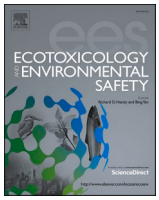
Ecotoxicology and Environmental Safety 268 (2023) 115677

Contents lists available at ScienceDirect

Ecotoxicology and Environmental Safety

journal homepage: www.elsevier.com/locate/ecoenv



The relationship between exposure to environmental noise and risk of atopic dermatitis, asthma, and allergic rhinitis

Yongho Lee [a,1], Seunghyun Lee [b,1], Seula Park [c], Seong-Kyu Kang [a,d], June-Hee Lee [e], Dong-Wook Lee [f], Won-Jun Choi [a,d,2], Wanhyung Lee [b,*,2]

[a] Department of Occupational and Environmental Medicine, Gil Medical Center, Incheon, Republic of Korea
[b] Department of Preventive Medicine, College of Medicine, Chung-Ang University, Seoul, Republic of Korea
[c] Department of Civil and Environmental Engineering, Seoul National University, Seoul, Republic of Korea
[d] Department of Occupational and Environmental Medicine, Gachon University College of Medicine, Incheon, Republic of Korea
[e] Department of Occupational and Environmental Medicine, Soonchunhyang University Hospital, Seoul, Republic of Korea
[f] Department of Occupational and Environmental Medicine, Inha University Hospital, Inha University, Incheon, Republic of Korea

ARTICLE INFO

Edited by Renjie Chen

Keywords:
Noise
Environmental noise
Environmental disease
Allergic disease
General population
Epidemiology

ABSTRACT

Background: Noise is defined as unwanted sound. It may induce negative emotions and mental health problems and may even lead to increased suicide risk. However, the impact of noise exposure on environmental diseases and disease severity is not well understood. This study aimed to elucidate the association between night-time noise exposure and the prevalence of environmental diseases in South Korea.

Methods: We conducted an analysis of the Environmental Disease Database provide by the National Health Insurance Service (NHIS) from 2013 to 2017. After spatially interpolating the noise data provided by the National Noise Information System (NNIS), night-time noise values in the district level were obtained by calculating the mean noise values at the administrative district level. The linear regression analyses were performed to test the association between the age-standardized prevalence ratio (SPR) and the night-time noise exposure in the district level.

Results: In areas with high night-time noise exposure (≥55 dB), the SPR for atopic dermatitis and allergic rhinitis were 1.0515 (95 % confidence interval [CI]:1.0508–1.0521) and 1.0202 (95 % CI:1.0201–1.0204), respectively, which were higher than those in the general population. The SPR for environmental diseases, including atopic dermatitis, asthma, and allergic rhinitis, was 1.0104 (95 % CI:1.0103–1.0105). Additionally, a significant linear association was observed between the level of nocturnal noise exposure and the total hospitalization period for atopic dermatitis ($\beta = 399.3$, $p < 0.01$).

Conclusion: We provide evidence of a significant association between night-time environmental noise and environmental diseases, particularly atopic dermatitis and allergic rhinitis. Furthermore, we observed a significant linear association between night-time noise exposure and the severity of atopic dermatitis.

1. Introduction

Environmental factors, such as air pollution and airborne allergens, may contribute to the development of allergic diseases, including atopic dermatitis, asthma and allergic rhinitis, through the sensitization of individuals with genetic predispositions (Jenerowicz et al., 2012). The prevalence of these allergic diseases, often referred to as environmental diseases, has increased in recent decades, and they are now considered major chronic diseases globally. Although reports from some Western countries suggest that the prevalence of environmental diseases has been declining in recent years, environmental diseases affect over 25 % of the population in industrialized countries and are increasing in prevalence in some developing countries due to urbanization and the loss of rural environments, representing a significant socioeconomic burden (Anandan et al., 2010; Kang et al., 2018).

As urbanization and industrialization accelerate, environmental

* Correspondence to: Department of Preventive Medicine, College of Medicine, Chung-Ang University, 84 Heukseouk-Ro, DongJak-Gu, Seoul, Republic of Korea.
E-mail address: wanhyung@gmail.com (W. Lee).
[1] Yongho Lee and Seunghyun Lee contributed equally to this work as first authors.
[2] Won-Jun Choi and Wanhyung Lee contributed equally to this work as corresponding authors.

https://doi.org/10.1016/j.ecoenv.2023.115677
Received 27 August 2023; Received in revised form 9 November 2023; Accepted 9 November 2023
Available online 17 November 2023

issues, such as aeroallergens, food, climatological factors, humidity, and stress factors, have been reported to negatively contribute to human health (Ogulur et al., 2021). Although genetic predisposition plays an important role in disease pathogenesis, environmental factors also play an important role in the development of these environmental diseases. Previous studies have reported the effects of physical environmental factors, such as aeroallergens and climatologic factors, on the respiratory and circulatory systems (D'Amato et al., 2020; Li et al., 2022). The pathophysiology of environmental diseases due to these environmental factors indicates that environmental allergens act on genome-inducing modifications, which function as important effectors of the inflammatory response and enhance the ability to produce specific immunoglobulin E (IgE) antibodies (Eguiluz-Gracia et al., 2020; Moreno, 2016).

