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Association of occupational noise exposure with hypertension: A cross-sectional study

Jin Wang MD[1] | Panpan Zhang MS[2] | Yaning Wang MS[1] | Hui Wang MS[1] | Yuan Gao MD, PhD[1] | Yurong Zhang MD, PhD[2]

[1]Department of Cardiovascular Disease, East District of the First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China

[2]Department of Neurology, the First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China

Correspondence
Yurong Zhang, MD, PhD, Department of Neurology, the First Affiliated Hospital of Xi'an Jiaotong University, No 277, West Yanta Road, Xi'an, Shaanxi, 710061 China.
Email: zhangyurong72@mail.xjtu.edu.cn

Abstract

The effects of chronic occupational noise exposure on hypertension are debated. We aimed to investigate the association between occupational noise exposure and the prevalence of hypertension. The cross-sectional data were collected from 2017 to 2018 using occupational physical examination data from a local aircraft manufacturing enterprise in Xi'an. We categorized occupational noise exposure as high (≥85 dBA) and low noise exposure (< 85 dBA). Logistic regression analysis was performed to evaluate the association between occupational noise exposure and hypertension, and associations were further evaluated using subgroup analyses for age, sex, and body mass index (BMI). Of the 4746 participants (median age, 43 years; 73.4% men), 9.57% (454/4746) had hypertension and 32.4% (1540/4746) were exposed to high noise. Compared to the participants with low occupational exposure to noise, the adjusted odds ratio (OR) for hypertension prevalence was 1.30 (1.05–1.62) for those with high occupational noise exposure. Subgroup analyses revealed that the noise-hypertension association only existed in young participants (OR, 1.70; 95% CI, 1.21–2.40). This study revealed a harmful association between high occupational noise exposure and hypertension in young adults. The study suggests occupational noise exposure as a target for worksite interventions to prevent hypertension.

KEYWORDS
hypertension, noise exposure, occupational noise, risk factors, workers

1 | INTRODUCTION

Hypertension is widely prevalent, affecting one billion people worldwide, and is responsible for more than 10 million deaths annually. It has been declared a global public health crisis by the World Health Organization.[1] The prevalence of hypertension among the Chinese population aged 18 years and over has risen from 18.8% to 27.5% from 2002 to 2018.[2] Extensive examination for hypertension is necessary due to its public-health importance and biological plausibility with other diseases.[3]

In addition to the traditional risk factors for hypertension, epidemiological data suggest that environmental factors such as noise are associated with a higher risk for hypertension.[4–6] Noise is one of the major environmental health problems and is defined as any unpleasant sound. It was reported that prolonged exposure to noise was associated with disease progression. Individuals are exposed to multiple noise sources daily from occupational and residential areas. Although individuals can habituate to noise exposures at a certain level, chronic noise exposures can still lead to changes in the autonomic nervous system and the endocrine system, triggered

oxidative stress, and dysregulated circadian rhythm, resulting in hypertension.[4]

However, research on the relationship between occupational noise exposure and hypertension is ambiguous.[5–11] Several studies have confirmed that occupational noise exposure may be a risk factor for hypertension[6–8,11]; however, some studies reported no association between the two.[9,10] A prospective study including 578 male workers from an aircraft manufacturing plant in Taiwan investigated the relationship between noise exposure and the 10-year risk of hypertension and found that prolonged exposure to occupational noise levels ≥85 dBA (A-weighted decibels) may increase blood pressure levels in men.[12] However, a study from an inception cohort of workers in a specialty metals manufacturing company showed that hypertension incidents did not significantly differ between groups by cumulative continuous or categorized noise exposure metric; however, the study was limited to control for other factors known to be associated with the risk of hypertension.[10] A systematic review and meta-analysis of 43 epidemiological studies published between 1970 and 1999 found that exposure to high occupational noise (generally measured as > 85 dBA) was associated with a large, clinically meaningful increase in the incidence of hypertension and concluded that a five dBA noise increase led to a moderate increase in hypertension.[13]

The association between occupational noise exposure and hypertension remains controversial because of the differences in study designs, exposure assessments, small sample sizes, and confounding control effects.[10,12,14] This study aimed to investigate the association between occupational noise exposure and hypertension using occupational physical examination data of workers from a local aircraft manufacturing enterprise in Xi'an.

2 | METHODS

2.1 | Study design and participants

A cross-sectional design was used in this study. Data were collected from 2017 to 2018 using occupational physical examination details of 4746 workers from a local aircraft manufacturing enterprise in Xi'an, China. The study was conducted in accordance with the Declaration of Helsinki and was approved by the ethics committee for medical research at the First Affiliated Hospital of Xi'an Jiaotong University, and the review board waived the requirement of written informed consent. Confidential patient information was deleted from the entire data set prior to analysis.

