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Risk factors in the genesis of sensorineural hearing loss in Finnish forestry workers

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ABSTRACT A detailed analysis of risk factors for the development of sensorineural hearing loss (SNHL) was carried out in 199 forest workers. The hearing threshold of both ears at 4000 Hz was measured, and the effect of age, exposure to noise, blood pressure, presence of vibration induced white finger (VWF), tobacco smoking, plasma LDL-cholesterol concentration, and consumption of drugs were evaluated by multiple linear regression analysis. Aging was the major risk factor, followed by exposure to occupational noise and the presence of VWF. Plasma LDL-cholesterol concentration and the use of antihypertensive drugs also correlated significantly with SNHL. These main factors were able to explain about 28% of the SNHL variance. Additional factors in the analysis, including smoking, systolic and diastolic blood pressure, and consumption of salicylates did not significantly contribute to the genesis of SNHL.

Despite the careful application of the equal energy principle in the analysis of populations exposed to noise, variability in the development of sensorineural hearing loss (SNHL) is a dominant feature. Whereas the hearing of some people is resistant to the harmful effect of noise, some are exceptionally vulnerable. It has been suggested that this individual resistance against occupational noise depends on heredity, diet, pigmentation, drugs, blood pressure, non-occupational exposure to noise, etc.[1-4] Recently we have shown a link between vibration induced white finger (VWF) and SNHL[5,6] suggesting that factors disturbing the peripheral circulation may influence cochlear function and hearing. The relative role of different factors in the genesis of SNHL is, however, poorly understood.

In the present study we have examined a population of workers exposed to noise and vibration. The purpose of the study was to evaluate the relative importance of selected risk factors in the aetiology of SNHL.

Subjects and methods

The investigation was carried out in 1986 in connection with a compulsory health survey in the county of Suomussalmi in north eastern Finland. The population in this area was stable and we have followed up these workers since 1972 in a longitudinal survey.[7] All the professional forest workers of the major employing company (National Board of Forestry) took part in the study. During a physical examination the ears were inspected, and workers with bilateral middle ear disease were excluded from the study. The study comprised altogether 199 professional forest workers. Their mean age was 43·1 years (range 25–60) and mean exposure to chain saw noise was 15·370 hours (range 0–32 200).

Hearing was tested in an acoustically treated but non-isolated room after absence of occupational exposure to noise for 15 to 48 hours using a pure tone audiometer (Maico Ma-19). A more detailed discription of the threshold evaluation is given elsewhere.[8,9] For the characterisation of individual SNHL mean hearing threshold at 4000 Hz was used. In cases of unilateral ear disease the hearing of the healthy ear was used.

A history of work habits (daily chain saw operating hours, alternative working tasks) were obtained annually during a longitudinal survey to get an estimation of exposure. The use of earmuffs, a history of vibration syndrome, and smoking habits were also recorded. Subjects were divided into six categories depending on the extent of their smoking.[9] If more than ten years had passed since giving up smoking the subject was classified as a non-smoker.

A complete medical examination was performed.[7] Blood pressure was measured in both arms in the

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recumbent position, and the mean value of the left and right arm for systolic and diastolic blood pressure measurements was taken after the subject had been supine for 20 to 30 minutes.

For each subject, serum high density lipoprotein-cholesterol (HDL-cholesterol) and total cholesterol levels were measured. Low density lipoprotein-cholesterol (LDL-cholesterol) content was calculated for risk analysis.

A history of head injuries, noisy activities during free time, annual consumption of salicylates and other antiphlogistic drugs, as well as the use of antihypertensive drugs, was recorded. Other possible sources of SNHL were noted. It was found that noisy free time activities or head injuries did not significantly contribute to SNHL and they are therefore not commented on in the present report.