At the same time, industrialization and urbanization inevitably generate noise as a by-product of their operations, which cannot be completely eliminated, thereby making it impossible to escape the noisy environment in human life. Numerous studies have extensively focused on the relationship between noise exposure and hearing loss due to the tiny hair cells responsible for transmitting sound signals to the brain (Hong et al., 2013; Shi et al., 2021). In contrast, previous studies have shown that noise exposure is related to non-auditory health effects, such as nervous system symptoms and mental health in Koreans (Lee et al., 2017; Yoon et al., 2014). Some studies have reported that noise exposure is associated with reproductive and digestive systems (Min and Min, 2018; Min and Min, 2017).

Recent studies have mentioned the probability that noise exposure plays a role in the incidence and prevalence of environmental diseases; however, limitations exist, such as the use of self-reported questionnaires to assess noise exposure or diagnose diseases, as well as participant selection being restricted to specific age groups or regions. Moreover, the findings from these studies exhibited inconsistency. Certain studies indicated a heightened prevalence or risk of allergic diseases in relation to nighttime noise exposure, whereas others yielded conflicting outcomes, suggesting an absence of a definitive association (Faraji et al., 2022; Kim et al., 2022; Wallas et al., 2020).

To fill these gaps, the primary aim of this study was to investigate whether exposure to environmental noise is associated with environmental diseases in Korea. Using the Environmental Disease Database of the National Health Insurance Service (NHIS) and noise data from the National Noise Information System (NNIS), we conducted a population-based cohort study to assess the association between environmental noise exposure and the prevalence of environmental diseases.

2. Methods

2.1. Data source

This study used data from the Environmental Disease Database of the National Health Insurance Service (NHIS) from 2013 to 2017. The Korean NHIS program is a mandatory public health insurance system that offers medical services to almost all Koreans and collects records of medical services and their utilization by all Koreans in its database. In recent years, there has been a growing interest in environmental factors, such as environmental pollution, climate change, and harmful substances, leading to an increase in related studies using big data. To analyze the impact of environmental factors in specific areas on the medical use for environmental diseases, such as allergic or respiratory diseases, the NHIS processed medical data by region (administrative district-city/county/district), date, and basic patient characteristics, including sex and age (provided in 5-year intervals), to create a separate Environmental Disease Database. However, to facilitate open data access for individual researchers, the data were constructed in the form of a statistical table with a low risk of personal identification. The details of the NHIS data have been described in a previous study (Yoo et al., 2021).

2.2. Outcome variables

In this study, we assessed several given variables, including sex (male and female), age, 5-digit postal code (administrative district-city/county/district), and daily medical service utilization of outpatients and inpatients. The environmental diseases represented in our study include atopic dermatitis, allergic rhinitis, and asthma. Atopic dermatitis (L20), allergic rhinitis (J30), and asthma (J45, J46) were identified using diagnostic codes from the 10th International Classification of Diseases (ICD-10).

2.3. Environmental noise and estimation of noise exposure

In this study, we investigated the impact of nocturnal noise exposure on environmental diseases between 2013 and 2017 using night-time noise data from the National Noise Information System (NNIS). The NNIS also provides information on metadata, measurement locations (specified with a 5-digit postal code), and statistics on environmental noise across the country. Noise measurement sites were selected as representative locations for noise levels in the target areas based on urban land use classifications, with three general spots and two roadside spots per area. Furthermore, measurements were conducted during two distinct periods: daytime (6 am–10 pm) and night-time (10 pm–6 am), with two nocturnal measurements taken at 11 pm and 1 am. Here, night-time noise data by manual methods were employed, which were obtained from a relatively higher number of sites than automatically measured data. Consequently, night-time noise data from 1678 measurement sites were converted to average values and processed for further analysis after excluding data from sites with inaccurate geographic coordinates.

Owing to the uneven spatial distribution and incomplete coverage of noise measurement sites in the entire target area, an estimation of noise levels in unmeasured areas was required. Empirical Bayesian Kriging (EBK), a widely adopted Geographic Information System (GIS) interpolation method, was used to evaluate the noise levels in missing areas. EBK is a spatial interpolation method based on the iterative process of subsetting and simulating; the process involves a semi-variogram model estimation from data, the new value simulation at each location using the estimated semi-variogram, a new semi-variogram model estimation from simulated data, and weight calculation of the semi-variogram (Krivoruchko, 2012). The EBK has been adopted for noise mapping because of its superiority in interpolating spatially-correlated data (Banerjee et al., 2016; Min and Min, 2018; Zambrano-Monserrate and Ruano, 2019). In particular, the EBK is an effective approach for interpolating nonstationary data for a large area because it ensures stable performance, even with small datasets, by accounting for both local and global variability (Krivoruchko, 2012; Krivoruchko and Gribov, 2020).

By using the EBK method for noise interpolation, a statistical noise surface covering the target area was created from the collected noise data. This statistical surface was composed of grids that stored the estimated noise values. Noise values by administrative district could be obtained by calculating representative values from the overlapped area on noise surfaces, where the mean of the noise values stored in the overlaid grids was calculated as the noise value for each district using zonal statistics. Fig. 1. shows the choropleth map used to depict the difference in noise levels by district using interpolated data.

The participants were divided into two groups based on their estimated noise exposure using the standard code of the administrative districts. The selection of a cutoff value of 55 dB was based on the WHO night-time noise guidelines (2009), which recommend a target value of 55 dB for interim night noise levels (Europe, 2009).

