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Noise frequency components and the prevalence of hypertension in workers

Ta-Yuan Chang [a,b,*], Chiu-Shong Liu [c], Li-Hao Young [a], Ven-Shing Wang [a], Shen-En Jian [a], Bo-Ying Bao [d]

[a] Department of Occupational Safety and Health, College of Public Health, China Medical University, Taichung, Taiwan, ROC
[b] Institute for Risk Assessment Sciences, Utrecht University, Utrecht, The Netherlands
[c] Department of Family Medicine, China Medical University Hospital, Taichung, Taiwan, ROC
[d] Department of Pharmacy, College of Pharmacy, China Medical University, Taichung, Taiwan, ROC

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ABSTRACT

Epidemiological studies have demonstrated a relationship between noise exposure and hypertension, but the association between hypertension and noise frequency components remains unclear. This cross-sectional study investigated the association between noise exposure at different frequencies and the prevalence of hypertension in 188 screw-manufacturing workers. Participants were divided into one high-noise-exposure group (≥80 A-weighted decibel, [dBA]; n = 68) and two reference groups, including 68 low-noise-exposure workers (75.8 ± 3.2 dBA) and 52 office workers (61.5 ± 0.5 dBA). Personal noise exposure and environmental octave-band analyses were performed at work. Multiple logistic regression models were used to estimate odds ratios (ORs) for hypertension between different noise-exposure categories after adjustment for potential confounders. Male workers exposed to noise levels at high frequencies of 2000, 4000 or 8000 Hz had a higher but non-significant risk of hypertension. Those exposed to ≥80 dBA for 2–4 years, 4–6 years and more than 6 years had a 4.43-fold (95% CI = 1.21–16.15), 1.21-fold (95% CI = 0.35–4.21) and 0.95-fold (95% CI = 0.16–5.60) risk of hypertension, respectively, compared with reference workers. A significant association was only observed in male workers exposed to ≥70 dBA at 4000 Hz for 2–4 years (adjusted OR = 4.22; 95% CI = 1.15–15.49) and was not found at other frequencies for any periods. These findings suggest that occupational noise exposure above 80 dBA for specific periods may be associated with hypertension, and noise frequency at 4000 Hz may have the greatest effect on hypertension.

1. Introduction

Many epidemiological studies have shown an association between occupational and environmental noise exposure and hypertension. Workers exposed to noise levels above 80 A-weighted decibels (dBA) have been reported to have a higher risk of hypertension (Chang et al., 2011b; Chang et al., 2009; Fogari et al., 1994; Sbihi et al., 2008; Zhao et al., 1991) and elevated blood pressure (Chang et al., 2003; Fouriaud et al., 1984; Green et al., 1991; Lee et al., 2009; Talbott et al., 1999; Tomei et al., 2000; Verbeek et al., 1987). Residents exposed to road traffic noise at a 24-h average equivalent sound level ($Leq_{,24\ h}$) above 55 dBA may have a higher odds ratio (OR) for hypertension (Barregard et al., 2009; Bodin et al., 2009; Chang et al., 2011a; de Kluizenaar et al., 2007; Jarup et al., 2008; Leon Bluhm et al., 2007). Residents exposed to aircraft noise of $Leq_{,24\ h} > 50$ dBA may have a higher risk of hypertension (Eriksson et al., 2010; Eriksson et al., 2007; Rosenlund et al., 2001).

The biological mechanism between noise exposure and hypertension may be that noise, a psychosocial stressor, activates the sympathetic and endocrine systems to induce a release of stress hormones (such as catecholamines, adrenaline, nor-adrenaline and cortisol), producing a transient elevation of blood pressure. Chronic and repetitive noise stimuli modify these otherwise normal responses to a permanent upward resetting of baroreceptors and lead to hypertension (Babisch, 2002; Ising and Kruppa, 2004; Spreng, 2000).

However, the association between noise frequency components and hypertension is unclear. Exposure to different frequencies of occupational noise may produce different adverse health effects. Previous studies have found that an audiometric notch at 3000, 4000 or 6000 Hz with recovery at 8000 Hz is a sign of occupational noise-induced hearing loss (ACOEM, 2003; May, 2000; McBride and Williams, 2001). High-frequency noise-induced hearing loss may be associated with the risk of hypertension (Chang et al., 2011b). Exposure to low-frequency noise (10–250 Hz) has been associated with annoyance in previous studies (Pawlaczyk-Luszczynska et al., 2010; Pawlaczyk-Luszczynska et al., 2003). Although annoyance is more relevant to environmental noise exposure than to occupational noise exposure, it also presents in industrial control rooms and office-like areas (Berglund et

Abbreviations: BMI, body mass index; 95% CI, 95% confidence interval; dBA, A-weighted decibel; DBP, diastolic blood pressure; HPD, hearing protective device; LA_{eq}, A-weighted equivalent sound level; OR, odds ratio; SBP, systolic blood pressure.

