A dose response relation for noise induced hypertension

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Abstract

The effect of industrial noise on the prevalence of hypertension was studied in a group of 1101 female workers in a textile mill in Beijing in 1985. Essentially the entire group had worked in specific workshops in this mill for all their working lives and all had worked for at least five years. The noise levels within the plant were assessed and appear to have been constant since 1954 resulting in well defined noise exposures for these workers. A cross sectional design was used in which blood pressures were determined and questionnaires administered to the workers over a two month period. As well as demographic information, data were gathered on personal and family history of hypertension, current use of prescription drugs, alcohol, tobacco, and salt in the diet. Logistic regression indicated that exposure to noise is a significant determinant of prevalence of hypertension, but third in order of importance behind family history of hypertension and use of salt. Each of the predictor variables exerted an independent influence on risk of hypertension. Cumulative exposure to noise was not an important dose related variable suggesting that, for those susceptible to the effect, hypertension was manifested within the first five years of exposure.

There have been many investigations of the relation between exposure to noise and hypertension in

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worker populations. The report by Parvizpoor[1] was one of the earliest to indicate that exposure to industrial noise was strongly associated with increases in the prevalence of hypertension. The more recent studies of Fouriaud *et al*,[2] Verbeek *et al*,[3] and Wu *et al*[4] present the most convincing confirmatory evidence of this positive association. In general, however, the published work indicating that noise is positively linked to hypertension suffers from incomplete data on other cardiovascular risk factors, inadequate control of confounding variables, or an inadequate quantification of noise exposure.

In several studies that suggest an association between noise and hypertension, noise induced hearing loss is used as a surrogate measure of cumulative exposure to noise.[5 6] This is also a common approach in studies which have concluded that no association exists between noise and hypertension.[7–9] As with the positive studies, those finding no association between noise and hypertension tend to be open to criticism on various grounds; inadequate exposure data or, in some cases, the use of hearing protection by the exposed groups being particular problems.[10–12]

A 1984 review on the cardiovascular effects of noise reported that 55 studies had assessed the relation between noise and blood pressure and about 80% reported some form of positive association.[13] These authors noted that "a paucity of quantitative data . . . makes it difficult to assess the strength of association or to derive a dose-response relation." A principal objective of the present study was to explore further the association between noise exposure and hypertension in a setting in which exposure was well defined and in which there was a reasonable expectation of describing any dose response relation free from the influence of known confounding variables.

Materials and methods

The subjects were 1101 female workers in a textile mill in Beijing. Each had worked in a single workshop in this mill for essentially their entire working life. All had been employed in the mill for at least five years. The workshops from which the subjects were drawn

Table 1 Distribution of age and working years of female textile mill workers by SPL group

SPL dB(A)	Age Mean (SD)	Working years Mean (SD)	Workers
104	38·51 (8·07)	19·76 (9·59)	164
96	37·20 (8·64)	18·23 (9·87)	294
86–90	33·93 (7·99)	14·18 (8·99)	428
75–80	33·90 (8·20)	14·59 (9·34)	215
Total	35·46 (8·43)	16·17 (9·64)	1101

were selected because of the spatial uniformity of sound pressure levels (SPLs) within each and to cover the range of noise exposure levels in the mill. All employees who worked the second shift in a selected workshop during the period of observation were offered an opportunity to participate unless they had worked in other workshops in the mill or outside of the mill for other than temporary assignments—that is, unless there had been the possibility of substantial noise exposure outside the workshop in which they were currently employed. The subjects were from six workshops and were divided into four exposure groups by sound pressure level as shown in table 1, which also contains the mean age and years worked in that environment for each group. Participation rates averaged 75% across the six workshops. When classified by exposure group, participation ranged from 70% in the 104 dB(A) group to 89% in the 75 dB(A) group. As might be expected with such a stable population, there is a very high correlation between age and years worked in the mill ($r = 0.966$).

The sound pressure levels in each workshop were measured with Bruel and Kjaer sound level meters types 2300 and 1625. Eighty measurements were made at fixed points within the working area of the six workshops. Time weighted average (TWA) exposures for workers in these various shops were estimated to range from 75 to 104 dB(A). The factory safety officer conducted a noise survey every two years and reported that the sound pressure levels in each workshop have been essentially steady since production started in 1954. His noise data were consistent with those collected during this study. In those groups where noise exposure is classified by a single SPL, the levels at various locations in the workshops did not vary from this value by more than two dB(A) and the TWA exposure of all workers was very close to the value given. In cases where the exposure group contained a range of TWA exposures, the group mean, as well as the range, was estimated from measurements of the spatial distribution of SPLs and observations of the time spent at various stations within the workshop. The noise spectrum was typical of textile mills and broad band in nature.