2.4. Statistical analysis

An indirect age-standardized prevalence ratio (SPR) and its corresponding 95 % confidence interval (CI) were estimated in the current

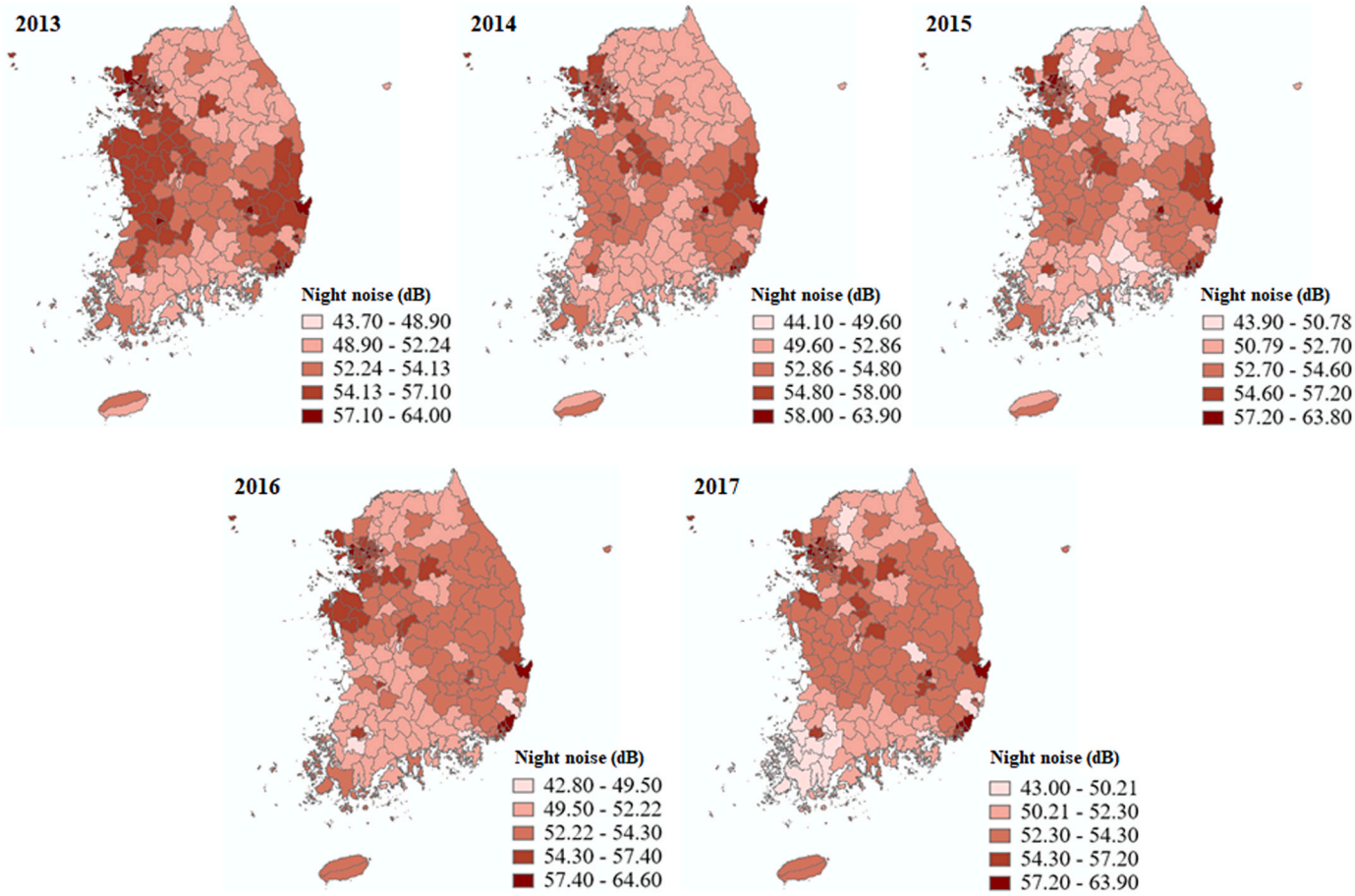


Fig. 1. Visualization of night-time noise distribution using natural breaks classification (2013–2017).

analysis using all study participants as the reference population. The SPR was calculated by determining the age-specific prevalence of environmental diseases using outpatient visits and the number of person-years for each 5-year age group in the population. The number of expected events of each disease was calculated by multiplying the disease prevalence in the reference population. To determine whether the SPR is higher or lower in a low night-time noise area, we calculated the ratio of the SPR in high night-time noise exposure to the SPR in low night-time noise exposure. To assess disease severity, linear regression was used to evaluate the relationship between night noise exposure

Table 1
Outpatient visits by patients with environmental diseases (2013–2017).

	Person-Year	Outpatient visits			
		Environmental diseases*	Atopic dermatitis	Asthma	Allergic rhinitis
Total	258,934,139	775,119,041 (100.0)	29,951,696 (3.9)	127,863,007 (16.5)	617,304,338 (79.6)
Sex					
Male	129,547,186 (50.0)	357,032,429 (46.1)	14,702,199 (49.1)	60,475,559 (47.3)	281,854,671 (45.7)
Female	129,386,953 (50.0)	418,086,612 (53.9)	15,249,497 (50.9)	67,387,448 (52.7)	335,449,667 (54.3)
Age					
0 – 19	51,576,808 (19.9)	347,006,426 (44.8)	14,937,812 (49.9)	61,565,381 (48.1)	270,503,233 (43.8)
20 – 39	73,549,192 (28.4)	117,032,690 (15.1)	4936,913 (16.5)	10,923,037 (8.5)	101,172,740 (16.4)
40 – 59	85,767,862 (33.1)	156,428,853 (20.2)	4859,350 (16.2)	19,997,010 (15.6)	131,572,493 (21.3)
≥60	48,040,277 (18.6)	154,651,072 (20.0)	5217,621 (17.4)	35,377,579 (27.7)	114,055,872 (18.5)
Night noise					
≥55 dB	85,036,540 (32.8)	251,953,259 (32.5)	10,126,792 (33.8)	38,632,816 (30.2)	203,193,651 (32.9)
<55 dB	173,897,599 (67.2)	523,165,782 (67.5)	19,824,904 (66.2)	89,230,191 (69.8)	414,110,687 (67.1)

