Effects of Occupational Noise Exposure on Blood Pressure

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We measured 24-hour ambulatory blood pressure and 16-hour noise exposure continuously for 20 automobile workers, and used linear mixed-effects regression models to estimate transient and sustained effects of noise exposure on blood pressure. The occupational noise levels of the high-exposure workers with 85 ± 8 dBA were significantly higher than those of the low-exposure workers with 59 ± 4 dBA ($P < 0.05$). We found a significant difference of 16 ± 6 mm Hg in sleep-time systolic blood pressure (SBP) existed between 2 exposure groups, and a marginal increase of 1 mm Hg SBP per 1-dBA increase in occupational noise exposure at a 60-minute lag time during work ($P = 0.07$). Occupational noise exposure had both transient and sustained effects on workers' SBP. (J Occup Environ Med. 2003;45:1289–1296)

Several comprehensive reviews have documented various adverse effects of noise exposure, including hearing impairment, annoyance, sleep disturbance, and hypertension.^{1–3} Blood pressure changes were mostly related to occupational noise exposure. Several epidemiologic studies had shown chronic noise exposure significantly increased blood pressure.^{4–10} Two experimental studies conducted in the laboratories had found workers' blood pressure increased after exposure to industrial noise levels greater than 95 dBA.^{11,12} Two field studies, which were originally designed to investigate acute and chronic effects of noise exposure on blood pressure simultaneously, found that occupational noise exposure had transient but no sustained effects on blood pressure.^{13,14} However, 1 animal study reported that noise exposure induced both acute and chronic effects on the rhesus monkey's blood pressure.¹⁵

In previous epidemiologic studies, only 1-time resting blood pressure measurements were used to represent individuals' blood pressure. Usually, environmental monitoring rather than personal data represented individuals' noise exposure. We believed insufficient measurements for noise exposure, blood pressure, and confounding factors usually restricted the findings of sustained effect by noise exposure in past epidemiologic studies. The lack of continuous measurements also limited the possibility of finding the effect of short-term noise exposure on blood pressure.

In this study, we conducted a panel study on automobile workers to in-

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investigate whether workers' blood pressure can be increased by their transient and sustained noise exposure in a working environment with high noise exposure.

Methods

Subjects

We performed a panel study on 20 male workers in an automobile manufacturing company in Taiwan in 2001. Our study subjects were randomly selected from all male employees who were working either in the production lines or in the office of the automobile company. Major operations in this company included forging and casting, press forging of stainless steel, engine manufacturing and assembly, body assembly, painting, peripheral element assembly, and roller and track trial testing. The 15 subjects with high noise exposure were from operational units of press forging, engine manufacturing and assembly, body assembly, roller and track trial testing. The environmental monitoring data in 2000 showed the noise levels were at 79 to 110 dBA and ototoxic chemicals were not present in these working environments. These study subjects were not wearing any hearing protection devices, but 6 of them were exposed to noise and vibration at the same time during the study. Another 5 subjects with low noise exposure were recruited from the staff working in a separate office building of the company.

According to the health checkup results in 2000 before the panel study, none of these 20 subjects were diagnosed with hypertension or other cardiovascular diseases. The checkup also provided important information related to hypertension such as subjects' heights and weights, resting blood pressure, total cholesterol, and triglyceride. Individuals' heights and weights were used to calculate body mass index (BMI). For the subjects working in the worksites with environmental noise levels above 85 dBA, audiometric

tests were performed at 500, 1000, 2000, 3000, 4000, 5000, and 6000 Hz for both ears annually. During the panel study, we used a questionnaire to collect additional risk factors of blood pressure such as individuals' age, employment duration, exercise habit, smoking history, alcohol consumption, and family disease history. The Institutional Review Board of the College of Public Health, National Taiwan University, approved this study, and written informed consent was obtained from each participating worker.

Ambulatory Blood Pressure Monitoring and Recording

We recorded each subject's blood pressure every 30 minutes during awake time (8:00 AM–11:00 PM) and every 60 minutes during sleep time (11:00 PM–8:00 AM) continuously by using a portable, noninvasive, automated blood pressure monitoring and recording system (DynaPulse model 5000A, Pulse Metric, San Diego, CA). The DynaPulse system can measure a subject's arterial pulsation signals, known as the arterial waveform, through a noninvasive cuff device. The systolic and diastolic blood pressure readings by DynaPulse have been validated against the traditional mercury sphygmomanometer measurements.^{16,17} Each subject carried a DynaPulse system for 24 hours to complete his continuous blood pressure monitoring. The blood pressure data of 15 high noise exposure subjects during the working period (8:00 AM–4:30 PM) were used to investigate the transient effect of occupational noise exposure on workers' systolic blood pressure (SBP) and diastolic blood pressure (DBP). All 20 participants' blood pressure measurements during the sleep period were used to study the sustained effect by occupational noise exposure.

