ORIGINAL ARTICLE

# Predictive validity of a retrospective measure of noise exposure

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**Aims:** To investigate the validity of measures of noise exposure derived retrospectively for a cohort of nuclear energy workers for the period 1950–98, by investigating their ability to predict hearing loss.
**Methods:** Subjects were men aged 45–65 chosen from a larger group of employees—assembled for a nested case-control study of noise and death from ischaemic heart disease—who had had at least one audiogram after at least five years' work. Average hearing loss, across both ears and the frequencies 0.5, 1, 2, and 4 kHz, was calculated from the last audiogram for each man. Previous noise exposure at work was assessed retrospectively by three hygienists using work histories, noise survey records from 1965–98, and judgement about use of hearing protection devices. Smoking and age at the time of the audiogram were extracted from records. Differences in hearing loss between men categorised by cumulative noise exposure were assessed after controlling for age, smoking, year of test, and previous test experience.
**Results:** There were 186 and 150 eligible subjects at sites A and B of the company respectively who were employed for an average of 20 years. Compared to men with less than one year's exposure to levels of 85dB(A) or greater, hearing loss was greater by 3.7 dB (90% CI −2.6 to 10.1), 3.8 dB (90% CI −2.6 to 10.3), 7.0 dB (90% CI 1.1 to 12.9) and 10.1 dB (90% CI 4.2 to 16.0) in the lowest to highest categories of cumulative noise exposure at site B. In contrast, at site A, the corresponding figures were −2.2 dB, −2.4 dB, −1.8 dB, and −4.4 dB, with no confidence interval excluding zero.
**Conclusions:** Noise estimation at one site was shown to have predictive validity in terms of hearing loss, but not at the other site. Reasons for the differences between sites are discussed.

Retrospective estimation of exposure is common in occupational epidemiology but often there is little opportunity to validate the resulting measure. However, if the exposure is already accepted to cause a particular adverse outcome, it may be possible to assess the *predictive validity* of the measure—that is, its ability to predict the adverse event. We developed a method[1] for retrospective estimation of noise exposure within an occupational cohort for use in a study of noise and cardiovascular mortality.[2] Since excessive noise can cause hearing loss,[3–5] and audiograms had been carried out on a sample of the cohort, an opportunity arose to investigate the predictive validity of the noise measure. We report here the results of that investigation.

## METHODS

For the mortality study[2] (referred to here as the main study) approximately 1200 cases and 1200 controls had been chosen from a cohort of male industrial employees based at either of two sites (A or B) of a nuclear energy company between 1950 and 1998. Noise exposure had been assessed retrospectively for these men (see below). The subjects for the validation study were a subset of these for whom there was sufficient audiometric data. There was little audiometric testing carried out before 1978. In that year, new and current employees were offered audiograms and new employees thereafter. In addition, subjects perceived to be at risk from noise exposure were offered periodic testing but we have no information on precisely how the decision was made. Around 70% of subjects in the main study who were employees of the company in or after 1978 had had an audiogram.

In the main study, average daily personal noise exposures measured on the A-weighted scale $L_{EP,d}$ (dB(A))—referred to hereafter as dB(A)—were estimated for all subjects throughout their employment at the company. The methods are fully described elsewhere.[1 2] Briefly, personnel records were used to characterise each subject's employment at the company as a series of non-overlapping time periods, within each of which job titles and job location (building) were fixed. The resulting information from all subjects was aggregated, for each site separately, to give a list of observed combinations of year × building × job title. Three hygienists then estimated 8-hour A-weighted environmental noise exposures for each combination at each site using historical information from company noise surveys together with professional judgement. Company policy on use of ear protection and judgement about likely adherence to policy over time were used to develop rules[1] for correcting the environmental exposure estimates to give estimates of ''personal'' noise exposure. In what follows, the term noise exposure refers exclusively to these corrected estimates. Each period of an individual subject's employment was then assigned a noise estimate according to site, year, building, and job title. Thus, their complete exposure history while at the company consisted of a series of time periods of varying length, T, within each of which 8-hour personal noise exposure, E, was assumed constant. Noise estimation was carried out ''blind'' to auditory data.