RISK EVALUATION BASED ON ROBINSON'S MODEL

Measured hearing loss was compared with estimated mean hearing loss evaluated using Robinson's model which gives the age corrected hearing level that is related to A-weighted equivalent noise level in the following way.[10]

$$HTL = 27{\cdot}5\left(1 + \tanh\frac{E_{Ai} - L_f + U_p}{15}\right) + U_p + C_f(N_i - 20)^2 \quad (1)$$

- HTL = actual hearing level exceeded by p per cent of the population
- E_{Ai} = $L_{Aeqi} + 10\log(T_i)$
- E_{Ai} = A-weighted noise immission level
- L_{Aeqi} = A-weighted equivalent dB level (8 h/d, 5 d/w)
- T_i = exposure time in years
- N_i = age in years
- C_f and L_f = coefficients depending on audiometric frequency
- U_p = constant depending on the selected percentage p.

This model was applied to the data in two different ways for comparison with actual HTL values measured among forest workers in Suomussalmi. Firstly, HTL was calculated using age, exposure time, and exposure level as independent factors for each individual. Exposure time (in years) was related to the total sawing time (in hours) assuming a working time of 1000 hours yearly. The exposure level depended on the period of earmuff use and calculated for each worker separately using logarithmic conversion. Secondly, the effect of age alone was examined by fixing the exposure level to the median value of the whole population and increasing age one year at a time from 20 to 60. The median exposure time was 16·0 years.

For risk evaluation, the audiometric frequency was 4000 Hz and the constants $C_f = 0{\cdot}012$ and $L_f = 112{\cdot}5$ dB. The values were calculated for median (p = 50%) and $U_p = 0$. Thus equation (1) becomes:

$$HTL = 27{\cdot}5\left(1 + \tanh\frac{E_{Ai} - 112{\cdot}5\,db}{15}\right) + 0{\cdot}012(N - 20)^2 \quad (2)$$

where $\tanh(x) = \dfrac{e^x - e^{-x}}{e^x + e^{-x}}$.

Statistics

In risk evaluation a multiple linear regression analysis was used. To determine the significance of regression the determination coefficients for each factor were tested with Student's *t* test. For individual factors the subjects were divided into two groups depending on the risk level of each factor. The difference in hearing between the groups was evaluated with age corrected SNHL. When p was less than 0·05 the result was considered statistically significant.

Results

LINEAR REGRESSION ANALYSIS

The measured hearing loss for the group was 27·3 dB at 4000 Hz. In the linear regression analysis aging was the most important single risk factor for SNHL and explained 25% of the variance in SNHL at 4000 Hz. Exposure to noise explained 9% of the variance in SNHL at 4000 Hz. A statistically significant correlation was also found with the presence of VWF, serum LDL-cholesterol concentration, and use of antihypertensive drugs, and SNHL at 4000 Hz (table). A combination of these risk factors explained about 28% of the variance observed in the SNHL at 4000 Hz. All these significant risk factors also correlated with age. Thus exposure ($r = 0{\cdot}612$, $p < 0{\cdot}001$), LDL-cholesterol ($r = 0{\cdot}34$, $p < 0{\cdot}01$), VWF ($r = 0{\cdot}251$, $p < 0{\cdot}05$),

Individual partial correlation coefficients, their cut-off level in risk analysis, and their effect on hearing (n = 199)

Factor	*Partial corr coeff*	*Cut off level*	*Effect on hearing (dB)*
Age	0·514	—	7·6*
Exposure	0·318	—	17·8*
VWF	0·202	Present or not	1·5
LDL-cholesterol	0·156	5·0 mmol/l	0·5
Salicylate	0·064	50 tablets a year	0·9
Smoking	0·100	Smoker or not	0·4
Antihypertensive drugs	0·165	User or not	1·0
Diastolic blood pressure	0·046	90 mm Hg	0·1

*Estimated according to Robinson's model.[10]

