

Does severe subacute noise exposure increase risk of new onset hypertension beyond conventional risk factors? A 30 000 person-years cohort study

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Objectives: Previous studies mostly focused on the relationship between chronic environmental noise exposure and hypertension but the effects of subacute (under 4 years) exposure with severe (>85 dB) noise exposure on clinical level hypertension have not been explored. This study aimed to reveal the association between severe noise exposure and hypertension.

Methods: The severe noise exposure group was recruited from a Common Data Model conducted for the Korean Participants Health Examination from January 2014 to December 2017. The use of antihypertensive drug and/or blood pressure of at least 140/90 mmHg was defined as new onset clinical hypertension. A multivariate Cox proportional hazard model was implemented to estimate hazard ratios and 95% confidence intervals (CI) by adjusting covariates including demographic, lifestyle, and other chemical exposure factors. Time-dependent Cox analysis and Landmark analysis were further performed as a sensitivity analysis.

Results: During the 29 332 person-years follow-up with 12 412 participants of the entire cohort, new onset hypertension occurred in 1 222 participants. The findings showed that severe noise exposure was associated with an increased risk of hypertension incidence in the entire cohort [final model hazard ratio 1.28 (95% CI 1.11–1.47)]. Other covariates did not attenuate the association after adjusting age and sex. Time-dependent Cox and Landmark analysis also showed significant results [hazard ratio 1.60 (95% CI 1.38–1.85) and hazard ratio 1.33 (95% CI 1.13–1.57)].

Conclusion: Severe noise with subacute exposure is significantly associated with hypertension development. Further studies should be implemented to clarify whether severe exposure to noise could be an important risk factor for hypertension.

Keywords: health check-up, hypertension, incidence, severe noise exposure

Abbreviations: ACGIH, American Conference of Governmental Industrial Hygienists; CDM, Common Data Models; KoHE, Korea Participants Health Examination

INTRODUCTION

Hypertension is one of the most prevalent chronic diseases worldwide. The National Center for Health Statistics estimated that about one in three adults in the United States develop hypertension [1]. South Korea also shows similar prevalence rates, with 34.6% for men and 30.8% for women [2]. Hypertension is associated with huge social costs as it is a major risk factor for serious conditions, such as cardiovascular diseases, heart failure, and chronic kidney diseases [3,4]. According to the *Journal of the American Heart Association*, hypertension creates a significant medical burden, amounting to a total estimated \$131 billion in the United States from 2003 to 2014 [5]. Identifying potential risk factors that could affect hypertension and preventing it in high-risk groups in advance will lower the prevalence of hypertension and medical costs.

Along with well known risk factors for hypertension, such as age, obesity, and smoking, exposure to physical and chemical hazards from work have been suggested as other risk factors [6]. Environmental noise exposure has been considered one of the most common hazards worldwide, especially in the workplace environment [7,8]. Noise exposure is known to affect cardiovascular disease as well as a variety of other health issues, both auditory and nonauditory [9]. Previous studies mostly focused on the

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relationship between environmental noise exposure and cardiovascular diseases. These studies included road traffic, railway, and aircraft noises as environmental noise exposure factors, and the level of exposure was almost 65 dB with the assumption of lifetime exposure [10–15].

Although some studies related to the impact of severe noise exposure (>85 dB) on cardiovascular diseases have been conducted, there is contention regarding this [16–18]. Although several studies have reported significant relationships between severe noise exposure and hypertension, these studies had methodological limitations. Most previous studies were based on self-reported exposures, small sample sizes, and cross-sectional designs [19–22]. Some recent studies used follow-up cohort methods; however, the results of these studies were inconsistent [16,23–25]. This implies that clarifying the relationship between severe noise exposure and hypertension is imperative. Furthermore, the independent effect of noise exposure beyond conventional cardiovascular risk factors, such as obesity, smoking, alcohol drinking, and other chemical exposure should be clarified.

