Original Contribution

Occupational Noise Exposure and Incident Hypertension in Men: A Prospective Cohort Study

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The associations between occupational noise exposure and hypertension remain controversial because of the differences in study designs, exposure assessments, and confounding controls. This prospective study investigated the relationship between noise exposure and the 10-year risk of hypertension. A cohort of 578 male workers in Taiwan was followed from 1998 to 2008. All subjects were divided into high-, intermediate-, and low-exposure groups on the basis of noise exposure assessment. Cox regression models were used to estimate the relative risks of hypertension after adjustment for potential confounders. During the 7,805 person-years of follow-up, 141 hypertension cases were identified. Significant increases of 3.2 (95% confidence interval (CI): 0.2, 6.2) mm Hg in systolic blood pressure and 2.5 (95% CI: 0.1, 4.8) mm Hg in diastolic blood pressure between the baseline and follow-up measurements were observed in the high-exposure group. Participants exposed to ≥85 A-weighted decibels (dBA) had a 1.93-fold (95% CI: 1.15, 3.22) risk of hypertension compared with those exposed to <80 dBA. There was a significant exposure-response pattern (P = 0.016) between the risk of hypertension and the stratum of noise exposure. Prolonged exposure to noise levels ≥85 dBA may increase males’ systolic and diastolic blood pressure levels. This association may translate into a higher incidence of hypertension.

blood pressure; hypertension; men; occupational noise; prospective studies

Abbreviations: CI, confidence interval; dBA, A-weighted decibel(s); DBP, diastolic blood pressure; RR, relative risk; SBP, systolic blood pressure; SD, standard deviation.

Chronic exposure to noise has been associated with cardiovascular disease, including ischemic heart disease (1), myocardial infarction (2–5), coronary heart disease (6, 7), and stroke (8). This association may exist because noise exposure activates the sympathetic and endocrine systems to affect the humoral and metabolic states of the human organism, producing the 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 (9).

The existence of an association between noise exposure in occupational settings and hypertension is still controversial. Occupational noise exposure has been associated with a sustained elevation of blood pressure or with a higher risk of hypertension in 2 retrospective cohort studies (10, 11), 2 repeated-measure studies (12, 13), and 9 cross-sectional studies (14–22). However, the results of other studies are inconsistent with these findings (23–31). Reasons for this inconsistency may include differences in study design, differences in exposure assessment, different degrees of ability to control for potential confounders, and various degrees of the use of hearing-protective devices at work.

One cohort study reported a relationship between occupational noise exposure and the incidence of hypertension (10). However, these results were limited by an exposure bias caused by no adjustments for the use of hearing-protective devices, and the association between noise exposure and blood pressure was not reported. In addition,
important risk factors for hypertension, such as body mass index, cigarette use, alcohol intake, regular exercise, salt intake, and a family history of hypertension (32, 33), were not considered. The objective of this study was to investigate the relationship between prolonged exposure to occupational noise and the 10-year incidence of hypertension by taking these important factors into account.

MATERIALS AND METHODS

Study population

This study was conducted by performing a follow-up study to a cross-sectional survey in an aircraft manufacturing plant in central Taiwan. The recruitment and selection of study subjects have been described in detail previously (21). Briefly, 790 male production line workers were recruited in 1998 and were invited to join again at the end of 2008. To ensure study subjects without hypertension at baseline, we excluded 10 workers because they reported having a physician diagnosis of hypertension and using antihypertensive medication according to a questionnaire survey in 1998. During the 10-year period, 86 workers retired and were lost to follow-up. In addition, 116 subjects were rejected because of the lack of follow-up results in 2008. Therefore, the study group comprised 578 male production line workers. There were no significant differences between the 578 participants and the 202 nonparticipants at baseline in terms of employment duration, body mass index, high-density lipoprotein level, triglyceride level, cigarette use, alcohol intake, and regular exercise. 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 in 2008, and written informed consent was obtained again from each participant.