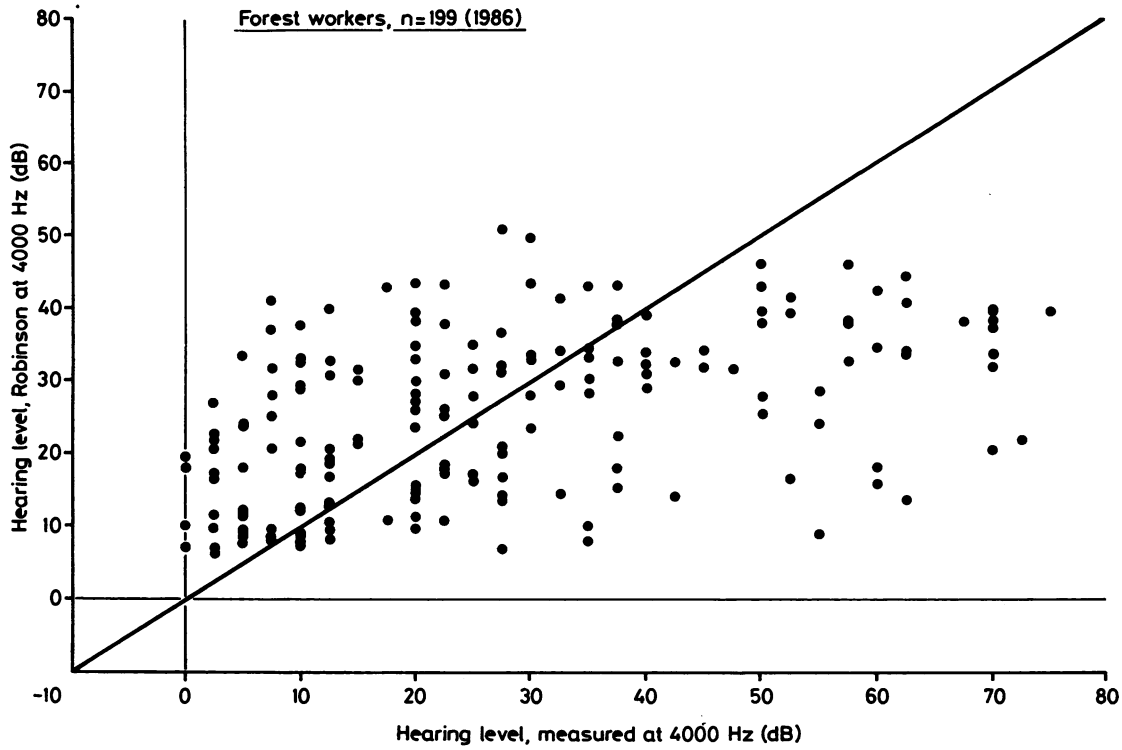

Fig 1 *Relation between estimated hearing loss according to Robinson's model[10] and measured hearing loss in forest workers.*

and use of antihypertensive drugs ($r = 0{\cdot}215$, $p < 0{\cdot}05$) were age dependent. The sensorineural hearing loss did not correlate significantly with the consumption of salicylates, diastolic and systolic blood pressure, and smoking.

The table gives the partial correlation coefficients for determination as well as the cut off level of each risk factor and their effects on hearing.

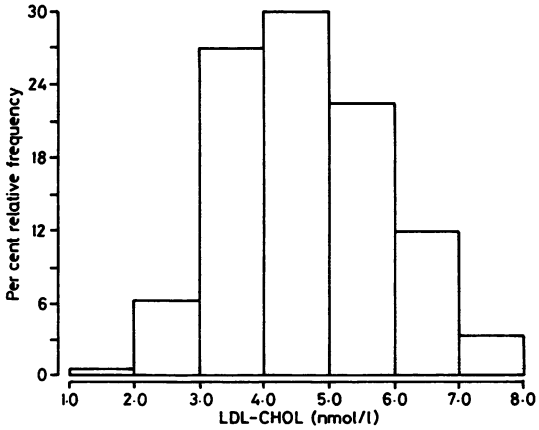

Fig 2 *Distribution of subjects according to plasma LDL-cholesterol concentration.*

EXPOSURE AND AGE

The mean A-weighted equivalent noise level measured outside the earmuffs was 96–103 dB(A) and the respective figures inside the earmuffs were 83–91 dB(A). The mean duration of exposure for noise was 16·0 years with a standard deviation of 6·4 years; the average percentage use of earmuffs was 79% during the exposure period. When the wearing time of earmuffs was taken into account, the median exposure level to noise inside the earmuffs was 94·7 dB.[9] A significant correlation was found between exposure to noise and SNHL at 4000 Hz ($r = 0{\cdot}317$, $p < 0{\cdot}001$). Aging, according to Robinson's model applied to the present data, explained 7·3 dB of hearing loss on average. When the effect of age was removed by using the age correction provided by Robinson,[10] the mean SNHL at 4000 Hz measured for the group was about 19·7 dB. This is about the same (17·8 dB) as predicted from a theoretical model calculated with fixed exposure time and level.[9] The results indicate that the combination of hand/arm vibration with noise does not contribute excessively to SNHL. Moreover, the