Auditory testing had been carried out in sound-attenuating booths in the medical departments at each site. We have no information about the equipment used or differences in equipment between sites or over time. Air conduction thresholds (dBHL), referred to here as hearing levels (dB), were measured for the pure tone frequencies 0.5 kHz, 1, 2, 3, 4, 6, and 8 KHz in each ear. Depression in hearing levels due to noise exposure is initially seen at a frequency of 4 KHz,[5] and with continuing exposure there is an extension to lower and higher levels. As our main outcome measure, we used hearing impairment, defined by the World Health Organization[6] as the average hearing level across the frequencies 0.5, 1, 2, and 4 KHz, and, in our case, further averaged across left and right ears.

The validation study was restricted to 45–64 year old subjects with at least one audiogram taken after a period of at least five years' continuous employment. There were few eligible subjects outside this age range and the restriction ensured a more homogeneous group in respect of an important potential confounder. A study based on within-person change in hearing thresholds was ruled out because less than a third of subjects had more than one audiogram record; hence the analysis was based on one audiogram per person only, generally the last one recorded. For a small number of subjects, the last audiogram had missing information at some frequencies; for these, the penultimate audiogram was used instead if one existed. If subjects had several separated periods of employment with the company, the last audiogram within the first period of employment was used.

Smoking has been linked to hearing loss.[7 8] All employees underwent annual medical examinations and information from the medical records on smoking habit and other variables had been retrieved for the main study. The information closest in time to the audiogram was used to classify subjects as non-smokers, ex-smokers, light (<15 cigarettes/day) or heavy (15+ cigarettes/day) smokers.

### Summary noise exposure measures

The threshold for the adverse effect of noise on hearing was assumed to be 85 dB(A). Noise exposure estimates for all periods between the start of employment and the date of the audiogram were extracted from the main study database for each subject; total years before the audiogram, during which noise was estimated at 85 dB(A) or more, was calculated and is referred to here as $TT_{85}$. Men with a $TT_{85}$ of less than one year were considered unexposed.

Two other indices of exposure, $M_{85}$ and $NIL_{85}$, were calculated for subjects with $TT_{85} \geqslant 1$. $M_{85}$, measured in dB(A), is a measure of the mean noise level across all periods when E ⩾85 dB(A). For each such period, the noise intensity, I (watts/$m^2$) was calculated from E using the formula, $I = 10^{-12} * (10^{E/10})$, and multiplied by the number of years, T. The mean intensity $\Sigma IT/TT_{85}$ across these periods was found; $M_{85}$ was calculated from mean intensity using the reverse transformation. The noise immission level,[9] $NIL_{85}$, is a measure of *cumulative* exposure found by applying the reverse transformation to cumulative intensity, ΣIT. It can be shown that $NIL_{85} = M_{85} + \log_{10}(TT_{85})$ implying that, for example, four years at 91 dB(A) is equivalent to eight years at 88 dB(A) or 16 years at 85 dB(A). To examine the predictive ability of intensity and duration separately, without the assumptions built into the $NIL_{85}$ scale, a high exposure group was also defined on the basis of high (above median) levels of $M_{85}$ and high (above median) levels of $TT_{85}$.

Estimated years at 85 dB(A) or higher might not reflect true exposure time if there was systematic underestimation or overestimation of exposure. The hygienists considered it unlikely that they had systematically underestimated noise. To allow for the possibility that the noise measures might systematically overestimate true exposure—in which case a threshold of 85 dB(A) on the *measured* scale would be too low—thresholds of 88 dB(A) and 91 dB(A) were also considered, and corresponding measures $TT_{88}$, $NIL_{88}$, $M_{88}$, $TT_{91}$ $NIL_{91}$, and $M_{91}$ calculated.

### Statistical analysis

Linear regression was used for all analyses with the dependent variable being hearing loss averaged across both ears and frequencies 0.5, 1, 2, and 4 kHz. The predictors in the regression model were age (continuous), current smoking category, calendar year (continuous) of the hearing test, previous test experience (that is, whether the subject was known to have had a previous audiogram), and one or more of the noise exposure measures. As age is a potentially strong confounder when considering the effect of cumulative noise measures on hearing levels, the form of the relation between hearing loss and age was investigated in preliminary analyses using non-parametric ''lowess'' plots [10] and non-linear, fractional polynomial models[11]. Inclusion of calendar year as a predictor was an attempt to account for cohort effects and/or changes in equipment over time; its relation with hearing level was examined using the same tools. Test experience was included to account for possible learning effects in audiometric responses.