Therefore, this study aims to elucidate the relationship between severe noise with subacute (4 years) exposure and hypertension incidence in participants. We controlled for several chemical exposures related to cardiovascular diseases in this study. We hope our comprehensive control of conventional cardiovascular risk factors with severe noise exposure will provide scientific evidence of association between noise exposure and hypertension.

METHODS

Data set and study population

In Korea, nine hospitals conducted Common Data Models (CDMs) for the Korea Participants Health Examination (KoHE). A CDM is a technique for facilitating data standardization and sharing among disparate local database systems [26]. General measurements, common questionnaires, special questionnaires, and night questionnaires are the four domains that the KoHE-CDM harmonizes. The Nebraska Lexicon, Systematized Nomenclature of Medicine-Clinical Terms, Logical Observation Identifiers Names and Codes, and the questionnaires were standardized into KoHE-defined coding, which was used to standardize the general terminology [26].

In the current study, participants were recruited from the Severance Hospital KoHE-CDM cohort based on the results of health check-ups conducted between 2014 and 2017. From 19 113 initially recruited participants, those who did not work at companies with severe noise exposure ($n=953$), have not been followed up since 2014 ($n=130$), and were diagnosed with hypertension or recorded high blood pressure at the time of the baseline health check-up in 2014 ($n=1889$) were sequentially excluded from this study. A total of 12 141 participants were finally enrolled; 97.7% of the participants were annually followed up with health check-ups. The follow-up period of participants was calculated by totaling the period between each health check-up. For the time-dependent Cox analysis, the period between each health check-up and variables at each check-up were also used.

Definition and evaluation of data

The primary outcome of this study was hypertension incidence. Hypertension was defined based on one of the following: participants who answered ‘yes’ on a questionnaire asking about their history of physician-diagnosed hypertension or use of antihypertensive drug; SBP equal to or greater than 140 mmHg during check-up; and DBP equal to or greater than 90 mmHg during check-up. History of physician-diagnosed hypertension or antihypertensive drug was first reported by a participant, then trained nurses and physician in the field of occupational environment double checked the response. The participant finally confirmed the history of hypertension after health check-up. The blood pressure was measured by trained nurses with an automatic blood pressure monitor. If the measured blood pressure was high, participants took a 10-min break and blood pressure was measured again.

Severe noise exposure was defined as when participants were exposed to noise of 85 dBA or more in their workplaces 6 h a day, according to Threshold Limit Values criteria provided by the American Conference of Governmental Industrial Hygienists (ACGIH) [27]. In Korea, every check-up institution has a database of exposure of each worker, including severe noise exposure. All exposure data were accessed using the participants’ exposure database. Participants who were exposed to noise at any interval period of the health check-ups were classified into the severe exposure group. Participants’ exposure data were evaluated by experts who specialize in assessing work environments. Noise levels in dBA were measured on a sound level meter, conforming, at a minimum, to the requirements of the American National Standards Institute Sound Level meters.

Covariates were obtained from self-reported questionnaires and health check-ups, including age, sex, waist circumference, exercise, smoking and alcohol history, family history of hypertension, and diabetes. Male participants with more than 85 cm or female participants with more than 80 cm of waist circumference were defined as having abnormal waist circumference [28]. Waist circumference was used as a scale for obesity. Participants who engaged in high-intensity or medium-intensity exercise more than twice a week were classified into the ‘exercise group’, whereas others were classified into the ‘nonexercise group’. Smoking status was stratified into three groups: non-smokers, ex-smokers, and current smokers, according to their response to the question asking whether they have ever smoked more than five packs of cigarettes (100 cigarettes) in their lifetime. Drinking history was divided into two groups: men who reported having more than seven drinks per week and women who had more than five drinks per week were defined as having a history of drinking; otherwise, they were defined as having no history of drinking. Waist circumference, smoking status, exercise, and drinking history are defined as lifestyle factors. Family history of hypertension was defined according to the response of participants on the self-reported question asking history of hypertension in their family. Participants who responded that they had a history of diabetes or had a fasting blood glucose of 126 or higher in a health check-up were defined as having diabetes.