* Environmental diseases (atopic dermatitis, asthma, allergic rhinitis)

levels and total hospitalization period for environmental diseases. Statistical significance was set at $p < 0.05$, and all statistical analyses were conducted using SAS version 9.4 (SAS Institute, Cary, NC, USA).

3. Results

The basic characteristics of the outpatient visits for environmental diseases, including atopic dermatitis, asthma, and allergic rhinitis, in South Korea between 2013 and 2017 are shown in Table 1. Over the five-year period, 775,119,041 outpatient visits were recorded from 258,934,139 person-years. The distribution of visits for specific diseases was as follows: atopic dermatitis, 29, 951, 696 (3.9 %); asthma, 127, 863, 007 (16.5 %); allergic rhinitis, 617,304,338 (79.6 %). The sex distribution of patients was nearly equal, and patients aged 0–19 years comprised 19.9 % of the total visits, those aged 20–39 years accounted for 28.4 %, those aged 40–59 years accounted for 33.1 %, and those aged 60 years or older comprised 18.6 %. Female patients and those aged 0–19 years accounted for a higher proportion of visits for each of the three diseases. In terms of environmental factors, 32.5 % of all outpatient visits were associated with night-time noise levels of 55 dB or higher. In areas with less than 55 dB of night-time noise, the proportion of outpatient visits was 67.5 %.

In areas with high noise exposure (≥ 55 dB), the mean value of night-time noise exposure was 57.4 dB, with a standard deviation (SD) of 2.0. The maximum, minimum, intermediate, and interquartile ranges (IQR) values were 63.8 dB, 55.1 dB, 56.8 dB, and 2.7 dB, respectively. In contrast, in areas with lower noise exposure (< 55 dB), the mean value of night-time noise exposure was 52.6 dB, with an SD of 1.5. The maximum, minimum, intermediate, and IQR values were 54.9 dB, 43.5 dB, 52.7 dB, and 2.1 dB, respectively.

The association between night noise exposure and outpatient visits for environmental diseases is presented with the age-standardized prevalence ratio (SPR) in Table 2. In areas with high noise exposure during the night (≥ 55 dB), the SPR (95 % Confidence Interval; CI) for environmental diseases was 1.0104 (1.0103–1.0105) for both males and females and 1.0945 (1.0943–1.0947) for females, which was significantly higher than that of the general population. The SPR for atopic dermatitis was 1.0714 (1.0704–1.0723) for males, 1.0316 (1.0307–1.0325) for females, and 1.0515 (1.0508–1.0521) for all sexes, while the SPR for allergic rhinitis was 1.0202 (1.0201–1.0204) for all sexes and 1.1169 (1.1167–1.1172) for females, which was significantly higher than that in the general population. However, no significant SPR results were found for patients with asthma compared to the general population. In addition to the SPR of diseases, the rate ratios (SPR of the high exposure group to the low exposure group to night noise) of environmental diseases, atopic dermatitis, and allergic rhinitis were higher than 1 in Table 3.

As shown in Fig. 2., based on simple linear regression analysis, there was no significant correlation between the level of exposure to night-time noise and the total hospitalization period for environmental diseases ($\beta = 7533.8$, $p = 0.14$), asthma ($\beta = 3870.5$, $p = 0.12$), or allergic rhinitis ($\beta = 3264.1$, $p = 0.22$). However, a significant linear association was observed between the level of exposure to night-time noise and the total hospitalization period for atopic dermatitis ($\beta = 399.3$, $p < 0.01$).

4. Discussion

Therefore, we investigated the association between night-time noise exposure and environmental diseases. Our study found that the age-standardized prevalence ratio (SPR) of certain environmental diseases was significantly higher than that in the general population when exposed to night-time noise. Specifically, the SPR was 1 % higher for environmental diseases, 5 % higher for atopic dermatitis, and 2 % higher for allergic rhinitis compared to the population with low exposure to night-time noise. These findings suggest a potential impact of night-time noise exposure on the development of these diseases. Furthermore, we investigated disease severity using the total hospitalization data for each disease and found a significant linear association between night-time noise exposure and atopic dermatitis.

To the best of our knowledge, no other studies on environmental noise have examined the risk of environmental or allergic diseases in the general population, although there have been some studies in children and adolescents. A recent study demonstrated an increased risk of allergic disease (hazard ratio [HR]=1.710; 95 % CI:1.424–2.052)

Table 3
The ratio of age-standardized prevalence ratio (SPR) for night-time noise exposure and environmental diseases (2013–2017).