Initial regression analyses investigated which threshold on the measured scale—85, 88, or 91 dB(A)—was appropriate. To do this, estimated years of exposure above 85 dB(A) for each subject were partitioned into three parts—years in the ranges 85–87.9 dB(A), 88–90.9 dB(A), and ⩾91 dB(A) respectively—and all three measures were entered into the model simultaneously. If there was no apparent association between hearing loss and estimated years of exposure in the range 85–87.9 dB(A) for example, this would suggest that the threshold should be 88 dB(A) or higher.

Thereafter, the aim was to investigate whether there was a correlation between hearing loss and measures of cumulative noise based on the chosen threshold, having allowed for age, smoking, year, and test experience. Continuous and categorical versions of $NIL_{85}$ were examined as were categories based on $M_{85}$ and $TT_{85}$. Categories were formed using quartiles or medians of continuous distributions for all subjects combined with rounding to the nearest whole number.

### Significance level and power

The level of a significance test corresponds to the probability of a type I error that ''an experimenter is willing to accept''.[12] Fisher noted that, ''No scientific worker has a fixed level of significance at which from year to year, and in all circumstances, he rejects hypotheses''.[13] Here we used two-tailed tests with a significance level of 0.10, and corresponding 90% confidence intervals. These reflected a view that the implications of a type I error in a validation study were less serious than, for example, when testing hypotheses about new causal factors.

Power calculations were based on an estimate of the expected difference in hearing impairment between unexposed and exposed subjects. Using data from the UK MRC National Study of Hearing,[3] we estimated the difference in *annual* loss, averaged across frequencies 0.5, 1, 2, and 4 kHz, between men considered unexposed to excessive noise (the ''screened group''), and an exposed group with unquantified, ''high'' exposure due to work or hobbies, to be around 0.31 dB/year. Therefore we expected that, in men with 20 years of high exposure for example, the loss would be on average 6 dB more than in unexposed men. Within group variability (standard deviation) in average hearing impairment was estimated at 13 dB for the present study. Given a true difference between exposed and unexposed of 6 dB, a comparison based on 81 men in each group would have 90% power to give a significant result at the 10% level using a two-tailed test.

## RESULTS

Four hundred and sixty five subjects (276 at site A and 189 at site B) from the main study had at least one audiogram. Of these, 105 were excluded because they had not been employed continuously in the company for at least five or more years before their last audiogram, a further 22 because they were under 45 years old, and two because they had one or more hearing levels missing, leaving 336 subjects (186 from site A, 150 from site B) for analysis. These had a median age of 57 years at the time of the audiogram and, prior to this, median years of employment in the company of 20 years (range 5–38).

**Table 1** Age, employment, hearing, and smoking characteristics of subjects

| | Site A | Site B |
|---|---|---|
| All subjects | n = 186 | n = 150 |
| Median (range) length of employment prior to audiogram (years) | 19 (5–38) | 25 (6–40) |
| Median (range) age at audiogram (years) | 57 (45–65) | 59 (45–65) |
| Median (range) of hearing level*, dB | 27 (9–81) | 25 (1–80) |
| Smoking, n (% of total) | | |
| Never | 34 (18%) | 24 (16%) |
| Ex-smoker | 55 (30%) | 47 (31%) |
| Current | 97 (52%) | 79 (53%) |

*Averaged across both ears and frequencies 0.5, 1, 2, 4 kHz.

The majority of audiograms were carried out in 1978 with a range of 1971–98. At site A, the audiogram used for analysis was the only audiogram carried out for the majority (79%) of subjects. The opposite was true at site B: 76% of subjects had had a previous audiogram. At both sites, just over half the subjects were current smokers and nearly a third were ex-smokers (table 1).

Eighty nine per cent (n = 300) of subjects were estimated to have been exposed to noise levels of 85 dB(A) or higher for at least a year before the audiogram. Employment time before the audiogram was similar for these and the unexposed subjects, with medians of 20 years (range 5–40) and 23 years (range 7–34) respectively. Among exposed subjects, the duration of exposure to levels above 85 dB(A)—that is, $TT_{85}$, ranged from one to 35 years with a median of 15 years; the median (range) of $M_{85}$ and $NIL_{85}$ were 89 (85–96) dB(A) and 100 (86 111) units respectively.

