Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers

T Mizoue, T Miyamoto, T Shimizu

Background: Evidence has accumulated concerning the adverse effects of smoking on hearing acuity, but it is not clear whether smoking modifies the association between exposure to noise and hearing loss.

Aims: To examine the synergistic effect of these variables on hearing.

Methods: Data used were derived from periodic health examinations for 4624 steel company workers in Japan and included audiometry testing and information on smoking habits. Occupational exposure to noise was determined based on company records. Logistic regression was used to examine the dose-response association between smoking and hearing loss. The Cochran-Mantel-Haenszel method was used to calculate the prevalence rate ratio (PRR) of hearing loss for each combination of smoking and noise exposure factors, taking non-smokers not exposed to occupational noise as a reference. The interaction between smoking and noise exposure was assessed using a synergistic index, which equals 1 when the joint effect is additive.

Results: Smoking was associated with increased odds of having high frequency hearing loss in a dose-response manner. The PRR for high frequency hearing loss among smokers exposed to occupational noise was 2.56 (95% CI 2.12 to 3.07), while the PRR for smokers not exposed to noise was 1.57 (95% CI 1.31 to 1.89) and the PRR for non-smokers exposed to noise was 1.77 (95% CI 1.36 to 2.30). The synergistic index was 1.16. Smoking was not associated with low frequency hearing loss.

Conclusions: Smoking may be a risk factor for high frequency hearing loss, and its combined effect on hearing with exposure to occupational noise is additive.

Methods

Data source

Data used were derived from periodic health examinations conducted during fiscal year 1999 on workers in a factory of a major Japanese steel company. In Japan, audiometry tests are provided for all employees, irrespective of noise exposure at work, as part of mandatory health examinations. In addition, governmental guidelines recommend the provision of full band auditory examinations for workers exposed to high levels of noise; the corresponding workplaces, where noise levels are expected to exceed 85 dB, are specified in the guidelines. Following these regulations and guidelines, the present company provides a pure tone air conduction audiometry tests at frequencies of 1000 Hz and 4000 Hz for workers not subject to significant noise exposure at work, and additionally at frequencies of 500 Hz, 2000 Hz, and 8000 Hz for those working in noisy environments. Hearing acuity was tested before work of the day by a trained nurse, using an audiometer (DA-24, Dana Japan Co. Ltd) in a booth designed specifically for the test. Health related lifestyle information, including smoking data, was obtained at the time of the health examinations, using a self administered questionnaire. Queries on smoking habit included smoking experience (never smoked, quitted, currently smoking) and, for quitters and current smokers, the number of cigarettes smoked per day and the duration of smoking in a year. If multiple examinations were conducted on a given worker during the study period, the results of the first examinations were used. As periodic health examinations are mandatory for all employees, a participation rate of nearly 100% may be expected for all workers, except for those on extended sick leave during the study period.

Exclusions

Female subjects were excluded, because of the low percentage of smokers among this group (4%). A retirement age of 60 is applied to most workers in the company, leaving 5459 male workers.

Abbreviations: CI, confidence interval; PRR, prevalence rate ratio
subjects aged less than 61 years. Further excluded from this group were individuals without auditory examination results (n = 225), those who provided incomplete information on their smoking histories (n = 3), and those who had quit smoking (n = 646), with some overlap among these exclusion criteria. This left 4624 subjects for the present analysis.

**Definition of exposure and outcome**

Non-smokers were defined as those who had never smoked in their lifetime. Workplace exposure to noise was determined according to the company records of workers who were selected for special auditory examination for the year. The present company prepares the examination if the subject is a smoker and/or current noise exposure; B, smoking) was assessed using the synergy index S, defined as:


in which \(\sim\) denotes the absence of a factor and RR denotes the rate ratio relative to the rate for \((A, B, \sim)\). The ratio S will be equal to unity under additivity, and will exceed unity if the observed rate ratio for joint exposure exceeds the magnitude predicted based on the sum of the rate ratios. All the above calculations were performed using SAS software.