Only second shift workers were studied (2.00 pm to 10.00 pm). The measurement of blood pressure and the administration of a questionnaire were carried out between 1.30 pm and 4.30 pm and before the beginning of the day's work. All data were collected from 8 July to 10 August 1985. Blood pressure was measured by 10 medical students twice in sitting subjects by mercury sphygmomanometer after 10 minutes of rest. The students were trained by one of us (ZY) using a dual stethoscope so that readings differing by less than 4 mm Hg were achieved between the trainer and each student. If two readings on any worker differed by more than 4 mm Hg in either systolic or diastolic pressure, measurements were repeated after further rest intervals until the difference met this criterion. The room in which the blood pressures were measured had sound pressure levels below 60 dB(A).

According to the recommendations of the World Health Organisation Expert Committee,[14] hypertension was defined as systolic pressure greater than or equal to 160 mm Hg, or diastolic pressure greater than or equal to 95 mm Hg, or both. The subjects were also classified as hypertensive if they were currently using antihypertensive drugs. This definition produced 79 hypertensive women from the workforce of 1101 (7·2%). Of these, 68 were currently using medication for hypertension.

The questionnaire included identifying information, occupational history, disease history, parent's hypertensive history, and information regarding the subject's history of antihypertensive drug use. Also, data were collected on smoking and drinking habits and the worker's perception of their own use of salt in the diet in relation to their coworkers. This was thought to be useful because the workers routinely ate their meals in a common dining hall and many were aware of the dietary habits of their fellow workers. Twenty one workers were smokers, three of whom were hypertensive, one in the 75 dB(A) exposure group and two in the 90 dB(A) group. Seven workers drank alcohol routinely. None were hypertensive. None of the workers reported that they had been diagnosed as hypertensive at the time they were first employed in the mill. Thirty two women were pregnant at the time of the study. None was hypertensive.

Results

Table 2 shows the crude hypertensive prevalence in four SPL groups. Hypertensive prevalence increases appreciably at the higher exposure levels. To explore this association in greater detail a logistic regression was carried out using hypertension, as defined above, as the dependent binary outcome variable. Logistic regression was used because of the high proportion of the hypertensive group who were currently on

Table 2 Sound pressure level and prevalence of
hypertension in female textile mill workers

SPL dB(A)	No with hypertension	Total	Hypertensive prevalence (%)
104	25	164	15·2
96	25	294	8·5
86–90	18	428	4·2
75–80	11	215	5·1
Total	79	1101	7·2

Table 4 Relative influence of predictor variables on
probability of hypertension: differences in likelihood values

Variable	Difference	p Value
SPL	4·061	0·044
Age	2·234	0·135
Working years	1·021	0·312
Salt use	7·752	0·005
Family history	8·520	0·004

medication for this condition and therefore without useful measures of blood pressure. The predictor variables were SPL, age, years worked, use of salt (low, normal, or high) and a history of hypertension in either parent (presence or absence). The possibility of interactions among the five predictor variables was extensively explored. No evidence was found of any substantial interactive influence and therefore an additive logistic model was employed in the following analysis. Table 3 contains the summary estimates for this analysis. The coefficients of SPL (p value = 0·047), self-reported use of salt (p value < 0·001), and parental history of hypertension (p value = 0·004) are all associated with the probability of hypertension as judged by significance values (p values) associated with the regression coefficients of less than 0·05. The signs of the coefficients indicate an increasing likelihood of hypertension with increasing noise exposure, with increasing use of salt, and with a parental history of hypertension.

The logistic model provides an opportunity to assess the impact of SPL on the probability of hypertension adjusted for the influences, if any, of other predictor variables.[15] The maximum likelihood estimates of the logistic model parameters (table 3) produces an adjusted SPL odds ratio of 1·031. Therefore an increase of, say, 30 dB(A) increases the odds of hypertension by $(1·031)^{30} = 2·479$ regardless of the values of the other predictor variables as long as they are held constant and the additive model is an adequate representation of the data. In terms of probability, a 50 year old worker, employed 20 years, with normal salt intake and no family history of hypertension, and who is exposed to 70 dB(A) has a

probability of being hypertensive of 0·089. By contrast, a worker with the same levels of the other predictor variables, but an exposure of 100 dB(A), has a probability of 0·195 of being hypertensive, again, based on the additive logistic model.