There were only nine unexposed subjects at site A and 27 at site B (table 2). Those exposed at site A had slightly higher mean exposure levels on average compared to exposed men at site B, but the estimated length of exposure was less; when duration and intensity were combined in the $NIL_{85}$ scale, the medians for exposed men at the two sites were the same. Median average hearing levels were also similar, at 27 dB and 25 dB for sites A and B respectively (table 1).

Non-parametric lowess plots suggested that the assumption of a linear decline in hearing levels with age among the 336 45–64 year old men was reasonable and the addition of other fractional polynomial age terms to a linear regression model did not significantly improve the fit. Therefore a linear relation was assumed. These same methods also supported the assumption of a linear relation between hearing levels and the year of the test if one subject—the only man tested in 1971 and with a very high hearing loss—was omitted. This subject's data were included in the final model with a separate predictor variable representing the year 1971 (results were very similar if this man was omitted entirely). Since early analyses suggested that the predictive validity of noise estimates varied between sites, results are presented separately for each.

At site B, total years of exposure to noise levels ⩾85 dB(A) was associated with an increase in hearing level of 0.22 dB/year (90% CI 0.04 to 0.40) per year of exposure, after controlling for the other variables. When, instead, the variables measuring years of exposure in the three decibel ranges, 85–87.9, 88–90.9, and ⩾91 dB(A), were added simultaneously to the model, the estimated increases per year of exposure were 0.20 dB/year (90% CI −0.02 to 0.43), 0.15 dB/year (90% CI −0.09 to 0.38) and 0.45 dB/year (90% CI 0.07 to 0.83) respectively (table 3). The near significant, positive association with estimated exposure in the range 85–87.9 dB(A) suggested that a threshold of 85 dB(A) was appropriate for further analyses. Although the biggest effect size (0.45 dB/year) was found for the highest exposure range, the estimated effects for the different ranges were not significantly different (p = 0.42).

When the cumulative exposure measure, $NIL_{85}$, was added to the model instead, the estimated increase in hearing loss per $NIL_{85}$ unit was 0.47 dB (90% CI −0.08 to 1.02). A comparison of hearing impairment in the four exposure subgroups, defined by approximate quartiles of the NIL scale for both sites combined, compared with those in the unexposed group, showed evidence of a dose-response relation. The increases in hearing loss, compared to the unexposed, were 3.7, 3.8, 7.0, and 10.1 dB, in the lowest to the highest $NIL_{85}$ categories respectively, with the 90% CIs for the last two excluding zero (table 3). A test of trend, where the exposure categories were represented by the numbers 1 to 4, gave p = 0.05.

An alternative categorisation of the exposed group was formed from crossing categories of mean exposure, $M_{85}$ and of duration of exposure $TT_{85}$, using the medians for both sites combined as cut-off points. The two categories of $M_{85}$ (<89 dB(A), ⩾89 dB(A)) differed by 3 dB on average (from 87 to 90) and the two categories of duration (1–14.9 years, ⩾15 years) by 12 years on average (from 9 to 21 years). Both aspects of exposure appeared to increase hearing loss at site B (table 3): in the lower duration group, those with mean levels above 89 dB(A) had a greater loss (5.5 dB) than those with mean levels between 85 and 89 dB(A) (2.3 dB), and among those with mean levels between 85 and 89 dB(A), more than 15 years' exposure was associated with a greater loss (8.4 dB) than 1–15 years' exposure (2.3 dB).

At site A, the estimated increases of hearing loss per year of exposure in the three decibel ranges, 85–87.9, 88–90.9, and ⩾91 dB(A) were −0.11 (90% CI −0.40 to 0.17), −0.25 (90%

**Table 2** Noise exposure characteristics of subjects by site

| | Site A | Site B |
|---|---|---|
| Exposed to noise ⩾85 dB(A) for at least a year | n (%) | n (%) |
| No | 9 (5%) | 27 (18%) |
| Yes | 177 (95%) | 123 (82%) |
| Exposed subjects only | Median (range) | Median (range) |
| $M_{85}$ (dB(A)) | 89 (85–96) | 88 (85–93) |
| Years above 85 dB(A) | 13 (1–29) | 17 (1–35) |
| $NIL_{85}$ | 100 (86–111) | 100 (87–106) |

