Occupational Exposure to Noise and Mortality From Acute Myocardial Infarction

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Background: Exposure to noise is highly prevalent in the workplace, and an etiologic association with cardiovascular disease has been hypothesized. Although there is evidence of hypertension among noise-exposed workers, evidence of heart disease has been less conclusive.

Methods: We identified a cohort of 27,464 blue-collar workers from 14 lumber mills in British Columbia who worked at least 1 year between 1950 and 1995 and who were followed up over the same period. Cumulative noise exposure was quantitatively assessed. Vital status was ascertained from the Canadian Mortality Database. We estimated standardized mortality ratios using the general population as referents, and we estimated relative risks using an internal low-exposure group as controls. To examine acute effects of noise, we assessed relative risks during subjects' working years in lumber mills. Because of the possibility of exposure misclassification as a result of hearing-protector use, we investigated a subgroup that had been employed before widespread use of protectors.

Results: During the follow-up period, 2510 circulatory disease deaths occurred. Relative risks for acute myocardial infarction mortality were elevated in the full cohort, with a stronger association in the subgroup without hearing protection. There was an exposure-response trend, with a relative risk in the highest exposed group of 1.5 (95% confidence interval = 1.1–2.2). The highest relative risks (2.0–4.0) were observed during subjects' working years. Smoking did not appear to confound these associations.

Conclusions: Chronic exposure to noise levels typical of many workplaces was associated with excess risk for acute myocardial infarction death. Given the very high prevalence of excess noise exposure at work, this association deserves further attention.

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Noise has been called the most ubiquitous of hazardous occupational exposures.[1] In the United States alone, 30 million workers are exposed to noise levels that are damaging to their hearing,[2] and worldwide the prevalence of overexposure to noise in the workplace is likely increasing.[3]

An association between exposure to noise and sensorineural hearing loss has been recognized for several centuries.[4] Recently, however, there has been a growing body of evidence that noise might also be associated with adverse health effects in body systems other than the ear, including ischemic heart disease.[5] Given the severity and prevalence of this disease and the high prevalence of noise exposure, this is potentially an important public health issue.

The link between noise and cardiovascular disease is thought to be stress-mediated. It is hypothesized that the normally transient physiological stress responses to noise of the sympathetic nervous and neuroendocrine systems become pathogenic when chronically or repeatedly activated. Thus, temporary increases in blood pressure might, through structural autoregulation, lead to permanent elevations and then hypertension; repeated oversecretion of cortisol in response to noise exposure may lead to visceral fat accumulation and to insulin resistance.[6,7]

The hypothesized model has been examined in a number of ways. In animals, noise consistently evokes the presumed physiological responses. Similarly consistent responses are seen in human experiments and, although people appear to habituate quickly in the laboratory, habituation is thought to be incomplete in nonexperimental settings.[8,9]

A large number of epidemiologic investigations, primarily focusing on blood pressure changes, have been published.[5] The results have been inconsistent, and the studies have been criticized for having numerous design limitations.[10] Overall, however, the findings point to small increases in systolic and diastolic blood pressure, and an increased risk of hypertension. Large longitudinal follow-up studies of other cardiovascular end points such as ischemic heart disease have been much less common.[11–14] The findings of these studies have also been equivocal, with relative risks ranging from below 1.0 to almost 4.0. Reports of the studies

noted many limitations (including small study size, inadequate control of confounding, and poor exposure assessment) as possible explanations for their weak associations and inconsistencies.[11,15] A particular problem related to the study of occupational noise-related disease is the widespread use of hearing-protective devices (ear "plugs" or "muffs") by those exposed to noise. Exposure reduction resulting from hearing-protection device use is not captured by personal noise dosimetry, and this deficiency may lead to systematic overestimation of exposure levels where such devices are used.