* Corresponding author at: Department of Occupational Safety and Health, College of Public Health, China Medical University, 91 Hsueh-Shih Road, Taichung 40402, Taiwan, ROC. Tel.: +886 4 22053366x6203; fax: +886 4 22079225.
E-mail address: tychang@mail.cmu.edu.tw (T.-Y. Chang).

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al., 1996; Pawlaczyk-Luszczynska et al., 2009). Low-frequency noise-induced annoyance may produce hypertension by causing emotional responses that interfere with work performance in jobs requiring selective attention or processing of high load of information (Babisch, 2002; Bengtsson et al., 2004; Pawlaczyk-Luszczyniska et al., 2005; Persson Waye et al., 2001; Persson Waye et al., 1997). Work noise annoyance was associated with an increase of diastolic blood pressure (DBP) in a community-based study (Lercher et al., 1993). To our knowledge, however, no study has investigated the association between noise frequency components and hypertension. The purpose of this study was to determine the association between occupational noise exposure at different frequencies and the prevalence of hypertension in industrial workers.

2. Materials and methods

2.1. Study population

This cross-sectional study was conducted with 281 employees of a screw-manufacturing plant in Taichung County at the end of 2009. Because personal information, including working history, lifestyle and health examination results, was required to establish the study population, only 202 volunteers (23–58 years old) with complete data were recruited in this study. There were no significant differences between the 202 participants and the 79 non-participants in terms of educational level, cigarette smoking, alcohol drinking, tea consumption, coffee consumption, regular exercise, working activity or family history of hypertension. To avoid interference from non-occupational exposure, nine workers were excluded due to a previous diagnosis of hypertension prior to beginning work in the factory with a questionnaire. In addition, five workers were excluded because they reported the use of hearing protective devices (HPDs) at work that might cause potential bias in exposure classification. Therefore, the study group comprised 136 production-line workers and 52 office workers. Production-line workers were exposed to occupational noise due to metal cutting, pressing, grinding, sand blasting, polishing and gear washing processes in manufacturing ball screws and linear guides. The present study was reviewed and approved by the Institutional Review Board of the School of Public Health, China Medical University before the study commenced, and written informed consent was obtained from each participant.

2.2. Blood pressure measurements and hypertension

All subjects were required to fast overnight before blood sampling and blood pressure measurements during annual health examinations in 2009. Subjects sat quietly for 10 min in a chair with the back supported, both feet flat on the floor and arms supported at the heart level during blood pressure measurements. A trained nurse used an automated sphygmomanometer (Ostar Model P2, Ostar Meditech Corp., Taipei, Taiwan) to measure subjects' blood pressure two times in both arms at 0700–0900 in the morning, before the subjects began work. The higher mean value of blood pressure measurements was used to represent individuals' blood pressure in the present study. The procedure for blood pressure measurements followed established guidelines (Chobanian et al., 2003; Pickering et al., 2005), and the nurse was blind to the exposure status of each worker during the blood pressure measurements. Subjects were defined as hypertensive if they reported a previous medical diagnosis of hypertension after beginning work at this factory, if their mean resting systolic blood pressure (SBP) was $\geq$140 mm Hg or if their mean resting DBP was $\geq$90 mm Hg. Height, body weight, total cholesterol level and triglyceride level were also measured in all subjects. Body mass index (BMI) was calculated as body weight (kg) divided by the square of height (m^2).

In addition, a self-administered questionnaire was used to identify potential confounders. These factors included age, gender, employment duration, cigarette smoking, alcohol drinking, tea consumption, coffee consumption, regular exercise, work activity and family history of hypertension. Cigarette smokers were defined as those who smoked cigarettes more than three days per week for at least six months; alcohol, tea and coffee drinkers were defined by the same criterion. Regular exercisers were defined as those who participated in a sporting activity at least three times per week for six months or more. Working activity was categorized into high and low levels based on a scoring system that counted each subject's time of sitting, walking, lifting heavy objects during working periods and the distance walked between the worksite and home (Hwang et al., 1997).

2.3. Noise exposure assessment and frequency analyses

Workplace noise levels were measured using an octave-band analyzer (TES-1358, TES Electronic Corp., Taipei, Taiwan) that can report 1-sec to 24-h continuous equivalent sound levels (Leq) in the range of 50–120 dBA and time-weighted-average (TWA) noise levels at frequencies of 31.5, 63, 125, 250, 500, 1000, 2000, 4000 and 8000 Hz. This equipment was calibrated with a sound-level calibrator (TES-1356, TES Electronic Corp., Taipei, Taiwan) prior to environmental monitoring. The 8-h TWA Leq and its frequency components were collected by industrial hygienists at 14 locations around the company. Each subject was assigned a specific value of noise exposure and various frequency components based on the Leq measured in that subject's workplace.