**RESULTS**

Fifty six per cent of male workers in the factory were current smokers and 29% were working in environments with potentially high levels of noise. Age and employment tenure were highly correlated each other (r = 0.95; p < 0.001). As table 1 shows, smokers were more likely to be exposed to noise at work, and were somewhat older than non-smokers. As table 2 shows, age was a strong determinant of hearing loss for all combinations of occupational noise exposure and smoking; as age increased, the prevalence of subjects with hearing loss increased markedly. The difference in hearing loss between frequencies of 1000 Hz and 4000 Hz, separately. Hearing loss was calculated according to age group (<40 years, 40–49 years, 50–60 years), occupational noise exposure, and smoking status. The age category was initially defined by 10 year intervals, but the first three categories (<20 years, 20–29 years, 30 to <40 years) were then combined because the corresponding prevalence of study defined hearing loss was no greater than 3% for both frequency levels. Logistic regression was used to examine the dose-response relation between smoking and hearing loss according to occupational noise exposure, controlling for age as a continuous variable. For this analysis, smoking categories were defined as follows: non-smokers, smokers consuming 1–14 cigarettes per day, smokers consuming 15–24 cigarettes per day, and smokers consuming 25 cigarettes or more per day. The Cochran-Mantel-Haenszel method was used to calculate the prevalence rate ratio (PRR), stratified by the above mentioned age groups, of hearing loss with a 95% confidence interval (CI) for each combination of smoking and noise exposure factors, taking non-smokers not exposed to occupational noise as a reference group. The interaction between the two factors (A, noise exposure; B, smoking) was assessed using the synergy index S, defined as:


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**Table 1** Characteristics of the study subjects according to occupational exposure to noise and smoking status

<table>
<thead>
<tr>
<th></th>
<th>Non-smoker (n=1693)</th>
<th>Smoker (n=2931)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td><strong>Occupational noise exposure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1321</td>
<td>78</td>
</tr>
<tr>
<td>Yes</td>
<td>372</td>
<td>22</td>
</tr>
<tr>
<td>Age y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;39</td>
<td>680</td>
<td>40</td>
</tr>
<tr>
<td>40–49</td>
<td>671</td>
<td>40</td>
</tr>
<tr>
<td>50–60</td>
<td>342</td>
<td>20</td>
</tr>
</tbody>
</table>

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**Table 2** Prevalence (numerator/denominator) of hearing loss* according to age, occupational exposure to noise, and smoking in 4624 male workers in a steel factory in Japan, 1999

<table>
<thead>
<tr>
<th>Occupational noise exposure</th>
<th>Smoking status</th>
<th>Age group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-smoker</td>
<td>39 y</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1000 Hz</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>Non-smoker</td>
<td>0.7 (4/560)</td>
</tr>
<tr>
<td></td>
<td>Smoker</td>
<td>1.0 (6/578)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>Non-smoker</td>
<td>0.8 (1/120)</td>
</tr>
<tr>
<td></td>
<td>Smoker</td>
<td>2.4 (8/329)</td>
</tr>
<tr>
<td>4000 Hz</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>Non-smoker</td>
<td>1.6 (9/560)</td>
</tr>
<tr>
<td></td>
<td>Smoker</td>
<td>1.4 (8/578)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>Non-smoker</td>
<td>0.8 (1/120)</td>
</tr>
<tr>
<td></td>
<td>Smoker</td>
<td>2.7 (9/329)</td>
</tr>
</tbody>
</table>

*Hearing threshold: 1000 Hz, >25 dB; 4000 Hz, >40 dB.
smokers and non-smokers was small at 1000 Hz, while it was large at 4000 Hz. Smokers exposed to noise at work showed the highest degree of hearing loss, while non-smokers working without significant noise exposure had the lowest, with individuals subject to either factor (smoking or exposure to noise) situated between these groups. For the age groups of 40–49 years and 50–60 years, hearing loss among smokers exposed to occupational noise can be estimated based on the additive model using the following equation:

\[ P_{sn} = P_{o} + (P_{s} - P_{o}) + (P_{n} - P_{o}) \]