According to the Occupational Safety and Health Act of Korea, several chemical and physical exposures including carbon monoxide, nitric dioxide, cyanide compounds, antimony compounds, carbon disulfide, trichloroethylene, ethylene glycol dinitrate, acetonitrile, methyl chloroform, dichlorofluoromethane, dichloromethane, nitroglycerin, vibration and high pressure or low pressure, and night shift are recognized as cardiovascular risk factors [29]. Therefore, we defined the presence of cardiovascular-related exposure as exposure to any of those factors because of their possibility of affecting blood pressure. Number of exposures related to cardiovascular risk was used for covariates in the adjusted Cox model. All cardiovascular-related exposures were also measured by experts who specialize in assessing work environments.

Statistical analyses

The differences between baseline health check-up data of participants with and without severe exposure were compared using independent *t* tests and the chi-square tests for continuous data and categorical data, respectively. A Kaplan–Meier plot of the proportion of the time to hypertension incidence by severe exposure was drawn.

Hazard ratios with 95% confidence intervals (CIs) of hypertension incidence were calculated by using a multivariate Cox proportional hazard model. Hazard ratios and 95% CIs were further calculated by multivariate time-dependent Cox proportional hazard models to reduce the immortal time bias. Landmark analysis was also

performed as a sensitive analysis [30]. The standard period for the Landmark analysis was set to 1 year. The aforementioned covariates were included in the relevant steps of the models in all analysis. The interaction variable between waist circumference and noise exposure on hypertension incidence was defined by with or without noise exposure and normal or abnormal waist circumference groups. The hazard ratio (95% CI) of the interaction variable for hypertension incidence was also calculated by a Cox proportional hazard model.

All statistical tests were two-sided, and a *P* value less than 0.05 was considered statistically significant. All statistical analyses were performed using the ‘survival’ package of R version 4.0.2 (R Foundation for Statistical Computing, Vienna, Austria).

Ethics statement

The study protocol was in accordance with the ethical guidelines of the 1975 Declaration of Helsinki and was approved by the Institutional Review Board of Severance Hospital (IRB: Y-2020–0011). The need to obtain informed consent from the participants was waived because of the retrospective nature of this study.

RESULTS

Baseline characteristics of the entire cohort are summarized in Table 1. 46.1% of the participants were men and the mean age was 37.95 (SD = 9.72). The total follow-up person-years

TABLE 1. Baseline characteristics of participants by occupational noise exposure

Variable	Nonexposure (n = 10 485)	Noise-exposure (n = 1656)	<i>P</i> value
Age			<0.001
Mean (SD)	37.33 (9.49)	41.92 (10.25)	
Sex			<0.001
Male	4089 (39.00%)	1509 (91.12%)	
Female	6396 (61.00%)	147 (8.88%)	
Smoking history			<0.001
Nonsmoker	7582 (72.31%)	659 (39.79%)	
Ex-smoker	1049 (10.01%)	359 (21.68%)	
Current smoker	1854 (17.68%)	638 (38.53%)	
Waist circumference			<0.001
Normal	9282 (88.53%)	1321 (79.78%)	
Abnormal	1203 (11.47%)	335 (20.22%)	
Exercise history			<0.001
Yes	4661 (44.45%)	855 (51.63%)	
No	5824 (55.55%)	801 (48.37%)	
Diabetes			<0.001
No	10184 (97.13%)	1580 (95.41%)	
Yes	301 (2.87%)	76 (4.59%)	
Drinking history			<0.001
No	8240 (78.59%)	898 (54.23%)	
Yes	2245 (21.41%)	758 (45.77%)	
Cardiovascular-related exposure			<0.001
No	6143 (58.59%)	850 (51.33%)	
Yes	4342 (41.41%)	806 (48.67%)	
Family history of hypertension			<0.001
No	8651 (82.51%)	1511 (91.24%)	
Yes	1834 (17.49%)	145 (8.76%)	
New onset of hypertension			<0.001
No	9569 (91.26%)	1350 (81.52%)	
Yes	916 (8.74%)	306 (18.48%)	

SD, standard deviation.