To assess subjects' noise exposure more accurately, personal noise monitoring was conducted simultaneously using a personal noise dosimeter (Logging Noise Dose Meter Type 4443, Brüel & Kjær, Nærum, Denmark.). This equipment can report 5-min continuous Leq at an exchange rate of 3 dBA and TWA noise levels. The range of 50–120 dBA was used to measure all subjects' noise exposure with 5-min readings over 8 h.

To examine the association between occupational noise exposure and the prevalence rate of hypertension, the personal 8-h TWA Leq was used to classify 136 production-line workers into a high-exposure group ($n=68$; noise level $\geq$80 dBA) and a low-exposure group ($n=68$; noise level $<$80 dBA). The cut-off value of 80 dBA was selected because it was the median in the distribution of personal noise measurements among production-line workers. The same approach was applied to classify production-line workers into the high- and low-exposure groups according to environmental noise exposure and workplace noise levels at various frequencies. In addition, 52 office workers were chosen as the other reference group in the present study. The variability of noise exposure distribution, obtained from personal and environmental measurements, was examined on grouping strategy (Kromhout et al., 1995; Seixas and Sheppard, 1996; Vermeulen and Kromhout, 2005). To investigate the effects of exposure duration, production-line workers were also classified into less than 2 years, 2–4 years, 4–6 years and more than 6 years based on the first, second and third quartiles in the distribution of employment duration.

2.4. Statistical analysis

The Shapiro–Wilk test was first used to determine the normality of the continuous variables of resting SBP and DBP, which were relevant to the prevalence of hypertension, as well as the independent variables of age, environmental and personal noise levels, employment duration, BMI, total cholesterol level and triglyceride level. Statistical p values for these variables were less than 0.001 among all participants, indicating a non-normal distribution. Therefore, multiple comparisons between the three groups were performed using the Kruskal–Wallis test for continuous variables and the Chi-square test for dichotomous variables. For those groups with significant differences, the Mann–Whitney test and the Chi-square test were used to compare the high-exposure group with the two reference groups.

Multiple logistic regression models were applied to calculate odds ratios (ORs) and 95% confidence intervals (CIs) in order to compare

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between-group differences in the prevalence of hypertension for each of octave band frequencies and for total TWA noise exposure after controlling for potential confounding factors. These confounders included significant factors in simple logistic regression models (i.e., sex, BMI and triglyceride level), significant variables between the three groups (such as sex, educational level, triglyceride level, cigarette smoking and working activity) and important risk factors of hypertension reported in previous literature, including age, cholesterol level, alcohol drinking, regular exercise and family history of hypertension (Chobanian et al., 2003; Hajjar et al., 2006). These variables were selected manually and stepwise into multiple logistic regression models because only two variables of gender ($p=0.052$) and BMI ($p=0.002$) were retained in the final step while using an automatic stepwise procedure. Sensitivity analyses estimated the effects of one continuous variable of occupational noise exposure (instead of categorical exposure) and two dichotomous variables of environmental noise measurements (instead of personal noise measurements) on the prevalence of hypertension. The SAS standard package for Windows version 9.1 (SAS Institute Incorporation, Cary, North Carolina, USA) was used for statistical analyses. The significance level was set at 0.050 for all tests.

3. Results

Table 1 summarizes the demographic characteristics and potential risk factors for the three groups of 188 subjects. Significant differences were identified in the mean values of resting SBP, resting DBP and triglyceride level as well as the proportions of male subjects, educational level, cigarette smoking and high working activity between the three groups. Workers in the high-exposure group had significantly higher mean values of resting SBP, resting DBP and triglyceride level as well as the higher proportions of male subjects, educational level<13 years, cigarette smoking and high working activity than those in the office group. In addition, high-exposure workers had a significantly larger proportion of high working activity compared with low-exposure workers.

Table 2 presents personal and environmental measurements of noise exposure as well as frequency components of environmental noise for the three groups. The grouping strategy based on personal sampling had lower between-group and within-group variances in the log-transformed noise exposure distributions compared with the grouping strategy based on environmental sampling (data not shown). Accordingly, the grouping strategy based on personal sampling had little higher resolution than one based on environmental sampling (99.3% vs. 99.2%). Significant differences were identified in the mean values of personal noise level, environmental noise level and frequency components at 125, 250, 500, 1000, 2000, 4000 and 8000 Hz between the three groups. High-exposure workers had significantly higher mean values of personal noise level, environmental noise level and frequency components at 500, 1000, 2000, 4000 and 8000 Hz than low-exposure workers and office workers. In addition, workers in the high-exposure group had significantly higher mean values of noise frequency components at 125 and 250 Hz than workers in the office.

Table 1
Demographic characteristics in three different study groups.