Blood pressure measurements and definition of hypertension

The procedure for baseline blood pressure measurements in 1998 was the same as that for the follow-up measurements in 2008. All subjects were required to fast overnight before blood sampling and blood pressure measurements during annual health examinations. Subjects sat for 10 minutes in a chair with back support before blood pressure was measured bilaterally by a trained nurse using an automated sphygmomanometer (Ostar Model P2; Ostar Meditech Corp., Taipei, Taiwan). The mean value of the 2 measurements was recorded to represent the individual’s blood pressure in the present study. Subjects were defined as hypertensive if they reported a diagnosis of hypertension given by physicians after 1998, if the mean value of their resting systolic blood pressure (SBP) was ≥140 mm Hg in 2008, or if the mean value of their resting diastolic blood pressure (DBP) was ≥90 mm Hg in 2008. Height, body weight, total cholesterol level, and triglyceride level were also measured in all subjects at baseline and follow-up. The body mass index was calculated as weight (kg)/height (m)2.

In addition, a self-administered questionnaire was used to collect potential confounders in 1998 and 2008. These factors included age, educational level, employment duration, cigarette use, alcohol intake, regular exercise, the use of antihypertensive medication, and the use of hearing-protective devices. Additional information (such as salt intake and a family history of hypertension) was included only in 2008. To avoid information bias, we defined the lifestyle habits for regular users specifically (21, 34). The use of hearing-protective devices included the percentage of time that the subjects wore hearing-protective devices (i.e., never use, <2 hours, 2–4 hours, >4–6 hours, and >6–8 hours working time) and the type of hearing-protective devices (i.e., earplugs, earmuffs, or both). High salt-intake workers were defined as those who reported ingesting food cooked with soybean sauce for more than one meal per day. A subject was defined as having a family history of hypertension if a positive answer was given to the question: “Have your parents or grandparents been diagnosed with hypertension by a physician in the past?” (21, 34).

Follow-up

We used employment personnel records to obtain each subject’s first date of employment at this company and assigned this date as the beginning of the follow-up period. The end of the follow-up period was defined as either the date of diagnosis by a physician or the date when the subjects who had not been diagnosed with hypertension by a physician were given their blood pressure examinations in December 2008. Among the 141 cases classified as hypertensive in this study, 56 cases were diagnosed with hypertension (30 cases were taking antihypertensive medication), and 85 cases were identified at the annual examination in 2008.

Noise exposure assessment

The procedure for noise exposure assessment in 1998 was similar to that for the follow-up measurements in 2008. We first identified 18 departments in this company and divided each department into different locations on the basis of the manufacturing processes by industrial hygienists and senior workers. After the walk-through survey, we measured the 15-minute time-weighted average equivalent sound level by using a sound analyzer (Model TES-1358; TES Electronic Corp., Taipei, Taiwan), which was calibrated with a sound-level calibrator (Model TES-1356; TES Electronic Corp.) before environmental monitoring. The short-term environmental sampling was performed at 337 locations that were possibly the loudest workplaces at this company. For the 121 sites exhibiting a 15-minute time-weighted average equivalent sound level of ≥65 A-weighted decibels (dBA), additional 8-hour time-weighted average measurements were conducted during September–December of 1998. All subjects were divided into one of similar exposure groups on the basis of the similarity and frequency of tasks performed, the agents and processes with which they worked, and the ways in which they performed the tasks (35). Each subject was assigned a specific value of noise exposure on the basis of the 8-hour time-weighted average equivalent sound level measured in his workplace.
To avoid an exposure bias due to the use of hearing-protective devices at work, we calculated each participant’s level of noise reduction according to the noise reduction rating of the hearing-protective devices that he wore (29 dB for earplugs and 25 dB for earmuffs), the protection levels of the hearing-protective devices (an average of 28% for earplugs and an average of 62% for earmuffs), a comfort factor of 0.5 (because of the minimal percentage of comfort related to compliance with usage of hearing-protective devices) (36), and the percentage of working time that he used the hearing-protective devices (36, 37). We used the hearing-protective device-adjusted value of the 8-hour Table 1. Baseline Characteristics of Study Participants, Taichung, Taiwan, 1998–2008