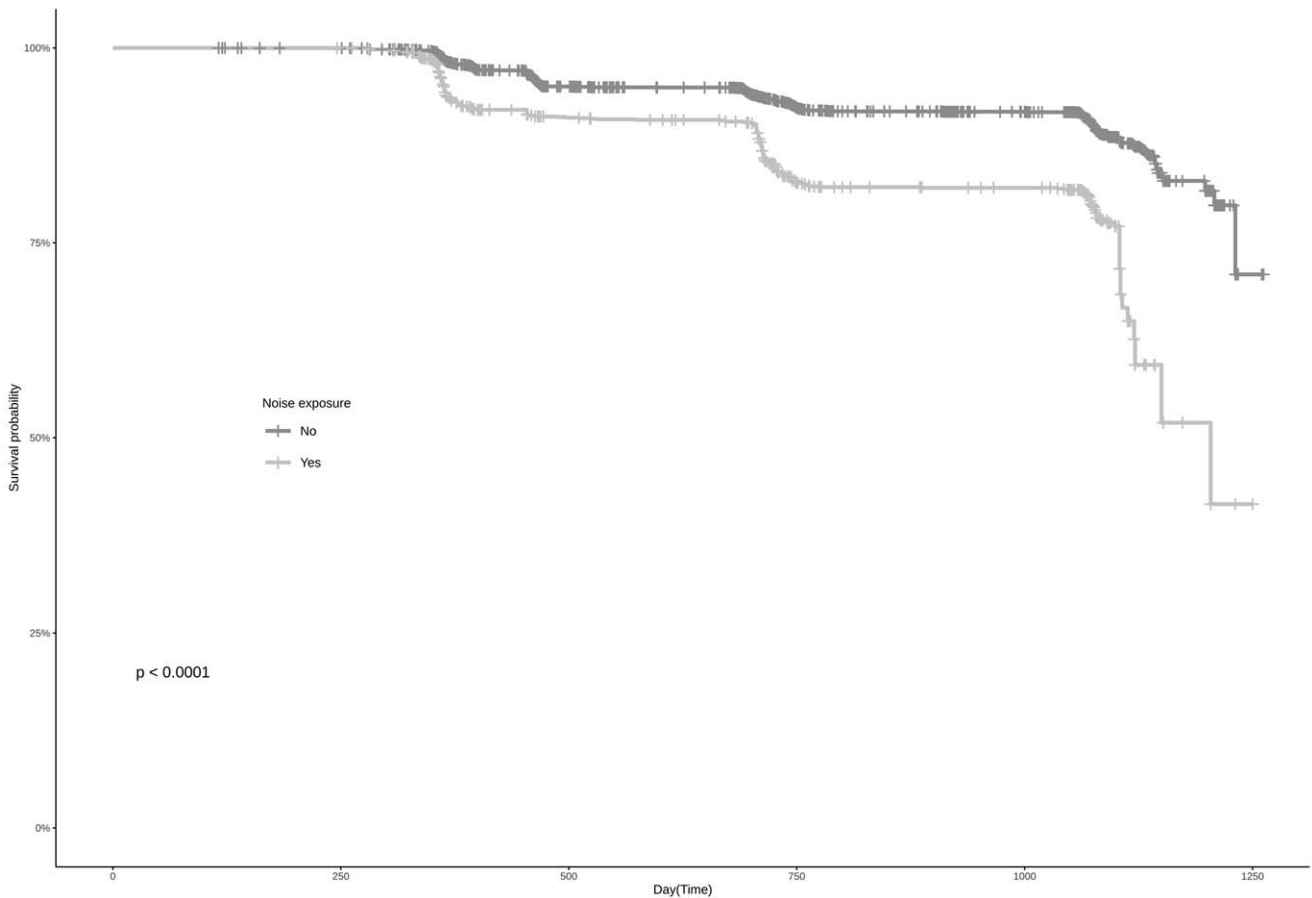


FIGURE 1 Kaplan–Meier plot of time to hypertension incidence by noise exposure.