To evaluate the magnitude of the individual influence of each variable on the risk of hypertension, the coefficients in the logistic regression equation must be standardised to account for differences in their units of measure. A set of commensurate values is achieved by the comparison of two likelihood values; one likelihood value measuring the influence of the entire set of variables (all five variables) and a reduced likelihood where one variable at a time is deleted from the model. The difference in these likelihood values measures the influence on hypertensive risk solely attributable to the deleted variable. Contrasting these two likelihoods for the 1101 female workers (table 4) shows that a family history of hypertension and high intake of salt have about equal and significant impact (8·52 and 7·75; p < 0·001) on the probability of hypertension, followed in order of importance by SPL at about half the magnitude (4·061; p = 0·044). Age and years worked have less influence and are not statistically significant (2·23 and 1·02; p > 0·05).

The influence on risk of hypertension associated with SPL is relatively unaffected by the other four predictor variables. Table 5 shows the influence on the logistic coefficient associated with SPL as other variables are added to the analysis. The SPL coefficients, column 1, remain essentially constant when the other variables are included in the analysis, particularly after age of the worker is taken into account. That is, the odds ratio associated with hypertension and SPL, considering no other factors, is $e^{0·052} = 1·053$ and changes little as other variables are added to the model. The final value with all five predictor variables entered is $e^{0·030} = 1·031$. Although the other variables are contributors to increases in risk of hypertension, they are independent (nonconfounding) influences with respect to the levels of SPL. In other words, the risk contributed by factors other than SPL is randomly distributed among the workers and plays a relatively minor role in estimating the relation between the exposure to noise and the occurrence of hypertension.

The ability of the logistic model to summarise the

Table 3 Estimated parameters for logistic regression model
for prevalence of hypertension among 1101 female textile mill
workers

Variable	Term	Coefficient	SE	p Value	Odds ratio
Constant	b_0	− 10·610	2·070	—	—
SPL	b_1	0·030	0·015	0·047	1·031
Age	b_2	0·096	0·063	0·126	1·101
Working years	b_3	0·054	0·055	0·323	1·056
Salt (high)	b_4	0·827	0·408	0·043	2·286
Salt (normal)	b_5	− 0·057	0·314	0·856	0·945
Family history	b_6	0·748	0·260	0·004	2·113

Table 5   Confounding influence on logistic regression: regression coefficients for a series of sequential analyses

SPL	Age	Working years	Salt (normal)	Salt (excess)	Hypertension history
0·052					
0·030	0·148				
0·029	0·079	0·064			
0·028	0·069	0·072	0·784	0·130	
0·030	0·096	0·054	0·827	0·057	0·748

multivariate relations under investigation can be judged by the goodness of fit of the values predicted by the logistic model to those found (table 6). The 1101 workers were distributed into 24 categories using three variables, hypertension, SPL, and age. The expected frequencies (column 4), based on the logistic model, and the observed frequencies (column 5), in table 6, show close agreement with the exception of one aberrant cell where five workers were obeserved and only 1·08 were expected. Formally, the χ^2 value for goodness of fit is 27·348 with 20 degrees of freedom producing a p value of 0·126. A further tabulation of the data would produce cell frequencies too small to analyse accurately and is unnecessary as the remaining variables have already been shown to have no important additional influence on the relation between SPL and the probability of hypertension.

Discussion
Figure 1 shows the effect of noise on the probability of hypertension based on the logistic model using the

coefficients given in table 3. In the figure, age is the parameter which differentiates the four curves (age = 30, 40, 50, and 60 years). Each element of the figure displays a different combination of use of salt and parental history of hypertension as indicated in the legend. Because of the high correlation between age and years worked, it is not possible to determine accurately the independent effects of these two variables; hence years worked does not appear in these figures. The choice of age is based on its known effects on prevalence of hypertension as well as the results of the regression that suggest it to be the more important variable, an issue that will be explored further below.

