A SYSTEMS APPROACH TO INDIVIDUAL HEARING CONSERVATION

Esko Toppila

Academic dissertation

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To all workers exposed to noise

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III Starck J, Toppila E, Pyykkö I (1999). Smoking as a risk factor in sensory neural hearing loss among workers exposed to occupational noise. Acta Otolaryngol 119:302-305.

IV Toppila E, Pyykkö I, Starck R (2000). Age and Noise-Induced Hearing Loss. Scand Audiol, Submitted for publication.

V Kaksonen R, Pyykkö I, Kere J, Starck J, Toppila E (2000). Hereditary hearing loss – the role of environmental factors. Acta Otolaryngol (Suppl) 542:70-72.

VI Starck J, Pyykkö I, Toppila E (1999). Management of a sophisticated hearing conservation program. AJIM (Suppl) 1:47-50.

ABBREVIATIONS

APV	Assumed Protection Value
ATF	Acoustic Test Fixture
CEN	Centre Européen de Normalisation
CHABA	Committee on Hearing, Bioacoustics and Biomechanics
dB	desiBel
dB(A)	A-weighted desiBel
DP	Distortion Product
DPOAE	Distortion Product OtoAcoustic Emission
HCP	Hearing Conservation Program
HL	Hearing Loss
HPD	Hearing Protective Device
IHCP	Individual Hearing Conservation Program
ISO	International Standardisation Organisation
L	Sound Level
L_{Aeq}	A-weighted Equivalent Sound Level
L_{eq}	Equivalent Sound Level
NIHL	Noise Induce Hearing loss
NIOSH	National Institute of Occupational Safety and Health
NRR	Noise Reduction Rate
OAE	OtoAcoustic Emission
OHL	Occupational Hearing Loss
OSHA	Occupational Safety and Health Administration
PPE	Personal Protective Equipment
PTS	Permanent Threshold Shift
S/N	Signal to Noise ratio
SNHL	SensoriNeural Hearing Loss
SPOAE	SPOntaneous OtoAcoustic Emission
TEOAE	Transient Evoked OtoAcoustic Emission
VWF	Vibration White Finger symptom

SUMMARY

The purpose of the present study was to design an individual hearing conservation program (IHCP) and evaluate the validity of the various components of the program. The study is focused on the evaluation of the effects of environmental, biological, and medical factors, as well as the effects of aging and hereditary hearing loss on NIHL. The results were used to develop the database and an inference engine for the IHCP. The study comprised of forest, shipyard, and paper mill workers, totaling 685 subjects. Audiograms were taken by a clinical audiometry in a sound-insulated booth. Medical histories of the workers, serum cholesterol levels and blood pressure readings were retrieved from charts or questionnaires. History on the use of analgesics and tobacco smoking was obtained. Noise exposure was measured simultaneously outside (L_{ANO}) and inside (L_{ANI}) the hearing protectors (HPD) for each worker. All data were entered into the IHCP NoiseScan.

L_{ANI} and impulsiveness of noise, presence of vibration-induced white fingers with elevated serum cholesterol level, elevated blood pressure, tobacco smoking, and use of analgesics contributed significantly to the extent of NIHL. At L_{ANI} levels less than 100 dB(A), biological and environmental factors dominated the effect of L_{ANI} in the etiology of NIHL. In one pedigree with non-symptomatic hereditary hearing loss, no definite association between environmental noise and hearing loss could be shown. Elderly subjects were more vulnerable to noise than younger ones.

To increase our knowledge on the individual development of NIHL, several factors linked to the hearing loss must be collected systematically. The large number of factors involved in NIHL require the use of systematic data collection and an organized database program, with specific expert sub-programs, such as that created in the present study. Noise exposure data must include occupational, leisure-time and military service noise for the entire lifetime. The program must incorporate data of environmental, biological, and hereditary factors, as well as medical conditions and diseases. The NoiseScan program will need continuous development. The aim is to create a modern IHCP that can be used for prediction of NIHL, for workers education, to better identify hazardous working places, and to permit the reliable assessment of controlling measures to improve the safety and efficiency of workplaces.

INTRODUCTION

Noise pollution is a pervasive byproduct of industry and densely populated regions, impacting the quality of life, both socially and medically (Alberti 1998). Almost 25% of Europe's population is exposed to transportation noise exceeding 65 dB(A**),** determined as 24 h average energy equivalent noise. In some countries more than one half of the population is exposed to transportation noise (Hinchcliffe 1998). When environmental noise exceeds 65 dB(A), sleeping is disturbed and the quality of waking hours compromised. Levels exceeding 85 dB(A) can cause hearing loss. Both in the United States and Europe, 30 million people are exposed to potentially hazardous levels of noise. Approximately 400 to 500 million people are at risk of developing noise-induced hearing loss (NIHL) (Alberti 1998).

NIHL is considered to be one of the most common occupational health hazards of any country. There are no global figures available for the prevalence of NIHL. Such figures, if they did exist, would lack validity in a rapidly changing industrialized world (Alberti 1998).

There are two fundamentally different ways that excessive noise may lead to cochlear injury, mechanical or metabolic (Lim and Melnick 1971). Noise at a very high intensity may mechanically alter or disrupt cochlear structures. Cellular distortion, disorganisation of stereocilia and possible ruptures of both cell membranes (McNeil 1993, NcNeil and Steinhardt 1997, Mulroy et al. 1998) and cochlear fluid barriers will cause immediate reduction of auditory sensitivity (Flock et al. 1999). Experimental evidence suggests a critical level around 125 dB SPL (Luz and Hodge 1971**)** below which the cause of damage is predominantly metabolic. Experimental data suggest that free radicals and other highly reactive endogenous substances play a significant role in noise-induced hearing loss. The mechanisms related to metabolic changes consist of oxidative stress, synaptic hyperactivity and altered cochlear blood flow (Miller et al. 1996, Yamasoba et al. 1998**,** Puel and Pujol 1998). This primarily affects outer hair cells of the cochlea, eventually resulting in apoptosis. This process is gradual and deterioration of hearing continues over a period of years.

When NIHL is moderate to severe, it leads to speech distortion, reduced word discrimination, increased noise intolerance and tinnitus. Reduced oral communication is a social handicap (Ward 1986). NIHL also reduces the perception of warning signals, environmental sounds and music. Consequently, NIHL may lead to social isolation, decreased

worker productivity and morale, and an increase of job-related accidents (Ward 1986).

NIHL is often defined by changes seen in the audiogram; its handicapping influence is seen by changes in the speech frequencies (0.5 kHz - 2 kHz). Threshold shifts in hearing show great variability across populations of noise-exposed subjects, indicating varying levels of susceptibility against the harmful affects of noise. This variation has been described using statistical models (ISO 1999-1990, Robinson 1971, NIOSH 1974). Models that include age, gender and noise exposure as parameters, are used to explain variations of changes in hearing threshold of large populations. Because the variation in hearing threshold values is great, these statistical models are not useful in predicting the development of NIHL of individual subjects. However, individual predictions of NIHL would be of utmost importance in industrial hearing conservation programs.

In addition to noise level, age, and gender, several other factors may contribute to the variation in the vulnerability to noise. Factors such as the characteristics of noise (Campo and Lataye 1992, Starck et al. 1988a), otoxic drugs and certain solvents (Starck et al. 1988a, Morata et al. 1991, Myers and Bernstein 1965), biological and human related factors (Humes 1984, Pyykkö et al. 1986, Borg et al. 1992) and genetic factors (Barrenäs 1998, Gates et al. 1998, Kaksonen et al. 1998). If new models were developed to include consideration of all contributing factors in assessing an individual's susceptibility variability could be reduced and would no longer hinder accurate prediction, prevention and treatment of NIHL.

The purpose of the study is to design an individual hearing conservation program (IHCP) and evaluate the validity of the various components that may contribute to NIHL.

REVIEW OF THE LITERATURE

The impact of hearing loss on man

To analyze the impact of hearing loss on man, it is important make a distinction between impairment, disability and handicap (WHO 1980). Impairment refers to functional abnormality. In NIHL impairment refers to alteration in auditory system, such as loss of hearing sensitivity or decreased frequency resolution. Hearing disability refers to the functional limitations caused by impairment in everyday activities, primarily where communication is concerned. The handicaps are the social consequences of impairment. In NIHL the handicap refers to social consequences of communication difficulties, such as social isolation and unemployment.

Hearing impairment may comprise the following symptoms (Hétu et al. 1995):
- The individual threshold of sound detection is decreased.
- The increase in loudness is distorted when the sound level increases.
- Difficulties in resolving neighboring sounds.
- Ability to detect gaps in an ongoing sound is reduced.
- Ability to localize the sound sources is reduced
- Persistent tinnitus

In working conditions workers with hearing impairment require a Signal to Noise (S/N) ratio up to 25 dB higher than those of normal listeners for detecting, recognizing and localizing the sound (Hétu et al. 1995). Due to the characteristics of the warning signals in industry and for the necessity to wear hearing protection, workers with hearing impairment are more prone to accidents than workers with normal hearing. Because of a loss of frequency resolution, the S/N ratio in communication must be up to 10 dB higher among hearing impaired listeners (Plomp 1986).

In daily communication subjects with NIHL experience disabilities in communication when they are facing less than ideal conditions, for example, on a phone, varying levels of background noise, reverberant rooms, and in group conversations (Hallberg and Barrenäs 1993, Hétu et al. 1995). Because the onset of hearing loss is deceptive, people tend to avoid these disabling situations. In the long run this avoidance process results in changes in the lifestyle of people with hearing impairment (Hallberg and Carlsson 1991).

The resulting handicap caused by NIHL affects the social and family life in different ways. The partner of a person with NIHL needs to pay

attention when communicating with the impaired family member. The verbal contact should be performed under visual conditions and the information content must be confirmed. The handicap affects the unimpaired family member by forcing them to keep the conversions brief. Other consequences may include setting higher volumes when watching television or listening to music, loud speech and the increased social dependence of the impaired partner (Hétu et al. 1993).