was 29 332, and the mean follow-up years per person was 2.42 years. There were 1509 (26.96%) male participants and 147 (2.25%) female participants with severe noise exposure. The median follow-up of 1656 participants with severe exposure was 2.92 years and the median of their exposure periods to noise was 1.93 years. The severe exposure group had a higher prevalence in the ex-smoker and current smoker group ($P < 0.001$). Furthermore, participants with abnormal waist circumference, diabetes, a drinking history, in the exercise group, having cardiovascular-related exposure, and new onset of hypertension were highly prevalent in the severe exposure group with statistical significance ($P < 0.001$). Family history of hypertension showed higher prevalence in the nonexposure group ($P < 0.001$).

During the follow-up period, 1222 (9.85%) participants developed hypertension. The noise exposure group had 916 participants, whereas 306 participants were in the nonexposure group. One hundred and fifty-two participants responded 'yes' to the question of physician-diagnosed hypertension history or to the history of using antihypertensive drug. One thousand and ninety-seven participants recorded SBP equal to or greater than 140 mmHg or DBP equal to or greater than 90 mmHg. Twenty-seven participants were counted from history of hypertension/drug and direct blood pressure measurement. A Kaplan–Meier plot of the proportion of

hypertension development by time is shown in Fig. 1. Survival rate significantly differed between the severe exposure group and nonexposure group with respect to the incidence of hypertension ($P < 0.0001$).

Table 2 summarizes the results of multivariate time-fixed Cox proportional hazard analysis in the entire cohort. The crude model used univariate analysis, and model 1 used age and sex as covariates. Diabetes and smoking history were added as covariates in model 2. The final model was adjusted using age, sex, waist circumference, diabetes, smoking status, number of exposures related to cardiovascular risk, family history of hypertension, exercise, and drinking history. The result showed that severe noise exposure is associated with a higher risk of hypertension, and hazard ratios remained significant after adjusting for other covariates in the final model [crude model: hazard ratio 2.37 (95% CI 2.08–2.69); final model: hazard ratio 1.28 (95% CI 1.11–1.47)].

Furthermore, a multivariate time-dependent Cox regression model was performed in a similar way. Waist circumference, smoking status, number of exposures related to cardiovascular risk, family history of hypertension, exercise, drinking history, and diabetes were used as time-varying covariates in the time-dependent Cox regression model. The final model 3 showed a significant association between severe noise exposure and incidence of

TABLE 2. Hazard ratios and 95% confidence intervals of hypertension in time-fixed Cox proportional hazard models

	Crude model	Model 1	Model 2	Final model
Noise exposure				
No	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
Yes	2.37 (2.08–2.69)	1.21 (1.06–1.39)	1.21 (1.06–1.39)	1.28 (1.11–1.47)
Sex				
Female		1.00 (reference)	1.00 (reference)	1.00 (reference)
Male		3.09 (2.70–3.53)	2.79 (2.38–3.26)	2.46 (2.09–2.90)
Age		1.05 (1.04–1.05)	1.04 (1.04–1.05)	1.04 (1.04–1.05)
Diabetes				
No			1.00 (reference)	1.00 (reference)
Yes			1.49 (1.20–1.86)	1.40 (1.13–1.75)
Smoking history				
Nonsmoker			1.00 (reference)	1.00 (reference)
Ex-smoker			0.99 (0.83–1.17)	0.91 (0.77–1.08)
Current smoker			1.29 (1.11–1.49)	1.17 (1.01–1.36)
Waist circumference				
Normal				1.00 (reference)
Abnormal				1.75 (1.54–2.00)
Exercise history				
Yes				1.00 (reference)
No				1.03 (0.92–1.16)
Drinking history				
No				1.00 (reference)
Yes				1.35 (1.19–1.53)
Family history of hypertension				
No				1.00 (reference)
Yes				1.55 (1.34–1.79)
Number of exposures related to cardiovascular risk				0.91 (0.84–0.99)

CI, confidence interval; HR, hazard ratio.