	Total	Male	Female
	SPR ratio (95 % CI) OR (95 % CI)		
Environmental diseases*	1.0154 (1.0152–1.0155)	1.0009 (1.0060–1.0011)	1.0277 (1.0275–1.0279)
Atopic dermatitis	1.0778 (1.0770–1.0786)	1.0787 (1.0777–1.0798)	1.0768 (1.0757–1.0778)
Asthma	0.9318 (0.9314–0.9322)	0.9157 (0.9151–0.9162)	0.9466 (0.9460–0.9471)
Allergic rhinitis	1.0301 (1.0299–1.0303)	1.0158 (1.0156–1.0161)	1.0418 (1.0416–1.0420)

CI, confidence interval
* Environmental diseases (atopic dermatitis, asthma, allergic rhinitis)

Table 2
Age-standardized prevalence ratio (SPR) and 95 % confidence intervals (CI) of environmental diseases exposed to high levels (≥ 55 dB) of night-time noise (2013–2017).

Diseases	Outpatient visits	Person-year	Hospital utilization	SPR	95 % CI
Environmental diseases*					
Total	251,953,259	85,036,540	296.29	1.0104	1.0103–1.0105
Male	114,808,120	42,342,711	271.14	0.9254	0.9253–0.9256
Female	137,145,139	42,693,829	321.23	1.0945	1.0943–1.0947
Atopic dermatitis					
Total	10,126,792	85,036,540	11.91	1.0515	1.0508–1.0521
Male	4967,799	42,342,711	11.73	1.0316	1.0307–1.0325
Female	5158,993	42,693,829	12.08	1.0714	1.0704–1.0723
Asthma					
Total	38,632,816	85,036,540	45.43	0.9524	0.9521–0.9527
Male	18,106,387	42,342,711	42.76	0.9113	0.9109–0.9117
Female	20,526,429	42,693,829	48.08	0.9918	0.9914–0.9923
Allergic rhinitis					
Total	203,193,651	85,036,540	238.95	1.0202	1.0201–1.0204
Male	91,733,934	42,342,711	216.65	0.9231	0.9229–0.9233
Female	111,459,717	42,693,829	261.07	1.1169	1.1167–1.1172

* Environmental diseases (atopic dermatitis, asthma, allergic rhinitis)

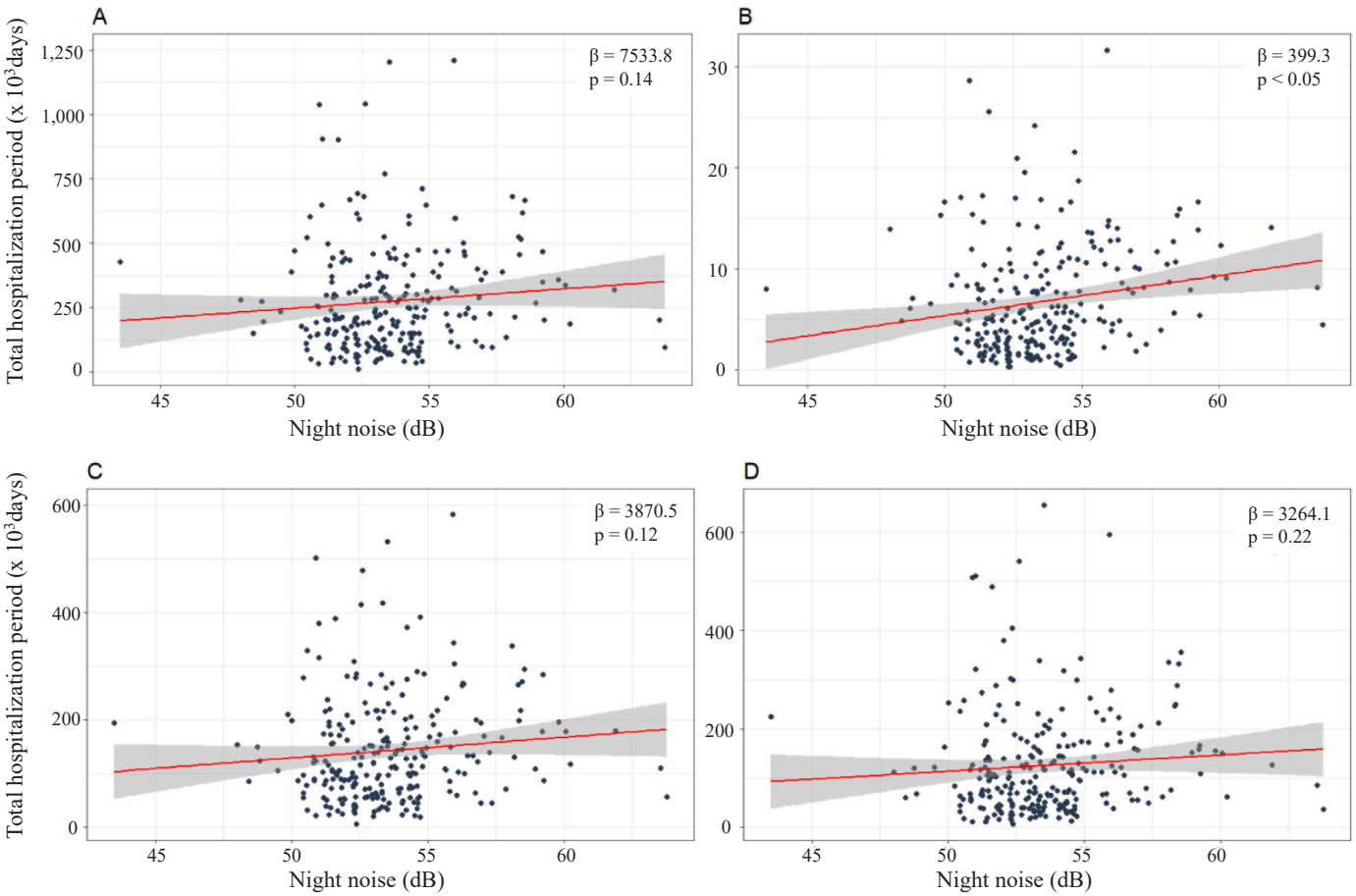


Fig. 2. Linear regression for night-time noise with total hospitalization period. (A) Environmental diseases (atopic dermatitis, asthma, allergic rhinitis). (B) Atopic dermatitis. (C) Asthma. (D) Allergic rhinitis.