The audiogram

The standard measure for hearing impairment is the audiogram, which is a written record of a person's hearing level measured with certain pure tones (Sataloff and Sataloff 1993a). In the audiogram pure tones at the following frequencies are used: 0.25, 0. 5, 1, 2, 3, 4, 6, 8 kHz (ISO R389-1964). In the audiogram 0 dB represents the average normal hearing of young people between the ages of 20 and 29 and was established from the data obtained in 1935-1936 (ASA-1951). In the sixties the audiometric 0 dB level has been adjusted to be approximately 10 dB more sensitive (ISO R389-1964**,** ANSI S3.6-1969).

The range for normal hearing is 0-25 dB (ISO 1999-1990). Sataloff and Sataloff (1993a) suggest, however, that a subject with a 15 dB hearing loss at most frequencies has a hearing deficit (Sataloff and Sataloff 1993c). The correlation of the audiogram with subjective evaluation and handicap varies between 0.2 and 0.5 (Barrenäs and Holgers 2000). The subjective evaluation of disability correlated somewhat better with the audiogram than the handicap.

Disability and handicap are expected as a result of a hearing threshold level exceeding the limits of normal hearing at speech frequencies of 0.5 kHz – 2 kHz (Sataloff and Sataloff 1993c). The shape of the audiogram can reveal the cause of hearing loss. NIHL is most profound at frequencies of 3 kHz - 6 kHz (Burns 1973). NIHL exists in both ears and is usually greater in the left ear (Pirilä 1991). It has been debated whether or not the audiogram is sensitive enough to monitor changes in the inner ear in the presence of NIHL. Experiments on animals have shown that a hair cell loss in cytocochleograms do not necessarily correlate with hearing loss measured in audiograms. Hamernik et al. (1989) reported that as many as 75% of the outer hair cells can be lesioned without causing a substantial change in HL in a certain frequency range.

The use of a clinical audiometer or a screening audiometer may cause considerable differences in the recorded hearing threshold values. Melnick (1984) proposed that when working with clinical audiometry, a

10 dB shift at any frequency is significant, but with screening audiometry a 15 dB step should be used. The automatic audiometer is more accurate than the clinical audiometer that uses 5 dB steps. Royster et al. (1980) showed that the variability in clinical audiometry is greater than in automatic audiometers. Consequently, they proposed the use of automatic audiometry in the screening of hearing in industry. The background noise of a sound-proof room is seldom measured and may exceed the permissible levels for hearing measurement. In industry the hearing sometimes has been measured in non-isolated but quiet rooms. The environment does not allow measurement of the 0 dB level in audiometry. Royster and Royster (1986) pointed out that calibration may not be adequately carried out, and therefore, the audiogram results may be biased. These authors proposed the normal controls with stable hearing to be mixed with the noise-exposed population in addition to relevant calibration. The instructions given by the technician may affect the accuracy of hearing threshold value evaluation in audiograms up to 10 dB (Hinchcliffe 1997).

The equal energy principle

As noise in the workplace tends to vary and workers are often exposed to different tasks with different noise levels, a method is needed to combine the different levels to single a number that is related to risk of hearing impairment. The equivalent noise level (L_{eq}) is the most commonly used one at present. It is the sound level which, when integrated over a specified period of time, would result in the same energy as a variable sound over the same time (Earshen 1986).

$$L_{eq} = 10 * \log\left(\frac{1}{T}\int_0^T \left(\frac{p(t)}{p_0}\right)^2 dt\right) \qquad [1]$$

where p(t) = sound pressure level
p_0 = reference sound pressure level ($2\ 10^{-5}$ Pa)
T = duration of exposure

If the exposure consists of several exposure periods they can be combined by using the following equation:

$$L_{eq,tot} = 10 * \log\left(\frac{\sum_i 10^{L_i/10} * T_i}{\sum_i T_i}\right) \qquad [2]$$

where T_i = duration of i^{th} exposure
L_i = level during i^{th} exposure

The total noise dose (L_{Ex}) is the total acoustical power that has entered the ear. It is calculated from the equivalent levels using the following equation.

$$L_{Ex} = L_{eq} + 10 * \log(\frac{T}{T_0}) \quad [3]$$

where L_{ex} = Exposure
L_{eq} = equivalent level
T = length of exposure usually in years
T_0= reference time usually 1 year

The vulnerability of the human inner against noise is frequency dependent. The mid-frequencies, 2-6 kHz, are the most damaging ones. The vulnerability decreases as the frequency decreases or increases. To take into account this frequency dependency, the so- called A-filter was created. The A-filter is a physical filter corresponding to the loudness curve of human ear at low sound pressure levels (IEC 651-1979). The A-filtered equivalent level is marked L_{Aeq}. The other filter used in noise risk evaluation is C-filter, which is a presentation of the loudness curve of human ear at high sound pressure levels. This filter is used in the risk assessment of impulse noise. The risk of impulse noise is often related to the C-weighted peak level, most often noted as $L_{C,peak}$.

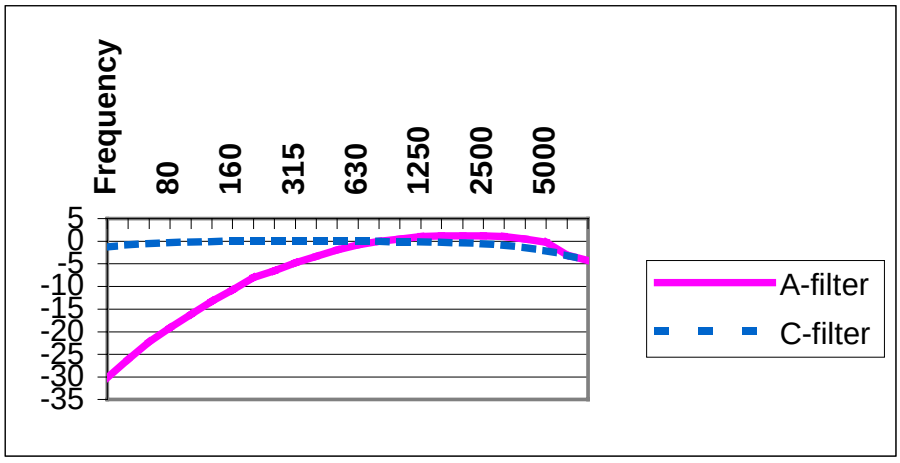


Figure 1. The characteristics of A- and C-filters.

Modeling NIHL

One of the first damage risk criteria based on exposure to steady-state noise, has been proposed by Kryter (1966). The damage risk criteria is composed from a group of curves which were based on laboratory experiments on the development of Temporary Threshold Shift (TTS). Data collected in 1955-1956 on permanent threshold shifts (PTS) in

workers exposed to industrial noise was also included. The Committee on Hearing, Bioacoustics and Biomechanics (CHABA) (Kryter 1965) used the data to exposure HL contour as a function of exposure. This was the first norm proposed for evaluation of hazardous noise.

The first large epidemiological study on the relationship between noise exposure and hearing loss was made by Baughn (1973). His studies from the early sixties' involved a large worker population (6835) under stable work locations and conditions with stable noise exposure (Baughn 1966, Baughn 1973). The exposure durations went up to 45 years with average noise exposure levels of 78, 86 and 92 dB. Baughn (1973) recommended that the hearing loss of subjects exposed to the 78 dB(A) noise would be considered as representing typical non-noise-exposed males. According to his data, it is possible that factory workers suffer more sociocusis and nosocusis than the general population.

Burns and Robinson studied 759 subjects of which 422 males were exposed to 4 classes of noise ranging from 87 dB(A) to 97 dB(A) (Burns & Robinson 1971). The maximum exposure was about 49 years. As controls 97 subjects not exposed to noise were included in the study. The population was screened to be otologically normal. The authors developed a mathematical generalization of the predicted hearing loss (Robinson and Shipton 1977, Robinson 1968). This model introduced the energy principle to enable the combination of different sound levels (Burns 1973). Hearing loss was divided into two parts: age dependent hearing loss (presbycusis) and NIHL. After correcting the model for age and gender, the distribution of hearing loss can be calculated by using the given formulas. The separation of presbycusis from NIHL leads to a predicted hearing loss that is smaller than those found in other models, partly because the material was rigorously and otologically screened (Suter 1994).

Passchier-Vermeer (1974) summarized the results of 19 smaller studies 12 of which have 50 or less cases. The data agrees well with the Robinson’s data at some frequencies but at other frequencies large differences were found. One reason was the deviation in the definition of audiometer zero level used on some of the studies (Glorig and Nixon 1960).

Johnson (1973) prepared a report for the US Environmental Protection Agency (EPA) on the prediction of NIPTS from exposure to continuous noise. This report is based on the data of Burns and Robinson (1971) and Passhier-Vermeer (1974). The data of Baughn (1966, 1973) was also used in evaluating the hearing loss of the non-exposed population. For this reason the hearing loss of the non-exposed population is somewhat

less in this report than in works by Burns and Robinson (1960) or Passhier-Vermeer (1974).

The National Institute for Occupational Safety and Health (NIOSH) in the USA conducted a study on industrial workers exposed to noise levels approximately 85, 90 and 95 dB(A) and control subjects exposed to levels below 80 dB(A) (NIOSH 1974). The study consisted of an otologically screened population of 792 noise-exposed subjects and 380 controls. Hearing loss was tabulated by a function determined by exposure level and duration. Using these tables, the occurrence of NIHL could be calculated by subtracting the control values from hearing threshold values measured in noise-exposed subjects.

The International Organization for Standardization (ISO), published in 1975, a standard for assessing occupational noise exposure for hearing conservation (ISO 1999-1975). The information on which the standard is based is not identified, but according to Suter (1994) the data of Baughn (1966, 1973) form the bases of this standard. The ISO-standard adopted the equal-energy principle for the combination of different sound exposures from the Robinson model. According to ISO tables 50% of non-noise-exposed people have a hearing loss, whereas Robinson & Sutton (1979) demonstrated a 10% and US public health services study (Glorig and Roberts 1965, Rowland 1980) a 20% prevalence of hearing loss for non-noise-exposed people. The ISO-model was corrected and a mathematical form for the hearing loss was given in order to produce the present standard model (ISO1999-1990).