Characteristic	Exposure group	Reference group		*P*-value
	≥80 dBA	<80 dBA	Office workers	
Subjects (no.)	68	68	52	
Age (years)				
Mean (SD)	32.4 (6.4)	31.9 (5.5)	33.4 (6.7)	0.479[a]
Employment duration (years)				
Mean (SD)	3.8 (2.7)	3.8 (3.3)	4.2 (3.3)	0.657[a]
Body mass index (kg/m^2)				
Mean (SD)	23.5 (3.8)	24.2 (4.1)	22.9 (3.6)	0.172[a]
Resting SBP (mmHg)				
Mean (SD)	129.2 (12.5)[c]	127.9 (10.8)	121.7 (15.9)	0.013[a]
Resting DBP (mmHg)				
Mean (SD)	80.0 (8.7)[c]	80.1 (10.2)	75.8 (11.4)	0.027[a]
Total cholesterol (mg/dl)				
Mean (SD)	187.3 (33.7)	197.0 (39.0)	186.4 (35.1)	0.317[a]
Triglyceride level (mg/dl)				
Mean (SD)	135.0 (84.0)[c]	124.6 (71.8)	92.1 (45.3)	0.005[a]
Gender				
Male (%)	66 (97.1)[e]	63 (92.7)	26 (50.0)	<0.001[b]
Educational level				
≥ 13 years	15 (22.1)[e]	19 (27.9)	39 (75.0)	<0.001[b]
Cigarette smoking				
Yes (%)	27 (39.7)[e]	28 (41.2)	7 (13.5)	0.002[b]
Alcohol drinking				
Yes (%)	12 (17.7)	11 (16.2)	6 (11.5)	0.641[b]
Tea consumption				
Yes (%)	34 (50.0)	33 (48.5)	29 (55.8)	0.717[b]
Coffee consumption				
Yes (%)	20 (29.4)	21 (30.9)	18 (34.6)	0.826[b]
Regular exercise				
Yes (%)	6 (8.8)	10 (14.7)	9 (17.3)	0.364[b]
Family history of hypertension				
Yes (%)	24 (35.3)	23 (33.8)	22 (42.3)	0.605[b]
Working activity				
High (%)	62 (91.2)[d,e]	43 (63.2)	4 (7.7)	<0.001[b]

dBA, A-weighted decibel; DBP, diastolic blood pressure; SBP, systolic blood pressure.
[a] Kruskal–Wallis test of differences between groups.
[b] Chi-square test of differences between groups.
[c] Mann–Whitney test of significant difference ($p<0.050$) compared with office workers.
[d] Chi-square test of significant difference ($p<0.05$) compared with the low-exposure (<80 dBA) group.
[e] Chi-square test of significant difference ($p<0.05$) compared with office workers.

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Table 2
Noise exposure and noise frequency components by study groups.

Variable	Exposure group	Reference group		*P*-value
	≥80 dBA	<80 dBA	Office workers	
Personal noise levels (dBA)				
Mean (SD)	82.7 (6.7)[b, c]	75.8 (3.2)	61.5 (0.5)	<0.001[a]
Environmental noise levels (dBA)				
Mean (SD)	82.3 (4.4)[b, c]	79.2 (4.7)	61.4 (0)	<0.001[a]
Frequency components				
31.5 Hz (dBA)				
Mean (SD)	32.6 (2.6)	32.9 (3.4)	33.3 (0)	0.711[a]
63 Hz (dBA)				
Mean (SD)	47.1 (6.1)	45.2 (4.8)	44.6 (0)	0.332[a]
125 Hz (dBA)				
Mean (SD)	54.8 (4.9)[c]	52.9 (6.5)	50.8 (0)	<0.001[a]
250 Hz (dBA)				
Mean (SD)	61.5 (4.8)[c]	59.7 (7.8)	53.9 (0)	<0.001[a]
500 Hz (dBA)				
Mean (SD)	67.8 (5.4)[b, c]	64.3 (7.5)	60.0 (0)	<0.001[a]
1000 Hz (dBA)				
Mean (SD)	71.4 (6.6)[b, c]	63.0 (5.6)	61.2 (0)	<0.001[a]
2000 Hz (dBA)				
Mean (SD)	70.3 (6.4)[b, c]	63.7 (5.1)	57.8 (0)	<0.001[a]
4000 Hz (dBA)				
Mean (SD)	72.3 (5.9)[b, c]	66.4 (5.5)	53.6 (0)	<0.001[a]
8000 Hz (dBA)				
Mean (SD)	72.2 (5.7)[b, c]	65.0 (6.4)	50.5 (0)	<0.001[a]

dBA, A-weighted decibel; SD, standard deviation.
[a] Kruskal–Wallis test of differences between groups.
[b] Mann–Whitney test of significant difference ($p<0.050$) compared with the low-exposure (< 80 dBA) group.
[c] Mann–Whitney test of significant difference ($p<0.050$) compared with office workers.