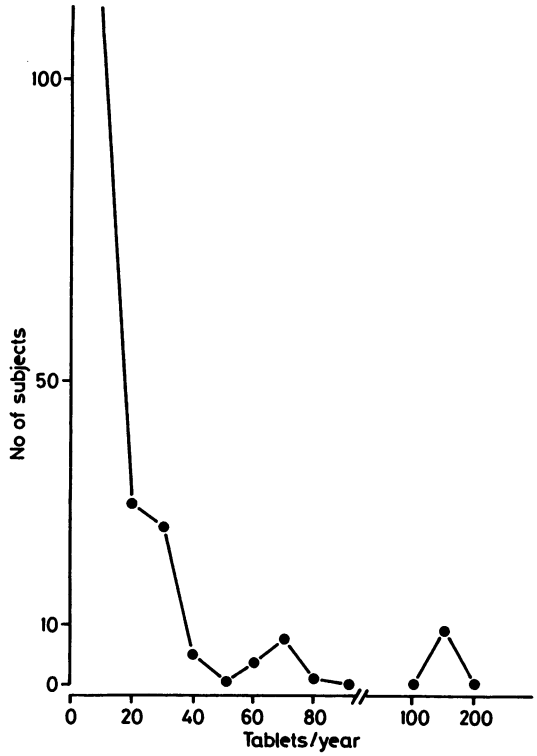

Fig 3 *Distribution of subjects according to annual salicylate consumption: one tablet contains 0·5 g of salicylate.*

measured data showed wide case to case variation when values were compared with calculated values derived from Robinson's model (fig 1). Measured values ranged from 0 to 70 dB, whereas Robinson's model ranged from 5 to 50 dB.

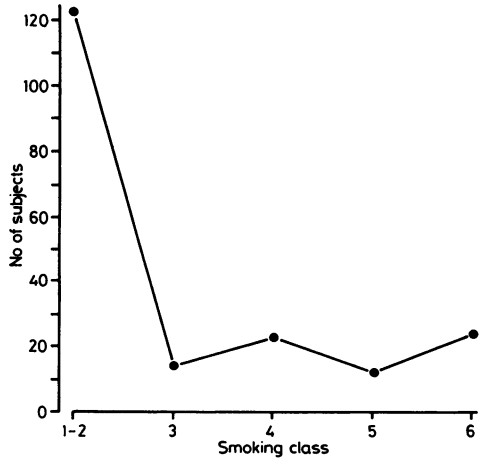

Fig 4 *Distribution of subjects according to smoking habits.*

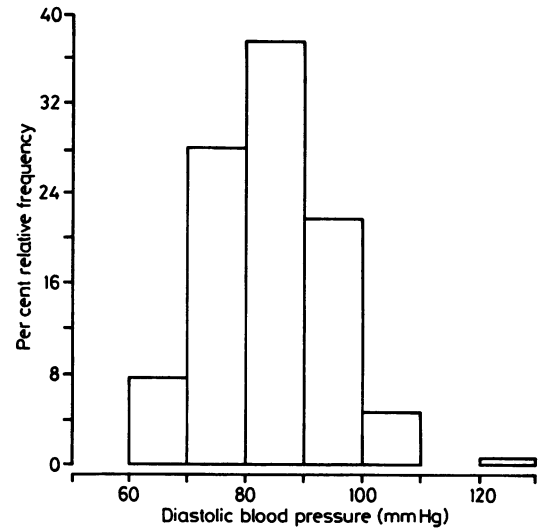

Fig 5 *Distribution of subjects according to diastolic blood pressure.*

PERIPHERAL VASCULAR DISTURBANCES

The vascular component of the vibration syndrome, known as VWF, correlated significantly with SNHL at 4000 Hz ($r = 0{\cdot}201$, $p < 0{\cdot}01$). When the subjects with VWF were removed, the average gain in age corrected hearing loss at 4000 Hz was 1·5 dB.