Exposure Measurements

We measured personal noise exposure continuously by using a personal noise dosimeter (Logging Noise Dose Meter type 4443, Brüel

& Kjær, Nærum, Denmark), which can report 5-minute continuous equivalent sound level (Leq) at an exchange rate of 3 dBA and time-weighted-average (TWA) noise dose. This dosimeter has 3 measurement ranges, which are 70–140 dBA, 50–120 dBA, and 30–100 dBA. The range of 50–120 dBA was used to measure the high-exposure group and the range of 30–100 dBA was to measure the low-exposure group. All subjects carried the Logging Noise Dose Meter to measure their personal noise exposure with 5-minute readings over their working period (8:00 AM–4:30 PM). One single TWA noise exposure level was obtained for each subject during the off-duty period (4:30 PM–11:00 PM). Noise exposure levels were not measured during the sleep period. To investigate the time-lag effect of acute noise exposure, 5-minute exposure measurements were transformed to 30-minute, 60-minute, and 120-minute time-moving-average segments for further analysis.

Statistical Analysis

We performed univariate comparisons between the 2 exposure groups by using *t* test for continuous variables and Fisher exact test for dichotomous variables. We used linear mixed-effects regression models to associate transient noise exposure with blood pressure increase by controlling important confounding factors of study subjects.¹⁸ The linear mixed-effects regression model has the advantages of dealing with the autocorrelation problem between consecutive blood pressure measurements and increasing statistical power by combining information across study subjects. In our mixed-effects models, personal noise exposure at the moving averages of 0 to 2 hours was generated from original 5-minute measurements. The fixed effect in our mixed-effects models contained covariance parameters of age, employment duration, BMI, smoking, alcohol consumption, fam-

TABLE 1
Brief Description of Subjects

Characteristics	High-Exposed Group	Low-Exposed Group	P Value
Subjects (no.)	15	5	
Age (years)*			
Mean (SD)	39 (7)	45 (8)	0.16
Employment duration (years)*			
Mean (SD)	14 (8)	17 (9)	0.58
Body mass index (kg/m ²)*			
Mean (SD)	24 (2)	26 (1)	0.22
Resting SBP (mm Hg)*			
Mean (SD)	123 (19)	121 (18)	0.85
Resting DBP (mm Hg)*			
Mean (SD)	83 (12)	84 (9)	0.98
Total cholesterol (mg/dL)*			
Mean (SD)	205 (89)	191 (14)	0.56
Triglyceride (mg/dL)*			
Mean (SD)	170 (94)	138 (50)	0.49
Regular exercise†			
Yes (%)	3 (20%)	2 (40%)	0.56
No (%)	12 (80%)	3 (60%)	
Smoking†			
Yes (%)	11 (73%)	2 (40%)	0.29
No (%)	4 (27%)	3 (60%)	
Alcohol consumption†			
Yes (%)	5 (33%)	1 (20%)	1.00
No (%)	10 (67%)	4 (80%)	
Hypertension family†			
Yes (%)	8 (57%)	3 (60%)	1.00
No (%)	6 (43%)	2 (40%)	
Material hearing impairment‡			
Number (%)	12 (80%)	NA	

* *t* test of the difference between the 2 exposure groups.

† Fisher exact test of the difference between the 2 exposure groups.

‡ Defined by the U.S. Occupational Safety and Health Administration's criteria, ie, an average hearing threshold level (HTL) of 25-dB HTL at 1000, 2000, and 3000 Hz.

SD, standard deviation; SBP, systolic blood pressure; DBP, diastolic blood pressure.

NA = Not available.

ily hypertension history, and noise exposure. Individual subjects were treated as a random effect in the model. The explanatory variables were chosen in a stepwise procedure to minimize Akaike's information criterion (AIC), which is a function of the log-likelihood function adjusted for the number of covariance parameters.¹⁹ The model with the lowest AIC value was selected as the best-fitted model. The mixed procedure containing fixed and random effects in SAS 8.1 version (SAS Institute Inc., Cary, NC) was used to perform the linear mixed-effects regression, and the statistical significance level was set at 0.05 in this study.