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Exposure Groups</th>
<th>Total Subjects (n = 578)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High (n = 152)</td>
<td>Intermediate (n = 221)</td>
<td>Low (n = 205)</td>
</tr>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td></td>
<td>No.  %</td>
<td>No.  %</td>
<td>No.  %</td>
</tr>
<tr>
<td>Age at entry, years</td>
<td>27.6 (4.6)</td>
<td>27.5 (5.4)</td>
<td>28.0 (5.6)</td>
</tr>
<tr>
<td>Employment duration, years</td>
<td>10.2 (5.0)b</td>
<td>9.0 (4.8)c</td>
<td>10.9 (6.0)</td>
</tr>
<tr>
<td>Body mass indexd</td>
<td>24.0 (3.0)</td>
<td>23.8 (3.1)</td>
<td>23.6 (2.9)</td>
</tr>
<tr>
<td>HDL, mg/dl</td>
<td>45.4 (6.9)</td>
<td>46.3 (10.6)</td>
<td>46.0 (8.3)</td>
</tr>
<tr>
<td>LDL, mg/dl</td>
<td>115.1 (30.4)</td>
<td>114.5 (30.3)</td>
<td>112.3 (28.8)</td>
</tr>
<tr>
<td>Triglyceride, mg/dl</td>
<td>142.2 (63.4)</td>
<td>143.6 (86.0)</td>
<td>156.7 (95.1)</td>
</tr>
</tbody>
</table>

**Education (≤12 years)**

<table>
<thead>
<tr>
<th>Education (≤12 years)</th>
<th>Mean (SD)</th>
<th>No.  %</th>
</tr>
</thead>
<tbody>
<tr>
<td>63 41.5e</td>
<td>106 48.0e</td>
<td>50 24.4</td>
</tr>
<tr>
<td>192 33.3e</td>
<td>219 37.9</td>
<td>&lt;0.001f</td>
</tr>
</tbody>
</table>

**Use of hearing-protective devices at work**

<table>
<thead>
<tr>
<th>Use of hearing-protective devices at work</th>
<th>Mean (SD)</th>
<th>No.  %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>39 25.7e, g</td>
<td>16 7.2e</td>
</tr>
<tr>
<td>&lt;2 hours’ working time</td>
<td>80 52.6</td>
<td>35 15.8</td>
</tr>
<tr>
<td>2–4 hours’ working time</td>
<td>31 20.4</td>
<td>39 17.7</td>
</tr>
<tr>
<td>&gt;4–6 hours’ working time</td>
<td>1 0.7</td>
<td>59 26.7</td>
</tr>
<tr>
<td>&gt;6–8 hours’ working time</td>
<td>1 0.7</td>
<td>72 32.6</td>
</tr>
</tbody>
</table>

**Abbreviations:** HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation.

- a: Kruskal-Wallis test of the difference among the 3 groups.
- b: Mann-Whitney test for a significant difference (P < 0.05) compared with the intermediate-exposure group.
- c: Mann-Whitney test for a significant difference (P < 0.05) compared with the low-exposure group.
- d: Body mass index: weight (kg)/height (m)^2.
- e: χ2 test for a significant difference (P < 0.05) compared with the low-exposure group.
- f: χ2 test for the difference among the 3 groups.
- g: χ2 test for a significant difference (P < 0.05) compared with the intermediate-exposure group.
time-weighted average equivalent sound level to classify participants into 3 exposure groups by selecting the median and third quartile as cutoff points in the distribution of noise exposure among all subjects. The 578 workers were subdivided into a high-exposure group (n = 152; noise level: ≥85 dBA), an intermediate-exposure group (n = 221; noise level: 80–<85 dBA), and a low-exposure group (n = 205; noise level: <80 dBA).