The crude rates of hypertension as a function of exposure to noise shown in table 2 suggested a dose response relation that, accounting for the influence of other predictor variables, is now confirmed and reflected in the figures. As shown above, an increase in noise level from 70 to 100 dB(A) roughly doubles the probability of hypertension for any combination of other variables. The absolute level of this change is, of course, highly dependent on the other predictor variables.

It is interesting to explore further the role of years worked as a contributor to risk of hypertension as it seems reasonable to postulate that the risk should be a function of cumulative exposure to noise. Cumulative exposure is a function of both the level and the duration of exposure, the latter being highly correlated with years worked in the present case. Some insight can be gained by comparing the probability of hypertension predicted by the model for low noise exposure to prevalence data for the

Table 6   Goodness of fit of values generated from the additive logistic model; log-odds = $b_0 + b_1$ (SPL) + b_2 (age)

| Hypertension	SPL	Age	Observed number	Expected number	Difference	χ^2 Contribution
Yes	104	45	19	12·878	6·122	2·910
Yes	104	35	5	2·589	2·411	2·245
Yes	104	25	1	0·508	0·492	0·477
Yes	96	45	20	15·942	4·058	1·033
Yes	96	35	5	3·731	1·269	0·432
Yes	96	25	0	0·898	−0·898	0·898
Yes	90	45	13	11·450	1·550	0·210
Yes	90	35	2	4·515	−2·515	1·401
Yes	90	25	3	1·615	1·385	1·188
Yes	77	45	6	4·860	1·140	0·267
Yes	77	35	5	1·081	3·919	14·208
Yes	77	25	0	0·620	−0·620	0·620
No	104	45	50	56·122	−6·122	0·668
No	104	35	47	49·411	−2·411	0·118
No	104	25	42	42·492	−0·492	0·006
No	96	45	84	88·058	−4·058	0·187
No	96	35	89	90·269	−1·269	0·018
No	96	25	96	95·102	0·898	0·008
No	90	45	74	75·550	−1·550	0·032
No	90	35	133	130·485	2·515	0·048
No	90	25	203	204·385	−1·385	0·009
No	77	45	46	47·140	−1·140	0·028
No	77	35	42	45·919	−3·919	0·334
No	77	25	116	115·380	0·620	0·003
Total			1101	1101·000	0·000	27·348
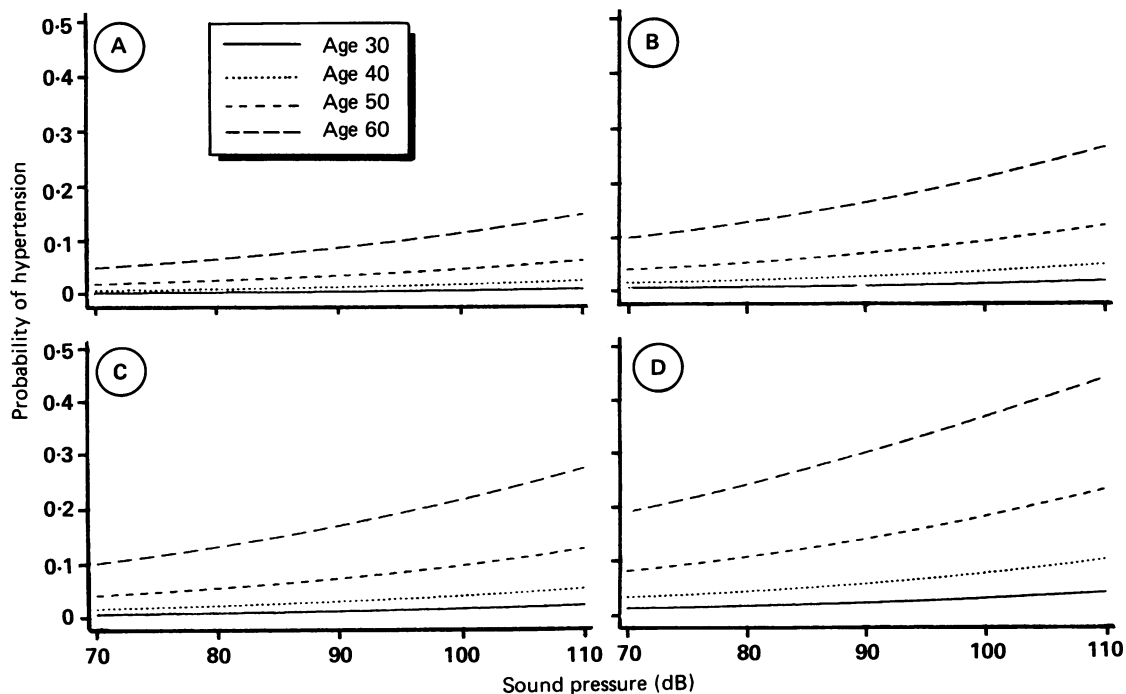
Figure 1 Prevalence of hypertension as a function of sound pressure level for four age groups and (A) normal dietary salt, no family history of hypertension, (B) high dietary salt, no family history of hypertension, (C) normal dietary salt, family history of hypertension, (D) high dietary salt, family history of hypertension.