In this article, we report the findings of a large retrospective study of chronic exposure to very high levels of occupational noise in association with acute myocardial infarction mortality. This cohort comprised blue-collar lumber mill workers who were followed for up to 45 years. Because of the large size of this cohort and the quality of employment records and of exposure data (including adjustments for hearing-protector use), we were able to address several weaknesses of earlier studies.

METHODS

Study Design and Subject Selection

We used records from 14 large softwood lumber mills in the province of British Columbia, Canada, to retrospectively identify 27,464 hourly-paid production and maintenance workers who had worked at least 1 year between January 1, 1950 and December 31, 1995. All mills had similar technology, processes, and product lines. The mills had been selected previously, based on their size and completeness of historical personnel records, for a study of cancer outcomes.[16] We identified deaths by probabilistic linkage to the Canadian Mortality Data Base (Statistics Canada, Ottawa). Vital status ascertainment through this database is 97.6% complete for deaths in Canada.[17] Vital status determination for subjects classified as alive, but who were not still actively employed, included pension and motor vehicle records, personal inquiries at union halls, and linkage to province-wide medical insurance registration data through the British Columbia Linked Health Data Project.[18]

Follow up began January 1, 1950 or on meeting the 1-year employment inclusion criteria, whichever was later. Follow up ended at death, or December 31, 1995. Lost-to-follow up was handled in 2 ways. For subjects for whom no social insurance number was known and who had never been linked to any external data source following enumeration, we ended follow up at their date of last employment (4% of all subjects). This is a typical procedure, because it requires no unverifiable assumptions.[19] However, individuals for whom a social insurance number was known, or who had been linked to an external data source following enumeration, were assumed to be alive at December 31, 1995 because they would have a high likelihood of linking to the national mortality database if deceased (12% of all subjects).

The study protocol was reviewed and approved by the Research Ethics Board of the University of British Columbia.

Exposure Assessment

Exposure assessment for retrospective studies is difficult, because of the paucity of historical exposure data and the absence of standardized methods. The extensive exposure assessment phase of this study will be published in detail elsewhere (Davies HW, unpublished data, 2004). Briefly, we collected 1900 full-shift personal noise dosimetry measurements for the participating mills. In addition, we gathered information through interviews and site visits and from building plans that described potential determinants of both current and past noise exposure. These data were used to build empiric regression models with which we predicted exposure levels for all jobs and time periods, including those lacking any exposure measurements. Estimates for over 3800 unique mill/job-title/time-period combinations were merged with subjects' work histories.

Two exposure metrics were used. Duration of exposure was defined as the number of years worked in jobs in which noise levels exceeded a specific threshold. Because the quantitative relation between noise and ischemic heart disease was not known, thresholds of is 85 A-weighted decibels (dB(A)), 90 dB(A), and 95 dB(A) were all examined. The common regulatory exposure limit is 85 dB(A), which is similar to the level associated with use of a power hand tool such as an electric drill. We also calculated cumulative exposure as a product of exposure intensity and duration. The resulting unit of "dB(A)-year" has a logarithmic scale [dB(A)-year = 10 log (sound × time)]; consequently, a doubling of either sound level or time results in an increase of approximately 3 dB(A).

Accounting for the Use of Hearing Protection

Individual-level data regarding hearing-protector use were not available. To examine the effect of exposure misclassification resulting from such use, we created a subgroup of 8668 workers who terminated their employment before June 30, 1970 (the "subgroup without hearing protection"). Interviews with senior mill workers indicated that before this date, there was minimal use of hearing protection in participating mills. In addition, we repeated analyses on the full cohort using exposure measurements arithmetically adjusted for hearing protector use. Adjustments were calculated from real-world hearing-protection efficacy data[20] and estimates obtained from annual hearing test data of the prevalence of hearing-protector use among BC sawmill workers.

Health Outcomes

We examined all ischemic heart disease mortality (International Classification of Diseases, 9th Revision [ICD-9] codes 410–414.9 and 429.2), acute myocardial infarction

(ICD revision 9 codes 410–410.9), and ischemic heart diseases other than acute myocardial infarction (ICD-9 codes 411–414.9 and 429.2) as recorded in the national mortality database.