2.2 | Study measures

Detailed demographic, clinical, and laboratory information of each participant was obtained.

A self-administered questionnaire was used to collect information regarding potential confounders, including age, sex, current smoking status (yes/no), alcohol drinking (yes/no), education (below high school, high school, or above high school), marital status (never married, married or living with partner, or widowed/separated/divorced), shift work (day shift, night shift, day-night shift, or non-shift), Perceived Stress Scale (PSS), and histories of diabetes mellitus, hypertension, coronary heart disease (CHD), cerebral vascular disease (CVS), hyperlipidemia, and hyperuricemia.

2.3 | Definitions

Body mass index (BMI) was calculated as the weight (kg) divided by the square of the height (m2). BMI was classified based on the criteria for the Asian population as follows: normal weight: BMI < 24, overweight: BMI 24–27.9, and obese: BMI ≥28 kg/m2.[15] Hyperlipidemia and hyperuricemia were categorized as normal (absence of both) and hyperlipidemia or hyperuricemia, separately. Cut-off points for lipid and uric acid were chosen according to international guidelines.[16,17] History of diabetes was based on participants' self-reported diagnosis of diabetes. Additionally, participants who did not report a diabetes diagnosis but had a fasting HbA1c greater than 6.4% were also considered to have diabetes. History of CHD and CVS was based on participants' self-reported diagnosis. Hypertension was defined as the use of anti-hypertensive medication, a previous diagnosis of hypertension given by physicians, or blood pressure measurements with the mean resting systolic blood pressure (SBP) ≥140 mm Hg, or the mean resting diastolic blood pressure ≥90 mm Hg.[18] Blood pressure was measured from the right arm of the participant after 5 min of sitting using an automated device (HBP-9021J, Omron, Japan) by a trained nurse three times within 5 min, and the mean BP was calculated.

As reported in the previous studies, occupational noise was defined as a dichotomous variable; low noise (< 85 dBA) and high noise exposures (≥85 dBA).[19,20] The noise exposure was examined by professional institutions and was included in the noise surveillance program. The procedure for noise exposure assessment was as follows: We identified 17 workshops in this enterprise and divided each department into different locations based on the manufacturing processes by workers. After the walk-through survey, the 15-min time-weighted average equivalent sound level was measured using a sound analyzer (MS-6700, SANZER ELECTRONICS CO., LTD, CHINA). All the participants were divided into one similar exposure group based on the similarity and frequency of the work performed, the processes with which they worked, and how they performed the tasks.[21] Each participant was assigned a specific value of noise exposure based on the 8-h time-weighted average equivalent sound level measured in the workplace.

2.4 | Statistical analysis

A descriptive analysis was performed. Continuous variables were described as medians with interquartile ranges (IQRs) or as means with standard deviations (SDs) if the variables had a normal distribution. Categorical variables were presented as numbers with percentages.

TABLE 1 Characteristics of study participants by noise

Characteristics	Total ($n = 4746$)	Low noise ($n = 3206$)	High noise ($n = 1540$)	p-Value
Age, in years (median, IQR)	43 (34–49)	42 (32–49)	39 (33–47)	.003
Men, n (%)	3483 (73.39)	2,177 (67.90)	1306 (84.81)	<.001
Shift Work, n (%)				.017
Day shift	480 (10.11)	311 (9.70)	169 (10.97)	
Night shift	327 (6.89)	239 (7.45)	88 (5.71)	
Non-shift	3865 (81.44)	2614 (81.53)	1251 (81.23)	
Day-night shift	74 (1.56)	42 (1.31)	32 (2.08)	
Education status, *n* (%)				.006
Below high school	180 (3.79)	114 (3.55)	66 (4.29)	
High school	476 (10.03)	294 (9.17)	182 (11.82)	
Above high school	4090 (86.18)	2798 (87.27)	1292 (83.90)	
Marital status, *n* (%)				<.001
Never married	618 (13.02)	493 (15.38)	125 (8.11)	
Married or living with partner	3956 (83.35)	2603 (81.19)	1353 (87.86)	
Widowed/separated/divorced	172 (3.62)	110 (3.43)	62 (4.03)	
Current smoking, *n* (%)	1643 (34.62)	920 (28.70)	723 (46.95)	<.001
Alcohol drinking, *n* (%)	1948 (41.05)	1,191 (37.15)	757 (49.16)	<.001
Body mass index (kg/m2)	24.0 (21.9–26.2)	23.8 (21.7–26.0)	24.5 (22.3–26.7)	<.001
PSS (median, IQR)	19 (14–22)	19 (13–21)	18 (12–21)	.406
Medical history, *n* (%)				
Diabetes	185 (3.90)	123 (3.83)	62 (4.03)	.752
Hypertension	454 (9.57)	275 (8.58)	179 (11.62)	.001
Cerebral vascular disease	143 (3.01)	65 (2.03)	78 (5.06)	<.001
Coronary heart disease	192 (4.05)	130 (4.05)	62 (4.03)	.962
Hyperlipidemia	1062 (22.38)	698 (21.77)	364 (23.63)	.149
Hyperuricemia	454 (9.57)	303 (9.45)	151 (9.81)	.698