associated with exposure to night-time noise after adjusting for confounding variables including age, sex, income, medical history, and air pollution (Kim et al., 2022). Additionally, another study found a significant association between noise annoyances in children and asthma prevalence (odds ratio [OR]=1.38; 95 % CI:1.04–1.82) after adjusting for parental medical history and education status (Bockelbrink et al., 2008). Previous research has emphasized the significance of considering factors that can affect allergic diseases; however, limitations exist, such as the use of self-reported questionnaires to assess noise exposure or diagnose diseases, and restricting participant selection to children. Moreover, in line with our results, some studies have not revealed a clear association between noise exposure and asthma in adolescents, in contrast to previous studies (Wallas et al., 2020). Psychological factors, such as anxiety and depression, have been established as contributors to the pathogenesis and exacerbation of asthma in previous research (Pietras et al., 2011). Furthermore, emotional stress is thought to induce asthma by perturbing the immune system through an increased production of endogenous opioids (Shavit and Martin, 1987; Shavit et al., 1985). Therefore, a more precise examination of the association between asthma and noise exposure should encompass the consideration of both psychological factors and socioeconomic status.

Our results revealed that noise exposure is significantly associated with atopic dermatitis and allergic rhinitis. These chronic noninfectious inflammatory diseases are thought to be caused by a combination of genetic, environmental, and immunological factors (Acharya et al., 2019; Chai et al., 2022). In the inflammatory response, atopic dermatitis and allergic rhinitis are generally considered T-helper (Th)2- dominant immune responses, with increased secretion of Interleukin (IL)-4, which promotes the production of IgE antibodies, as well as the recruitment and activation of other immune cells, such as eosinophils and mast cells (Iwasaki et al., 2021). A recent study showed that exposure to noise at night changed the inflammatory response, increasing IL-4 levels (Tait et al., 2019). IL-4 downregulates filaggrin, loricrin, and involucrin levels in keratinocytes and exacerbates skin barrier function. In addition to its role in regulating IgE production in B cells, IL-4 directly stimulates itch-sensory neurons, leading to itching (Furue, 2020; Oetjen et al., 2017). Moreover, chronic IL-4 secretion enhances the action of Interferon (IFN)-γ and Tumor Necrosis Factor (TNF)-α in inducing CXC Motif Chemokine Receptor 3 (CXCR3) agonistic chemokines, which recruit more T cells into inflamed skin, thereby lead to disease chronicity (Albanesi et al., 2000).

In addition to cell-related responses, cortisol levels are another possible mechanism underlying atopic dermatitis and allergic rhinitis. Cortisol is a steroid hormone produced by the adrenal cortex in response to stress and it regulates the immune system and inflammation (Oakley and Cidlowski, 2013). Indeed, some evidence suggests that salivary cortisol response significantly increases with noise exposure (Pouryaghoub et al., 2016; Selander et al., 2009). Cortisol can be elevated in response to inflammation that triggers the condition and contributes to a cycle of stress and inflammation, which can exacerbate the symptoms of the condition. In response to acute stressors, such as noise, the HPA (hypothalamus pituitary adrenal) axis elevates cortisol levels, leading to a significant increase in Th1 cytokines, such as IFN-r, and initiating a cellular immune response (Buske-Kirschbaum et al., 2006; Dhabhar, 2000; Marshall, 2004). However, in the case of chronic stress, the body's reaction to stress diminishes, transitioning from a cellular to humoral immune response. This shift results in elevated basal cortisol levels and heightened Th2 cytokines, such as IL-4 and IL-5, consequently favoring a

Th2- dominant immune response (Buske-Kirschbaum et al., 2002; Buske-Kirschbaum, 2009; Elenkov and Chrousos, 2002). The prevalence of this Th2 cell response can contribute to the development of allergic diseases, underscoring the importance of managing an individual's chronic stress as a critical factor in both the prevention and management of allergic diseases (Angelucci, 1994; Branchi et al., 2004; Weaver et al., 2004). Notably, in women, salivary cortisol levels significantly increase with noise exposure (Selander et al., 2009). It offers a possible explanation of our results that female patients exhibited a higher prevalence of allergic rhinitis compared to the lower noise exposed group, indicating a possible sex susceptibility to allergic diseases. In model organisms, gene expression varies extensively between females and males. These sex-specific genetic effects might have contributed to the sex differences in the occurrence of allergic rhinitis because there are underlying differences in the inflammatory pathways to different allergens between females and males (Barrenäs et al., 2008). Further research is needed to obtain a more detailed understanding of the relationship between atopic dermatitis, allergic rhinitis, and noise exposure.