Results

Table 1 summarizes the demographic characteristics and health risk factors by noise exposure levels for 20 study subjects. There were no significant differences between high and low noise exposure workers for age, employment duration, BMI, resting SBP, resting DBP, total cholesterol, triglyceride, regular exercise, smoking, habitual drinking, and a family history of hypertension ($P > 0.05$). Although there is no statistically significant between-group difference in the prevalence of smoking, it is worth pointing out that the prevalence of smoking is very high in the high-exposure group

(73%) compared with the low-exposure group (40%). There were 12 workers in the high-exposure group meeting the criteria of material hearing impairment defined by the U.S. Occupational Safety and Health Administration, which is an average hearing threshold level (HTL) of 25-dB HTL at 1000, 2000, and 3000 Hz. Their average hearing thresholds were 50-dB HTL at 4000 Hz and 51-dB HTL at 6000 Hz, respectively.

An exemplary graph of concurrent measurements of noise exposure and SBP for 1 high-exposure worker and 1 low-exposure worker is shown in Figure 1. The 24-hour SBP profiles of the high-exposure worker were consistently higher than those of the low-exposure worker. For both workers, the variation in SBP was more profound during the working period (8:00 AM–4:30 PM) than either the off-duty period (4:30 PM–11:00 PM) or the sleep period (11:00 PM–8:00 AM).

During the study subjects' 24-hour monitoring periods, we obtained a total of 477 available blood pressure measurements and 1785 personal noise readings, including 1765 segments of 5-minute Leq during the working period and 20 TWA data during the off-duty period. Data completeness was 87% (1785 of 2060) for noise exposure measurements and 61% (477 of 780) for blood pressure measurements. The missing data averaged at 14 (range, 6–24) for noise measurements and 16 (range, 7–24) for blood pressure measurements. Table 2 summarizes SBP, DBP, and personal noise exposure measured over 24 hours, the 8.5-hour working period, the 6.5-hour off-duty period, and the 9-hour sleep period for workers with high and low noise exposure during the study. As expected, the full-shift TWAs (Leq) of high-exposure workers (85 ± 8 dBA; mean $\pm$ standard deviation) were significantly higher than those of low-exposure workers during the working period (59 ± 4 dBA). The 15 high-exposure workers' full-shift TWAs (Leq) were also

Fig. 1. Measurements of noise exposure and systolic blood pressure in the 2 different cases. * One single time-weighted-average noise level obtained for each subject during the off-duty period (4:30 PM–11:00 PM).

comparable to their environmental noise levels measured in October 2000 (79–110 dBA). By contrast, the full-shift TWAs (Leq) during the off-duty period had no significant difference between the high- and low-

exposure groups (61 ± 7 dBA vs. 56 ± 12 dBA).

The distribution of the current exposure showed that the full-shift TWAs (Leq) were all above 70 dBA for the 15 high-exposure workers at

work during the study. Moreover, there were approximately 90% measurements above 75 dBA, 49% measurements above 85 dBA, and 9% measurements above 95 dBA. By contrast, approximately 99% of the

TABLE 2

Mean Values $\pm$ Standard Deviations of SBP, DBP, and Personal Noise Exposure Over 24-Hour, Working Time, Off-Duty Time and Sleep Time in the High- and Low-Exposure Groups

	Working Time (8:00 AM–4:30 PM)	Off-Duty Time (4:30 PM–11:00 PM)	Sleep Time (11:00 PM–8:00 AM)	24-Hour Average
SBP (mm Hg)				
High-exposure group (no.)*	127 $\pm$ 24 (125)	122 $\pm$ 16 (112)	110 $\pm$ 16 (121)	120 $\pm$ 21 (358)
Low-exposure group (no.)	108 $\pm$ 17 (52)	114 $\pm$ 15 (41)	100 $\pm$ 16 (26)	108 $\pm$ 17 (119)
P value†	<0.01	0.11	0.01	<0.01
DBP (mm Hg)				
High-exposure group (no.)	75 $\pm$ 20 (125)	73 $\pm$ 12 (112)	64 $\pm$ 11 (121)	71 $\pm$ 16 (358)
Low-exposure group (no.)	68 $\pm$ 13 (52)	70 $\pm$ 16 (41)	62 $\pm$ 13 (26)	67 $\pm$ 14 (119)
P value†	0.05	0.05	0.21	0.02
Personal noise (dBA)				
High-exposure group (no.)	85 $\pm$ 8 (1301)	61 $\pm$ 7 (15)	NA	NA
Low-exposure group (no.)	59 $\pm$ 4 (464)	56 $\pm$ 12 (5)	NA	NA
P value‡	<0.01	0.21		

* Number of measurements for SBP, DBP, and personal noise exposure.