**Table 3** Association between hearing level* and noise measures by site

| | Site A (n = 186) Effect size (90% CI) | | Site B (n = 150) Effect size (90% CI) | |
|---|---|---|---|---|
| Years at | | | | |
| 85–87.9 | −0.11 (−0.40 to 0.17) dB/year | | 0.20 (−0.02 to 0.43) dB/year | |
| 88–90.9 | −0.25 (−0.49 to 0.00) dB/year | | 0.15 (−0.09 to 0.38) dB/year | |
| ⩾91 dB(A)) | −0.01 (−0.32 to 0.30) dB/year | | 0.45 ( 0.07 to 0.83) dB/year | |
| $NIL_{85}$ (continuous) | −0.02 (−0.38 to 0.33) dB/NIL unit | | 0.47 (−0.08 to 1.02) dB/NIL unit | |
| $NIL_{85}$ (categorical) | | | | |
| Unexposed = reference category | 0 | n = 9 | 0 | n = 27 |
| 85–97.9 | −2.3 (−9.6 to 4.9) | n = 47 | 3.7 (−2.6 to 10.1) dB | n = 28 |
| 98–99.9 | −2.4 (−9.8 to 4.9) | n = 36 | 3.8 (−2.6 to 10.3) dB | n = 24 |
| 100–101.9 | −1.8 (−9.2 to 5.6) | n = 41 | 7.0 (1.1 to 12.9) dB | n = 36 |
| ⩾102 | −4.4 (−11.5 to 2.8) | n = 53 | 10.1 (4.2 to 16.0) dB | n = 35 |
| Categories of $M_{85}$ (dB(A)), T (years): | | | | |
| Unexposed = reference category | 0 | n = 9 | 0 | n = 27 |
| <89, <15 | −2.8 (−10.0 to 4.5) | n = 5 | 2.3 (−3.9 to 8.5) dB | n = 30 |
| ⩾89, <15 | −2.0 (−9.3 to 5.2) | n = 50 | 5.5 (−1.0 to 12.1) dB | n = 23 |
| <89, ⩾15 | −2.9 (−10.5 to 4.7) | n = 30 | 8.4 (2.8 to 14.1) dB | n = 44 |
| ⩾89, ⩾15 | −3.9 (−11.1 to 3.3) | n = 47 | 8.9 (2.5 to 15.3) dB | n = 26 |
| $NIL_{91}$ (continuous) | 0.11 (−0.40 to 0.63) dB/NIL unit | | | |
| $NIL_{91}$ (categorical) | | | | |
| Unexposed (never above 91 dB(A) for 1 year) | 0 | n = 110 | | |
| <99 | −0.1 (−3.8 to 3.6) | n = 39 | | |
| ⩾99 | 1.6 (−2.2 to 5.3) | n = 37 | | |

*Averaged across both ears and frequencies 0.5, 1, 2, and 4 kHz.

CI −0.49 to 0.00), and −0.01 (90% CI −0.32 to 0.30) respectively (table 3). Thus, there was no evidence that estimated years of exposure to noise above 85 dB(A) predicted hearing loss. There was also little evidence of a relation with $NIL_{85}$: in the analysis using the continuous $NIL_{85}$ variable, the estimated increase in hearing loss per $NIL_{85}$ unit was −0.02 dB (90% CI −0.38 to 0.33). In the corresponding categorical analysis, no exposed group had a higher hearing level on average than the unexposed group.

A possible explanation of these results is that noise was overestimated at site A, in which case analyses based on a higher threshold might show a better predictive relation. In further analyses, a new threshold was set at 91 dB(A), and only the 76 subjects at site A who had at least one year of exposure above 91 dB(A) were considered exposed. The measure $NIL_{91}$ was calculated for these subjects. The estimated increase in hearing loss per unit on this scale was 0.11 dB (90% CI −0.40 to 0.63). In a categorical analysis, using only two approximately equal sized categories divided at the median, those with a $NIL_{91}$ of 99 or higher had a 1.6 dB (90% CI −2.2 to 5.3) greater hearing loss than those whose exposure never exceeded 91 dB for at least one year.

Other analyses (not shown) considered the relationship between noise estimates and hearing level at 4 KHz only. These analyses had less statistical power than before because of the greater degree of between subject variability at a single hearing level, and few results were statistically significant. However, for site A, the association between $NIL_{91}$ and hearing loss seemed somewhat stronger at this frequency, the estimated increase per $NIL_{91}$ unit being 0.45 dB (90% CI −0.24 to 1.14). Hearing loss among those with a $NIL_{91}$ level of 99 or more was 4.6 dB (90% CI −0.3 to 9.6) greater than those whose exposure never exceeded 91 dB(A) for a year or more.