Abbreviations: IQR, interquartile range; PSS, perceived stress scale.

One-way analysis of variance and chi-square tests were conducted to compare the continuous and categorical variables, respectively. All participants' prevalence of hypertension was estimated according to their age (20–29.9, 30–39.9, 40–49.9, ≥50 years). Logistic regression analysis was performed to evaluate the associations between occupational noise and hypertension. Variables were entered in the multivariable logistic regression models if the p-value was ≤.15 in the univariable analysis in Table 1. Three logistic regression models were constructed to assess the association between noise and hypertension. Model 1 was unadjusted. Age and sex were adjusted in Model 2. Model 3 was adjusted for age, sex, BMI, education level, marital status, shift work, and hyperlipidemia. In addition, we performed stratified analyses by sex (men versus women), age (< 45 vs. ≥45 years), and BMI (< 24 kg/m2, 24–27.9 kg/m2, and ≥28 kg/m2) based on Model 3. The statistical analysis was performed using SPSS version 22.0 (SPSSInc., Chicago, IL, USA). A two-tailed test was performed; the significance was defined as $p < .05$.

3 | RESULTS

Of the 4746 participants, 9.57% (454/4746) had hypertension and 32.45% (1540/4746) were exposed to high occupational noise. The prevalence of hypertension increased substantially with age: 2.6% in the age group 20–29.9 years, 18.7% in 30–39.9 years, 37.4% in 40–49.9 years, and 47.2% in ≥50 years, respectively (Figure 1).

Characteristics of all the study participants and by noise exposure are presented in Table 1. The median age of the total participants was 43 years (interquartile range, 34–49 years), and 3,483 (73.39%) of the participants were men. Compared to the participants exposed to low noise, participants exposed to high noise were more likely to be younger, men, current smokers, drinking, and married or living with partner. In addition, greater BMI and less high school or above education were more common in participants with high noise. These participants also tended to have shift work and hypertension.

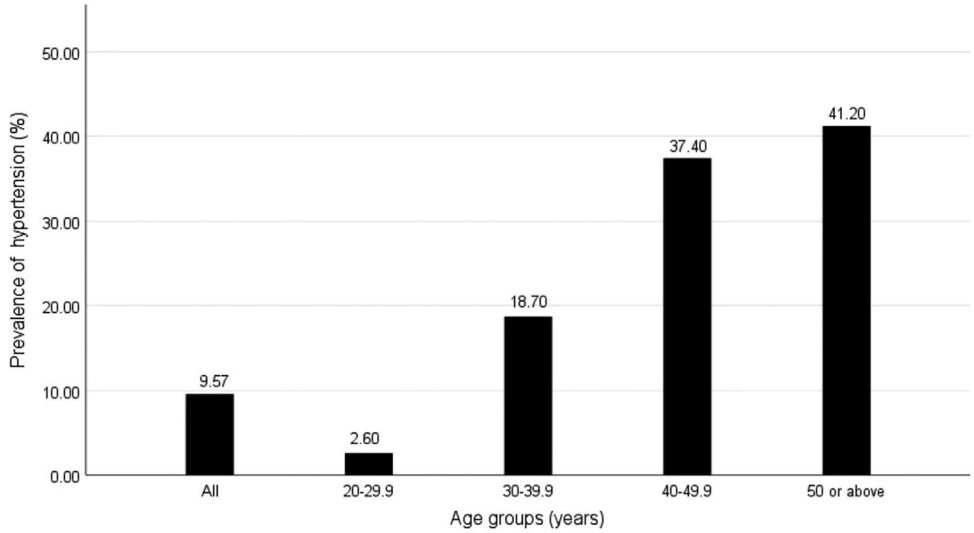

FIGURE 1 Prevalence of hypertension in different age groups. The data is expressed in percentage (%)

TABLE 2 Unadjusted and adjusted associations between noise and hypertension

Variables	Model 1 OR (95% CI)	*p*-Value	Model 2 OR (95% CI)	*p*-Value	Model 3 OR (95% CI)	*p*-Value
Noise	1.40 (1.15–1.71)	.001	1.44 (1.18–1.77)	<.001	1.30 (1.05–1.62)	.017

Note: Model 1 adjusted for none. Model 2 adjusted for age, sex. Model 3 adjusted for age, sex, BMI, education, marital status, hyperlipidemia, and shift work.