### Statistical analysis

We first used the Shapiro-Wilk test to determine the normality of continuous variables. The Kruskal-Wallis test was then used to perform multiple comparisons of continuous variables among the 3 groups for the nonnormal distribution, and a 1-way analysis of variance was used for the same comparison of continuous variables that were normally distributed. We also used the χ² test to compare the difference in dichotomous variables among the 3 groups. For those groups with significant differences, the Mann-Whitney test (or t test) and the χ² test were used to compare the high- and intermediate-exposure groups with the low-exposure group for continuous and dichotomous variables.

To compare individual differences in SBP and DBP between the baseline and follow-up measurements, we used the linear mixed-effect regression models for each exposure group (38, 39). The fixed effects in the mixed model included all variables in the final model at baseline and the use of antihypertension medication in 2008. Individual subjects were used as a random effect. We used the first-order autoregressive model for covariance structures because of the minimizing value of Akaike’s Information Criterion in both SBP and DBP measurements (38, 39).

To avoid the information bias in observed person-years due to the availability of only 1 blood pressure measurement within the past 10 years, we identified hypertensive cases by questionnaire, blood pressure measurements, and total hypertensive cases used as the outcomes to perform the regression analyses. We used Cox proportional hazard regressions and calculated relative risks with 95% confidence intervals to compare the differences in incidence of hypertension among groups while controlling for potential confounders.

We used a manual stepwise regression to build the final model because only one variable of body mass index (P = 0.005) was retained in the final step using an automatic stepwise procedure. A basic model was examined first that included age at baseline for biological plausibility and 2 dummy variables of exposure categories. The basic model was then enlarged to include 2 variables (i.e., body mass index and employment duration) that were significantly associated with the risk of hypertension in the simple Cox regression models. The final model included all variables in the extended model and important risk factors of hypertension reported in the previous literature (32–33, 40), including socioeconomic status (using educational levels or job positions as surrogates), cigarette use, alcohol intake, and regular exercise. In the sensitivity analysis, the 2 variables of salt intake and family history of hypertension that had been collected only in 2008 were included in the model. The SAS standard package for Windows, version 9.2 (SAS Institute, Inc., Cary, North Carolina), was used for the statistical analyses. The significance level was set at 0.050 for all tests.

### RESULTS

Table 1 summarizes the demographic characteristics of 578 participants at baseline. Significant differences were identified among the exposure groups in the mean value of employment duration, the number of individuals with an educational level of ≤12 years, and whether individuals used hearing-protective devices at work. Workers in the high- and intermediate-exposure groups were more likely to have an educational level of ≤12 years and were less likely to never use hearing-protective devices at work than those in the low-exposure group. In addition, high-exposure workers had a significantly higher mean of employment duration and were less likely to never use hearing-protective devices at work compared with the intermediate-exposure workers. In contrast, intermediate-exposure workers had a significantly lower mean of employment duration than did the low-exposure workers.

Table 2 shows the measurements of occupational noise exposure for the 3 groups. Significant differences were
identified in the mean noise levels among the 3 groups either before or after an adjustment for hearing-protective device use. The high- and intermediate-exposure groups were exposed to significantly higher mean values of noise compared with the low-exposure group both before and after an adjustment for hearing-protective device use.

Table 3 shows the changes in blood pressure between the baseline and follow-up measurements in the 3 groups. Although there were no significant differences in SBP and DBP among these 3 groups at baseline or follow-up, the SBP of both the high-exposure ($P = 0.035$) and the intermediate-exposure ($P < 0.001$) groups significantly increased between the baseline and follow-up measurements in the linear mixed-effect models. In addition, the DBP of high-exposure workers significantly increased between the baseline and follow-up measurements ($P = 0.042$) after controlling for potential confounders.

