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Original Research Paper

Assessing the Effects of Chronic Noise Exposure and Disrupted Wildlife Soundscape on Cardiovascular Health and Hypertension in Urban Residents

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Key Words

Chronic noise exposure, Cardiovascular health, Hypertension, Sleep disruption, Stress hormones, Blood pressure, Urban residents, Wildlife soundscapes.

Abstract

Excessive exposure to environment-related noise, particularly in cities, is also being increasingly considered as a serious risk factor that has led to cardiovascular diseases (CVDs), such as hypertension. The review is devoted to the cardiovascular effects of chronic noise exposure, where the mechanisms involved in the progression and onset of hypertension in urban residents in relation to noise exposure are highlighted. Research has always indicated that noises during the night can interfere with sleep patterns, increase the level of stress hormones, and cause the activation of the sympathetic nervous system, which in turn results in an increase in levels of blood pressure. Moreover, disruptive effects on the glucose metabolism and lipid profiles caused by noise conditions have been associated with unpleasant outcomes in the cardiovascular system. The effect is especially severe among the older generation, males, and less socio-economically advantaged populations, as they might be more susceptible to it, as they have more cardiovascular risk factors at baseline. There is growing information that noise pollution may also disrupt the circadian rhythms in the body, which is even more dangerous in terms of increased hypertension. Chronic urban noise also disrupts natural wildlife and animal soundscapes, reducing exposure to calming acoustic environments created by birds, insects, and other species that help regulate human stress responses. This diminished contact with natural animal auditory cues may further intensify cardiovascular strain and contribute to hypertension progression. The results demonstrate the importance of implementing public health measures to minimize noise levels especially in congested cities so as to curb the risk of cardiovascular health. More studies are needed to know how exactly the biological process works and to create interventions that should lead to the decrease of the burden of noise-induced hypertension.

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Introduction

The noise in the city has become a common environmental stressor, which has a huge impact on the health and well-being of people living in urban areas (Meng et al., 2022). Noise pollution, especially that which is produced by traffic, industry, and other sources, is becoming one of the most identified risk factors to cardiovascular diseases (CVDs) (Peng et al., 2023). Hypertension, which is a condition that is closely associated with cardiovascular dysfunction, is one of the most frequent and worrying effects of prolonged noise exposure (Lee et al., 2023). Research has indicated that noise interferes with physiological functions in raising the levels of stress hormones, sleeping disruption, and activation of the sympathetic nervous system, all of which lead to high blood pressure (BP) (Krittanawong et al., 2023; Qiu et al., 2022; Rogers et al., 2024; Haufe & Leeners, 2023). Moreover, noise has also been associated with disruption of metabolic functions, in terms of blood glucose and lipid levels, which adds to cardiovascular risks, which is further aggravated by noise (Zhang et al., 2025; Nakshine et al., 2022; Desai et al., 2024; Zhang et al., 2017). Although the connection between environmental noise and hypertension and cardiovascular dysfunction is becoming more evident, there still remains a huge gap in the realization of the extent to which noise affects the human body, especially when it comes to an urban setting (D'Souza et al., 2021). In addition to these physiological mechanisms, chronic noise exposure alters natural sound environments by masking wildlife vocalizations and disrupting acoustic communication among birds, mammals, and other urban animal species. Research shows that anthropogenic noise interferes with animal behavioral patterns, territory signaling, and stress responses, leading to diminished natural soundscapes in cities (Shannon et al. 2016). These animal-generated soundscapes are known to support human psychological recovery and autonomic regulation, and their loss may heighten stress reactivity and cardiovascular load. Reduced exposure to restorative animal sounds such as bird calls and insect choruses limits opportunities for auditory relaxation, further intensifying the cardiovascular impacts of urban noise. This ecological and animal-behavioral dimension emphasizes the need to understand noise exposure within broader environmental disturbances. The connection between long-term exposure to urban noise and its impact on hypertension has not been fully explored, as well as its interaction with other environmental and socio-economic factors (Eckrich et al., 2021). In addition, the possibility of the noise exposure promoting the emergence of treatment-resistant hypertension makes the issue more complicated, as it makes it difficult to manage and control the population at risk. The objective of this paper is to discuss the link between prolonged exposure to noise and cardiovascular health, and specifically, hypertension (Dusi, 2024; Sadulla, 2025). The main goals are to determine the impact of the urban noise on our blood pressure rates, examine its correlation with the occurrence of hypertension in urban dwellers, and find out how the noise influences the hypertension control and

treatment adherence rate (Warembourg et al., 2021; Poulsen et al., 2021; Shannon et al., 2016). Also, the research aims at pointing out the susceptibilities of various population groups, such as older adults and individuals with lower socio-economic statuses, who could be at a greater risk of cardiovascular effects through exposure to noise (Gupta & Verma, 2025; Sadulla, 2025).