Table 3 summarizes the association between occupational noise exposure and the risk of hypertension between two different subpopulations. In simple logistic regression models, three variables, male gender, BMI ≥ 23 kg/m^2 and triglyceride level ≥ 100 mg/dl, were significantly associated with the prevalence of hypertension ($p<0.050$). However, there were no significant differences in the risk of hypertension between the high-exposure group and the two reference groups in multiple logistic regression models. The same results were found when workers were classified into smoking and non-smoking subgroups (data not shown). In sensitivity analyses, one continuous variable of occupational noise exposure was positively associated with the increased risk of hypertension, but it was not statistically significant (adjusted odds ratio = 1.01; 95% confidence interval = 0.95–1.07, $p = 0.820$). The effect estimates of using environmental sampling on the prevalence of hypertension were similar to those using personal sampling, but these results were also not significant (both p values > 0.050).

Fig. 1 shows the association between noise frequency components and the risk of hypertension among male production-line workers (n = 129), classified by median values of noise levels. Workers exposed to noise levels ≥ 70 dBA at 2000 Hz (adjusted OR = 1.92, 95% CI = 0.76–4.82), 4000 Hz (adjusted OR = 2.05, 95% CI = 0.82–5.12) or 8000 Hz (adjusted OR = 2.34, 95% CI = 0.89–6.16) had a higher risk of hypertension compared with the reference workers after controlling for potential confounders. No significant results in any octave band frequencies were found in the present study.

To investigate the association between the effects of noise exposure and employment duration on the prevalence rates of hypertension, ORs for male production-line workers exposed to noise levels ≥ 80 dBA for 2–4 years, 4–6 years and more than 6 years were compared with those in the reference group, including 63 workers exposed to <80 dBA and 19 workers exposed to ≥80 dBA for less than 2 years. As shown in Fig. 2, male production-line workers exposed to ≥80 dBA for 2–4 years (noise levels of 83.4 ± 2.7 dBA; adjusted OR = 4.43, 95% CI = 1.21–16.15) had a significantly higher risk of hypertension than did the reference group (noise levels of 77.6 ± 4.4 dBA) after adjusting for potential confounders. However, there were no statistically significant associations between workers exposed to noise levels greater than 80 dBA at other employment durations and the prevalence of hypertension. The examination of the duration of employment as a continuous variable showed that it

Table 3
Associations between noise exposure and prevalence of hypertension.

Variable	Hypertension (%)	Simple logistic regression model	Multiple logistic regression model[a]
		Crude OR (95% CI)	Adjusted OR (95% CI)
Subpopulation 1			
≥80 dBA group vs. <80 dBA group	17 (25.0)/14 (20.6)	1.29 (0.58, 2.87)	1.37 (0.56, 3.36)
Subpopulation 2			
≥80 dBA group vs. office workers	17 (25.0)/7 (13.5)	2.14 (0.82, 5.64)	1.11 (0.17, 7.08)

dBA, A-weighted decibel; OR, odds ratio; 95% CI, 95% confidence interval.
[a] Multiple logistic regression model adjusted for significant factors in simple logistic regression models (i.e., gender, body mass index and triglyceride level (≥100 vs. <100 mg/dl)), significant variables between the three groups (such as gender, educational level (≥13 years vs. <13 years), triglyceride level, current smoking and working activity (high vs. low)) and important risk factors reported in previous literature (i.e., age, cholesterol level (≥200 vs. <200 mg/dl), alcohol drinking, regular exercise and family history of hypertension).