The standard model –ISO 1999

The ISO-model (ISO 1999-1990) uses three input parameters: age, exposure to noise, and gender in the evaluation of NIHL. Exposure to noise is evaluated using the equal energy principle. Based on these parameters the distribution of NIHL can be calculated. The variation is large; for men the difference between 10% and 90% percentile of hearing loss is 60 dB when the subjects are exposed to a noise level of 100 dB(A) for 30 years (Fig 2). According to the ISO-model women are somewhat less vulnerable to noise than men.

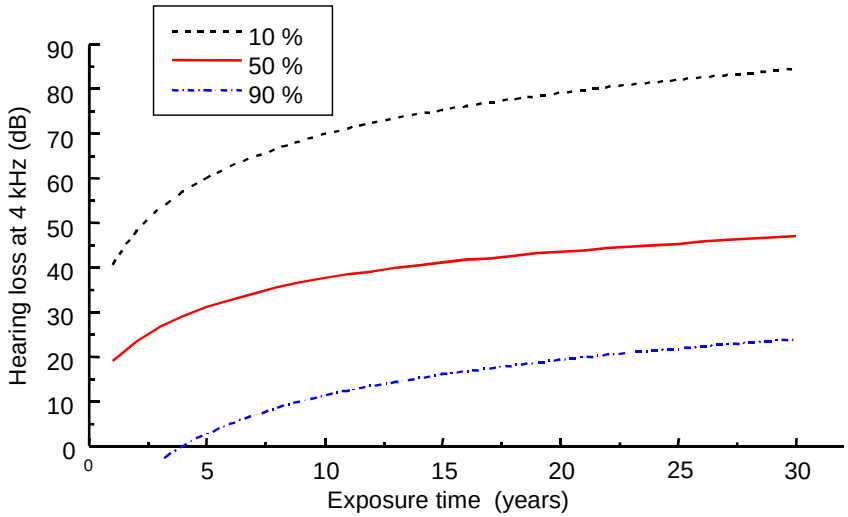


Figure 2. The hearing loss of a male worker exposed to 100 dB(A) noise as a function of time.

The ISO standard (ISO 1999-1990) is used to estimate the noise-induced hearing loss (NIHL). According to the standard, the permanent threshold shift (PTS) is due to the combination of aging and noise. The effect of aging H_Q is according to the standard as follows:

$$H_Q = a(Y-18)^2 + kS_u \qquad \text{When } 0.05 < Q < 0.5 \qquad [4]$$

$$H_Q = A(Y-18)^2 - kS_i \qquad \text{When } 0.50 < Q < 0.95 \qquad [5]$$

Where a= frequency dependent coefficient given by standard (Table 1 annex A)
$S_u = b_u + 0.445(Y-18)^2$
$S_i = b_i + 0.356(Y-18)^2$
b_u and b_i are genre and frequency dependent coefficient given by the standard (Table 2 Annex A)
Q is the selected fractile
Y is age in years

In this formula the first term is the mean age-dependent hearing loss and the second term is variation. The formula is valid only when Y >18.

The hearing loss due to noise (N_Q) is calculated according to the standard as follows:

$$N_Q = (u + v\log_{10}(T))(L_{EX,8h} - L_0)^2 + k(X_u + Y_u \log_{10}(T))(L_{EX,8h} - L_o)^2 \qquad [6]$$
when $0.05 < Q < 0.50$

$$N_Q = (u + v\log_{10}(T))(L_{EX,8h} - L_0)^2 - k(X_i + Y_i \log_{10}(T))(L_{EX,8h} - L_o)^2 \qquad [7]$$
when $0.50 > Q > 0.95$

Where u and v are frequency dependent coefficients
T is exposure time in years and greater or equal to 10
L_0 is frequency dependent limit value. If $L_{EX,8h}$ is < L_0 the term=0
X_u, Y_u, X_i and Y_i are frequency dependent coefficients
Q is the fractile
$L_{EX,8h}$ is the mean daily exposure

These terms are combined as follows:

$H' = H+N+HN/120$ where H' is the PTS [8]

According to the standard at low noise levels (below 90 dB A), age is a much more important factor than noise (Fig 3). Noise and age become equally important at levels above 100 dB(A).

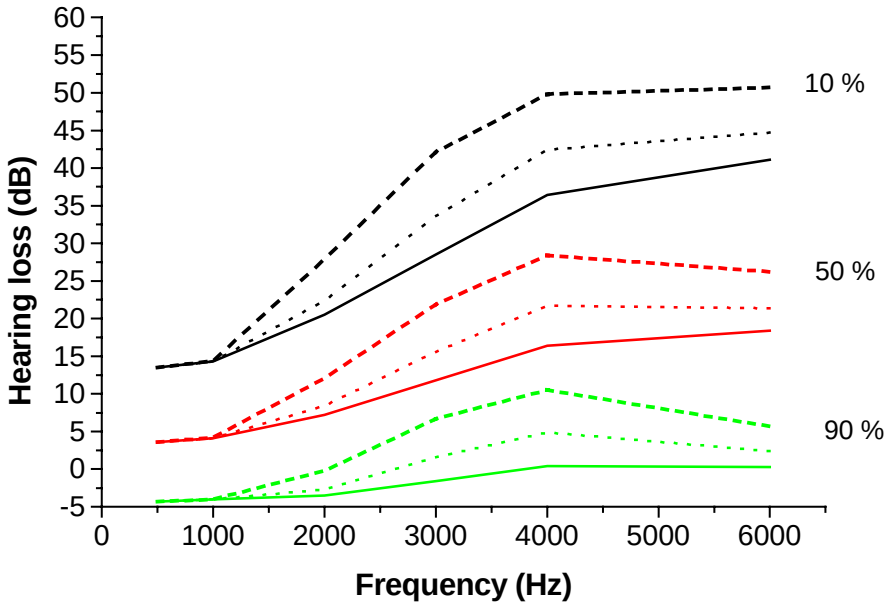


Figure 3. Hearing loss plotted against audiometric frequency. The 10%, 50% and 90% fractiles of PTS of a 50 year-old man according to the standard ISO 1999-1990. Solid line indicates age-related deterioration of hearing (presbycusis), dotted line 85 dB daily exposure for 30 years and dashed line 90 dB daily exposure for 30 years.

The ISO-standard is intended to estimate the NIHL of the population, free from auditory impairment for other reasons. The standard may also be used for estimating the permanent effects of noise on the perception of

everyday acoustic signals. Although the standard is intended for population studies, it is not very accurate at the individual level. However, it can be used to evaluate the probability of NIHL in individual subjects. In this case the fractiles are drawn with the known exposure and the audiograms are printed above, to provide a comparison on how well noise can explain the PTS.

The large variation has been explained by several factors like pitfalls in the equal energy principle, other noise exposure, confounding biological and environmental factors and individual susceptibility factors (Borg et al. 1992, Campo and Lataye 1992, Pyykkö et al. 1988).

Comparison of the different models

The action levels in the European countries are not risk limits. Table 1 shows the percentage of population with a NIHL greater than 25 dB at speech frequencies (500, 1000 and 2000 Hz) after 40 years of exposure to noise of common action levels in European countries. The NIOSH model has the greatest risk percentages due to the fact that it is using 3000 Hz instead of 500 Hz. The EPA and ISO models are in good agreement.

Table 1. Estimated percentages of the population at risk of exceeding an average hearing threshold level of 25 dB at 500, 1000 and 2000 Hz as a function of average noise exposure for 40 years according to three different models.

Noise level dB(A)	**80**	**85**	**90**
ISO (1990) (%)	0	10	21
EPA (%)	5	12	22
NIOSH (%)	3	15	29

Evaluation of exposure

The evaluation of noise exposure is based on noise level or noise dose measurements. In noise level measurements a noise level meter is installed in the relevant place and the mean noise level is measured over an appropriate time period (Michael and Michael 1993). The equivalent A-weighted sound pressure level calculation is based on the exposure times and energy level of noise during exposure period. The total noise exposure is obtained by summing up all exposure periods. The procedure is somewhat simpler with noise dose measurements. Dosimeters are mounted on the worker and the dose is measured over a representative

period. The accuracy of these measurements depends on several factors like the calibration of the measuring device (Michael and Michael 1993), the accuracy of the instruments (ISO 9612-1997), how representative the measurement periods are (ISO 9612-1997), and the selection of the measurement place, among others. At worst, these factors may reduce the accuracy of the measurement by as much as ± 8 dB.

The equal energy principle provides a good approximation for the vulnerability of ear in steady state noise as in process industry. However, the time domain characteristics of noise have been shown to affect the harmfulness of noise; the risk of NIHL is higher in the occupations where workers are exposed to impulse noise. In several occupations the impulses are so rapid that they contribute only a minimal amount to the energy content of noise. For example in impulsive noise among shipyard workers, there was a 10 dB higher hearing loss than could be predicted by the model. The observed hearing levels were very consistent with the model for forest workers, where the noise was not impulsive (Starck et al. 1988). Pauses in exposure allow for some recovery, and the resulting hearing loss is not as great as is proposed by the equal energy principle in animal experiments (Campo et al. 1992). Among paper mill workers, the hearing loss among those who used hearing protective devices (HPDs) on average, 50% of the time, was less than the HL among those who never used HPD. The difference could not be explained by the small change in exposure (Starck et al. 1996). The authors concluded that even temporary use of HPDs may provide relatively good protection against HL.

The ear may also become toughened against noise in certain conditions. In animal studies it has been shown that exposure to non-traumatizing noise before a traumatizing noise is introduced, decreases the NIHL (Canlon et al. 1988). This effect has been shown recently in humans (Waraich et al. 1998).

A HPD can reduce the exposure significantly. The nominal attenuation, recommended by the manufacturers, varies from 11 dB to 35 dB, depending on the HPD and the frequency contents of the noise (www.eisosh.org). This nominal attenuation is obtained if the usage rate is more than 99% of the exposure time (EN 458-1993) if the condition of the HPD is good (Pekkarinen 1987). However, the use of manufacturers' data for the evaluation of attenuation has been questioned by the following studies:

- Based on studies made by several authors, Berger (1983) concluded that the Noise Reduction Rate (NRR) index overestimates the performance of hearing protectors. He suggested that 10 dB should be subtracted from the NRR values given by the manufacturers, although

the actual differences varied from 8 to 18 dB for earplugs and from 5 to 18 dB for earmuffs. He demonstrated that the reduction of NRR is caused by a lower mean attenuation and an increased standard deviation.