Key Contributions

- Presentation of Empirical Evidence: The paper presents empirical evidence of large cohort studies on the relationship that exists between long-term urban noise exposure and hypertension.
- Two Cohort Analyses: The analysis of the study is based on the data of two dissimilar cohorts in Chicago, Illinois, which provides a wide overview of the issue of city noise and its impact on cardiovascular health.
- Impact on Blood Pressure: The paper discusses the effect of an extended exposure to noise in cities on the levels of systolic and diastolic blood pressure among the urban residents.
- Hypertension Management Challenges: In the study, I would focus on the issues of hypertension management in urban settings with the prevalence of noise exposures.
- Noise as a Modifiable Risk Factor: The article reiterates the fact that environmental noise should be viewed as a modifiable risk factor of hypertension, and prophylactic and curative interventions may be put in place.

The paper is divided into four sections. Section 2: Materials and Methods includes the research design, the selection of participants, and the data collection process, as it will examine the two cohort studies (CHAP and MESA) that will be analyzed in this paper. It explains the way in which the exposure to noise in the city was evaluated by the use of spatial prediction models and the way in which the level of blood pressure was measured and classified. Part 3: Results and Discussion reports the results, indicating the high level of connection between the increased level of noise in the city and the elevated level of blood pressure, and the predisposition to the development of hypertension that resists treatment. These findings are interpreted in the discussion by examining possible mechanisms involved, such as stress and sleeping problems, and the difficulties in controlling the hypertension in urban areas. It further recommends the need to incorporate environmental noise as a constituent of the health measures of the populace. Lastly, Section 4: Conclusion includes the summary of the main findings and the necessity of policies that can help to decrease noise exposure as a risk factor that can be modified in a cardiovascular sense. Future research recommendations and strategies for mitigating noise are also given.

Materials and Methods

In this research, data were used in 2 large prospective cohort studies, which were the Chicago Health and Aging Project (CHAP) and the Multi-Ethnic Study of Atherosclerosis

(MESA). The study was planned to determine the linkage between chronic exposure to noise in the city and the cardiovascular health, i.e., hypertension, among older adults. The sections below explain the study population, approaches to noise exposure assessment, approaches to blood pressure measurement, plans of data analysis, and ethical considerations.

Study Population

The CHAP study enrolled participants at the age of 65 years or above and recorded those who were in Chicago, Illinois, to explore the risk factors for long-term diseases like hypertension and cardiovascular disease. In our study, we have used data from the CHAP cohort observed over the past 2000 patients aged 65 years and above. In the same way, the MESA study has sampled the 45-84 age group of people in six metropolitan areas in the United States with an orientation on subclinical cardiovascular disease. This analysis also involved the MESA data of the Chicago-based participants (n=691). The overall sample to be used in this study consisted of 6,764 participants in both cohorts, and 16,462 repeated measurements of blood pressure measures were taken in a mean follow-up of 4 years.

Noise Exposure Assessment

Long-term exposure to noise in urban areas was the most important variable of interest. This was estimated with the help of a spatial prediction model that was coded for the Chicago area. In 2006 and 2007, there were two intensive noise measurement campaigns, in which 5-minute grab samples of A-weighted decibel (dBA) values were taken at 136 locations in the city. A-weighted decibel scale takes into consideration the sensitivity of the human ear to different kinds of frequencies, and thus, the readings are the perceived sound levels. The model took into account geographic covariates like land use, land proximity to sources of transportation (highways, airports, and railways), and urban infrastructure as predictors of noise exposure at the residence of the participant. These forecasts were checked against field data in 2016 and were found to be in good agreement with time. The average levels of noise exposure calculated in decibels (dBA) during the last 5 years prior to the visit were calculated using the long-term residential history of the participants. Each participant was estimated to have a spatially resolved noise exposure depending on the geographic location where the participant stayed.

Figure 1 demonstrates the spatial prediction model that was used to predict the exposure to noise in Chicago. It reveals how the data flow occurs, starting with the main factors of input, including land use, roads, airports, and railways, which leads to the ultimate noise forecast, expressed in decibels (dB(A)). The model uses the following environmental variables in computing long-term residential noise exposure to individuals under study.