SERUM LIPID CONCENTRATION

We found a significant correlation between serum LDL-cholesterol level and SNHL ($r = 0{\cdot}155$, $p < 0{\cdot}05$). Removal of subjects with raised LDL-cholesterol improved the average age corrected SNHL at 4000 Hz with 0·5 dB (fig 2).

SALICYLATES

The effect of salicylate consumption on SNHL was not significant ($r = 0{\cdot}100$, p = NS). When heavy users of salicylates (50 tablets or more a year) were removed from the group, the age corrected hearing was improved by 0·9 dB (fig 3).

SMOKING

Smoking was not significantly correlated with SNHL at 4000 Hz ($r = 0{\cdot}100$, p = NS). When smokers were removed the average age corrected hearing level at 4000 Hz improved by 0·4 dB (fig 4).

BLOOD PRESSURE

Neither the systolic ($r = 0{\cdot}029$, p = NS) nor the diastolic blood pressure ($r = 0{\cdot}046$, p = NS) correlated with SNHL. The increase in diastolic blood pressure explained only 0·1 dB of the reduction of the age corrected hearing level (fig 5).

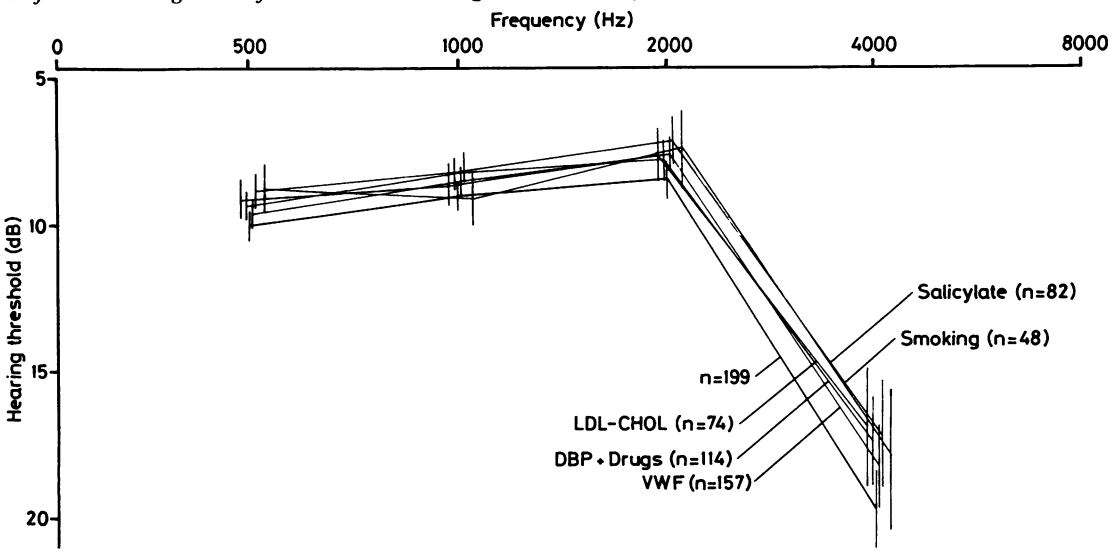

Fig 6 *Sensorineural hearing loss in 199 forest workers at different frequencies. Hearing level is shown after exclusion of different risk factors. Number of subjects remaining in analysis is shown in parentheses. VWF = Vibration induced white finger; neuropathy = subjects with history of vibration neuropathy; LDL-CHOL = raised cholesterol level; drugs = users of antihypertensive drug; smoking = smokers; DBP = raised diastolic blood pressure. Vertical bars indicate SD.*

ANTIHYPERTENSIVE TREATMENT

We found a positive correlation between treatment of antihypertensive drugs and SNHL ($r = 0{\cdot}165$, $p < 0{\cdot}05$). Possibly, therefore, the lack of an association between raised blood pressure and SNHL was masked by treatment. When the subjects with antihypertensive treatment were removed, the mean age corrected hearing level was improved in the remaining subjects by 1·0 dB.