The associations among the different groups and the risk of hypertension are summarized in Table 4. The multiple Cox proportional hazard regression models showed that workers exposed to 86.9 (standard deviation (SD), 2.2) dBA and those exposed to 83.0 (SD, 1.3) dBA had a 1.93-fold and a 1.75-fold relative risk of hypertension, respectively, compared with those exposed to 71.9 (SD, 9.0) dBA after an adjustment for potential confounders. There was a significant dose-response relationship between the levels of noise exposure experienced by the 3 groups and the risk of hypertension using total cases as the outcomes (adjusted relative risk (RR) = 1.35, 95% confidence interval (CI): 1.06, 1.73). Similar results were found while using job positions (managers, engineers, administrators, and technicians) instead of educational levels in the models. In addition, the intermediate-exposure group had a significantly higher risk of measured hypertension than did the low-exposure group.

In sensitivity analyses, the association between occupational noise exposure and the risk of hypertension persisted for both the high-exposure (adjusted RR = 1.95, 95% CI: 1.16, 3.28) and the intermediate-exposure (adjusted RR = 1.74, 95% CI: 1.08, 2.80) groups after the addition of the 2 variables of salt intake and family history of hypertension to model 3. Furthermore, an increasing exposure-response trend was found (adjusted RR = 1.37, 95% CI: 1.07, 1.75) ($P = 0.014$) in this sensitivity analysis. No significant differences were found among the 3 exposure groups with regard to salt intake ($P = 0.173$) or family history of hypertension ($P = 0.656$).

**DISCUSSION**

We found a positive relationship between occupational noise exposure ≥85 dBA at baseline and the 10-year incidence of hypertension. The findings were consistent with the results in a retrospective study that reported a significantly higher risk of hypertension in sawmill workers exposed to ≥85 dBA for more than 30 years (10). Although their findings were limited to a misclassification of noise exposure because of no adjustment for the use of hearing-protective devices at work, the 2 longitudinal studies reached similar results to show an increased risk of hypertension following long-term chronic
occupational noise exposure. Moreover, the present study overcame the lack of individual risk factors of hypertension in the previous study (10) to provide strong evidence that hypertension was associated with occupational noise exposure. We suggest that the currently regulated threshold for occupational noise exposure may expand the prevention of noise-induced hearing loss (41) to the risk of hypertension, which is a leading cause of cardiovascular diseases.

We also observed an exposure-response pattern between noise-exposure levels and the risk of hypertension. Our results were concordant with the findings in a cross-sectional study showing that increasing noise exposure from 75 to 104 dBA was associated with an increasing prevalence of hypertension in female textile mill workers (16). One cohort study reported a significantly increasing risk of hypertension with cumulative noise exposure ranging from 95 dBA × years to >115 dBA × years among male workers (10). The same exposure metrics (i.e., 4 categories from <85 dBA × years to ≥95 dBA × years) were applied to our analyses, but no significant dose-response relationship (adjusted RR = 1.03, 95% CI: 0.87, 1.21) was found in the Poisson regression. The possible reason for different exposure conditions resulting in the same dose-response association might be that workers did not change jobs and tasks in present and previous studies (16) and that there was turnover in another cohort (10).

In addition, the inverse-U-shaped dose-response relationship for measured hypertension indicated a role of exposure duration in the etiology of the disease. Although we used Cox regressions to account for varying time of exposure, the magnitudes of effects on measured hypertension and diagnosed hypertension in the high-exposure group were similar (i.e., around 2-fold), yet the effect halved from 2.22 to 1.16 in the intermediate-exposure group were similar (i.e., around 2-fold), yet the effect halved from 2.22 to 1.16 in the intermediate-exposure group. These findings may indicate a “plateau” effect of exposure intensity that is independent of exposure duration. Therefore, when the exposure intensity is not the highest, the exposure duration is likely the second component of exposure that “kicks in” the dysregulation of the nervous and hormonal systems, leading to the disease.