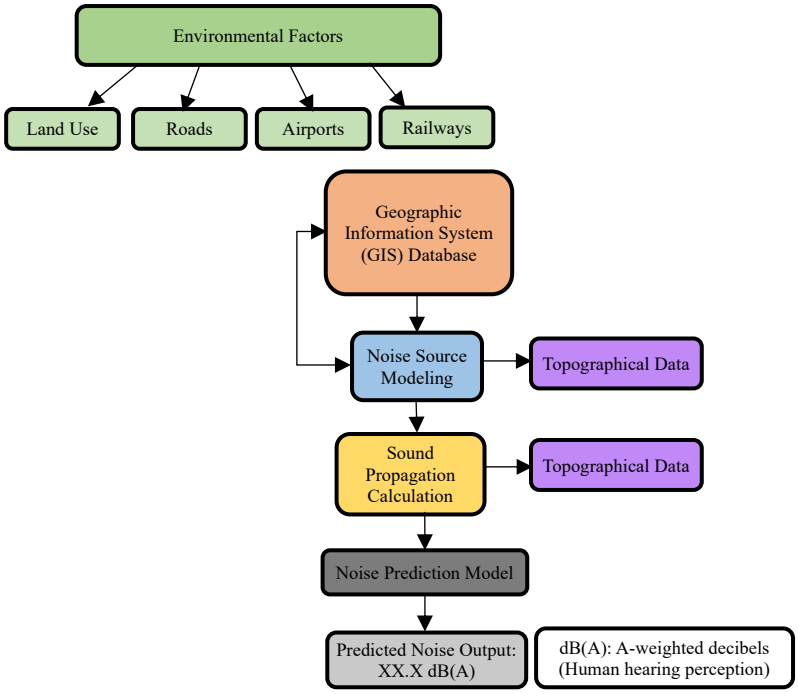


Figure 1: Noise Exposure Model for Chicago

Blood Pressure Measurement

The measurement of blood pressure (BP) was taken when the participants visited the research centers or during in-home measuring, depending on the study protocol. Measurement of BP came following a 5-minute rest period for the participants in the seated position. To measure the systolic blood pressure (SBP) and the diastolic blood pressure (DBP), two consecutive measurements were made at every visit, with an average of the measurements calculated and taken as the systolic blood pressure or diastolic blood pressure. Those who had a BP of SBP 140mmHg and DBP 90mmHg were considered to be hypertensive. The individuals under antihypertensive medication were also analyzed, as their levels of BP were imputed by the medications they were prescribed and adequate characteristics. In order to measure the level of hypertension management, the subjects were characterized into three variables, namely normal BP (SBP <140 mmHg and DBP <90 mmHg), uncontrolled hypertension (SBP 140 mmHg or higher and DBP 90 mmHg or higher, with or without medication use), and apparent treatment-resistant hypertension (aTRH) (those taking more than one antihypertensive medication and yet hypertensive). The main area of interest was the relationship between the exposure to noise over the long term and the BP levels among these groups.

Covariates and Confounders

Both cohorts had data on possible confounders collected during subsequent visits and at baseline. They were the following variables: Products:

Demographic: Sex, age, race, ethnicity.

Socioeconomic Status (SES): SES score of Income, Education, and Neighborhood.

Lifestyle Factors: Smoking condition, alcohol consumption, exercise, and diet.

Comorbidities: A history of chronic diseases, including diabetes, obesity, and heart attacks in the past.

Air Pollution: The estimated spatiotemporal model-based ambient concentrations of nitrogen oxides (NOx) and fine particulate matter (PM 2.5).

Light Pollution: The level of light pollution per year was also logged, as this would be a confounding variable in the relationship between noise and BP.

There were other environmental variables that were incorporated in the models, such as air pollution, to consider the possible confounding variables. Traffic noise is usually associated with these pollutants, and the effect of their possible impact on BP had to be controlled.

Statistical Analysis

Multivariate linear mixed models were used to determine the relationship between noise exposure and the levels of BP. These models explained repeated measures of BP by the same subjects. As not all of the participants were taking any antihypertensive drugs, the blood pressure levels have been imputed in these cases, taking into consideration their age, sex, race, and the type of antihypertensive drug/ drugs. In the case of categorical outcomes, including those of uncontrolled hypertension and apparent treatment-resistant hypertension (aTRH), the logistic regression models were employed. The pooled model was conducted using each cohort (CHAP and MESA) independently and combined. The models were also corrected in case of confounding factors such as demographic variables, SES, as well as co-exposure to air pollutants. The terms of interaction were also put to the test to see if they were affected by age, sex, and race. Nonlinearity of noise-BP association was measured in b-splines in the secondary analyses, and sensitivity analyses were performed to test the strength of the findings to alternative model specifications and corrections to the possibility of bias. Inverse probability weighting was used to address the possibility of selection bias in the final models.