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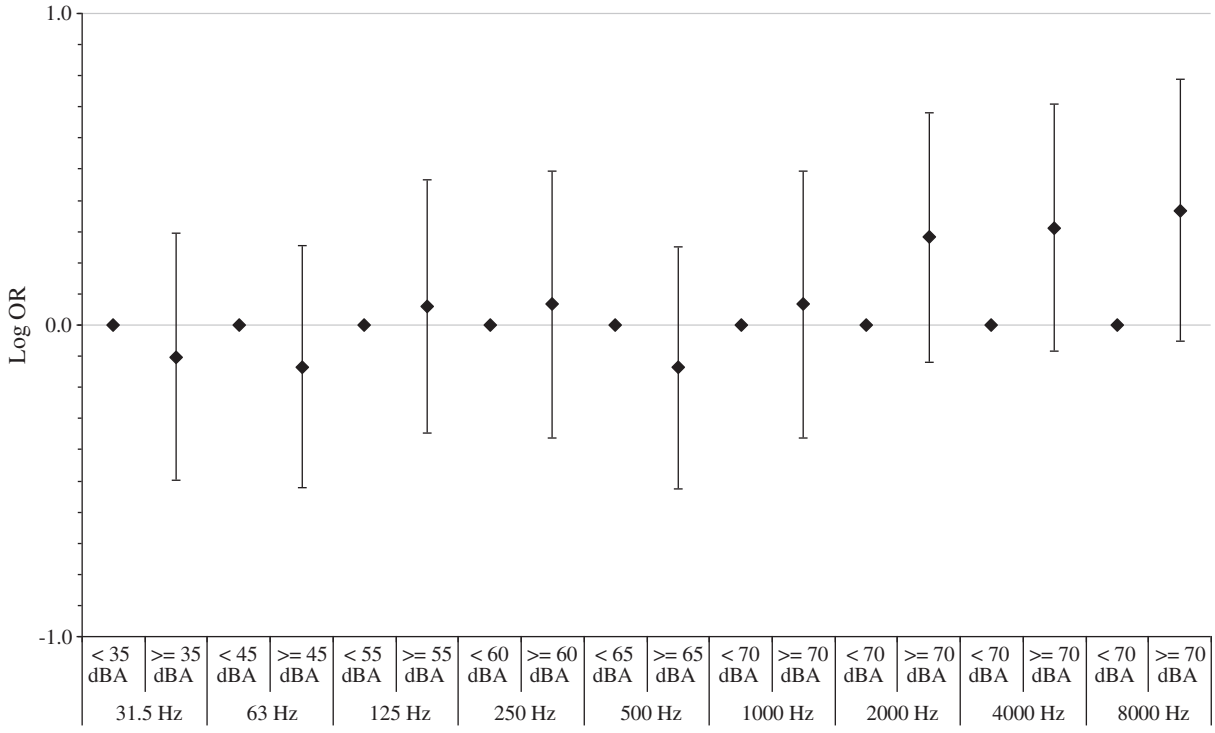

Fig. 1. Odds ratios and 95% confidence intervals for hypertension by median values of noise levels at different frequencies in male production-line workers (n = 129). All analyses adjusted for age, body mass index, cholesterol level, triglyceride level, educational level, current smoking, alcohol drinking, regular exercise, working activity and family history of hypertension.

was positively associated with the risk of hypertension among workers exposed to noise levels above 80 dBA, but this result was not significant in the multiple logistic regression model (adjusted odds ratio = 1.02; 95% confidence interval = 0.87–1.20, $p = 0.785$).

Such inverse "V-shaped" associations were found among male production-line workers exposed to noise levels ≥ 70 dBA at 1000, 2000, 4000 or 8000 Hz for 2–4 years, 4–6 years and more than 6 years, as shown in Fig. 3. Only workers exposed to ≥ 70 dBA at 4000 Hz for 2–4 years (noise levels of 75.1 ± 2.3 dBA at 4000 Hz; adjusted OR = 4.00, 95% CI = 1.04–15.42) had a significantly higher risk of hypertension compared with the reference group (noise levels of 67.0 ± 6.1 dBA at 4000 Hz) after controlling for potential confounding factors (Fig. 3 (c)). These inverse "V-shaped" associations might demonstrate the healthy worker effect in the present study.

4. Discussion

The present study is the first to show the association between noise frequency components and the prevalence of hypertension. We found that exposure to occupational noise at high frequencies (i.e., 2000, 4000 and 8000 Hz) was associated with an increased risk of hypertension. In particular, workers exposed to ≥ 70 dBA at 4000 Hz for 2–4 years had a significantly higher OR of hypertension compared with workers exposed to < 70 dBA at 4000 Hz and those exposed to ≥ 70 dBA at 4000 Hz with seniority less than 2 years. One possible reason for this finding may be that high-frequency noise exposure has a greater effect than low-frequency noise exposure on the release of stress hormones through the activation of the sympathetic and endocrine systems. An experimental study reported that

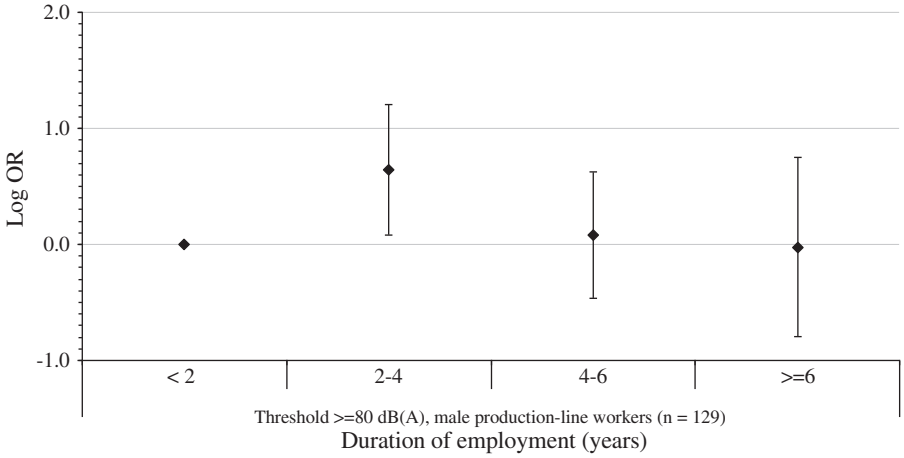

Fig. 2. Odds ratios and 95% confidence intervals for hypertension by duration of employment in male workers. All analyses adjusted for age, body mass index, cholesterol level, triglyceride level, educational level, current smoking, alcohol drinking, regular exercise, working activity and family history of hypertension.