- Pfeiffer (1992) observed that with poor fitting, the mean attenuation was lowered 2.3 - 5.7 dB for earmuffs, 13.3 dB for foam plugs, and 5.9 - 8.7 dB for glass earplugs. A good installation improved the attenuation by 3.8 dB for foam plugs and 4.8 dB for glass earplugs compared to the poor installation case.
- Casali et al. (1991) found that under working conditions the laboratory data overestimated the performance of foam plugs by 5.7 - 8.3 dB and of premolded earplugs by 6-10 dB. In their study the laboratory results provided a better estimation for earmuffs, the difference being about 2 dB.
- Merry et al. (1992) studied the effect of the fitting procedure on the attenuation of plugs. When the test conductor gave substantial fitting assistance, the attenuation was about 8 dB higher than when the user fitted themselves according to written instructions from the package. The user fit method best approximated the field data.

However, the use of mean attenuation to characterise the effectiveness of HPDs may be somewhat misleading. Based on a study among paper mill workers it was observed that the distribution of attenuation composed of two partly overlapping gaussian distributions (Toppila 1998). The other one corresponded well to the attenuation data given by the manufacturers. The authors concluded that it is possible to obtain protection, which corresponds to the manufacturers' data if the protectors are in good condition, the user is motivated, and the usage rate is 100%.

The HPDs attenuate industrial impulse noise even more effectively than steady state continuous noise. This is due to the high frequency contents of impulses, which are attenuated effectively in earmuffs. Even though the earmuffs reduce the impulse noise rate, workers in the metal industry are still exposed to more impulsive noise than workers in paper mills and forestry (Starck et al. 1988).

Other noise exposure

Shooting and hunting increase the risk of hearing loss (Pekkarinen et al. 1993). Forest workers who were exposed to gunfire noise had an additional 10 dB hearing loss than those who had only occupational exposure to chain saw noise (Pekkarinen et al. 1993). NIHL occurs at a younger age in the military than in other groups of workers exposed to excessive noise (Ylikoski et al. 1995). In branches of the military where large caliber weapons are used, the risk is especially high, as is the

development of NIHL. Hearing protection has proved to be less effective here, due to the non-linearity of the attenuation against very high peak levels and the low frequency components of large caliber weapons (Ylikoski et al. 1987, Starck et al. 1987, Starck and Pekkarinen 1992).

Exposure to gunfire noise is difficult to assess, since there is no standard method available to evaluate its effect on the inner ear. The existing measurement methods can be divided into two categories; the peak level methods and energy attenuation methods. With the peak level methods (Pfander 1975, CHABA 1968**,** ACGIH, 1997) the risk for hearing loss is related to the peak level and duration. These methods do not provide a way of combining different gunfire exposures or gunfire exposure with work noise exposure to a single exposure index. The latest approach is to apply the energy attenuation of the impulse in risk assessment (Dancer et al. 1996, Patterson and Johnson 1996, ANSI 2000).

The most frequent exposure in free time is exposure to music. The highest music exposure rates are from rock music. Noise levels in a concert or a disco may be around 100 dB (Smith et al. 2000). Thus, only one attendance a week causes an exposure exceeding the occupational action limit. Similar levels are reported in the users of portable cassette recorders (Airo et al. 1996). In classical music the levels are lower but the musicians still have a risk of hearing loss. Among musicians the use of HPDs are low, but use is increasing, notably during rehearsals (Sataloff et al. 1993). The role of music in NIHL is not well understood. In studies conducted among young people, no changes in the audiogram have been found (Davis et al. 1998). It has been suggested that the effect of music exposure would show up later. This is in accordance with recent studies of Davis et al. (1998), where people exposed to music had more frequent and severe tinnitus than people with less exposure to music. The severity of tinnitus was shown to correlate with hearing loss (Davis et al. 1998a).

Other exposure

The effect of tobacco smoking on hearing loss is controversial. Smoking has been found to cause hearing loss (Gruckshanks and Klein 1998, Rosenhall et al. 1993). Thus, it could be found as a risk factor for NIHL. Some authors have reported that smokers have an increased risk of NIHL (Barone and Peters 1987). However, many authors (Drettner et al. 1975, Friedman et al. 1969, Pyykkö et al. 1988, Fuortes and Tang 1995) were not able to demonstrate that smoking could be a significant risk factor in NIHL. This may be due to the fact that the effect of smoking may be obscured by other risk factors such as aging, blood pressure and VWF, or

cessation of smoking, and therefore, confounding the statistical analysis (Pyykkö et al. 1988).

The acute or toxic effects of non-steroid analgesic drugs on hearing loss, is well documented in the literature, but little is known about its long-term effects. After high doses of salicylates, very few morphological changes occur in the inner ear (Myers and Bernstein 1965). Hawkins (1967) was one of the first investigators to demonstrate that salicylates reduce cochlear blood flow by causing capillary narrowing. The narrowing of vessels appears to be caused by swollen endothelial cells and possibly pericyte contraction (Smith et al. 1985). In humans the critical ototoxic salicylic level is high (Graham and Parker 1948), corresponding to the ingestion of 10-15 g of salicylic acid a day (Grifo 1975). The acute symptoms of a hearing deficit are characterized by a sudden onset, but this reverses within one to ten days (Myers and Bernstein 1965).

Acute exposure to noise seems to potentiate the hearing loss induced by salicylates. Eddy et al. demonstrated in acute experiments on chinchillas that a temporary threshold shift produced by combined noise (85 dB) and salicylates (20-40 mg/100 mg) was significantly greater (55 dB) than that produced by noise (35 dB) or salicylates (30 dB) alone. So far, it is not known whether salicylates in combination with environmental noise would promote a permanent NIH (Pyykkö et al. 1989).

Ethyl benzene is a very potent ototoxic chemical in rats (Cappaert 2000), but guinea pigs are dramatically less susceptible to its ototoxic effects. Cappaert also found that a synergistic interaction between noise and ethyl benzene can occur, particularly in outer hear cell counts.

The exposure to solvents has been known to cause a hearing loss. In the paper mill a larger proportion (23%) of the employees in the chemical section exposed to organic solvents had a pronounced HL despite lower noise levels (80–90 dB), compared to workers in a non-chemical environment who had noise levels of 95–100 dB (Bergström et al. 1986). A combined exposure to toluene and noise increased the risk of hearing loss by 11 times among rotogravure printing workers (Morata et al. 1991). In this study, exposure to noise or toluene alone increased the risk of NIHL by four and five times, respectively. The effect of solvents depends on the solvent concentration (Mäkitie 1997). Sass-Kortsak et al. (1995) did not observe any interaction between noise and low-level styrene exposure in the fiber-reinforced plastics manufacturing industry. In the glass-fiber reinforced plastic industry in the Netherlands, it has been found that at higher levels of styrene there is a significant change in hearing threshold at high frequencies (Muijser et al. 1988).

Individual susceptibility to NIHL

Several biological factors have been studied in their role to aggravate NIHL. In population surveys, advanced hearing loss in non-exposed populations have been attributed to biological and environmental factors (Hinchcliffe 1973). Nevertheless, the data on NIHL in carefully controlled studies show considerable case-to-case variation, indicating that individual susceptibility also plays a significant role (Chung 1982, Pyykkö et al. 1989). Factors such as elevated blood pressure (McCormic et al. 1982, Pyykkö et al. 1989), altered lipid metabolism (Rosen and Olin 1965), the vibration white finger (VWF) (Pyykkö et al. 1986, Pyykkö et al. 1988), and genetic factors (Gates et al. 1999) are believed to contribute to NIHL.

An association between elevated blood pressure and NIHL has been described by some researchers (Johansson and Hansson 1977, Andren et al. 1980), but the relationship has not been found in all studies (Drettner et al. 1975). Animal studies have indicated that arterial hypertension accelerates age-related hearing loss (McCormic et al. 1982, Borg 1982). An antihypertensive medication may partly mask the effect of elevated blood pressure on NIHL (Pyykkö et al 1989).

Skin pigmentation seems to affect the vulnerability to NIHL. A study among African-Americans showed a somewhat better average in hearing threshold levels than caucasians (Royster et al. 1980). This has been attributed to higher levels of melanocytes and its protective capability in the inner ear against noise damage (Barrenäs and Lindgren 1991, Barrenäs 1998).

Many authors have found a significant and relatively large difference in vulnerability between men and women (Berger et al. 1978, ISO 1999-1990). These results may be explained by women's smaller exposure to free time noise, especially to gunfire. In a recent study where these factors were controlled more accurately, no difference was found (Davis et al. 1998b).

The role of age

Age is one of the factors that emerge in risk analysis; in many cases it overrules the exposure data (Pyykkö et al. 1986, Royster and Royster 1986, Pyykkö et al. 1989, Franks et al. 1989). This does not mean that age, in itself, would cause hearing loss (Robinson and Sutton 1979, Robinson 1988). Several factors have been suspected to underlie the causes of presbycusis, such as hypertension, dietary habits, drugs and

social noise exposure. For example, Rosen et al (1964) and Hinchcliffe (1973) suggest that if all the environmental and disease processes could be controlled, no prominent age-related hearing impairment could be demonstrated. Driscol and Royster (1984) concluded in their study on the etiology of SNHL and aging that the existing databases are contaminated by environmental noise, and therefore there is an overestimation of the effect of age on hearing. Stephens (1982) examined consecutive presbycusis patients who were seeking rehabilitation and found out that in 93% of these cases there was an underlying cause for presbycusis. In a prospective study on the causes of hearing loss in the elderly, Lim and Stephens (1987) found out that 83% of the cases had a disease condition that was associated with the hearing loss. About 30% of the subjects took medication known to be ototoxic. Humes (1984) made a critical review on the causes of hearing loss and discovered several confounding factors that affect age-related hearing loss.