Figure 6 gives the mean age corrected audiogram for the group at frequencies of 500–4000 Hz after stepwise elimination of subjects owing to different risk factors. Removal of the different risk factors described above improved the average age corrected hearing level at 4000 Hz by 2·9 dB. At 200 Hz the improvement was 1·6 dB.

Discussion

In Finland noise induced SNHL is the leading occupational hazard in the 1980s and accounts for about 2000 new cases annually—that is, 0·1% of the working population. In the present study we compared our noise exposure data with population data on normal hearing reported by Robinson and Sutton.[11 12] According to Robinson it is not generally realistic to compare the hearing of a noise exposed population with an age matched "otologically normal" baseline, since the noise exposed population will include those with adventitious hearing loss as well as those with noise related loss.[13]

The age related hearing loss accounted in the present study for an average of 7·3 dB. When, based on this data, the noise induced hearing loss was calculated, the measured noise induced hearing loss (19·2 dB) was close to that predicted from Robinson's model (17·8 dB). In risk assessment based on Robinson's model we used a median percentile criterion that gives the risk of hearing impairment expressed as percentage of population exceeding the specified hearing threshold. The amount of age correction differs in the available models for SNHL—for example, the ISO standard proposal would give on average 3·5 dB greater age correction for a population with a mean age of 43 than Robinson's model does.[14] The variance caused by risk factors cannot be eliminated by more severe age correction.

EFFECT OF AGING ON NOISE INDUCED HEARING LOSS

In the present study age, according to linear regression analysis, accounted for 25% of the variance at 4000 Hz but there is no indication that age should be construed as a cause of sensorineural hearing impairment. More probably, as individuals get older they will have been exposed to, or suffered from, factors responsible for such impairment.[15]

The hearing loss caused by aging is gradual and

forms a part of the progressive functional deterioration associated with the degeneration of sensory organs.[16 17]

Several factors have been suspected as contributory factors to presbycusis, including hypertension, diet, drugs, and exposure to social noise.[1 18–22] In the present study the age of the workers had a high correlation with factors provoking SNHL. Thus exposure to noise could explain 25% of the variance of age related SNHL. LDL-cholesterol was significantly correlated with age, as was antihypertensive treatment and VWF. The older subjects suffered more often from pain than did the younger and consequently used more salicylates. Thus presbycusis is contaminated by several factors each of which impedes hearing, but through somewhat different mechanisms. The histopathological findings presented by Schuknecht[17] may all be caused by these different, age related environmental confounding factors. Based on the present study we tend to disregard age as the primary reason for hearing impairment in modern society.

INTERACTION OF NOISE AND VIBRATION

The forest workers in the present study were exposed to noise of L_{Aeq} 97–102 dB and weighted vibration of 2–18 m/s^2 in their work.[5] We were unable to confirm that combined exposure to noise and hand/arm vibration generates more SNHL than exposure to noise alone.

So far, relatively few studies have considered the combined effects of noise and vibration. One study on tractor drivers showed more SNHL than could be predicted on the basis of the drivers' exposure to noise.[23] This was assumed to be the consequence of exposure to vibration. A hazardous interaction between noise and vibration has also been proposed by Taniewski and Banaszkiewics[24] who found a connection between VWF and SNHL, but there was no conclusive evidence for an interaction between hand/arm vibration and aggravated SNHL. Moreover, in the present study we found that Robinson's model for SNHL estimated rather accurately the measured SNHL, when the effect of age and exposure to noise, including the protection efficiency of earmuffs, were considered in the calculation.