† Linear mixed-effects regression models adjusted for age, employment duration, body mass index, smoking, drinking alcohol, and hypertension family were used to test the between-group differences.

‡ t test was used to test the difference between the 2 exposure groups.

SBP, systolic blood pressure; DBP, diastolic blood pressure.

Fig. 2. The transient effect of noise exposure on blood pressure in the high-exposed group by linear mixed-effects models after adjusting for age, employment duration, body mass index, smoking, drinking alcohol, and hypertension in the family. * Marginally significant ($P = 0.07$).

TABLE 3

The Association Between Occupational Noise Exposure and Sleep-Period Blood Pressure

Models	SBP (mm Hg)		DBP (mm Hg)	
	β ($\pm$ Se β)	P Value	β ($\pm$ Se β)	P Value
High exposure vs. low exposure	16 ($\pm$ 6)	0.02	6 ($\pm$ 5)	0.22
Hypertension family (yes/no)	13 ($\pm$ 5)	0.02	5 ($\pm$ 4)	0.25
Employment duration (years)	2 ($\pm$ 1)	0.01	1 ($\pm$ 1)	0.12
Body mass index (kg/m ²)	2 ($\pm$ 1)	0.12	2 ($\pm$ 1)	0.06
Alcohol (yes/no)	1 ($\pm$ 5)	0.88	4 ($\pm$ 4)	0.36
Age (years)	-1 ($\pm$ 1)	0.21	0 ($\pm$ 1)	0.55
Smoking (yes/no)	-1 ($\pm$ 6)	0.87	-1 ($\pm$ 5)	0.80

SBP, systolic blood pressure; DBP, diastolic blood pressure.

full-shift TWAs (Leq) were measured between 50 and 70 dBA for the 5 low-exposure workers. These distributions in the full-shift TWAs (Leq) indicated that the high and low noise-exposure groups in our subjects could be classified by an occupational noise exposure at 70 dBA. In fact, the full-shift TWAs (Leq) were mostly greater than 75 dBA for the high-exposure group and were all less than 65 dBA for the low-exposure group.

The high-exposure workers' SBP were significantly higher than the low-exposure workers' ones by averaging 12 mm Hg over 24 hours, 19 mm Hg over the working period, and 9 mm Hg over the sleep period. The high-exposure workers' DBP were also significantly higher than the low-exposure workers' ones by averaging 4 mm Hg over 24 hours and 7 mm Hg over the working-period. Over the off-duty period, the DBP was marginally different ($P = 0.05$),

but the SBP was not different ($P = 0.11$) between the high- and low-exposure groups. Over the sleep time, the DBP was not different between the high- and low-exposure groups either.

The concurrent measurements of noise exposure and blood pressure during the working period among the high-exposure workers were used to estimate the transient effect of noise exposure. Figure 2 shows the SBP and DBP changes by various time-lagged noise exposure after adjusting for age, employment duration, BMI, smoking, drinking, and family hypertension history in the linear mixed-effects models. The mixed-effects models showed workers' SBP and DBP increased transiently at 30 and 60 minutes and returned to base-lines at 120 minutes after noise exposure at work. Our models showed that there was a marginally significant increase of 1 mm Hg SBP at 60-minute lag for the 1-dBA increase in noise exposure for autoworkers with occupational exposure between 70 and 108 dBA ($P = 0.07$).