Exposure to occupational noise was positively associated with hypertension. The age and sex-adjusted OR (95% CI) of hypertension with occupational noise exposure was 1.44 (1.18–1.77) ($p < .001$) (Table 2). The OR of hypertension was similar following additional adjustment for education, smoking, alcohol drinking, BMI, history of hyperlipidemia, and shift work (aOR, 1.30; 95% CI, 1.05–1.62; $p = .017$).

In the stratified analyses, significant positive associations between noise and hypertension were found in participants under 45 years of age (OR = 1.56, 95% CI: 1.12–2.18) as well as overweight participants (OR = 1.51, 95% CI: 1.10–2.07). Furthermore, the association between noise and hypertension was more pronounced in participants under 45 years of age (p for interaction = .001). The adjusted odds ratios (OR) between noise exposure and hypertension was 1.25 (95%CI: .99–1.57) for males and 1.78 (95%CI: .90–3.51) for females, and the p-value for interaction was not significant (p for interaction = .269) (Table 3).

4 | DISCUSSION

This cross-sectional study revealed a positive association between high occupational noise exposure and hypertension in young adults. Participants exposed to high occupational noise (≥85 dBA) had a 28% higher likelihood of having hypertension compared to those exposed to low occupational noise (< 85 dBA). Our findings, in conjunction with previous studies, supported that high occupational noise exposure may play an important role in hypertension development; therefore, these findings should be considered in future interventions for the prevention and management of hypertension.

The prevalence of hypertension in this study was lower than that of the Chinese national survey report (27.5% in total, 4.0% in 18–24-year-olds, 6.1% in 25–34-year-olds, and 15.0% in 35–44-year-olds).[2,22] A study reported that the prevalence of hypertension was 38.3% in those aged 40–49 years, 53.9% in those aged 50–59 years, and 67.9% in those aged 60–69 years old in rural north China.[23] The difference may lie in the fact that most of the participants in this study were younger, had higher education status (86.2% had above high school) and had regular physical examinations every year, which possibly led them to have better economics status, a more nutritionally balanced diet, better health knowledge, and lower hypertension prevalence.

Hypertension is amenable to early prevention and treatment; therefore, modifiable factors should be identified. Noise is one of the modifiable risk factors for hypertension; however, studies assessing the relationship between occupational noise exposure and hypertension have shown inconsistent results.[14] Therefore, additional research is required in this area. The present study provided evidence that high occupational noise (≥85 dBA) was associated with hypertension in young adults in China. A cohort study reported a relationship between occupational noise exposure and the incidence of hypertension; however, important risk factors for hypertension, such as BMI, cigarette smoking, and alcohol intake, were not considered.[10] The present study investigated the relationship between occupational noise and the prevalence of hypertension by considering more important factors. A

TABLE 3 Adjusted associations between noise exposure and hypertension by age, sex, and BMI

Variables	Sample size (*n*)	Multivariable-adjusted Model OR (95% CI)	*p*-Value	*p*-Value (interaction)
By sex				.269
Men	405/3483	1.25 (.99–1.57)	.057	
Women	49/1263	1.78 (.90-3.51)	.097	
By age				.001
≥45 year	168/2871	1.11 (.83–1.49)	.486	
<45 year	286/1875	1.56 (1.12–2.18)	.009	
By BMI				.328
Normal weight	132/2340	1.34 (.92–2.03)	1.121	
Overweight	216/1775	1.51 (1.10–2.07)	.011	
Obese	106/621	.93 (.58–1.49)	.753	

Note: The results were adjusted for all covariates (age, sex, BMI, education, marital status, hyperlipidemia, and shift work) except the corresponding stratification variable.
Abbreviations: BMI, body mass index;CI, confidence interval; OR, odds ratio.