Statistical Analyses

We used PC Life Table Analysis System to calculate standardized mortality ratios (SMRs).[21] STATA version 7 (STATA Corp., College Station, TX) was used for all other statistical analyses. All person-years-at-risk calculations were begun 1 year after first employment. Mortality referent rates were based on the general British Columbia population for the years 1950 to 1995 (Statistics Canada, Ottawa). We calculated 95% confidence intervals (CIs) for the SMRs assuming that the observed effect followed a Poisson distribution. Exposure-response relations within the lumber mill cohort were examined using Poisson regression, with adjustment for age, calendar year, and South Asian ethnicity. Age and calendar year were entered in 10-year categories and pooled if cells contained fewer than 5 deaths. We conducted tests for linear trend by entering the exposure category as a continuous variable in Poisson regression analyses. South Asian subjects were known to be primarily Sikh and therefore likely to differ substantially from the rest of the cohort with respect to smoking and alcohol habits. This ethnic group has also been shown to be at increased risk for cardiovascular disease.[22] South Asians were identified based on their names. This technique was validated in a subcohort of 1959 subjects employed in 1979 (the peak year of employment in the industry) and interviewed in 1996–1997.[23] Sensitivity of this approach was 99% and specificity was 100%.

The temporal relation between noise exposure and outcome was examined by restricting follow up to subjects' working years in some analyses. Follow up was truncated at death, or 1 month after termination of employment (to capture subjects who terminated employment as a result of ill health and died shortly thereafter), whichever occurred first. These restricted analyses included the entire cohort and did not result in changes to cumulative exposures achieved by subjects.

Smoking

Although individual-level data were not available for all subjects, we compared smoking habits in the 1996–1997 interview subset with the general British Columbia population (based on a random sample of 18,030 respondents)[24] and among different noise-exposure groups within the interview subset. We also examined the relative risk for lung cancer among the noise-exposure groups because if smoking were confounding a relation between noise and ischemic heart disease, we would expect to see increased mortality rates with increasing noise in other smoking-related diseases.

RESULTS

This study cohort was highly exposed to noise, with a mean full shift noise exposure of 92 dB(A). The mean age at entry into the cohort was 30 years, and mean follow-up duration was 24 years (Table 1). Mean duration of employment in the cohort was 10 years; the year first employed ranged from 1909 to 1994 (mean = 1964). There were a total of 5850 deaths in the full cohort, with a mean age at death of 66 years (interquartile range, 57–77). Although the subgroup without hearing protection represented only one third of the subjects, it included 60% of the deaths (3477) and therefore provided considerable statistical power. Mean age at death in the subgroup was 68 years (interquartile range, 59–79).

The SMR for deaths from all causes in the full cohort was below 1 (Table 2), as would be predicted for a healthy working population.[19] This healthy worker effect was not as pronounced for heart disease. SMRs for each disease in the subgroup without hearing protection were generally higher than in the full cohort, consistent with it being an older group in which the healthy worker effect is expected to diminish.

Patterns of total mortality by level of exposure were also consistent with the healthy worker effect (results not shown). Increasing duration of exposure above thresholds of 85, 90, and 95 dB(A), as well as increasing cumulative exposure, were associated with flat or reduced total mortality trends. Negative trends were stronger in the full cohort; attenuation in the subgroup without hearing protection is

TABLE 1. Demographic Characteristics for the British Columbia Lumber Mill Workers Cohort and a Subgroup of Lumber Mill Workers Not Using Hearing-Protection Devices

	Full Cohort (n = 27,464)	Subgroup Without Hearing Protection (n = 8668)
Age at entry (years); mean (25th–75th percentile)	30 (21–36)	33 (22–43)
Follow-up duration (years); mean (25th–75th percentile)	24 (17–32)	28 (20–40)
Duration of employment (years); mean (25th–75th percentile)	10.4 (2.4–16.2)	8.2 (2.4–10.8)
Year first employed; mean (25th–75th percentile)	1964 (1953–1975)	1952 (1948–1959)
South Asian ethnicity (%)	5.9	2.1