where \( P_{sn} \) is the prevalence of hearing loss at 4000 Hz for smokers exposed to occupational noise, \( P_{s} \) represents hearing loss among smokers not exposed to noise, and \( P_{n} \) represents hearing loss among non-smokers exposed to noise. As table 3 shows, a dose-response relation was observed between smoking and high frequency hearing loss; odds ratio increased with higher numbers of cigarettes smoked per day. The trend association was more evident among those who had worked in noisy environments. Adjustment of alcohol drinking did not materially change the results (data not shown). Low frequency hearing loss was not associated with smoking. As table 4 shows, the PRR of hearing loss was estimated for each combination of smoking and noise exposure factors, using non-smokers not exposed to occupational noise as a reference group. Smoking and noise exposure each increased PRR for non-smokers not exposed to occupational noise as a reference group. Smoking and noise exposure for each worker was determined based on the occupational noise exposure for each worker was determined based on the occupational noise exposure for each worker was determined based on the occupational noise exposure for each worker was determined based on the occupational noise exposure for each worker.

## DISCUSSION

In the present study of workers in a Japanese steel factory, smoking was significantly associated with an increased risk of high frequency hearing loss, and the combined effect of smoking and exposure to occupational noise was comparable to the sum of the independent effects of each factor.

Inconsistent findings have been reported on the association between smoking and hearing loss. The present finding of a dose-response relation at 4000 Hz indicates that smoking is a risk factor for high frequency hearing loss. Although the mechanism of the adverse effects of smoking on hearing is not clear, it is hypothesised that smoking may damage hair cells though an ischaemic mechanism—that is, by reducing blood flow to the cochlea or by increasing carboxyhaemoglobin.\(^\text{3,11}\) Experimental studies have found nicotinic-like receptors in hair cells, implying direct ototoxic effects of nicotine on hair cell function.\(^\text{12}\) In contrast, we observed no association between smoking and low frequency hearing loss, a finding similar to the previous results by Nakanishi et al, where a close association was determined between smoking and hearing loss at high frequencies, but not at low frequencies. Inner ear cells responsible for high frequency hearing acuity might be easily damaged through ischaemic mechanisms, as they are located at the end of nutrient arteries. In addition, a study showed that high frequency hearing loss was associated with high shear blood viscosity,\(^\text{13}\) which may be caused by smoking. The differential impact of smoking on hearing according to frequency may partly explain the inconsistency of results among studies that linked smoking with average hearing thresholds at a variety of frequencies.

Prince and Matanoski found that the combined effect of smoking and noise was greater than the expected independent effects.\(^\text{15}\) In the present study, however, although smokers working in environments with significant noise had the highest risk of hearing loss at 4000 Hz, the prevalence and the PRR of hearing loss was nearly equal to values estimated based on an additive model. Furthermore, the synergistic index of 1.16 also supports the hypothesis that the joint effects of noise exposure and smoking on hearing are additive. In other words, smoking does not enhance the effect of noise on hearing but instead acts independently.

The advantages of the present study over previous studies are as follows. First, occupational noise exposure for each worker was determined based on company records. Second, the large sample size, together with a high participation rate, allowed us to evaluate the risk of hearing loss among all combinations of smoking and occupational noise exposure factors. However, the present study had the following limitations. First, the cross-sectional nature of the study limits the extent of causal inferences that may be drawn from the findings. However, very few workers were relocated as a result of hearing loss; thus, the selection bias arising from such a mechanism should be minimal. Furthermore, the finding of a dose-response relation between
The present results did not show any synergistic effect between smoking and exposure to noise, but this finding does not negate the importance of smoking in terms of protecting workers from hearing loss. As measures reducing worker exposure to noise have been progressively introduced in occupational settings, the relative importance of other aetiological factors (such as smoking) should increase. In fact, among the present subjects in the steel factory, hearing loss at 4000 Hz among smokers without significant noise exposure was comparable to hearing loss among non-smokers working in noisy environments. In addition, because blue collar workers in general smoke more than office workers and are more likely to be exposed to noise at work, they form a high risk population in terms of hearing impairment. From a public health perspective, multidisciplinary measures minimising exposure to noise and discouraging smoking should be effected for such high risk groups, to help maintain hearing acuity as workers age.

Conclusions
The present finding indicates, together with previous findings, that smoking is an independent risk factor for high frequency hearing loss. Furthermore, the combined effects of smoking and exposure to noise on hearing may operate in an additive manner. In contrast, smoking was not associated with low frequency hearing loss. Hearing loss among workers can therefore be viewed as a work related and lifestyle related impairment, necessitating a coordinated remedial approach.

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REFERENCES