The association between occupational noise exposure and sleep-period blood pressure is summarized in Table 3. Based on a dichotomous noise exposure variable (high vs. low), our regression models showed that occupational noise exposure was significantly associated with SBP ($P = 0.02$) but not associated with DBP ($P = 0.22$) after controlling other risk factors. Workers with TWA occupational noise exposure of 85 ± 8 dBA had 16 ± 6 mm Hg increments in sleep-time SBP compared with workers with TWA occupational noise exposure of 59 ± 4 dBA. In addition, we also identified that sleep-time SBP were significantly increased for workers with family hypertension history and longer employment duration ($P < 0.05$). Although there was a significant correlation between age and employment duration ($r = 0.883$, $P < 0.01$), we found that the interaction term of these 2 variables was not significant when it was added to our regression models ($P = 0.10$).

Discussion

We demonstrated that using recently available personal monitoring instruments to measure individuals' noise exposure and blood pressure continuously could reveal the transient and sustained effects of occupational noise exposure on blood pressure. Our study showed that 24-hour continuous measurements of blood pressure were key to reveal the effects of noise on blood pressure. There was a considerable discrepancy between 2 blood pressure data of Table 1 and Table 2. There were explanations for this seeming discrepancy. The values in Table 1 were obtained as single measurements by using a nonsystematic protocol in a clinic setting. In contrast, the present study collected repeated measurements over a span of 24 hours on each individual using a systematic protocol and different measurement instruments. Our study showed that the ambulatory monitoring technique was able to identify between-group

differences in blood pressure that were not evident with conventional clinical measurements, including identification of subjects with prehypertension (SBP in 120–139 mm Hg) for the high-exposure group and normal blood pressure status (below 120 mm Hg) for the low-exposure group, respectively. Accordingly, the long-term effect of noise was better evaluated by using 24-hour blood pressure measurements. In addition, the investigation on acute effect of noise became feasible because continuous blood pressure was measured concurrently with noise exposure in this study.

Our findings suggested the transient effect of increasing SBP could be induced by noise exposure at lower noise levels and shorter time lags. Previous studies found that industrial noise exposure above 85 dBA had a transient effect of increasing SBP.^{13,14} Our study reported such a transient effect could also occur at noise levels as low as 75 dBA. Our study also found the transient effect of noise exposure on SBP had the lagged effect maximally at 1 hour, which was shorter than the 2 or 3 persistent hours reported in a previous study.¹⁴ However, such transient effect can be overestimated by lacking information about workers' activities during the ambulatory blood pressure monitoring period such as heavy dynamic exercise during working hours and during off-duty time.

Increasing sleep-time SBP by TWA occupational noise exposure at 85 ± 8 dBA indicated that noise exposure could also have a sustained effect on blood pressure because noise exposure differed significantly by 26 dBA during working time but was not significantly different during the off-duty period between the high-exposure and the low-exposure groups. By contrast, Babisch et al. reported inconsistent findings of chronic effect for people with lower noise exposure levels, 70 dBA, and smaller difference in noise exposure levels between high- and low-

exposed groups, 15 dBA.^{20,21} Such comparisons indicate there could be threshold exposure levels for noise-induced hypertension.

However, we might overestimate the sustained effect of noise exposure on SBP without excluding all potential confounders completely in this study. We included some but not all important risk factors of hypertension that might lead to confounding the sustained effect of noise exposure on blood pressure. Uncontrolled important risk factors of hypertension included serum cholesterol (high-density lipoprotein and low-density lipoprotein), blood glucose, uric acid, creatinine, potentially ototoxic drugs, salt in diet, number of cigarettes smoked per day, and daily alcohol intake and coffee consumption among our study subjects. For example, the high-exposure workers were almost twice as likely to be smokers, although the difference in smoking prevalence was not statistically significant. Therefore, the sustained difference in SBP between 2 exposure groups could still possibly be contributed by lifestyle differences.

Our findings also support that people with longer employment durations and with a family hypertension history are associated with abnormal blood pressure reported in previous studies.^{6–10} Although our statistical model has already controlled age, it should be noted that such analysis might not be sufficiently robust with 20 subjects. Therefore, the possibility of confounding effect on hypertension by age could not be completely excluded.

Two possible mechanisms in the genesis of hypertension resulting from noise exposure are through sympatheticotonia-induced endothelial lesion and stress-induced hormone release. For the former 1, acute noise exposure can activate sympathetic reflex immediately,^{11,22,23} then accelerate the development of structural changes in the cardiovascular system, and finally induce a sustained elevation of blood pres-

sure.^{4,11,23} On the other hand, several studies have shown higher stress hormone levels in relation to chronic noise exposure.^{24,25} A stress-related activation of neuroendocrine pathways through high cortisol niveaus can give rise to a metabolic syndrome with hypertension and other cardiovascular effects as end points. Both pathways are reasonable approaches in the genesis of hypertension resulting from noise exposure.