Genetic factors

Research using contemporary molecular biological tools have provided insights into the genetic factors involved in the deterioration of hearing. Genetic hearing loss is divided into hereditary or sporadic gene transformations (Morton 1991). The hearing loss may appear in a syndromic form having specific symptoms or signs that are relatively easy to detect. It may also appear in non-syndromic form, without specific symptoms or signs and are often difficult to separate from NIHL. Non-syndromic form often increases with aging. The genetic background of non-syndromic hearing loss is quite heterogeneous, and to date, 33 different gene loci for non-syndromic hearing loss have been localized (7 autosomal recessive hearing loss, 11 autosomal dominant hearing loss, 1 x-linked HL and 6 mitochondrial mutations) (Van Camp and Smith 2000). From these gene mutations the connexin 26 (Cx26) mutation is most frequent and can be observed in 3% of the population (Green et al 1999). In recessive form the Cx26 mutation is observed in 50% of the population (Green et al. 1999). In the extension of the Framingham study, a good correlation was found with early onset of hearing loss and extent of presbycusis within the family (Gates et al. 1999). In males the relationship was not as evident as with females, which could be linked to environmental noise as a confounding factor (Gates et al. 1999).

There are insufficient data available on the relationship between NIHL and genetic background. Such data could be crucial in explaining the great variability of noise vulnerability in population studies. The results of the Framingham (Gates et al. 1999) study indicate that genetic factors play a significant role in the development of age-dependent hearing loss and consequently in NIHL. In future subjects with indications of

genetically induced hearing loss, they might be tested for a possible defect in the Cx26 gene and possibly also in some mitochondrial defects. The number of new known gene mutations is constantly increasing and the current situation can be verified by looking at the home page for genetic hearing loss (Van Camp et al. 2000).

Legislative approach to protection of workers

In the European community the protection against noise is controlled by directives 86/188/EEC which set the requirement of the workplaces, 89/656/EEC which set the requirements concerning the use of personal protective equipment (PPE) and 89/686/EEC which set the requirement to test the PPE.

Testing of HPDs

The directive 89/686/EEC concerning personal protective devices, sets (in annex II) the basic health and safety requirements for personal protective devices. Based on these requirements the development of standard series EN 352 was started. At present EN 352 –1 for earmuffs, EN 352-2 for earplugs and EN352-3 for helmet mounted earmuffs are available.

When testing in accordance with EN352-1 the mechanical tests also serve as preconditioning. The earmuffs are cycled 1000 times with a 25mm movement in the width of headband, the cup rotation and size are evaluated and a drop test is performed. Optionally the drop test can be performed after preconditioning to a temperature of –25 C. The headband force and pressure of cushions are measured. The change in headband force is measured after a conditioning where the protectors are set in a 40 C water bath for 24 hours. Optionally, the protectors may be used during the water immersion. The acoustical tests comprise of an objective test, according to ISO 4869-3, and a subjective test according to ISO 4869-1. Finally the flammability of protectors is tested.

The objective test, intended for quality control purposes, is an insertion loss measurement and is made using an artificial test fixture. The subjective measurement is made using 16 test persons as a threshold measurement with and without hearing protectors. The H-,M-,L- and SNR-indices, and assumed protection values (APV) are given as results. The H-, M- and L-indices describe the attenuation performance in industrial noise tuned to high, medium and low frequencies. The SNR index describes the performance in average industrial noise. The APV evaluate the performances of the protectors at octave bands with

frequencies from 125 Hz to 8000 Hz. All the figures have a statistical character, that is, 84% of people get better protection than that indicated by the indices. In addition, this standard sets requirements for the users' information and it's availability. The comfort index issue, which is strongly inherent in annex II of the directive 89/686/EEC, is not covered in the standard. This issue has been studied by many groups (Lataye et al. 1983, Ming-Young et al. 1991, Mimpen et al. 1987), but so, far none of the methods are generally approved.

Use of HPDs

The attenuation results obtained using EN 352-1 should be used in the selection of hearing protectors. The selection criteria are given in standard EN 458-1993, covering the selection, use and maintenance of protectors. According to EN 458-1993 the selection of hearing protectors should be made in such a way that usage rate is as high as possible. The EN 458-1993 recommends that protectors be as light as possible while still being able to provide enough protection. To do this the sound pressure level inside the protector must be evaluated. Bearing in mind that the indicated attenuation is obtained only by 84% of the users, EN 458-1993 recommends that several models be made available and the users should select from them.

EN 458-1993 gives four methods of how these indices can be used to evaluate the level inside the HPD. In the octave band method the octave spectrum of noise is measured. From each octave band the APV is subtracted to get the noise spectrum inside the HPD. Finally, the levels inside the HPD are added up to obtain the noise level inside the protector (L_A). In the HML method the A- and C-weighted noise levels (L_A and L_C) are measured. The difference (L_C-L_A) provides an estimate of the noise frequency characteristics. Positive values indicate low-frequency noise and negative values high-frequency noise. Based on the difference and given H-, M, and L-values an estimate of the attenuation of HPD can be obtained.

In the HML-check method the noise is divided into low, medium and high frequency noise. In the case of low-frequency noise the L-value and in the case of medium and high frequency noise the M value is used as an estimate of the attenuation of the HPD. The last method is the SNR method. In this method the SRN-value is directly subtracted from the noise level.

The octave band method is the most accurate, and the HML-method is almost as accurate. The HML-check method provides a reasonable estimate for attenuation. The SNR-method gives a reasonable estimate in

a typical industrial environment, but considerable underestimation occurs in low frequency noise.

The attenuation of each protector is rated according to the following table.

Table 2. The rating of the attenuation according to EN 458-1993. L_{AL} is the national action level (85 dB in Finland) and L' is the effective noise level inside the HPD.

Rating	**Criteria**
Insufficient:	$L' > L_{AL}$
Satisfactory:	$L_{AL}-5 < L' < L_{AL}$
Good:	$L_{AL}-10 < L' < L_{AL}-5$
Satisfactory:	$L_{AL}-15 < L' < L_{AL}-10$
Overprotection:	$L' < L_{AL}-15$

In working conditions the attenuation of HPDs also depends on environmental factors. In a cold environment, as in forest work, the hardening of the cushion rings causes a slight but systematic worsening in the attenuation. In the winter forest workers use helmet liners, which in some cases nullifies the attenuation of the hearing protectors (Starck and Pekkarinen 1987). Worn out cushions and reduction in spring force also affect the attenuation to such an extent that it is difficult to assess protectors in continuous use.

Protection against noise

The approach to the protection of workers described in the directive 86/188/EEC is based on the identification of the risks in the workplace (Fig 4). Risk assessment must be done by qualified personnel. If there is risk of NIHL, the employer must develop a HCP. In HCP the first task is to evaluate the sources of noise and the possibilities to reduce the levels by technical means. If reduction of the noise source is not possible, the workers should be provided with HPDs and the workers should be informed about the risks and the correct use of the selected HPDs in an appropriate way.

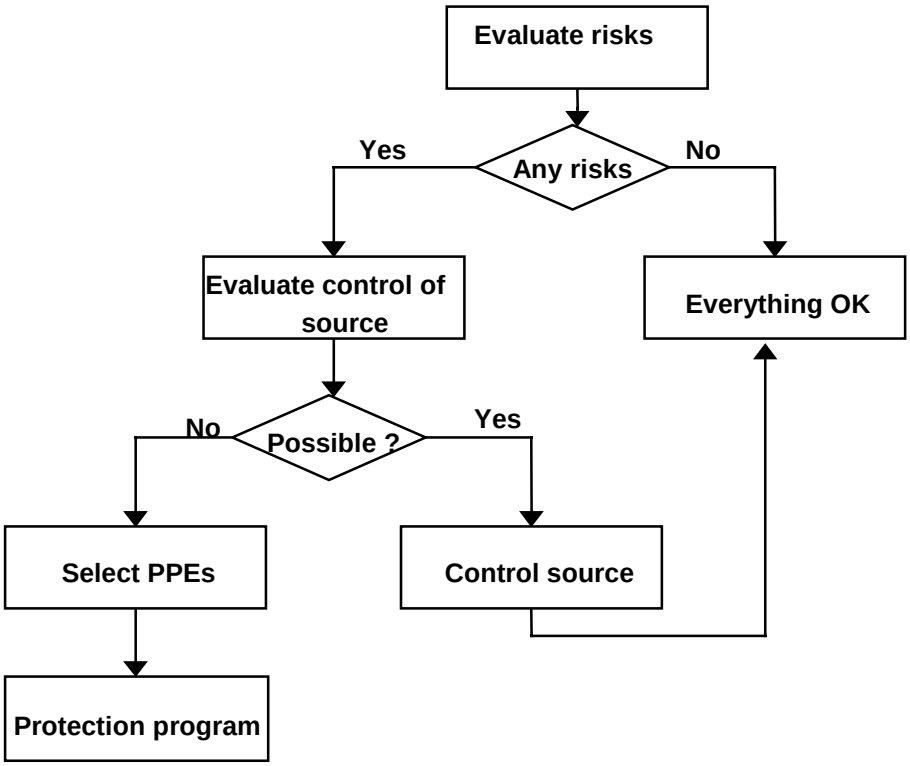


Figure 4. General approach to the hearing protection program for workers who are at risk to develop NIHL.

These guidelines are not sufficient for practical purposes. The following problems must be solved:

- How to guarantee that the HPDs are used properly
- How to discover risky workplaces or tasks
- Addressing the counter measures against the relevant noise source, especially if the greatest exposure occurs in free time is difficult

By solving these questions the minimal legal requirements of a HCP will be achieved. A good HCP contains additional elements. These elements are added to increase the power of the HCP, which will be discussed in the next chapter.

Hearing conservation program

The primary goal of a HCP must be the prevention or, at least, limitation of NIHL associated with exposure to industrial noise (Royster et al. 1982). Other goals may be formulated in addition to this primary goal, such as reduction of employees' stress and absenteeism, and reduction of work place accidents.

The components of an effective hearing conservation program are as follows (Stewart 1994):

1. measurement of work-area noise levels
2. identification of over-exposed employees
3. reduction of hazardous noise exposure to the extent possible through engineering and administrative control
4. provision of HPD if other controls are inadequate
5. initial and periodic education of workers and management
6. motivation of workers to comply with HCP policies
7. professional audiogram review and recommendations
8. follow-up for audiometric changes
9. detailed record-keeping system for the entire HCP
10. professional supervision of HCP

One observes that many of these above-mentioned tasks are not well defined. The exposure evaluation is not a simple straightforward task, and the comparison of audiograms is not easy, due to large variations in NIHL and the strong effect of age.