The significant increases in SBP between the baseline and follow-up measurements were also observed among workers exposed to 80–<85 dBA and those exposed to ≥85 dBA. These findings were consistent with the results in a cohort study showing that male workers exposed to ≥85 dBA and using 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 in office workers (11). Additionally, workers exposed to ≥85 dBA exhibited a significant increase in DBP between the baseline and follow-up measurements as well as a significantly higher DBP at follow-up compared to

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**Table 4. Associations Between Different Noise Exposure Levels and Incidence of Hypertension, Taichung, Taiwan, 1998–2008**

<table>
<thead>
<tr>
<th>Outcomes by Different Noise Exposure Levels, dBA</th>
<th>No. of Cases</th>
<th>Incidence</th>
<th>Crude RR</th>
<th>Model 1&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Model 2&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Model 3&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosed hypertension&lt;sup&gt;d&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;80</td>
<td>18</td>
<td>6.3 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1</td>
<td>Referent</td>
<td>1</td>
<td>Referent</td>
</tr>
<tr>
<td>80–&lt;85</td>
<td>19</td>
<td>6.3 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1.00</td>
<td>0.52, 1.90</td>
<td>1.00</td>
<td>0.53, 1.91</td>
</tr>
<tr>
<td>≥85</td>
<td>19</td>
<td>9.9 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1.64</td>
<td>0.86, 3.12</td>
<td>1.65</td>
<td>0.87, 3.15</td>
</tr>
<tr>
<td>P&lt;sub&gt;trend&lt;/sub&gt;</td>
<td></td>
<td></td>
<td>0.149</td>
<td>0.141</td>
<td>0.051</td>
<td>0.051</td>
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<tr>
<td>Measured hypertension&lt;sup&gt;e&lt;/sup&gt;</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;80</td>
<td>26</td>
<td>9.0 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1</td>
<td>Referent</td>
<td>1</td>
<td>Referent</td>
</tr>
<tr>
<td>80–&lt;85</td>
<td>40</td>
<td>13.2 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1.48</td>
<td>0.90, 2.42</td>
<td>1.49</td>
<td>0.90, 2.43</td>
</tr>
<tr>
<td>≥85</td>
<td>19</td>
<td>9.9 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1.19</td>
<td>0.66, 2.15</td>
<td>1.20</td>
<td>0.66, 2.16</td>
</tr>
<tr>
<td>P&lt;sub&gt;trend&lt;/sub&gt;</td>
<td></td>
<td></td>
<td>0.452</td>
<td>0.438</td>
<td>0.101</td>
<td>0.136</td>
</tr>
<tr>
<td>Total hypertension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;80</td>
<td>44</td>
<td>15.3 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1</td>
<td>Referent</td>
<td>1</td>
<td>Referent</td>
</tr>
<tr>
<td>80–&lt;85</td>
<td>59</td>
<td>19.5 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1.28</td>
<td>0.87, 1.89</td>
<td>1.28</td>
<td>0.87, 1.90</td>
</tr>
<tr>
<td>≥85</td>
<td>38</td>
<td>19.9 × 10&lt;sup&gt;−3&lt;/sup&gt;</td>
<td>1.38</td>
<td>0.89, 2.13</td>
<td>1.39</td>
<td>0.90, 2.15</td>
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<tr>
<td>P&lt;sub&gt;trend&lt;/sub&gt;</td>
<td></td>
<td></td>
<td>0.134</td>
<td>0.125</td>
<td>0.012</td>
<td>0.016</td>
</tr>
</tbody>
</table>

Abbreviations: ARR, adjusted relative risk; CI, confidence interval; dBA, A-weighted decibel(s); RR, relative risk.

<sup>a</sup> The Cox proportional hazards regression adjusted for biological plausibility (i.e., age at baseline) as the basic model.

<sup>b</sup> The Cox proportional hazards regression adjusted for age at baseline and significant factors in simple Cox regression models (such as body mass index and employment duration) as the extended model.

<sup>c</sup> The Cox proportional hazards regression adjusted for all variables in model 1, model 2, and important risk factors reported in previous literature (i.e., educational level, cigarette use, alcohol intake, and regular exercise) as the final model.

<sup>d</sup> Subjects reported that a physician had previously given them a diagnosis of hypertension.