Our study presents novel findings regarding the impact of night-time noise exposure on environmental diseases, utilizing a large sample of hospital data and a long-term noise exposure assessment among the general population. Nevertheless, this study had some limitations. First, individual characteristics such as income, medical history pertaining to allergic diseases or respiratory conditions, and other environmental factors such as air pollution were not included in this study. The lack of information regarding these factors, which could influence the risk of allergic diseases, is a limitation. Exposure to air pollution, in particular, is an important factor in the development of allergic diseases in industrialized societies, and should be considered as an integral part of any study of environmental diseases (Lee et al., 2013; Takizawa, 2011). Therefore, our results should be interpreted cautiously. To confirm our findings, further studies should be conducted, which include more detailed information on participant characteristics and consider noise and air pollution as exposure factors. Second, estimating noise exposure based on residential addresses and outdoor measurements, which assume indoor noise levels, may have introduced bias. Although previous studies have shown a good correlation between indoor and outdoor noise levels (Naim et al., 2018; Ryu and Jeon, 2011), limitations remain due to noise estimation using GIS spatial analysis for areas without noise information. Because noise measurement points are designated to represent noise levels considering factors such as population and area, our kriging method, implemented for areas with a lower priority for measurement, may not accurately reflect noise information. In addition, the energy of noise decreases as the distance from the measurement point increases, and obstacles such as mountains or buildings can make measurements or estimates less representative. Hence, there is uncertainty in the estimation of night-time noise, and this uncertainty should be compensated for by increasing the number of noise measurement points to enhance the resolution of the measurements.

5. Conclusion

We found that nighttime noise exposure ($\geq$ 55 dB) was significantly associated with environmental diseases, especially atopic dermatitis and allergic rhinitis. In terms of disease severity, nighttime noise exposure showed a linear association with atopic dermatitis. Our study has demonstrated that noise exposure could potentially impact the occurrence and severity of allergic diseases, in addition to the well-established auditory health effects. Therefore, further investigations are necessary to strengthen the substantiation of these results.

Ethics approval and consent to participate

The data from NHIS were anonymized before being released to the authors. All participants provided written informed consent. The Institutional Review Board of the Gil Medical Center, Gachon University, approved this study (IRB number: GCIRB2021–380).

Funding

None.

CRediT authorship contribution statement

Yongho Lee and Seunghyun Lee: Conceptualization, Methodology, Investigation, Writing –original draft. Seula Park: Methodology, Writing –review. Won-Jun Choi, Seong-Kyu Kang, June-Hee Lee and Dong-Wook Lee: Investigation and validation–review. Wanhyung Lee: Supervision, Methodology, Writing-review and editing. All co-authors reviewed and edited the paper for clarity.

Declaration of Competing Interest

The authors have no conflict of interest to report.

Data Availability

The datasets used and/or analyzed during the current study are accessible from the corresponding author upon reasonable request.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.ecoenv.2023.115677.

References

Acharya, D., et al., 2019. Factors associated with atopic dermatitis and allergic rhinitis among residents of two municipal areas in South Korea. Medicina 55, 131.
Albanesi, C., et al., 2000. IL-4 enhances keratinocyte expression of CXCR3 agonistic chemokines. J. Immunol. 165, 1395–1402.
Anandan, C., et al., 2010. Is the prevalence of asthma declining? Systematic review of epidemiological studies. Allergy 65, 152–167.
Angelucci, L., 1994. The hypothalamus-pituitary-adrenocortical axis: epigenetic determinants, changes with aging, involvement of NGF. Neurochem. Int. 25, 53–59.
Banerjee, P., et al., 2016. GIS based spatial noise impact analysis (SNIA) of the broadening of national highway in Sikkim Himalayas: a case study. AIMS Environ. 3, 714–738.
Barrenäs, F., et al., 2008. Gender differences in inflammatory proteins and pathways in seasonal allergic rhinitis. Cytokine 42, 325–329.
Bockelbrink, A., et al., 2008. Environmental noise and asthma in children: sex-specific differences. J. Asthma 45, 770–773.
Branchi, I., et al., 2004. Epigenetic control of neurobehavioural plasticity: the role of neurotrophins. Behav. Pharmacol. 15, 353–362.
Buske-Kirschbaum, A., et al., 2002. Stress-induced immunomodulation is altered in patients with atopic dermatitis. J. Neuroimmunol. 129, 161–167.
Buske-Kirschbaum, A., et al., 2006. Endocrine stress responses in TH1-mediated chronic inflammatory skin disease (psoriasis vulgaris)—do they parallel stress-induced endocrine changes in TH2-mediated inflammatory dermatoses (atopic dermatitis)? Psychoneuroendocrinology 31, 439–446.
Buske-Kirschbaum, A., 2009. Cortisol responses to stress in allergic children: interaction with the immune response. Neuroimmunomodulation 16, 325–332.
Chai, W., et al., 2022. Allergic rhinitis, allergic contact dermatitis and disease comorbidity belong to separate entities with distinct composition of T-cell subsets, cytokines, immunoglobulins and autoantibodies. Allergy Asthma Clin. Immunol. 18, 1–12.
D'Amato, G., et al., 2020. The effects of climate change on respiratory allergy and asthma induced by pollen and mold allergens. Allergy 75, 2219–2228.
Dhabhar, F.S., 2000. Acute stress enhances while chronic stress suppresses skin immunity: the role of stress hormones and leukocyte trafficking. Ann. N. Y. Acad. Sci. 917, 876–893.
Eguiluz-Gracia, I., et al., 2020. The need for clean air: the way air pollution and climate change affect allergic rhinitis and asthma. Allergy 75, 2170–2184.
Elenkov, I.J., Chrousos, G.P., 2002. Stress hormones, proinflammatory and antiinflammatory cytokines, and autoimmunity. Ann. N. Y. Acad. Sci. 966, 290–303.
Faraji, M., et al., 2022. Exposure to road noise and asthma prevalence in adults. Environ. Sci. Pollut. Res. Int. 1–8.
Furue, M., 2020. Regulation of filaggrin, loricrin, and involucrin by IL-4, IL-13, IL-17A, IL-22, AHR, and NRF2: pathogenic implications in atopic dermatitis. Int. J. Mol. Sci. 21, 5382.
Hong, O., et al., 2013. Understanding and preventing noise-induced hearing loss. Dis. Mon. 59, 110–118.