TABLE 2. Summary Standardized Mortality Ratios (SMRs) for Full Cohort and Subgroup not Using Hearing Protection

	Full Cohort (n = 27,464)		Subgroup Without Hearing Protection (n = 8668)	
Cause of Death*	**No. of Deaths**	**SMR (95% CI)**	**No. of Deaths**	**SMR (95% CI)**
All causes	5850	0.95 (0.93–0.98)	3477	1.0 (1.0–1.1)
Circulatory diseases	2510	0.98 (0.94–1.0)	1624	1.1 (1.0–1.1)
Ischemic heart disease	813	0.96 (0.90–1.0)	603	1.1 (1.0–1.2)
Acute myocardial infarction	907	1.0 (0.97–1.1)	517	1.1 (1.0–1.2)

Reference group is the general population of British Columbia. Adjusted for age, and calendar year.
*Based on ICD revision at time of death.

consistent with it being a more mature group (all members terminated employment before 1970).

Table 3 shows SMRs for acute myocardial infarction by duration of exposure at each of the 3 threshold levels. SMRs in the full cohort were close to 1 except for subjects exposed above 95 dB(A) for 20 or more years, among whom SMRs were slightly elevated. SMRs were higher in the subgroup without hearing protection, with similar elevations in the highest exposure groups. Lower relative risks in the full cohort are consistent with the use of hearing protection by cohort members; an unmeasured reduction in subjects' exposure to noise would result in low-exposed workers being misclassified as high, thus attenuating the risk estimates. Table 4 shows SMRs for acute myocardial infarction by cumulative exposure. Again, SMRs in the full cohort are all close to 1, whereas in the subgroup without hearing protection, SMRs increase with increasing exposure level, reaching 1.3 in those highest exposed.

Comparisons within the study population are shown in Figures 1 and 2; subjects exposed for less than 3 years were used as the reference. In the full cohort, there was a general pattern of increasing relative risk for acute myocardial infarction with increasing duration of exposure and increasing exposure threshold, but this was not consistent (Fig. 1A). The highest relative risk was 1.3 in workers exposed for 30 or more years above 95 dB(A). In the subgroup without hearing protection, however, the same general pattern existed and was much stronger, with a positive exposure-response relation at all threshold levels (Fig. 1B). Relative risks for those exposed for 20 or more years were approximately 1.3, 1.3, and 1.5 for

TABLE 3. Association of Deaths Resulting from Acute Myocardial Infarction and Duration of Noise Exposure Above Thresholds*

	Threshold 85 dB(A)			Threshold 90 dB(A)			Threshold 95 dB(A)		
Duration of Exposure	**Person-Years**	**No. of Deaths**	**SMR (95% CI)**	**Person-Years**	**No. of Deaths**	**SMR (95% CI)**	**Person-Years**	**No. of Deaths**	**SMR (95% CI)**
Full cohort (n = 27,464)									
<3 years	232,305	151	1.1 (0.94–1.3)	323,024	293	1.1 (0.95–1.2)	499,452	535	1.0 (0.92–1.1)
3–9 years	237,371	189	0.94 (0.81–1.1)	200,504	184	0.96 (0.83–1.1)	101,033	137	1.1 (0.88–1.2)
10–19 years	111,896	201	1.1 (0.94–1.2)	87,569	175	1.0 (0.88–1.2)	46,054	122	1.0 (0.84–1.2)
20–29 years	57,651	221	1.1 (0.93–1.2)	40,458	169	1.1 (0.95–1.3)	16,203	75	1.2 (0.92–1.5)
>29 years	28,589	145	0.98 (0.83–1.2)	16,258	86	1.0 (0.81–1.3)	5070	38	1.3 (0.92–1.8)
Subgroup without hearing protection (n = 8668)									
<3 years	104,531	126	1.2 (0.97–1.4)	135,219	204	1.1 (0.94–1.2)	201,444	354	1.1 (0.96–1.2)
3–9 years	94,803	150	1.1 (0.90–1.3)	77,724	129	1.1 (0.90–1.3)	30,815	69	1.1 (0.88–1.4)
10–19 years	30,083	108	1.1 (0.89–1.3)	22,903	98	1.2 (0.96–1.4)	10,315	57	1.3 (0.97–1.6)
>19 years†	17,050	133	1.2 (0.97–1.4)	10,621	85	1.2 (0.95–1.5)	3895	37	1.4 (0.98–2.0)