hypertension [hazard ratio 1.60 (95% CI 1.38–1.85)]. Time-fixed Cox regression with Landmark analysis were also conducted with a 1 year criterion. The final model also showed a significant relationship between noise exposure and incidence of hypertension [hazard ratio 1.33 (95% CI 1.13–1.57)]. All the results of hazard ratios and 95% confidence intervals of hypertension incidence by occupational noise exposure in the three statistical analyses are summarized in Table 3. Hazard ratios and 95% CIs of all the covariates in time-dependent Cox models and time-fixed Cox models with Landmark are summarized in Supplementary Tables S1, <http://links.lww.com/HJH/B809> and S2, <http://links.lww.com/HJH/B810>.

The hazard ratios (95% CI) for hypertension incidence were 1.73 (1.45–2.05), 1.96 (1.69–2.27), and 2.66 (2.09–3.38) in nonexposure with abnormal waist circumference, noise exposure with normal waist circumference, and noise exposure with abnormal waist circumference groups, respectively, compared with the nonexposure with the normal waist circumference group as the reference group. Figure 2 shows the plot of hazard ratios with 95% CIs. The hazard ratio (95% CI) of noise exposure with abnormal waist circumference was no greater than the sum of hazard ratios in the others.

DISCUSSION

Our results reveal that severe noise exposure with even a subacute period significantly increases new onset hypertension risk with a cohort of 2.42 follow-up years per person on average. The relationship was still significant after adjusting for lifestyle (exercise, smoking and alcohol drinking), obesity, family history of hypertension, and even toxic chemical exposures. To minimize the immortal time bias, time-dependent Cox proportional hazard models were further performed, and significant relationship was found between noise exposure and incidence of hypertension. The results of time-fixed Cox models with Landmark performed as a sensitive analysis strengthened the association.

Covariates used for adjustment in the final Cox model were age, sex, waist circumference, the number of exposures related to cardiovascular risk, family history of hypertension, smoking status, exercise history, drinking history, and diabetes. These covariates are significant risks or relief factors for hypertension and variables that could be assessed by simple measurements, point-of-care tests, and questionnaires [2,29,31–41]. The relationship of severe noise exposure and risk of hypertension was still significant

TABLE 3. Hazard ratios and 95% confidence intervals of hypertension incidence by occupational noise exposure of each model

Statistical methods	Crude model	Model 1	Model 2	Final model
Time-fixed Cox regression	2.37 (2.08–2.69)	1.21 (1.06–1.39)	1.21 (1.06–1.39)	1.28 (1.11–1.47)
Time-dependent Cox regression	2.94 (2.56–3.37)	1.59 (1.38–1.84)	1.58 (1.37–1.82)	1.60 (1.38–1.85)
Time-fixed Cox regression with Landmark	2.34 (2.02–2.71)	1.22 (1.05–1.43)	1.22 (1.05–1.43)	1.33 (1.13–1.57)

CI, confidence interval; HR, hazard ratio.