Iwasaki, N., et al., 2021. Th2 cells and macrophages cooperatively induce allergic inflammation through histamine signaling. PLoS One 16, e0248158.
Jenerowicz, D., et al., 2012. Environmental factors and allergic diseases. Ann. Agric. Environ. Med. 19.
Kang, S.-Y., et al., 2018. Time trends of the prevalence of allergic diseases in Korea: a systematic literature review. Asia Pac. Allergy 8.
Kim, A.-R., et al., 2022. What is the role of night-time noise exposure in childhood allergic disease? Int. J. Environ. Res. Public Health 19, 2748.
Krivoruchko, K., 2012. Empirical bayesian kriging. ArcUser Fall. 6, 1145.
Krivoruchko, K., Gribov, A., 2020. Distance metrics for data interpolation over large areas on Earth's surface. Spat. Stat. 35, 100396.
Lee, S., et al., 2017. Symptoms of nervous system related disorders among workers exposed to occupational noise and vibration in Korea. J. Occup. Environ. Med. 59, 191–197.
Lee, S.-Y., et al., 2013. Allergic diseases and air pollution. Asia Pac. Allergy 3, 145–154.
Li, S., et al., 2022. Association between exposure to air pollution and risk of allergic rhinitis: a systematic review and meta-analysis. Environ. Res. 205, 112472.
Marshall, G.D., 2004. Neuroendocrine mechanisms of immune dysregulation: applications to allergy and asthma. Ann. Allergy, Asthma Immunol. 93, S11–S17.
Min, J.-y, Min, K.-b, 2018. Cumulative exposure to nighttime environmental noise and the incidence of peptic ulcer. Environ. Int. 121, 1172–1178.
Min, K.-B., Min, J.-Y., 2017. Exposure to environmental noise and risk for male infertility: a population-based cohort study. Environ. Pollut. 226, 118–124.
Moreno, M.A., 2016. Atopic diseases in children. JAMA Pedia 170, 96. -96.
Naim, F., et al., Assessment of residential exposure to aircraft, road traffic and railway noise in London: relationship of indoor and outdoor noise. Proceedings of Euronoise, 2018.
Oakley, R.H., Cidlowski, J.A., 2013. The biology of the glucocorticoid receptor: new signaling mechanisms in health and disease. J. Allergy Clin. Immunol. 132, 1033–1044.
Oetjen, L.K., et al., 2017. Sensory neurons co-opt classical immune signaling pathways to mediate chronic itch. Cell 171, 217–228 e13.
Ogulur, I., et al., 2021. Advances and highlights in biomarkers of allergic diseases. Allergy 76, 3659–3686.
Pietras, T., et al., 2011. Analysis of the correlation between level of anxiety, intensity of depression and bronchial asthma control. Post. Dermatol. Alergol. 28, 15–22.
Pouryaghoub, G., et al., 2016. Effect of acute noise exposure on salivary cortisol: a randomized controlled trial. Acta Med. Iran. 657–661.
Ryu, J.K., Jeon, J.Y., 2011. Influence of noise sensitivity on annoyance of indoor and outdoor noises in residential buildings. ApAc 72, 336–340.
Selander, J., et al., 2009. Saliva cortisol and exposure to aircraft noise in six European countries. Environ. Health Perspect. 117, 1713–1717.
Shavit, Y., et al., 1985. Stress, opioid peptides, the immune system, and cancer. J. Immunol. 135, 834s–837s.
Shavit, Y., Martin, F.C., 1987. Opiates, stress, and immunity: animal studies. Ann. Behav. Med. 9, 11–15.
Shi, Z., et al., 2021. Occupational hearing loss associated with non-Gaussian noise: a systematic review and meta-analysis. Ear Hear 42, 1472.
Tait, J.L., et al., 2019. The inflammatory response to simulated day and night emergency alarm mobilisations. PLoS One 14, e0218732.
Takizawa, H., 2011. Impact of air pollution on allergic diseases. Korean J. Intern. Med. 26, 262.
Wallas, A.E., et al., 2020. Noise exposure and childhood asthma up to adolescence. Environ. Res. 185, 109404.
Weaver, I.C., et al., 2004. Epigenetic programming by maternal behavior. Nat. Neurosci. 7, 847–854.
Yoo, S., et al., 2021. Data resource profile: the allergic disease database of the Korean National Health Insurance Service. Epidemiol. Health 43.
Yoon, J.-H., et al., 2014. Occupational noise annoyance linked to depressive symptoms and suicidal ideation: a result from nationwide survey of Korea. PLoS One 9, e105321.
Zambrano-Monserrate, M.A., Ruano, M.A., 2019. Does environmental noise affect housing rental prices in developing countries? Evidence from Ecuador. Land Use Policy 87, 104059.