Reference group is the general population of British Columbia.
Adjusted for age and calendar year.
*Standardized mortality ratios (SMRs) for full cohort and subgroup not using hearing protection.
†20–29 years and ≥ 30 years combined as a result of small number of observed deaths.

TABLE 4. Association of Deaths Resulting from Acute Myocardial Infarction and Cumulative Noise Exposure*

	Full Cohort (n = 27,464)			Subgroup Without Hearing Protection (n = 8668)		
Cumulative Exposure (dB(A)-year)	**Person-Years**	**No. of Deaths**	**SMR (95% CI)**	**Person-Years**	**No. of Deaths**	**SMR (95% CI)**
<100.0	314,128	226	0.99 (0.88–1.1)	133,556	174	1.0 (0.89–1.2)
100.0–104.9	155,837	228	1.0 (0.89–1.2)	58,940	136	1.0 (0.88–1.2)
105.0–109.9	116,303	231	1.1 (0.95–1.2)	37,133	120	1.2 (1.0–1.5)
110.0–114.9	63,998	165	1.0 (0.89–1.2)	14,646	71	1.3 (1.0–1.6)
≥115	18,479	60	1.1 (0.82–1.4)	3071	19	1.3 (0.81–2.1)

Reference group is the general population of British Columbia.
Adjusted for age and calendar year.
*Standardized mortality ratios (SMRs) full cohort and subgroup not using hearing protection.

85, 90, and 95 dB(A) thresholds, respectively; *P* values for exposure–response trends were 0.04, 0.03, and 0.01, respectively. For each duration level, relative risks also showed an increasing trend with increasing threshold.

Similar results were seen for acute myocardial infarction risk and cumulative exposure (Fig. 2). Although there was no apparent association in the full cohort, a linear trend of increasing relative risk with increasing cumulative exposure was evident in the nonhearing-protected subcohort ($P = 0.001$), reaching 1.6 in those exposed at or above 115 dB(A)-year (the reference group was those exposed below 100 dB(A)-year).

We repeated the internal analyses on the full cohort after arithmetically adjusting for hearing-protector use. There was no change in the relative risks for acute myocardial infarction when considering duration of exposure. However, consistent increases in relative risks were found for the cumulative exposure metric; although excess risk was not as great as for the subcohort analyses (the highest relative risk was 1.3 at ≥115 dB(A)-year), there was a positive linear trend ($P = 0.02$).

Relative risks for other ischemic heart disease deaths (ICD-9 = 411–414, 429.2) were also elevated, but less strongly or consistently, with relative risks of 1.3–1.4 in those exposed for 20 or more years above 85 dB(A) (results not shown).

We found the highest relative risks occurred during subjects' working years, when they were presumably still exposed to noise. Relative risks of 2.0 to 4.0 were observed among workers exposed for 20 years or longer (Table 5). There were strong trends for increasing relative risk with increasing duration at all 3 noise levels.