<sup>e</sup> Subjects had a mean value of resting systolic blood pressure of ≥140 mm Hg or a mean value of resting diastolic blood pressure of ≥90 mm Hg.
with those exposed to <80 dBA. These findings provided the evidence to explain the association between occupational noise exposure above 85 dBA and the higher risk of hypertension.

The strengths of the present study include a cohort-study design, detailed assessments of personal exposure histories, and comprehensive controls for most potential confounders of hypertension. In addition, occupational noise exposure adjusted for the use of hearing-protective devices may avoid the misclassification of study subjects and an overestimation of noise exposure to produce the consistent results associated with road traffic noise exposure in environmental epidemiologic studies (34, 42).

Testing for the validity of noise exposure assessment was also conducted to check the precision and accuracy of a predictive model, and noise levels accounted for the use of hearing-protective devices. Although information from departments, processes, job positions, and noise measurements collected in 1998 was used to establish a predictive model of noise levels, the adjusted $R^2$ was 0.93 in a multiple linear regression. In a comparison of environmental measurements at 11 locations in 2004, the bias and precision in this model were 2.4 (SD, 4.9) dBA to produce an accuracy of 5.5 dBA, which showed an overestimation due to engineering controls over time. In contrast, the bias and precision of 2-stage noise measurements accounting for the use of hearing-protective devices among study subjects were 1.5 (SD, 2.0) dBA to have an accuracy of 2.5 dBA. Such comparisons indicated that assessment of exposure by using the worst-case scenario might be inaccurate while still being precise.

One limitation of this study is the overestimation of individual cumulative levels in occupational noise exposure. Noise levels measured 10 years ago have obviously been reduced in response to the requirement for the improvement of engineering controls in the workplace. Therefore, workers assigned to the high-exposure group in 1998 might be exposed to <85 dBA in 2008. A comparison of environmental noise levels between the baseline and follow-up in all subjects showed that the mean value of noise levels measured in 2008 (77.6 (SD, 7.6) dBA) was significantly lower than that measured in 1998 (81.6 (SD, 8.5) dBA) (paired t test, $P<0.001$). The overall decrease of 5.2 (SD, 9.3) dBA had apparent decreases in the processes of peripheral element assembly ($-9.7$ (SD, 16.4) dBA) and forging and casting ($-7.3$ (SD, 7.7) dBA) that might reduce the proportion of the use of hearing-protective devices from 73.3% to 27.7% and from 85.7% to 61.9%, respectively. Because no subjects changed their jobs or tasks, the systematic overestimation of cumulative noise exposure was equally distributed across different groups, and the nondifferential misclassification of exposure might attenuate the estimates toward the null. However, our findings still observed the significant relationship between occupational noise exposure and the risk of hypertension.

The other limitation is that noise exposure, blood pressure, and other important risk factors of hypertension were not measured continuously over the past 10 years. The significantly higher risk of hypertension among the workers exposed to $\geq 85$ dBA might be because they experienced the greatest changes in lifestyle. However, this reason seems improbable because the prevalence rates of risk factors related to hypertension, such as cigarette use (58%), and alcohol intake (63%), declined significantly (27% and 21%, respectively; both $P'$s $<0.001$) in the high-exposure group at the follow-up examination. In addition, blood pressure was measured twice over the past 10 years. This is our first attempt to longitudinally observe the occupational noise exposure and the incidence of hypertension among these workers. The 4-year period of follow-up will continue in 2012 to circumvent limitations imposed by lifestyle changes over time and to have additional measurements of noise exposure and blood pressure.

In summary, the present study showed that occupational exposure to noise levels of $\geq 85$ dBA was associated with the development of hypertension and that there was a dose-response relationship between noise exposure and the 10-year incidence of hypertension. The association between occupational noise exposure of $\geq 85$ dBA and the risk of hypertension might be due to the increases of SBP and DBP observed in the present study. The currently regulated threshold for occupational noise exposure may prevent the adverse health effects from noise-induced hearing loss to the development of hypertension.

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