Several hearing conservation programs (HCP) have been launched in order to better understand the effect of occupational noise on the human ear (Royster et al. 1980, Melnick 1984). Some recent HCPs utilize data base analysis programs comparing data on the noise emission level, and including evaluation of factors other than work place noise (Royster and Royster 1986, Franks et al. 1989). These programs may take into consideration, for instance, the association of aging, non-occupational noise, and medical history (Franks et al 1989). Other researchers use models based on risk analysis in which the relative importance of various factors, as well as workplace noise, are considered (Pyykkö et al. 1986, Pyykkö et al. 1989).

Although individual models for the development of NIHL have been provided (Royster et al. 1980, Royster and Royster 1986) the studies have not been very successful so far. One reason may be the inaccuracies in the evaluation of the exposure data, in the usage rate of hearing protectors or in estimations of sosiocusis and of nosocusis, especially in the detection of genetic factors.

To compare people of different ages, an age correction is usually made. The age correction according to ISO 1999 has some exceptions (case 5, ISO 1999-1990). If the hearing loss exceeds 40 dB at any frequency, the age correction will not be applied. Thus, at lower hearing thresholds the effect of age on hearing loss is no longer additive. The interaction of noise-induced hearing loss and presbycusis does not yet seem to be well established (Rössler 1994). The uncertainty of age correction might be

diminished by selecting an internal control group. Usually a group that would be otologically screened and exposed to similar environmental stressors other than noise, is not available. Robinson (1988) focused on the problem to evaluate the noise-induced hearing loss in an industrial population. He concludes that it is not generally realistic to compare such a population with an age-matched "otologically normal" baseline, since a noise-exposed population will include adventitious hearing loss as well as noise-related components. The use of a well-documented baseline for data comparison makes it possible to estimate hearing loss in different geographic areas by using standard forms.

One major problem in HCPs is establishing individual base line values. Royster and Royster (1986) demonstrated a significant improvement of age-corrected audiograms when the subjects were annually tested over six years. The improvement was interpreted to be due to the training effect, but depended on the noise emission level. Also, those with prominent hearing loss had less training effect. Royster and Royster (1986) proposed that the audiogram showing the best hearing at frequencies of 500 - 6000 Hz should form the base line level. Thus any audiometric evaluation used in a hearing conservation program should be based on a serial audiogram and the database should include some expert programs to validate the data in order to establish base line values for hearing and also to calculate hearing loss.

User education and training

The use of HPDs gives best results with motivated users. Low motivation to wear HPDs is seen as low usage rates and low true attenuation values (Foreshaw and Cruchley 1982). A successful motivation can be obtained via appropriate education and training. The users must be informed about the effects of noise and the risks at work (89/188/EEC). Best results are obtained if personal audiometric data is used (Lipscomb 1994). This means that the education must be given privately. Users need training on maintenance, installation and use of HPDs. The attenuation of protectors work well only if they are well maintained (EN 458-1993). Good maintenance consists of cleaning, changing of replaceable parts like cushions and overall monitoring of the state of the HPD. Installation must be done before entering the noisy area (EN 458-1993). If earplugs are used special attention to the proper installation technique must be paid (Berger et al. 1983, Foreshaw and Cruchley 1982).

Although it is possible to obtain highly motivated users with proper education and training, the motivation tends to decrease over time. To avoid this, the education and training must be repeated consistently (Lipscomb 1994).

Early indicators

Pure tone audiometry is not a sensitive measure of hearing impairment, but it is easy to perform. Candidates for a more sensitive routine measurement method have been investigated during the past decades. These methods are based on new instruments like otoacoustic emission or high-frequency audiometry or on other symptoms like tinnitus.

Tinnitus

Tinnitus is a term used to describe perceived sounds that originate within the person (Sataloff et al. 1993). In the United States, 32% of all adults acknowledge having had tinnitus at some time in their life (NCHS 1968). These symptoms are more common in people with otologic problems. Fowler (1912) reported that 85% of patients with HL had tinnitus. Heller and Bergman (1953) reported a smaller reading of 75%. Tinnitus is not only related to HL, but other diseases such as otosclerosis (Glasgold and Altmann 1966) and acoustic neurinoma (House and Brackmann 1981), where 80% experience tinnitus.

Tinnitus is often experienced after an exposure to a very sudden loud noise, such as an explosion or gunshot (Savolainen and Lehtomäki 1996). In most instances, the tinnitus is accompanied by a high-tone HL. The tinnitus usually disappears in a few days. If permanent hearing loss has occurred, tinnitus may persist for many years (Sataloff et al. 1993). According to McShane et al. (1988) the prevalence of continuous tinnitus is 34% in a population for up to 10 years. The prevalence increased to about 50% in a population exposed for 11-30 years**.** Axelsson and Prasher (2000) evaluate that 20-40 % of people exposed to occupational noise have permanent tinnitus. The occurrence of continuous tinnitus among people exposed to impulse noise is 63-70 % (Alberti 1987).

Tinnitus is often related to the functional dissociation of hair cells (Ceranic et al. 1998) and a correlation to hearing loss exists (Davis et al 1998). Unfortunately, tinnitus is a fairly common complaint in populations without noise exposure and is aggravated by factors like loss of sleep, job interference, psychological problems and other stress factors (Morril 1986).

Otoacoustic emission

The term otoacoustic emission (OAE) refers to sounds emitted by the ear (Kemp 1979). The emitted sounds may be helpful in the early identification of SNHL caused by occupational noise exposure. In the normal ear the spontaneous otoacoustic emission (SOAE) is present virtually continuously in the absence of deliberate acoustic stimulation. Even after subtle lesion the SPOAE seems to disappear (Furst et al. 1992)). Three OAE forms exist; all of which are evoked by particular stimuli. The transient evoked otoacoustic emission (TEOAE) is elicited by brief stimuli, such as clicks and tone pips. The distortion product (DP) is elicited by nonlinear interaction of two simultaneous, long-lasting pure tones (Avan and Bonfils 1993). The evoking tones are referred to as the f_1 and f_2 primaries in humans, the largest DPOAE occurs at the frequency equivalent to $2f_1 - f_2$.

The contralateral inhibition of distortion product (DPI) is recognized as the reduction in the amplitude of evoked OAE in one ear upon stimulation of the opposite ear. OAE is vulnerable to known noxious agents to the inner ear, such as ototoxic drugs, intense noise and hypoxia, which are all known to affect the cochlea. They are absent in frequency regions with cochlear hearing losses greater than 35dB. The type of OAE that is most commonly used for clinical purposes is evoked by transient stimuli such as clicks and is referred to as a transient evoked OAE features making the measurement of TEOAE attractive for use as a screening procedure. Hitherto, a hearing loss is thought to affect to TEOAE at middle and DP at high frequencies. For NIHL OAE may be sensitive to discriminate subgroups of individuals whose cochlear pathology is biased by poor outer hair cell function (Oeken 1998). However, a settled analysis of efficacy of OAE in NIHL is still controversial (Cheng 2000).

High frequency audiometry

High frequency audiometry refers to threshold testing at frequencies from 8 kHz to 20 kHz. It is assumed to help in early detection of hearing loss revealing hearing impairment before it is detectable at frequencies normally measured. In NIHL improvement in hearing may be seen at 10 kHz, 12 kHz and 14 kHz. In age-related hearing loss this is not observed (Sataloff and Sataloff 1993b).

The high frequency audiometry starts to deteriorate quite early by the age of 18-24 years. (Hallmo et al. 1994). Thus, the high-frequency audiometry can be used for early detection of NIHL. This method seems

to work only among young persons with normal hearing before any noise exposure changes due to environmental noise in hearing (Bartsch et al. 1989, Osterhammel 1979). The use of high frequency audiometry is limited by its reliability and its ability to reproduce results with another high frequency audiometer (Chery-Groze et al. 1994).

PURPOSE OF THE STUDY

The purpose of the study is to design an individual hearing conservation program and evaluate the validity of the various components of the program. The study is focused especially on the following topics:

1. To create a database and an interactive user interface to collect data from workers exposed to noise (I)
2. To evaluate the effect of individual risk factors on NIHL (II, III)
3. To evaluate the effect of age on development of NIHL (IV)
4. To evaluate the role of environmental factors in hereditary hearing loss (V)
5. To develop an individual hearing conservation program (VI)

SUBJECTS AND METHODS

Subjects

The data for part of study I-IV and VI was collected in cross-sectional studies on: 1) forest workers (N=124) in 1995 in the northern part of Finland, 2) papermill workers (N=406) in 1995 located in the south-eastern part of Finland, and 3) shipyard workers (N=176) in 1990 located in the southern part of Finland. The data for forest workers was obtained during a follow-up study in 1972-1995. For paper mill and shipyard workers, data was obtained from the occupational health care centers of the different enterprises. For this study the data comprised detailed work exposures in different occupations and the use of HPDs. Complete work histories were obtained for 675 workers. The work histories were divided into work periods, during which the exposure and occupation of the worker was unaltered. The total number of work periods was 1873 from the years 1953 to 1995. The mean duration of work periods was 5.6 years ± 6.4 years and the duration ranged from 1 month to 35 years. The work periods were classified based on their end date into categories with 5-year intervals. The first category consisted of work periods ending in 1951-1955 and the last category from the periods ending in 1991-1995.

Table 3. The mean and standard deviation of exposure inside the hearing protector (L_{ANI}), outside the hearing protectors (L_{ANO}) and age of the study groups.