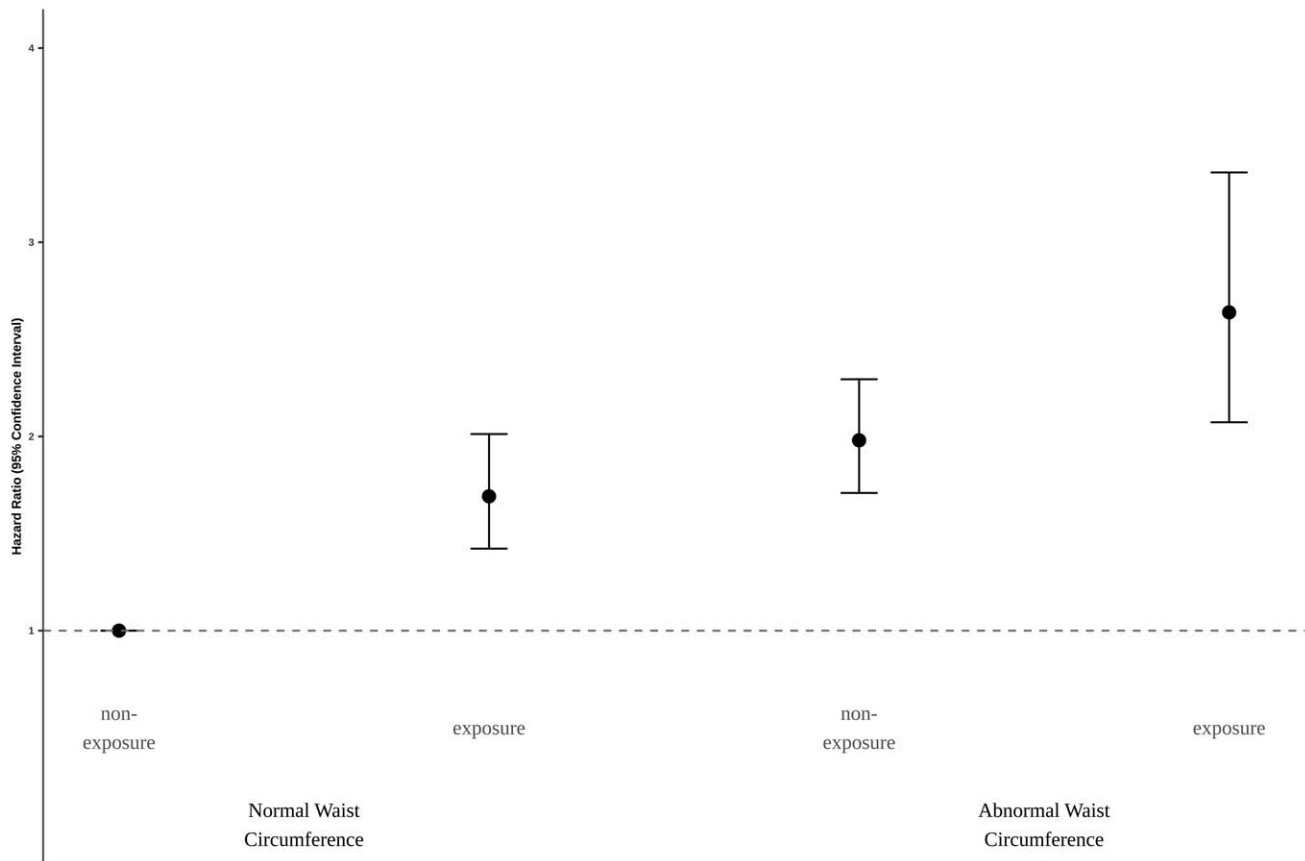


Figure 2. Hazard Ratio of hypertension stratified by noise exposure and obesity

FIGURE 2 Hazard ratio of hypertension stratified by noise exposure and obesity.

after adjusting for these covariates. Despite these findings of some degree of association, noise exposure is generally only considered a risk factor for hearing-related diseases, and is not yet included as a cardiovascular-related risk factor in South Korea [29]. The nonauditory health effects of noise exposure should be explored through further studies in South Korea.

Furthermore, participants with abnormal waist circumference and noise exposure show nearly 2.6 times higher hazard ratio compared with normal waist circumference participants in the nonexposure group. This implies that the obese population is vulnerable when they are exposed to noise; therefore, more careful management is needed in vulnerable populations who have traditional risk factors for cardiovascular diseases including hypertension.

Several studies have attempted to elucidate the relationship between severe exposure and hypertension, although not to the extent of studying the relationship between environmental noise exposure and hypertension. These studies' results are somewhat controversial. Stokholm *et al.* [25] conducted a 7-year Danish cohort study with 145 190 participants from 625 companies and the result showed no association between noise exposure and the development of hypertension. However, this study did not recruit participants with noise exposure of more than 86 dBA. Tessier-Sherman *et al.* conducted a cohort study of a

metal manufacturing company with 2052 participants from 1996 to 2012 [16]. Cox proportional hazard models were performed, and the results also showed no increased risk of incident hypertension with exposure to severe noise.