study conducted among Iron and Steel Enterprise Workers reported that occupational noise was associated with essential hypertension (aOR, 1.726).[24] A cohort study revealed that male workers exposed to ≥85 dBA noise, who use hearing-protective devices, had a mean increase of 3.8 mm Hg in SBP over 9 years of follow-up, which is a significantly higher increase than that observed among office workers in Korea.[25] A meta-analysis of 11 primary studies on the relationship between occupational noise exposure and the risk factors of cardiovascular disease in China showed that the risk of developing high blood pressure among workers exposed to noise was 2.55 times higher than that of the controls.[26] Another meta-analysis with 147,820 participants from three studies showed that workers exposed to noise ≥85 dBA had a 7% higher risk of hypertension incidence than those with no or low occupational exposure to noise (< 85 dBA); however, the evidence was low quality.[27] Some studies have reported a negative association between occupational noise exposure and hypertension. A meta-analysis from three studies (one case-control and two cohort studies), which assessed the association of occupational noise exposure with hypertension, published between 1982 and 2020, showed limited evidence of the harmfulness of occupational noise exposure; no relationship was found between long working hours and shift work with hypertensive disease.[12,24,28] The strength of the evidence was considered "inadequate evidence of harmfulness" because of the limited number of studies and the insignificance of the evidence.[29]

The health effects of occupational noise exposure depend on the sound's duration, repetition, intensity, and frequency, which may be modified by several factors, including individual susceptibility, ethnicity, sex, and other physical, chemical, and biological risk factors.[27] Therefore, we performed stratified analyses for sex, age, and BMI. We found that high occupational noise exposure was significantly associated with hypertension in young adults, but there were no differences in the association by sex or BMI. However, the reduction in sample size after stratification may have led to potential bias, so we need to validate this results in a larger sample size. Inconsistent with the previous study, a positive association between prolonged exposure to high noise levels (≥85 dBA) and blood pressure levels were found among men in Taiwan.[12] The association between noise exposure and isolated diastolic hypertension was particularly strong in participants aged < 50 years and men; however, no significant association was found between noise exposure and other hypertension subtypes.[7] The differences may be because hypertension in workers is the result of a complex set of factors involving the work environment and psychosocial factors such as exposure time, the use of hearing protection devices (HPDs), job stress, and social support; none of which are explained or intervened by a single risk factor. Furthermore, certain hypertension risk factors aggravate or attenuate other risk factors.[30] Therefore, the present study adjusted potential confounders, including demographic, clinical information, job stress, and shift work.

The potential mechanism that links noise exposure to hypertension is that prolonged exposure to noise affects the oxidative parameters of total antioxidant capacity, total oxidant status, and DNA damage.[31] There are three physiological outcomes related to occupational noise exposure: individual characteristics, stress indicators, and physiological risk factors. There is an association between occupational noise exposure and hypertension because noise exposure activates the sympathetic and endocrine systems to affect the humoral and metabolic states of the human organism, producing an increase in blood pressure and the changes in other biological risk factors (such as blood lipids and glucose levels) that promote the development of hypertension and cardiovascular diseases.[32]

This study has several limitations. First, it is a cross-sectional study; therefore, causality cannot be determined and residual confounding cannot be completely ruled out. Second, the study was conducted among currently employed workers. Older workers are more likely to have hearing loss and thus are not sensitive to occupational

noise. Third, although participants in high noise exposure areas were equipped with HPDs, we did not have individual-level information regarding the use of HPDs. Consistent with most previous studies, results were limited by an exposure bias caused by no adjustments for HPDs.[10,14] Finally, as in the previous study,[33] the limited information in the included studies precluded the possibility of prehypertension analysis or more information. Future studies are needed to clarify these observed associations.

However, it should also be noted that our study has several strengths. First, we have maintained consistency in developing noise estimates, and the same protocol was used for developing noise contours for all 17 workshops. This addresses one of the limitations often mentioned for meta-analyses investigating potential noise–hypertension associations that use noise and outcome data developed under different protocols and models. Furthermore, by restricting our analysis to a single enterprise with well-established occupational health and medical data collection procedures, we minimized the likelihood of potential biases in such data due to between-enterprises differences.

5 | CONCLUSIONS

This study demonstrated a harmful association of occupational noise exposure with hypertension. Occupational exposure to noise levels of ≥85 dBA was associated with hypertension in young adults. Future studies with prospective designs and clinical trials are needed to confirm the results of the current study.

AUTHOR CONTRIBUTIONS

Yurong Zhang and Jin Wang conceptualized and designed the study. Yurong Zhang did the statistical analyses, and Jin Wang collected the data and drafted and revised the manuscript. Panpan Zhang performed the statistical analysis and interpreted the results. Yaning Wang, Hui Wang, Yuan Gao, and Yurong Zhang critically edited the manuscript. All authors have read and agreed to the published version of the manuscript.

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CONFLICT OF INTEREST

None.

ORCID

Yurong Zhang MD, PhD https://orcid.org/0000-0002-1873-5019

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