## DISCUSSION

We found evidence that estimated noise exposure above 85 dB(A), based on a retrospective assessment exercise[1], was predictive of hearing loss among men at one of two sites. Both total years above 85 dB(A)—without distinguishing noise levels further—and a cumulative measure, $NIL_{85}$, based on level and duration, were predictive, and separate analysis suggested that very high exposures (89 dB(A) and above) had a greater effect than intermediary levels (85–89 dB(A)). In contrast, estimated noise levels above 85 dB(A) at the other site were not predictive, although there was a slight suggestion that a higher threshold of 91 dB(A) on the measured scale might have been more appropriate. Important features of the methods common to both sites are noted below before discussing possible reasons for the difference between sites.

The subjects of this study are unlikely to be representative of the underlying cohorts. Selection was opportunist: to be eligible, subjects had to have been chosen for a cardiovascular mortality study, and to have had an audiogram at work. Subjects in this validation study had higher average noise exposures across employment than other subjects in the main study—by about 1.5 dB(A) on average—as might be expected if testing was selective. Overrepresentation of those with higher noise exposures is unlikely to have biased the relation between estimated noise exposure and hearing loss because representative exposure is unnecessary in this respect. The *range* of exposures mirrored that in the main study[2] which is important if the validation is to be generalised to it.

For both studies historical noise surveys of buildings were used, where available, to form a judgement about exposure of subjects in that building; if no survey had been carried out, additional rules[1 2] based on job title and year were used to make estimates. In the main study, 65% of the total person-years of assessment were based directly on noise survey reports; in this study the corresponding figure was 75%, implying that the present subjects were more likely to have worked in a building for which there was a noise survey. This is not surprising: the likelihood of a noise survey would tend to be greater for buildings where noise was expected to be high, and subjects in such buildings might be more likely to accept an audiogram test.

In theory, a validation study based on within-subject change in hearing level would have been preferable to the present design, being less susceptible to confounding by factors that vary between subjects. However there were only 96 qualifying subjects with two or more audiograms of which only 19 were employed at site A. Furthermore, initial analyses suggested there might have been a ''learning effect'' during

the first audiogram whereby subjects took longer to respond to the stimuli due to unfamiliarity. This was evidenced by a systematic (and statistically significant) difference between hearing levels in right and left ears at the first audiogram, with the left ear being better at one site and right at the other. This phenomenon was not seen in repeat tests. An initial learning effect would have attenuated measures of within-subject change in hearing, and hence further reduced the statistical power.

Given a learning effect, it would also have been preferable to exclude first audiograms from the present design but this would also have had reduced power considerably. Instead, to reduce any potential for bias, previous test experience (yes/no) was included as a predictor of hearing loss in the regression models. Although we had no information on equipment or personnel involved in testing hearing we tried to detect any systematic changes over time in the measurement process by non-linear models and plots. There was no evidence of abrupt changes such as might be caused by equipment breakdown or miscalibration, but there was a steady decline in hearing thresholds which could be explained by a cohort effect.

We treated age and smoking as potential confounders in the analysis, although no significant differences between non-smokers and other smoking groups were found. Other potential confounders of the relation between hearing and *assessed noise exposure* during this employment, which were not allowed for in the analyses, include wax in the ear, hobbies that involve high noise exposures, and military service. Also, although many subjects had long service at the company (the average duration of employment prior to the hearing test was 20 years) noise exposure during other employment should also be considered. One might postulate, for example, that the within-company estimates of noise exposure in this study were really invalid, but a positive (spurious) association at site B was nevertheless found because of a positive correlation between these estimates and noise exposure outside the company. While this possibility cannot be discounted, there is no particular reason to believe in such a correlation.

One interpretation of the difference found for the two sites is that the method was valid for site B but that the noise estimation process failed at site A. Alternatively one could argue that it reflects a general failure of the retrospective exposure assessment method. The same hygienists were responsible for estimation at both sites but the work for site A was carried out first. It is conceivable that there was a learning process with less error when they reached site B. Interestingly, their prior expectation had been that estimation for site A would be superior because there was a greater amount of noise survey information there: survey information was used directly for 85% of person-years of employment at site A, compared to 64% at site B.