We observed no appreciable differences in smoking status (ie, current, former, never) between the 1996–1997 interview subgroup and the general British Columbia population, after adjusting for age and South Asian ethnicity. Within the 1996–1997 interview subgroup, there were no major differences in duration of employment, cumulative exposure to noise (dB(A)-year), or duration of exposure above 95 dB(A) among subjects in the 3 smoking categories. Similarly, there was no difference in mean pack-years smoked for the different exposure categories. We found no evidence for increased risk of lung cancer with increasing noise exposure; in fact, in the full cohort of 24,646, lung cancer risk showed strong negative exposure-response trends.

DISCUSSION

In our study of noise exposure among sawmill workers, we found increased relative risks of acute myocardial infarction mortality in individuals chronically exposed to noise when compared both with members of the general population and with workers in the same cohort who had lower exposures. Increasing relative risks were found with higher integrated cumulative exposure to noise and with increasing duration of noise exposure. Relative risks were greatest during subjects' working years.

An association between noise and cardiovascular disease is biologically plausible, and etiologic models of stress and heart disease have been proposed and extended to noise and heart disease.[25] Although experimental studies generally support the hypothesis, only weak associations have been found in human observational studies. Many of these have been community studies of road or air traffic noise exposure, in which noise levels were relatively low (50–70 dB(A)) compared with occupational exposure levels.[14,26] Larger relative risks have been reported in studies of occupationally exposed populations. In a 1997 study of acute myocardial infarction survivors in Berlin,[12] subjects self-reporting high levels of noise at work had a relative risk of acute myocardial infarction of 3.8, with a positive exposure-response relation. Other occupational studies have found no association,[11,13]

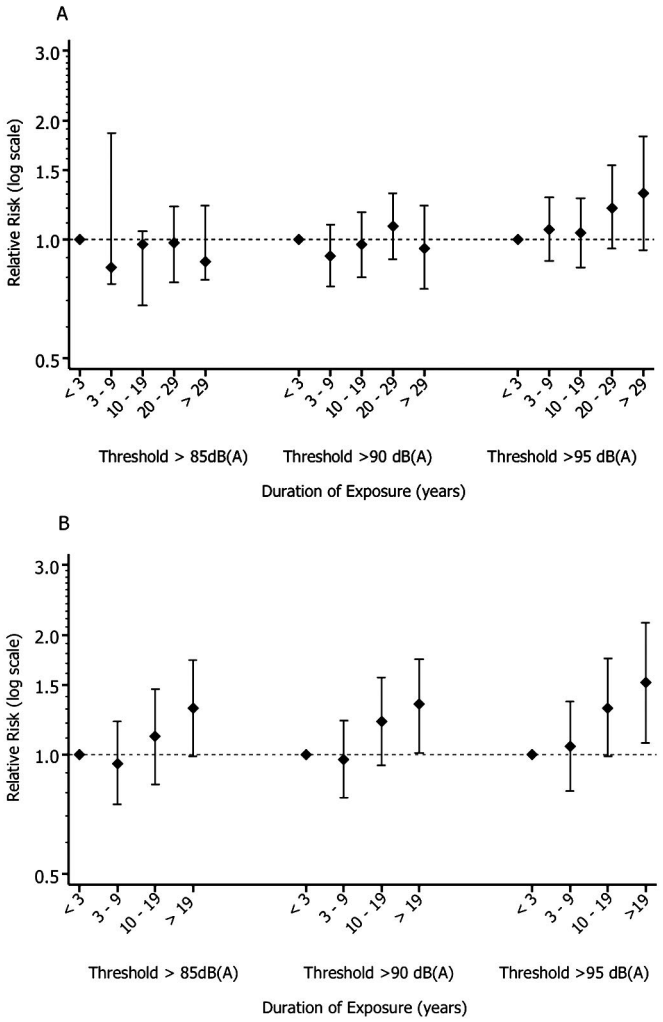

FIGURE 1. Relative risk estimates and 95% confidence intervals for acute myocardial infarction mortality by duration of exposure above threshold levels of 85, 90, and 95 dB(A). (**A**) Full cohort (n = 27,464). (**B**) Subgroup without hearing protection (n = 8668). Reference group, those exposed less than 3 years at threshold. Adjusted for age, calendar year, and South Asian ethnicity.