EFFECT OF VWF AND SNHL

In previous studies we[5 6 8 9] and others[25 26] have found a correlation between VWF and SNHL. The reason for the potentiating effect of VWF and SNHL is not known, but we have speculated that vibration may cause "vasospasm" in the cochlea through autonomous vascular reflexes.[8]

Possibly, during exposure to noise and vibration, the sympathetic nervous system participates in the control of the circulation of the inner ear—for example, by disturbing the local compensatory changes in capillary flow during high local energy demands, as occurs during exposure to noise. Vascular changes in the circulation of the inner ear provoked by the activation of sympathetic reflexes could aggravate SNHL in this manner. The mechanism that causes excessive SNHL in people with VWF may be analogous to the development of disturbed finger circulation—that is, a continuous bombardment of central autonomic vasoconstrictor reflexes on the malfunctioning local vascular flow.[27] Nevertheless, the role of the autonomic nervous system in controlling the cochlear circulation is still vague.[28 29]

EFFECT OF SMOKING

Cigarette smoking is widely accepted as one of the risk factors related to vascular disease, particularly to coronary heart disease, but its role in hearing loss is controversial. Studies by Weston,[30] Zelman,[31] and Chung *et al*[4] showed a positive correlation between tobacco smoking and loss of hearing. In risk evaluation Chung *et al* showed that compared with non-smokers, heavy smoking increased SNHL at 4000 Hz by 6 dB.[4] The mechanism is uncertain but it has been suggested that higher carboxyhaemoglobin levels[32] may reduce the available oxygen for the organ of Corti.[4] The direct ototoxic effect of nicotine has also been suggested.[33]

In line with some other investigators[34 35] we were not able to show that smoking is a significant individual risk factor for SNHL. According to the linear regression analysis smoking cannot be more than a minor confounder[36] and decreased hearing by only 0·4 dB.

EFFECT OF CARDIOVASCULAR FACTORS ON SNHL

Rosen *et al*[18] and Rosen and Olin[37] suggest that cardiovascular risk factors are closely related to SNHL. In these and some other studies SNHL seems to be related to serum levels of fatty acids and cholestrerol.[18 37 38] In the present study we confirmed that the serum LDL-cholesterol concentration correlated significantly with SNHL. A combination of information of triglyceride level or cholesterol ratio may provide additional data on the genesis of SNHL.[39 40]

Reports on the effects of hypertension are still controversial. Although a correlation with raised arterial blood pressure and SNHL has been shown[41 42] the reciprocal connection is unknown. It has been proposed that exposure to noise may cause an increase in blood pressure that may lead to aggravated hearing loss. Furthermore, not all studies have shown a relation between raised arterial blood pressure and SNHL.[4 34] In animal studies there is, however, an indication that arterial hypertension aggravates age related hearing loss.[43–45] In the present study we could not confirm that raised diastolic blood pressure sig-

nificantly contributes to SNHL, but this presumably was due to treatment, since treatment with antihypertensive drugs correlated significantly with SNHL.

EFFECT OF SALICYLATES ON HEARING

There is still uncertainty of the effect of salicylates on hearing. What seems to be well established is that salicylates produce loss of hearing and that this loss is reversible. After high doses of salicylates few morphological changes occur in the inner ear.[46] Hawkins was one of the first to show that salicylates reduce cochlear blood flow by causing capillary narrowing,[47] which appears to be produced by swollen endothelial cells and possibly pericyte contraction.[48] In man the critical ototoxic salicylaemic level is remarkably high[49] and corresponds to the ingestion of 5–10 g of salicylic acid.[50] The acute symptoms of hearing deficit are characterised by sudden onset followed by recovery within 1–10 days.[46] Such doses of salicylates were not reported by any of the forest workers in our study.

Salicylates seem to potentiate the hearing loss induced by acute exposure to noise.[51] Eddy *et al* showed in acute experiments on chinchillas that the temporary threshold shift produced by combined noise and salicylate was significantly greater (55 dB) than that produced by noise (35 dB) or salicylate (30 dB) alone.[52] It is not known whether continual and prolonged salicylate intake in combination with environmental noise would promote SNHL in man. In the present study the moderate use of salicylates did not appear to aggravate SNHL in the working population. Nevertheless, in the age corrected hearing level, the exclusion of salicylate consumers from the rest of the forest workers caused a gain in the average hearing level of 0·9 dB. Such an increase, though not statistically significant, suggests that even moderate use of salicylates in conjunction with environmental noise may in some subjects be hazardous to cochlear function.

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