Results and Discussion

This paper compared the relationship between chronic urban noise exposure and blood pressure (BP) among the sample of two cohort studies, including the Chicago Health and Aging Project (CHAP) and the Multi-Ethnic Study of Atherosclerosis (MESA). The total number of participants (6,764: 6073 CHAP and 691 MESA) in the analysis consisted of 16,462 repeated readings of blood pressure in an average follow-up time of 4 years. The participants were subjected to different levels of noise in the home environment with the exposure levels of 51 to 81 dBA.

Characteristics of the Study Population

Table 1 will give a summary of the baseline characteristics of the participants. The average age of the respondents was 72.7 years, and the participants in the CHAP cohort were a little older (mean age 73.8 years) than those in the MESA cohort (mean age 63.2 years). It is also indicated in the table that the systolic blood pressure (SBP) of CHAP participants (mean 135.2mmHg) was higher than the systolic blood

pressure (SBP) of MESA participants (mean 123.4mmHg), probably because CHAP participants were older. Likewise, the trend was the same in the values of diastolic blood pressure (DBP), where the mean values of CHAP and MESA participants were at 80.1 and 75.4mmHg, respectively. The exposures to noise were also relatively high, and the average one was 60.5 dBA in the CHAP cohort and 59.4 dBA in the MESA cohort.

Table 1: Baseline Characteristics of the Study Population

Variable	Mean (SD)	CHAP (n = 6073)	MESA (n = 691)
Age (years)	72.7 (8.0)	73.8 (7.0)	63.2 (10.1)
Systolic BP (mmHg)	134.0 (18.6)	135.2 (18.9)	123.4 (20.1)
Diastolic BP (mmHg)	79.8 (10.4)	80.1 (10.5)	75.4 (10.2)
Noise Exposure (dBA)	60.5 (5.4)	60.8 (5.6)	59.4 (5.3)

The demographic data of the members of both cohorts are displayed in Table 1, as well as the level of noise exposure. The CHAP participants were older and had higher blood pressure than the MESA participants, with the average levels of noise exposure being 60 dBA in the two cohorts.

Noise Exposure and Blood Pressure

Table 2 indicates that there was a correlation between the increments in the Diastolic and Systolic blood pressure and the elevation in noise exposure. We found out that on a 10 dBA rise in residential noise, systolic (95% CI, 0.1 to 2.2) and diastolic (95% CI, 0.6 to 1.7) BP rose by 1.2 and 1.1 mmHg respectively in the research sample. These findings were similar in both the CHAP and MESA cohorts and it was also indicative that there is a strong and uniform impact of chronic noise exposure on blood pressure.

Table 2: Association Between Noise Exposure and Blood Pressure Changes

Noise Exposure (dBA)	Systolic BP Change (mmHg)	Diastolic BP Change (mmHg)
10 dBA Increase	1.2 (0.1–2.2)	1.1 (0.6–1.7)

This table 2 shows approximated changes in systolic and diastolic blood pressure, with every 10 dBA residential noise exposure, and the 95% confidence limits of the same. The level of noise, both systolic and diastolic blood pressure were significantly higher with the increase in noise exposure implying that chronic noise exposure is one of the factors contributing to high blood pressure levels.

Hypertension and Treatment-Resistant Hypertension (aTRH)

Exposure to noise also was associated with uncontrolled hypertension and apparent treatment-resistant hypertension (aTRH). Each rise of 10 dBA of residential noise had odds of

20% of increasing the odds of aTRH (odds ratio: 1.2, 95% CI, 1.01.4). The implication of this association is that the control of hypertension, despite medication, may be complicated with the increase in noise levels. The correlation was similar in both groups but it was a little higher in MESA which could be explained by the fact that the participants of MESA were younger than those of CHAP.

Table 3: Odds of Apparent Treatment-Resistant Hypertension with Increased Noise Exposure

Outcome	Odds Ratio per 10 dBA Increase	95% Confidence Interval
Apparent Treatment-Resistant Hypertension (aTRH)	1.2	1.0–1.4

This Table 3 indicates the odds ratio of developing apparent treatment-resistant hypertension (aTRH) with an increase of 10 dBA in the residential noise exposure. As a result, a strong correlation was found, which suggests that an increase in the level of noise can be the cause of developing the treatment-resistant hypertension.