Parameter	Paper mill N=406	Shipyard N=176	Forest work N=124	All N=706
L_{ANI} (dB)	98 ± 10	100 ± 9	99 ± 6	99 ± 9
L_{ANO} (dB)	104 ± 6	109 ± 6	110 ± 4	107 ± 6
Age (y)	38 ± 9	36 ± 9	45 ± 8	40 ± 9

For study V, data from a large Finnish family with early onset sensorineural HL was collected. Data from the five-generation family were traced and 104 living members were included in the study. Audiograms were measured in 60 and blood samples for genetic linkage studies were taken from 83 family members. Family members were considered to be affected only if they had bilateral sensorineural HL below the 75th percentile of an age and sex-dependent control audiometric curve of ISO class A, and if the medical history and known risk factors were not able to explain the amount of HL. The pattern of

inheritance in this HL family was autosomal dominant. From the 22 affected individuals, 11 were females and 11 were males. The HL among affected family members was symmetric, sensorineural and varied from mild to severe. The type of audiogram varied from flat to deep sloping in high frequencies.

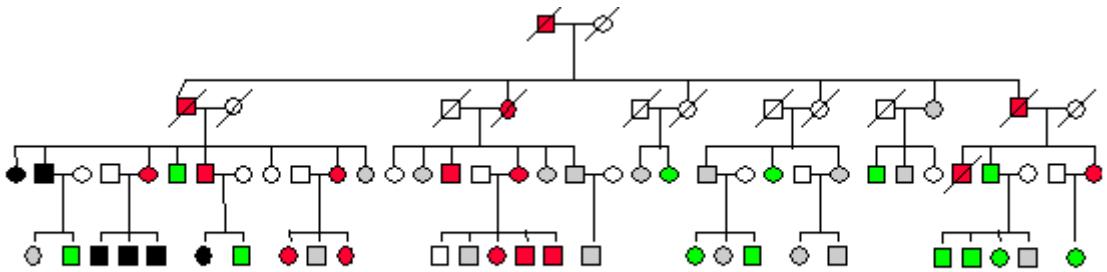


Figure 5. The Finnish family with postlingual autosomal dominant sensorineural hearing loss, o female, □ male, • affected emale, ❁ affected male : slash diseased.

Methods

The model

The components of an Individual Hearing Conservation Program (IHCP) are shown in Fig 6. The model consists of three sections; exposure, individual susceptibility and output parameters. The exposure section consists of noise exposure and other interacting exposures. The section of biological factors contains the hereditary factors and medical condition of the subjects. The output section shows the effects of noise that can be measured.

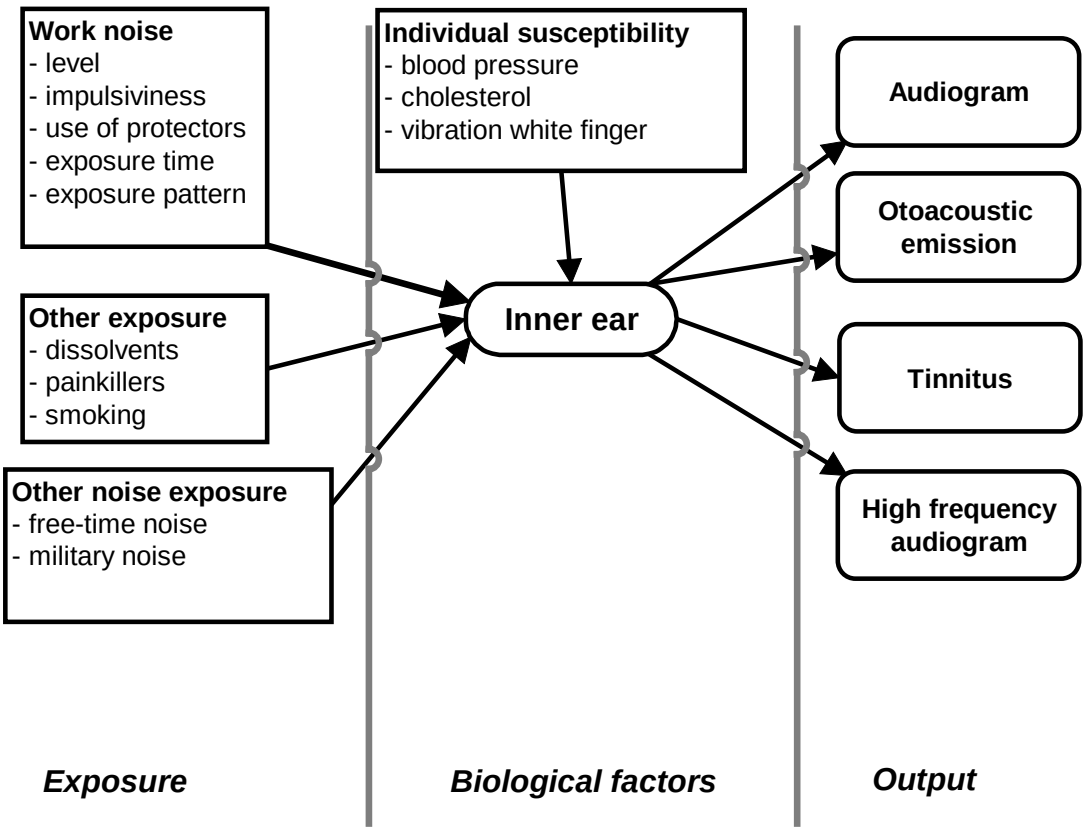


Figure 6. The elements of IHCP.

Exposure evaluation

Noise exposure evaluation

The noise exposure was measured as A-weighted equivalent level by qualified industrial hygienists. The measurements were completed by miniature microphone measurements consisting of simultaneous measurements outside and inside the HPDs using a portable 2-channel noise dose meter during a normal working day. These measurements were made with 21 paper mill workers, 28 shipyard workers and 20 forest workers. The sampling period was 10 minutes in each measurement. The workers used their own hearing protectors, which were of the same type in each work place. The real world attenuation of HPD was calculated as the difference between mean sound pressure levels outside and inside of the protector for the study groups. Mean noise level in the paper mill was 93 dB, 93-95 dB in the shipyard, and 95-97 dB in the forest. The mean attenuation of the HPDs was 15 dB in forest work, 17 dB in the paper mill, and 20 dB in the shipyard. The usage rate was collected using a written questionnaire. Workers were asked to evaluate whether they use HPDs always, often, half-of the time, sometimes or never. In

calculations the steps were estimated to correspond to 100%, 75%, 50%, 25 % and 0% usage rates, respectively.

In the present study no data about exposure to music or other free-time noise exposure was included. Information about gunfire noise and the total number of shots was collected using questionnaires in steps of ten. The usage rate of HPDs during free-time exposure was rated as always, nearly always, sometimes, seldom and never.

Military gunfire exposure was divided into two categories, large caliber weapons and hand-held guns. Information on the number of shots and the use of HPDs was collected similar to the free-time gunfire exposure. The number of shots was classified into three categories: 1-10, 11-100, 101-1000.

Using the period data, the noise emission level (L_{ANO}) and noise immission level (L_{ANI}) was calculated using the following formulas:

$$L_{ANO} = 10 * \log(\sum_i (10^{L_{Aeqi}/10} + 10 * \log(T_i)$$

$$L_{ANI} = 10 * \log(\sum_i (10^{(L_{Aeqi} - A_i')/10} + 10 * \log(T_i) \quad \text{where}$$

$$A_i' = L - (10 * \log(10^{L_i/10} * (1-c) + 10^{((L_i - A_i)/10)} * c))$$

where L_{AEqi} is the A-weighted equivalent noise level during i:th work period,
A_i' is the effective attenuation of HPD during i:th work period
T_i is the length of the i:th work period in years, and
LOG is base 10 logarithm.
L_i is the sound pressure level during i:th work period
A_i is the nominal attenuation of HPD during i:th work period
c= usage rate (%)/100

Evaluation of other exposure and individual susceptibility factors

The medical histories of the workers were collected from local occupational health care centers. Information about medication and prescribed pain medication was collected. Serum cholesterol levels and blood pressure measurements of the past three years, and details regarding the use of non-prescription analgesics and tobacco smoking were included. In the case of missing data on exposure or nosocusis, a new questionnaire or list of questions was sent out.

The role of hereditary factors

The data on individual risk factors for hearing loss were collected via questionnaires and fed into the NoiseScan program (56 out of 60 returned their questionnaires).

The questionnaire contained information on ear diseases, ear operations, neurological diseases, vertigo, hereditary syndromic signs, metabolic disorders, blood pressure, cholesterol and use of drugs. Smoking and exposure to solvents were also queried. Data from occupational, leisure-time, and military noise exposure was included.

Audiograms

Audiograms were collected from the local occupational health care center for the paper mill and shipyard workers, and they were also measured for the forest workers.

Evaluation of aging factors

To study the effect of age andother contributing factors, a scoring system was used. The cut-off values for diastolic and systolic blood pressure and cholesterol level weredetermined from the median values of the population studied (Table 4). For eachindividual value exceeding the cut-off point, one score was given. Regular analgesic use had one score. Tobacco smoking also received one score, even if the subject had stopped smoking. The final risk score was the sum ofthe different scores.

Table 4. Cut-off values for scoring blood pressure and cholesterol.

Risk factor	**Age (y)**	**Systolic blood pressure (mmHg)**	**Diastolic blood pressure (mmHg)**	**Cholesterol (mmol/l)**
Cut-off value	40	132	90	5.7

RESULTS

5.1. Database for hearing conservation program (I)

The database contains information about medical conditions, diseases promoting hearing loss, hereditary factors, noise exposure, and use of HPD (Fig 7). The data input is performed primarily with a mouse into the spreadsheets; keyboard input is seldom needed. Written guidelines for the user are provided.

The input data is divided into three categories: dynamic, semistatic and static. Static data, such as social security numbers or ear operations, receive a value, which is constant. Semistatic data includes data, the value of which can be changed, though previous values are not needed, such as blood pressure, cholesterol or weight. For dynamic data, the whole history is recorded. Dynamic data includes, audiograms, use of HPDs, and noise exposure.