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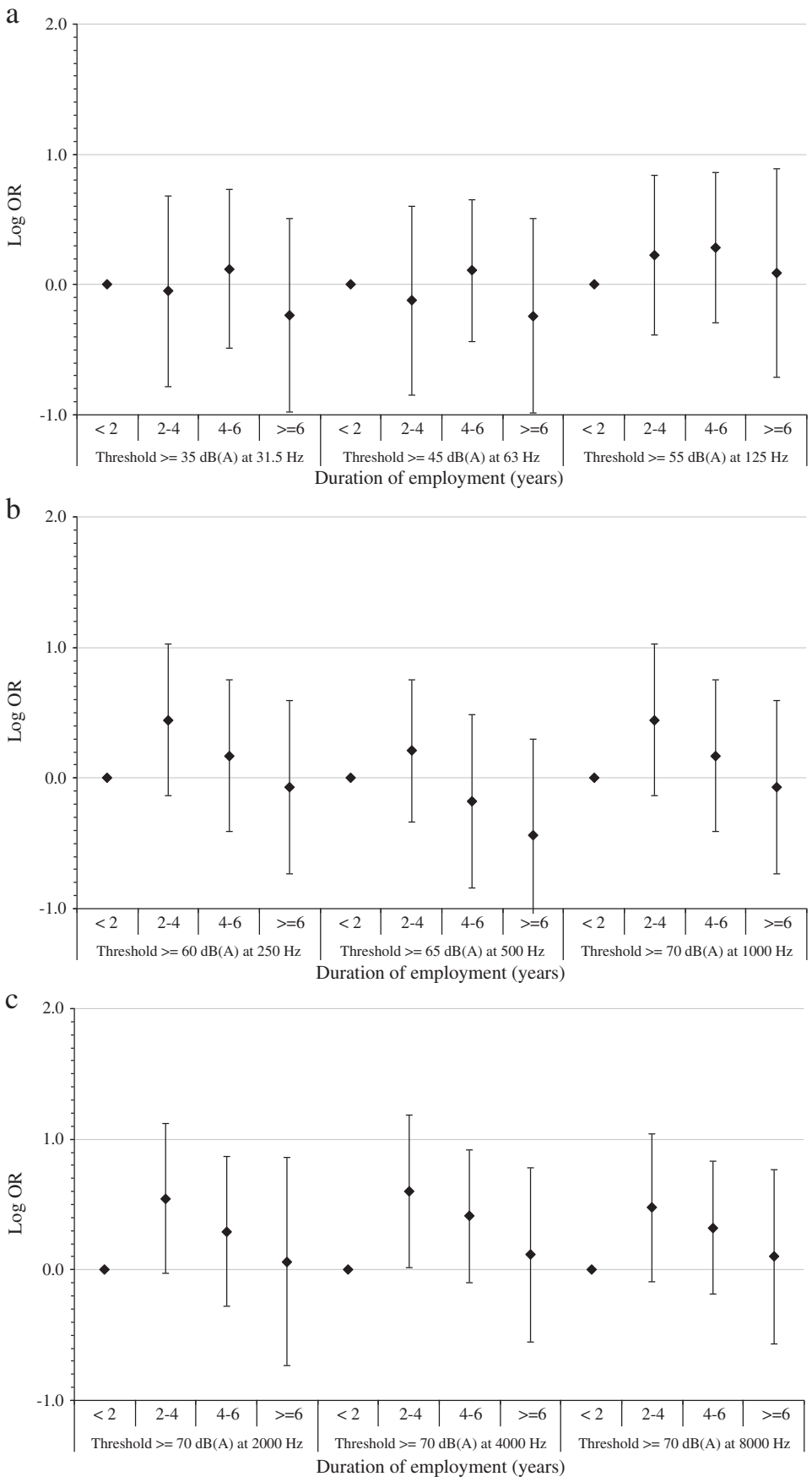

Fig. 3. Odds ratios and 95% confidence intervals for hypertension at different frequencies by duration of employment in male workers. (a) Low frequencies; (b) medium frequencies; (c) high frequencies. All analyses adjusted for age, body mass index, cholesterol level, triglyceride level, educational level, current smoking, alcohol drinking, regular exercise, working activity and family history of hypertension.