In contrast, Lin *et al.* [23] conducted a 17-year cohort study with 2459 participants, measuring the hazard ratio of incident hypertension with time-dependent exposure to severe noise and suggested that a noise increase of 5 dBA is significantly correlated with an increased hazard ratio of hypertension. Although this was not a meaningful statistic in comparison with the noise-unexposed group, this study showed a correlation between noise exposure and hypertension through noise volume. Chang *et al.* performed a study with a cohort from an aircraft manufacturing plant in central Taiwan. In total, 578 male participants were recruited and the results revealed that participants exposed to noise of 85 dBA or more had a 1.93-fold (95% CI 1.15–3.22) increased risk of hypertension compared with participants exposed to less than 80 dBA [24]. This study has some similarities to the setting of the current study; however, it has limitations of a small number of participants and that the industry field could not be generalized.

To reduce selection bias, the current study filtered participants with the inclusion criteria that participants should work in the same company that employed the participants in the severe exposure group. Through this process,

participants within similar environments could be enrolled in the study. Furthermore, the number of exposures related to cardiovascular risk was included in the adjusted variables so that confounding bias, which could occur related to those exposures, was reduced.

Emotional stress reactions and unconscious physiological stress are thought to be potential mechanisms of noise and hypertension [42]. Chronic noise exposure can also cause a homeostatic imbalance, and affect metabolism and the cardiovascular system, which can result in increased blood pressure, blood lipid concentrations, blood viscosity, and blood glucose concentrations [9,42]. Animal studies with albino rats demonstrate that exposure to high-intensity noise results in increased plasma levels of stress hormones, such as corticosterone, adrenaline, and noradrenaline [43]. Moreover, physiological stress caused by noise exposure may also increase indulgence in unhealthy behaviors, such as smoking and alcohol consumption and indirectly result in an increased risk of cardiovascular diseases, including hypertension [17].

This study has several strengths. First, it had a large number of recruited participants in the KoHE-CDM cohort with a follow-up. The average follow-up period of these participants was adequate, and every variable was followed up appropriately. Second, in addition to time-fixed Cox proportional hazard models, various statistical methods including time-dependent Cox proportional hazard analysis, Landmark analysis, and combined effect analysis were used to clarify the correlation between noise exposure and risk of hypertension incidence. Temporal relationship between noise exposure and increased risk of hypertension was achieved through those analyses. Third, the study controlled possible confounders in various ways. The bias that could be caused by company factors was minimized by including many companies and excluding companies that do not expose participants to noise. Moreover, our models adjusted other exposures related to cardiovascular effects, which was not performed in other studies.

Nevertheless, this study has some limitations. First, health check-up data are associated with healthy worker effects. However, hypertension is usually asymptomatic and rarely causes workers to resign from their job. Furthermore, participants were followed up for less than 4 years, which is a short period of time, to minimize the healthy worker effects. Second, severe noise exposure data were not specific in terms of volume and frequency of noise. However, experts in measuring working environments classified participants precisely in accordance with the ACGIH guidelines. Severe noise exposure was defined as when participants were exposed to noise of 85dBA or more in their workplaces for 8 h a day. Therefore, the participants' data can be considered as a comprehensive evaluation of noise exposure. Third, there was a lack of data pertaining to the presence of hearing protection device or hearing loss prevention programs, disease history related to hearing, and previous work history, which could lead to bias. However, this is not a systematic but a random error, and there were enough participants to compensate for any bias. Finally, medical check-up data could not reflect the exact data of hypertension diagnosis, which could cause bias related to unspecified period lengths. Despite the

uncertainty of diagnosis date, 97.7% of participants were annually followed up so that bias could be minimized.

In conclusion, our current study highlighted that severe noise exposure is associated with the increased risk of hypertension, even when considering other well known risk factors including lifestyle factors and other exposures. Our combined effect analysis deciphers the characteristics of participants who are vulnerable to toxic environmental exposure. Further studies should be implemented to clarify whether severe exposure to noise could be considered independently of well known cardiovascular risk factors in public and environmental health issues.

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Conflicts of interest

There are no conflicts of interest.

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