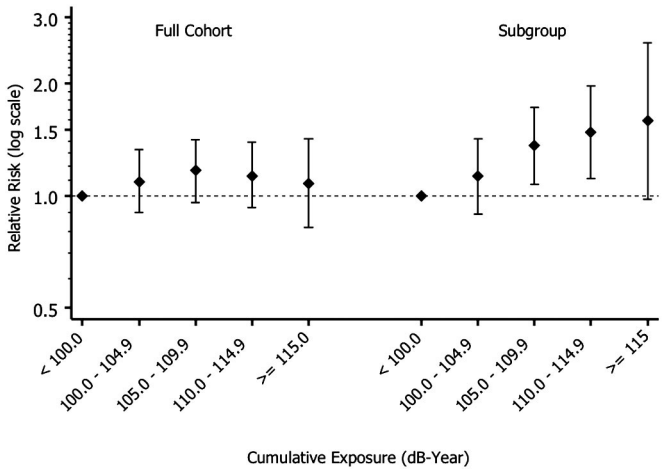

FIGURE 2. Relative risk estimates and 95% confidence intervals for acute myocardial infarction mortality by cumulative exposure (dB(A)-year). Full cohort (n = 27,464) and subgroup without hearing protection (n = 8668). Reference group, those exposed for less than 100 dB(A)-year at threshold level. Adjusted for age, calendar year, and South Asian ethnicity.

although their results may have been biased by failure to adjust for use of hearing-protector devices.

Subjects in our study were highly exposed to noise over long periods. Summary SMRs for heart disease did not show the cardiac disease risk deficit that is usually associated with a healthy worker effect,[27] although relative risks for other chronic diseases in the cohort were reduced (eg, all respiratory disease relative risk = 0.84, and all digestive disease relative risk = 0.82). SMRs indicated greatest risk of acute myocardial infarction in those exposed at the highest levels of noise when compared with the general population of British Columbia. Stronger associations were found using internal analyses with the lowest-exposed subjects as a comparison group. Such analyses of this socioeconomically homogeneous cohort should have reduced the potential for confounding by socioeconomic factors and by other important cardiovascular risk factors (such as smoking) that are associated with socioeconomic level. Such analyses also reduced the healthy worker effect.

Much higher relative risks were evident for subjects during their employed years. This might indicate that noise-related ischemic heart disease effects are chronic but reversible (ie, after termination of employment, risk decreases with time away from noise). Alternatively, it might suggest that noise presents an acute hazard in addition to a chronic hazard, perhaps by triggering an acute coronary event through transient elevation in blood pressure. Such a mechanism has been previously proposed for other stressors such as anger, perhaps interacting with circadian effects.[28]

We observed lower relative risks for ischemic heart disease (ICD-9 code 411–414.9 and 429.2) mortality other than for acute myocardial infarction. This might reflect true differences in disease mechanisms or might be the result of misclassification of outcome. Studies have shown acute myocardial infarction to be more validly coded that other ischemic heart diseases as a cause of death on the death certificates.[29]

Individual-level lifestyle data were unavailable for most cohort subjects. We were able to examine smoking (a strong risk factor for ischemic heart disease) only indirectly using a randomly selected subset of subjects. Nevertheless, this analysis indicated that smoking was not a confounder of

TABLE 5. Association of Deaths Resulting from Acute Myocardial Infarction Mortality and Duration of Exposure Above Noise Thresholds During Subjects' Working Years* for Full Cohort (n = 27,464)