Discussion

The findings validate that chronic exposure to urban noise is closely correlated with the high systolic and diastolic blood pressure. The relationship of noise and BP was seen in both CHAP and MESA cohorts even though the demographics of the participants varied age, and geographical region. Precisely, a 10 dBA rise in noise exposure played a role in 1.2 mmHg rise of systolic BP and 1.1 mmHg rise of diastolic BP. These findings are in line with mounting evidence that suggests the association between environmental noise and hypertension and also highlights its significance as a significant, yet neglected, cardiovascular risk factor. Another aspect that is also brought to light in the study is how noise exposure affects hypertension treatment-resistant (aTRH), which is a complication making it difficult to effectively treat hypertension. As implied by our results, noise exposure might be a contributing factor in the increased risk of developing aTRH and hence more difficult to develop controlled BP despite the use of antihypertension. The odds ratio of increase in aTRH by 20% with a 10 dBA change in environmental noise highlights the necessity of specific treatment of people who are exposed to high levels of environmental noise, especially under urban settings. Biological processes that lead to the connection between hypertension and noise exposure are most likely to rely on a number of physiological processes. Prolonged exposure to noises may also stimulate the sympathetic nervous system thereby accelerating the release of catecholamines like adrenaline resulting in vasoconstriction and rise in heart rate resulting in higher blood pressure. Moreover, long term exposure to noise has been observed to disrupt sleep patterns and alter circadian rhythms, which are some of the factors that lead to the amplification of blood pressure levels (Park et al., 2021). Moreover, oxidative stress and stress caused by noises may lead to endothelial dysfunction, which is one of the primary processes leading to the structural progression of hypertension and cardiovascular disorders. These results are of significance to the public health.

Since a good percentage of the urban population are subjected to the noise of the environment, especially in highly populated regions, noise pollution reduction may become a useful approach to decreasing the incidence of hypertension and its adverse effects (Krishnan et al., 2025). The urban planning to curb the traffic noise, the noise control policies, and the insulation of the buildings to reduce the noise infiltration should be regarded as public health strategies through which the urban noise can be mitigated. Also, the measures to promote noise-reducing technologies and policies, focused on high-noise zones, might also prove successful.

Limitations and Future Directions

Although the research findings provide strong arguments that urban noise is a risk factor in hypertension, there are a number of weaknesses that have to be noted. The amount of noise exposure was estimated as residential location, and we failed to consider the difference in exposure levels at different times of the day or seasons. This would cause certain misclassification bias to the findings since the amount of noise exposure can vary across the day or year. In addition, the study failed to identify the impacts of various forms of noise sources separately (e.g., road traffic, industrial noise, aircraft noise), as they might have varying effects on cardiovascular health. The studies in the future need to concentrate in source specific noise and how this type of noise varies with time in the long run. Also, the direct effect of noise mitigation on the hypertension and cardiovascular health outcomes would be better determined with the assistance of interventional work that concentrates on the strategies of noise reduction, such as sound insulation or noise control interventions in urban settings. There is a need to do further research on biological processes (including genetic vulnerability, stress biomarkers, and vascular effects) to further understand the effect of noise exposure on hypertension and its associated cardiovascular incidences.

Conclusion

This paper has presented strong arguments to confirm that chronic exposure to noises in the urban environment is a major risk factor to high blood pressure and non-responsive hypertension to treatment. We have found that residential noise exposure is related to a 10 dBA increase in systolic blood pressure and a 1.1 mmHg increase in diastolic blood pressure, and these findings are similar in two large cohort studies. Also, the study reveals the augmented chances of apparent treatment-resistant hypertension (aTRH) manifestation, where odds of the hypertension augmented by 20 percent with a 10-dBA aggrandation in noise exposure. These findings are important especially considering that exposure to urban noise, especially the high-density community resides, is prevalent, hence the need to integrate noise pollution as an adjustable cardiovascular risk factor. Urban noise reduction in terms of policy change, urban planning and noise mitigation strategies may help dramatically reduce the hypertension burden and cardiovascular health outcome in urban populations. Through additional research on the mechanisms by which noise exposure is associated with hypertension and interventional

studies to minimize noise exposure, it is necessary to reinforce the recommendations to individuals on the significance of maintaining good health and enhancing the prevention of hypertension, especially among the vulnerable groups. The disruption of wildlife soundscapes adds an important environmental layer to the cardiovascular burden of urban noise exposure. Natural acoustic environments contribute to stress recovery, and their loss may amplify sympathetic activation and hinder blood-pressure regulation. Restoring and protecting wildlife-rich acoustic habitats in cities could complement noise-reduction strategies and strengthen cardiovascular resilience. Integrating ecological considerations into noise-management policies may therefore yield broader public-health benefits. To sum up, reducing noise in cities is not merely a compulsory measure to enhance health of people but rather a working way of facing the increasing risk of hypertension and heart diseases in cities.

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