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a temporary elevation of saliva cortisol levels occurred in healthy male college students after pure tone exposure of 3000 Hz at 75 dBA (Yamamura et al., 1982). Another reason might be that men had a higher auditory sensitivity to high-frequency noise exposure than to low-frequency noise exposure. A previous study showed a dose–response association between high-frequency hearing loss at 4000 or 6000 Hz bilaterally and the risk of hypertension (Chang et al., 2011b). The notch at 4000 Hz in hearing loss has been considered the damage of outer hair cells within the cochlea. However, the link of this mechanism within the cochlea between hypertension and noise exposure at 2000 or 8000 Hz is not clear due to the lack of measurements for stress biomarkers. Future studies are advised to investigate this association at different noise frequencies.

Our results revealed that workers exposed to occupational noise with seniority of 2–4 years had a high prevalence of hypertension regardless of noise exposure at 2000, 4000 or 8000 Hz. Because subjects with a previous diagnosis of hypertension prior to working in the factory were excluded, this finding might imply that the higher risk of hypertension was observed only after occupational noise exposure over an empirical induction time (induction and latent periods). However, the risk of hypertension declined as seniority increased. One possible reason for this finding might be that these workers demonstrated healthier behaviors. For example, workers exposed to ≥70 dBA at 4000 Hz more than 6 years had lower proportions of cigarette smoking (30.8% vs. 42.3%) and alcohol drinking (15.4% vs. 19.2%) compared with the reference group. Another reason might be that production-line workers with noise-induced hypertension were transferred to the office. For instance, 2 out of 7 hypertension cases among office workers (28.6%) had employment durations of more than 6 years.

We also observed that workers exposed to noise levels ≥80 dBA for 2–4 years had a significantly higher risk of hypertension than the reference group. This finding was based on measurements of personal noise exposure and the exclusion of the use of HPDs at work. Fogari et al. (1994) reported that metallurgical workers exposed to environmental noise levels exceeding 80 dBA with employment durations of 11.6 ± 5.3 years had a significantly higher prevalence of hypertension than those exposed to less than 80 dBA. Chang et al. (2009) found that synthetic leather workers (55.6% using HPDs at work) exposed to 84.1 ± 2.3 dBA with employment durations of 7.4 ± 1.3 years had a significantly higher OR for hypertension compared with those exposed to 72.8 ± 2.2 dBA. These consistent results imply that exposure to occupational noise levels above 80 dBA might be associated with a risk of hypertension, although the noise exposure was less than the regulatory limit of 90 dBA in Taiwan.

The present study used results from personal sampling to classify subjects in an attempt to prevent non-differential misclassification of exposure, which might bias the risk estimate toward the null value (Checkoway et al., 2004). The exclusion of subjects who wore HPDs at work overcame an exposure bias that might produce a non-significant difference in the prevalence of hypertension between the noise-exposure and reference groups. It may be more effective to monitor occupational noise levels and related frequency components simultaneously to evaluate the association between noise frequency characteristics and hypertension.

A cross-sectional design of a temporal problem can potentially limit the causal inference between noise frequency components and the risk of hypertension. Although study subjects without a previous diagnosis of hypertension were recruited, the exposure history of participants prior to employment at this factory was unknown, which limited the ability to elaborate upon the between-group differences in the prevalence of hypertension due to occupational noise exposure.

Furthermore, inverse "V-shaped" associations between occupational noise exposure and employment duration might reveal the healthy worker effect, which is one of the main drawbacks in cross-sectional studies. Because employees working in December 2009 were recruited as study subjects, some workers exposed to noise levels above 80 dBA may have previously quit their jobs due to hypertension or noise-induced cardiovascular diseases. Therefore, the remaining workers may have been healthier employees or those more tolerant to noise exposure.

Additionally, the absence of time-activity logs in personal noise measurements may have limited the source identification and excluded some peak values at workplaces that are useful information to reduce workers' noise exposure in this study.

Finally, some confounders were not considered as covariates in the data analyses. These confounders are hypothesized to be associated with noise exposure and are important risk factors of hypertension, including low-density lipoprotein cholesterol and glycemia (i.e., blood glucose) (Babisch, 2002; Laurenzi et al., 1990; Lemne et al., 1994; Sowers et al., 1993). These unmeasured factors might contribute to the overestimate of the noise-exposure effect on the prevalence of hypertension.

5. Conclusions

The present study showed an association between occupational noise exposure during specific periods and the prevalence of hypertension in screw-manufacturing workers. This association was pronounced in noise frequency components at 4000 Hz. Occupational noise exposure at different frequencies may be associated with the variation in the risk of hypertension. Both exposure intensity and frequency characteristics should be considered to investigate the relationship between noise exposure and the development of hypertension in future studies.

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