	85 dB(A) Threshold			90 dB(A) Threshold			95 dB(A) Threshold		
Duration of Exposure	**Person-Years**	**No. of Deaths**	**RR (95% CI)**	**Person-Years**	**No. of Deaths**	**RR (95% CI)**	**Person-Years**	**No. of Deaths**	**RR (95% CI)**
<3 years				89,144	15†	1.0	163,871	27†	1.0
3–9 years	142,093	8†	1.0	85,146	5	0.36 (0.13–0.99)	55,455	18	1.8 (0.99–3.3)
10–19 years	71,924	26	3.9 (1.7–8.8)	55,592	22	1.5 (0.74–2.9)	29,324	18	1.9 (1.1–3.5)
>19 years	45,753	47	4.0 (1.8–9.3)	29,888	39	2.0 (1.0–3.7)	11,119	18	2.7 (1.4–4.9)
	P for trend = 0.003			*P* for trend = 0.004			*P* for trend = 0.001		

Adjusted for age, calendar year, and South Asian ethnicity.
*Follow up restricted to subjects' working years plus 1 month.
†Reference group for 85 dB(A) threshold analysis <10 years combined because of low numbers in reference group. Reference group for 90 dB(A) and 95 dB(A), threshold analyses <3 years.

the observed relation between noise and heart disease. Information on other individual risk factors (such as family history of heart disease, high serum cholesterol, and abdominal obesity) was similarly unavailable. However, for these factors to confound the relation with acute myocardial infarction, they would have to have been correlated with noise exposure; there is no reason to believe that such correlations existed. Other cardiotoxic exposures in the lumber mill environment include carbon monoxide, temperature extremes, shift work, psychosocial job strain, and physical stress. The last 2 had been previously investigated in this cohort, and no association with cardiovascular disease was found.[30] The others were evaluated by an occupational hygienist who visited all participating mills and were considered unlikely to confound the observed relation because none was correlated with noise exposure.

Despite a comprehensive exposure assessment process, some misclassification was inevitable. The majority of this bias would be independent of health outcome status and can therefore be assumed to be nondifferential; this pattern would attenuate the observed risk. Work histories were available only for the time that subjects worked in mills included in the study's sampling frame. This would lead to an underestimation of cumulative exposure (and to a degree inversely proportional to the time employed in the cohort sawmills), thus again attenuating the observed risk.

In the present study, we found higher relative risks in a subgroup in which hearing-protector devices were not used. Real-world hearing protection reduces noise exposure between 1 and 17 dB, depending on type of hearing protector, fit, and consistency of wear.[20] Use of hearing protection would therefore have the effect of reducing exposure overall and thus lowering the risk of acute myocardial infarction. However, because their use is not reflected in the usual noise exposure measures, resulting exposure overestimation would also bias the observed effects toward the null. Relative risks for acute myocardial infarction were increased in reanalyses of the full cohort after exposures had been arithmetically adjusted for hearing-protector use. These increases were seen only for the cumulative exposure metric and were not as great as in the subgroup without hearing protection. This inconsistency may be the result of the presumable large degree of exposure misclassification introduced by our relatively crude adjustments; these adjustments were based on population prevalence of hearing-protection practices and estimates of average noise attenuation provided by a range of hearing-protective devices.

The results of this study suggest that noise exposure may be an important risk factor for acute myocardial infarction in a lumber mill environment. However, the observed noise exposure, in terms of magnitude, spectral frequency, and impulsive content, are found in many industrial environments and in nonindustrial workplaces.[31] Thus, the health impact of chronic exposure to very high levels of noise may be significant. During the follow-up period of this study, there were 907 acute myocardial infarction deaths. Using the attributable fraction and assuming a causal relation between noise and acute myocardial infarction, we estimate that 58 of the 255 deaths occurring among those who worked for more than 20 years at 90 dB(A) could have been averted or delayed if noise levels had been reduced to that of the lowest-exposure group. Similarly, approximately 30 of 181 other ischemic heart disease deaths may have been averted or delayed. These excess deaths are twice the number of fatal injuries in sawmills during the same period. It is ironic that in a safety-conscious industry that puts great effort into accident prevention, greater reductions in work-related mortality might have been achieved by efforts to lower workers' exposure to noise.

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