systemic consequences. The evidence synthesized in this review demonstrates that chronic acoustic exposure induces sustained activation of the HPA axis, sympathetic adrenergic signaling, oxidative stress, endothelial dysfunction, and inflammatory responses.

These interconnected mechanisms contribute to increased cardiovascular risk, metabolic dysregulation, sleep disturbance, and neurocognitive impairment.

Although global health authorities have established exposure thresholds and monitoring frameworks, effective risk reduction depends on the integration of mechanistic evidence with region-specific exposure assessment. In the context of the Republic of Armenia, the absence of comprehensive longitudinal noise mapping limits precise quantification of exposure–response relationships and population-level health burden.

Strengthening environmental monitoring infrastructure and aligning preventive strategies with established cardiovascular and neuroendocrine mechanisms may facilitate reduction of cumulative allostatic load in urban populations. Addressing chronic acoustic exposure through evidence-based mitigation approaches represents a relevant component of environmental health protection and long-term cardiometabolic disease prevention.

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ՔՐՈՆԻԿ ԷԿՈԼՈԳԻԱԿԱՆ ԱՂՄՈՒԿԸ՝ ՈՐՊԵՍ ԿԵՆՍԱԲԱՆԱԿԱՆ ՍՏՐԵՍՈՐ. ԱԶԴԵՑՈՒԹՅԱՆ ՄԵԽԱՆԻԶՄՆԵՐԸ ԵՎ ՏԱՐԱԾԱՇՐՋԱՆԱՅԻՆ ՀԱՄԱՏԵՔՍՏԸ ՀԱՅԱՍՏԱՆԻ ՀԱՆՐԱՊԵՏՈՒԹՅՈՒՆՈՒՄ

Ձայնային աղտոտվածությունը գնալով ավելի շատ է դիտարկվում որպես հզոր կենսաբանական սթրեսոր, այլ ոչ թե լոկ էկոլոգիական անհարմարություն: Սույն ակնարկում համադրված են գիտական տվյալների լայն շրջանակ, որոնք լուսաբանում են, քրոնիկ աղմուկի ազդեցության պաթոֆիզիոլոգիական մեխանիզմների վերաբերյալ արդի գիտական տվյալները՝ շեշտադրելով դրա դերը հիպոթալամուս–հիպոֆիզ–մակերիկամային առանցքի և սիմպաթիկ նյարդային համակարգի ակտիվացման գործում: Տևական ակուստիկ սթրեսը հանգեցնում է նեյրոէնդոկրին խանգարումների՝ խթանելով ռեակտիվ թթվածնային միացությունների գերարտադրությունը, էնդոթելային դիսֆունկցիան և համակարգային բորբոքումը, որոնք սիրտ-անոթային, նյութափոխանակային և կոգնիտիվ հիվանդությունների զարգացման հիմնական խթանիչներն են: Վերլուծելով Հայաստանի Հանրապետության (հատկապես Երևանի) ուրույն ուրբանիստական միջավայրը, որտեղ քարաշեն ճարտարապետությունն ու բարձր խտությունը ֆիզիկապես ուժեղացնում են

ակուստիկ ազդակները՝ ուսումնասիրությունն ընդգծում է բնակչության շրջանում կայուն ալոստատիկ բեռի մեծացումն ու ֆիզիոլոգիական խոցելիությունը: Հոդվածն ամփոփում է, որ քրոնիկ աղմուկի ազդեցության մեղմացումը կրիտիկական նշանակություն ունի օրգանիզմի ֆիզիոլոգիական վերականգնման համար՝ առաջարկելով հանրային առողջապահության պրակտիկայում ներդնել նեյրոկենսաբանական ցուցանիշները՝ աղմուկով հարուցված համակարգային պաթոլոգիաները կանխելու նպատակով:

Л. Э. БАЛАЯН, А. Г. МКРТЧЯН, А. М. ОВАННИСЯН, Т. О. САРГСЯН,
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ХРОНИЧЕСКИЙ ЭКОЛОГИЧЕСКИЙ ШУМ КАК БИОЛОГИЧЕСКИЙ СТРЕССОР. МЕХАНИЗМЫ ВОЗДЕЙСТВИЯ И РЕГИОНАЛЬНЫЙ КОНТЕКСТ ВОЗДЕЙСТВИЯ ШУМА В РЕСПУБЛИКЕ АРМЕНИЯ

Шумовое загрязнение окружающей среды все чаще рассматривается не просто как экологическое неудобство, а как глубокий биологический стрессор. В данном обзоре синтезированы современные научные данные о патофизиологических механизмах хронического воздействия шума с акцентом на его роль в активации гипоталамо-гипофизарно-надпочечниковой оси и симпатической нервной системы. Продолжительный акустический стресс вызывает нейроэндокринную дисрегуляцию, провоцируя избыточную выработку активных форм кислорода, эндотелиальную дисфункцию и системное воспаление, которые являются основными факторами развития сердечно-сосудистых, метаболических и когнитивных нарушений. Анализируя специфическую городскую среду Республики Армения (г. Ереван), где каменная архитектура с высокой отражающей способностью и высокая плотность застройки физически усиливают акустические стимулы, исследование подчеркивает формирование устойчивой аллостатической нагрузки (allostatic load) и повышенную физиологическую уязвимость населения. В заключении статьи отмечается, что снижение уровня хронического шумового воздействия имеет критическое значение для физиологического восстановления организма, и обосновывается необходимость интеграции нейробиологических параметров в стратегии общественного здравоохранения для предотвращения системных патологий, индуцированных шумом.