There are a number of other possible explanations for the lack of a predictive relation at site A. For example, the problem could lie in the quality of the hearing test data: if there is sufficient random measurement error in an outcome or exposure measure, genuine relationships can be obscured. The majority (79%) of the audiograms used for analysis at site A were first audiograms and, as already noted, there was some evidence that results from the first audiogram were less reliable indicators of hearing loss. Another problem was the small number of ''unexposed subjects'' at site A, which would have limited the power of categorical analysis.

There was a slight suggestion that noise levels might have been systematically overestimated at site A. If this was true then, provided that the ranking of worker's exposures was correctly captured, the exposure estimates could still be predictive of disease if the appropriate threshold was used and there were sufficient exposed subjects. Another possibility is that exposure measurement error was not systematic, but that there was underestimation for those assigned to the low exposure group. This could come about, for example, if the assumptions about the use of hearing protection were invalid.

Considerable efforts have been made to delineate best principles for retrospective assessment of exposure in epidemiology,[14] yet the process has been likening to solving a mystery[15] and the search of new methods and opportunities is ongoing.[16] The difficulty of the task will vary with the exposure, the design, the subjects, and time span: our study spanned almost 50 years of company history at both sites, with each subject being assessed for a ubiquitous exposure under an average of five job × building combinations. We were fortunate to have had an opportunity to perform a validation study at little cost, albeit based on imperfect data. The existence of this study which, in our opinion, supports the exposure assessment for one site at least, will considerably strengthen the conclusions that can be drawn from our main investigation into the effect of noise on cardiovascular disease.

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## REFERENCES

1 **Burgess G**, Dippnall M, Ravandi M, *et al*. Retrospective noise estimates for British Nuclear workers using an alternative approach. *Ann Occup Hyg* 2004;**48**:117–27.
2 **McNamee R**, Burgess G, Dippnall WM, *et al*. Occupational noise exposure and ischaemic heart disease mortality. *Occup Environ Med* 2006;**63**:813–19.
3 **Davis A (ed)**. *Hearing in adults*. London: Whurr, 1995.
4 **Palmer KT**, Griffin MJ, Syddall HE, *et al*. Occupational exposure to noise and the attributable burden of hearing difficulties in Great Britain. *Occup Environ Med* 2002;**59**:634–9.
5 **Lutman ME**. What is the risk of noise-induced hearing loss at 80, 85, 90 dB(A) and above? *Occupational Medicine* 2000;**50**:274–5.
6 **WHO**. Facts about deafness. Geneva, Switzerland: World Health Organization, Available at http://www.who.int/pbd/deafness/facts/en/ (accessed January 2006).
7 **Mizoue T**, Miyamoto T, Shimizu T. Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers. *Occup Environ Med* 2003;**60**:56–9.
8 **Palmer KT**, Griffin MJ, Syddall HE, *et al*. Cigarette smoking, occupational exposure to noise and self-reported hearing difficulties. *Occup Environ Med* 2004;**61**:340–4.
9 **Lawton B**, Robinson D. *A concise vocabulary of audiology and allied topics. Part 1: vocabulary*, Institute of Sound and Vibration Research, 1999.
10 **Hastie TJ**, Tibshirani RJ. *Generalised additive models*. London: Chapman and Hall, 1997.
11 **Royston P**, Altman D. Regression using fractional polynomials of continuous covariates: parsimonious parametric modelling (with discussion). *Applied Statistics* 1994;**43**:429–67.
12 **Armitage P**, Colton T. *Encyclopedia of biostatistics*, Volume 3.H-MEA. Boston: John Wiley and Sons).
13 **Fisher RA**. *Statistical methods and scientific inference*. Edinburgh: Oliver and Boyd, 1956.
14 **Nieuwenhuijsen MJ (ed)**. *Exposure assessment in occupational and environmental epidemiology*. Oxford: Oxford University Press, 2003.
15 **Kromhout H**. Assessing historical exposure is like solving a mystery. *Occup Environ Med* 2005;**62**:429–30.
16 **Johansen K**, Tinnerberg H, Lynge E. Use of history science methods in exposure assessment for occupational health studies. *Occup Environ Med* 2005;**62**:434–41.