general population. The prevalence of hypertension in the population of urban Beijing was estimated as a function of age from the data of Wu et al[16] and is plotted in fig 2 together with the model predictions from the present study when the noise exposure is estimated, via the logistic model, at 70 dB(A) to be as
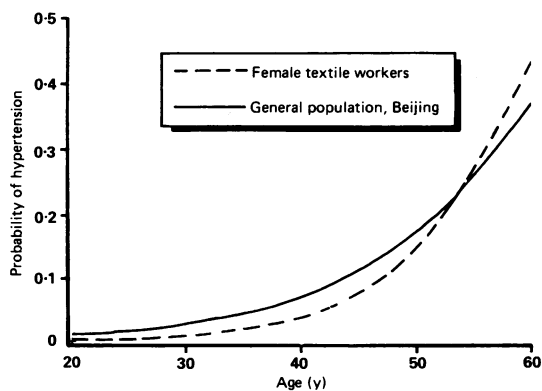

Figure 2 Prevalence of hypertension as a function of age estimated from the data of Wu et al[16] for the general population of Beijing and extrapolated to 70 dB(A) noise exposure using a logistic model fitted to the data of the present study.

directly comparable as possible with the general population data. As can be seen from fig 2, the data indicate a slightly higher prevalence of hypertension in the general urban population of Beijing than that found in this study up to about age 65. This may be due to a number of factors including the healthy worker effect, the fact that our study subjects are all women, or because the blood pressure measurements were taken in midsummer. In any case, this comparison tends to confirm that the model is essentially correct in attributing most of the temporal increase in hypertension to age and that years worked does not have a strong effect on hypertensive prevalence in the study population.

Exposure to noise is known to produce transient increases in blood pressure.[17] The cross sectional design of this study, therefore, might have captured transient increases that persisted between the end of one shift and the beginning of another. Wang et al[18] found an increased prevalence of hypertension, however, in a group of retired female textile mill workers exposed to noise at above 100 dB(A) contrasted with age matched controls drawn from the same population, but exposed to low noise levels. This finding suggests that the end result of repeated high noise exposures leads to an increased probability of a permanent increase in blood pressure

and is consistent with the experimental findings of Peterson *et al*[19] in rhesus monkeys.

The minimum time of exposure to noise among this group of workers was five years. Hence, the lack of association between hypertension and years worked is also consistent with the hypothesis that the effect of noise on hypertension becomes a permanent effect within five years of the beginning of exposure. If this were true, it would explain the ambiguity in published work concerning the association between noise induced hearing loss and hypertension because significant noise induced hearing loss can be expected to take place over a period in excess of five years, except at the highest exposure levels. Whatever the case, longitudinal studies are required to clarify this issue.

The most important determinants of blood pressure not measured during this study were individual height and weight. Recalling that workers were assigned to each workshop at the time of hiring, and stable thereafter, we know of no reason to suspect a height and weight placement bias which might confound the association of hypertension with level of noise exposure.

In conclusion, our results indicate that industrial exposure to noise is an important determinant of risk of hypertension in working populations. The logistic model estimates that a 30 dB(A) increase in SPL roughly doubles the risk of hypertension, independent of the other risk factors measured. The overall risk of hypertension, of course, is strongly dependent on the presence of other predictor variables, in particular, family history of hypertension, dietary intake of salt, and age. Cumulative exposure to noise, as measured by years worked in a particular noise environment, did not appear to be an important predictor of risk of hypertension. This might be due to the manifestation of this effect within the first five years of exposure, which we would not have detected, or to an inability to separate the effects of age and years worked because of the collinearity between these factors in this population.

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