This is a panel study, and possible limitations with a cross-sectional design often are missing in this situation. Weaknesses with a cross-sectional design with lack of exposure information and temporality problem with different confounders were some main topics discussed in past reviews.^{26,27} For example, we have greatly minimized temporality problem by measuring personal noise exposure and ambulatory blood pressure continuously instead of using 1 single measurement of noise exposure and resting blood pressure to investigate the transient and sustained effects of noise exposure on SBP and DBP in this study.

Although the number of study subjects was small, the numbers of available measurements of noise exposure and blood pressure were relatively large. There were 477 available blood pressure measurements and 1785 personal noise readings during the 20 study subjects' 24-hour monitoring period. Accordingly, a small number of study subjects with a large time series data sample size would not affect the determination of transient effect but might limit the extrapolation of the sustained effect observed among autoworkers in this study to the workers in other industrial settings.

One limitation of this study is the problem of missing data, which existed in blood pressure measurements but not in noise exposure measurements. Because the DynaPulse system is based on measuring a subject's arterial waveform to report blood pressure, it is sensitive to the subject's arm movements. The heavy

dynamic exercises during working hours and frequent changes of body posture during the sleep period are 2 possible reasons of missing data for blood pressure measurements. Differences in these activities between subjects resulted in inconsistent distribution of missing data of blood pressure across different cells in Table 2. The ratios of available measurements between high- and low-exposure groups were 4.7 (121:26) in the sleep time, 2.7 (112:41) in the off-duty time, and 2.4 (125:52) in the working time. The comparisons of blood pressure between different exposure groups and working periods might be compromised by the difference in the availability of data between subjects. The mixed-effects models were constructed to analyze data with different observational length data between study subjects. Therefore, difference in the availability of data between subjects would not compromise the validity of our statistical analyses in determining the transient and sustained effects of noise exposure on blood pressure.

The other limitation of this study is the truncated noise exposure measurement between 30 and 50 dBA for the high-exposure group during the off-duty period. This is because it was impractical for us to adjust the dosimeter's ranges of our study subjects after their working periods in our study. Ideally, the high-exposed subjects should have been provided separate dosimeters for our off-duty sampling with the measurement range that was identical to that for the low-exposed subjects. However, there is likely to be little, if any, effect from such measurement bias on the primary data analysis, which focuses on the relationship between blood pressure and work-period noise exposure. It should be noted that our choice of the practical approach could introduce a potential bias if the truncated data during the off-duty period were used for our primary data analysis. Therefore, we recommend that future investigators

be aware of this potential bias and consider using different sets of dosimeters for measuring noise levels during working and off-duty periods.

Although the weakness of the low number of subjects and potential differences between the groups of importance for the development of elevated blood pressure were not completely excluded, our findings generally supported the conclusion that there was a marginally transient effect and significantly sustained effect in normotensive adult autoworkers at occupational noise exposure above 70 dBA. For the transient effect, a 1-mm Hg increase in SBP was marginally associated with a 1-dBA increase in noise exposure. For the sustained effect, a 16-mm Hg increase in SBP was significantly associated with daily exposure to 85 dBA TWA at work. Such findings are particularly important for an aging working population because blood pressure control was key to promoting adult workers' health. Accordingly, the current noise exposure guidelines of the American Conference of Governmental Industrial Hygienists (ACGIH)²⁸ and the International Organization for Standardization (ISO),²⁹ which is based solely on hearing loss, might not be adequate for worker protection. We, therefore, recommend conducting further studies on blood pressure in relation to occupational noise and including the blood pressure effects in the future revision of noise exposure guidelines.

This was the first time that personal noise exposure was measured and the audiometric evaluation results were analyzed for the high-exposure group in this company. We found the full-shift TWA noise exposure exceeded the 8-hour TWA of 85 dBA, which required employers to provide hearing protection devices (HPD) to workers in Taiwan. We also found that workers in the high-exposure groups could have noise-induced hearing loss besides noise-induced blood pressure increases. After we found out these situations in the workplace, a hearing-conservation program has been recom-

mended to the company immediately. We are still working closely with the company to evaluate this conservation program's effectiveness of protecting worker's health.

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