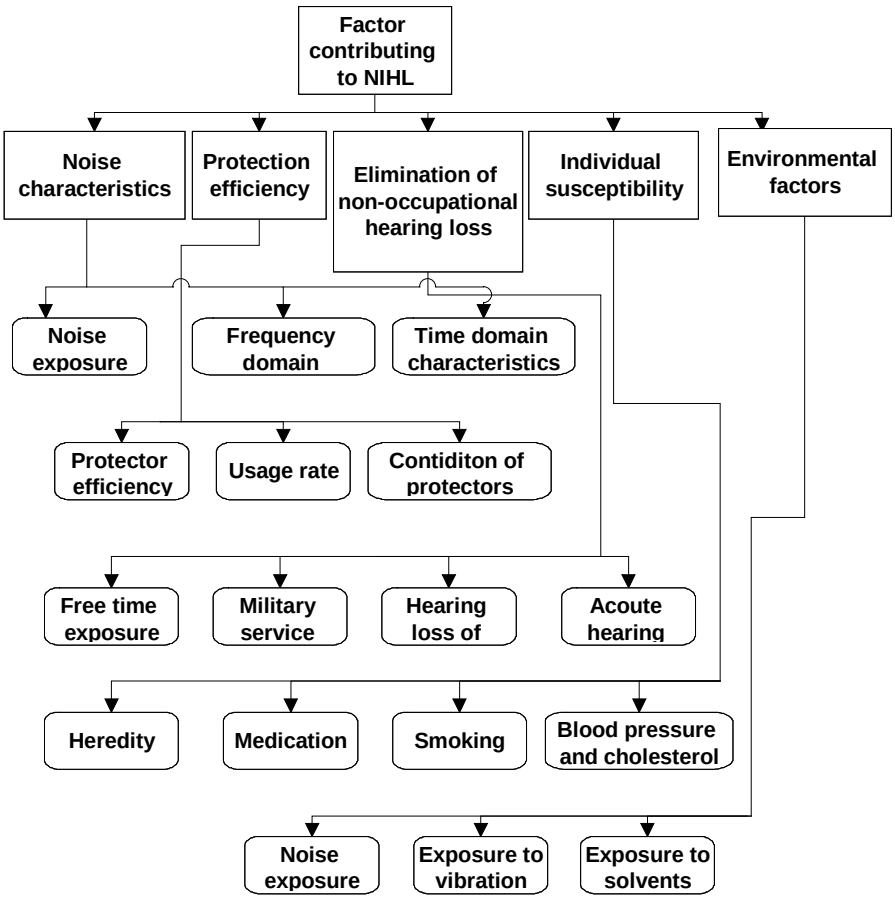


Figure 7. Organization of the database.

To collect a lifetime working history, a new data sheet is provided for each work task, noise exposure period, or occupation. Thus, a subject may have an unlimited number of noise exposure data sheets. The noise level and frequency content of noise is divided into low-medium or high-medium frequency content.

In leisure-time noise, special attention is paid to shooting noise and to work with chain saws. For military noise the caliber of the weapon and the number of shots are evaluated. The type and usage rate of hearing protectors are also queried.

Ear diseases and ear operations are queried separately. If the answer to either of these questions is yes, a new screen opens and the type of operation or ear pathology is requested in more detail. Neurological diseases and possible central nervous system infections are queried, and if the history is positive, a screen with detailed questions is provided. Questions pertaining to explosions or head injuries are also included.

Questions about vertigo (an indication of an inner ear disease that may cause SNHL), tinnitus and exposure to aminoglycosides are queried. To evaluate genetic factors, subjects are asked about hearing problems in their family. The type of general health informationcollected consists of cholesterol, hypertension, presence of vibration-induced white finger syndrome, use of analgesics, and exposure to solvents.

The number of audiogram sheets can be unlimited. The examination year, quality of the soundproof room, calibration of the audiometry, and type of audiometry are also queried. Speech audiometry result can be input, for each ear. Finally data concerning previous tympanic membrane perforations, otorrhea, middle ear infections or conduction impairments are input.

After all factors are entered, the program produces a life time exposure and prints out possible confounding factors. The program also validates the shape ofthe audiogramagainst this history and examines asymmetry of hearing to guide the user in assessing a diagnosis of NIHL.

5.2. Individual risk factors in the development of noise-induced hearing loss (II)

Exposure: The L_{ANO} and L_{ANI} for the workers were 107 dB ± 6 dB and 99 dB ± 9 dB, respectively. In L_{ANO} the distribution was bimodal, indicating the presence of two populations. The higher exposure level consists of forest and shipyard workers, and the lower exposure level of paper mill workers. In L_{ANI} the difference in exposure between

populations disappeared due to differences in the usage rates and attenuation of HPD**s**.

A significant correlation existed between hearing threshold level and L_{ANI} ($r = 0.316$, $p<0.001$) and L_{ANO} ($r = 0.305$, $p<0.001$) at 4 kHz. The L_{ANI} could explain about 2 dB HL of permanent threshold shift and the L_{ANO} about 1.8 dB HL at 4 kHz. The total permanent threshold shift for the subjects was 21.5 dB HL at 4 kHz.

The impulse noise group consisted of shipyard workers, and the steady state noise groupconsisted of forest and paper mill workers. The impulsiveness of noise caused an extra 12 dB HL permanent threshold shift ($t=2.97$, $p<0.05$) in shipyard workers. In these groups the mean L_{ANI} level was 100.2 dB.

Serum cholesterol level: A significant correlation between the serum cholesterol level and hearing level ($r = 0.194$, $p<0.01$) at 4 kHz was found. No correlation was found between the high density cholesterol and hearing level.

Blood pressure: Both the systolic and diastolic blood pressures correlated significantly with hearing levels at all frequencies studied, and the highest correlation was observed at 4 kHz for systolic ($r = 0.249$, $p<0.001$) and for diastolic ($r = 0.204$, $p<0.001$) blood pressures. The systolic blood pressure correlated significantly with the total cholesterol ($r = 0.132$, $p<0.05$), cessation of smoking ($r = 0.145$, $p<0.01$), and regular use of analgesics ($r = 0.139$, $p<0.01$).

Smoking: The effects of smoking were confounded by subjects who had stopped. Current smoking did not correlate with hearing level. The subjects were classfied into never-smokers and those who had given up smoking, and current smokers. This analysis indicated that never-smokers had significantly better hearing at 4 kHz ($r = 0.138$, $p<0.001$).

Analgesics: The use of analgesics were analyzed separately for non-prescription drugs and prescribed drugs, as was frequency of use. The total use of analgesics correlated with hearing level at 4 kHz ($r = 0.331$, $p<0.001$). The use of both prescription-free and prescribed analgesics tended to correlate with NIHL ($r = 0.118$, $p = 0.06$, $r = 0.188$, $p = 0.04$).

Interaction with hearing loss: The combined interaction between total cholesterol, blood pressure and noise exposure (Fig 8) was analyzed. In the low-exposure group, subjects with high cholesterol had significantly worse hearing at 4 kHz ($F=14.2$, $p<0.05$) than those with low cholesterol and low systolic blood pressure values. Subjects with high noise exposure, elevated cholesterol, and elevated systolic blood pressure had

worse hearing at 4 kHz than subjects with high noise exposure, but low cholesterol and low systolic blood pressure ($F=9.2$, $p<0.05$). Elevated blood pressure was a significant risk factor in the low-exposure group ($F=6.98$, $p<0.01$).

The interaction between the use of analgesics, smoking and exposure to noise was further explored. The analgesics significantly contributed to a threshold shift (Fig 9) in the high-exposure group ($F=2.9$, $p=0.01$). Results showed that smoking does not significantly interact with analgesics.

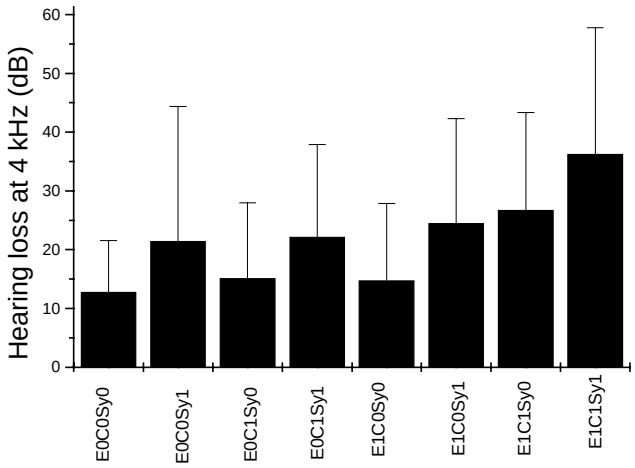


Figure. 8. Effects of different factors on NIHL at 4 kHz in workers after dividing the subjects into low-risk and high-risk groups based on median values. Abbreviations: E = noise exposure; C = low density serum cholesterol level, Sy = systolic blood pressure, 0 = below median, 1 = above median.

Modeling NIHL A general linear model analysis was applied. In the model, mean hearing of both ears at 4 kHz was used as a dependent variable. The independent variables included L_{ANI} ($p<0.05$), systolic blood pressure ($p<0.05$), regular use of analgesics ($p<0.001$), total cholesterol ($p<0.05$) and tobacco smoking ($p<0.05$). Added to the model were two combined interactions consisting of (a) L_{ANI}, systolic blood pressure and total cholesterol, and (b) L_{ANI} , tobacco smoking, and regular use of analgesics. It explained 36.4% of the variance of the hearing loss, which was statistically significant ($F=14.42$, $p<0.001$).

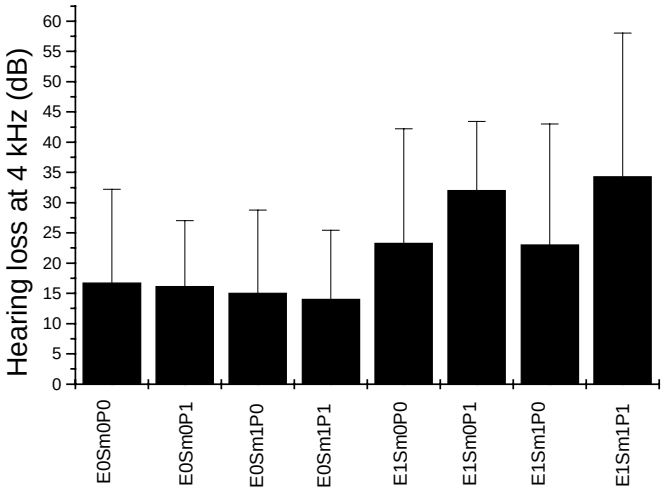
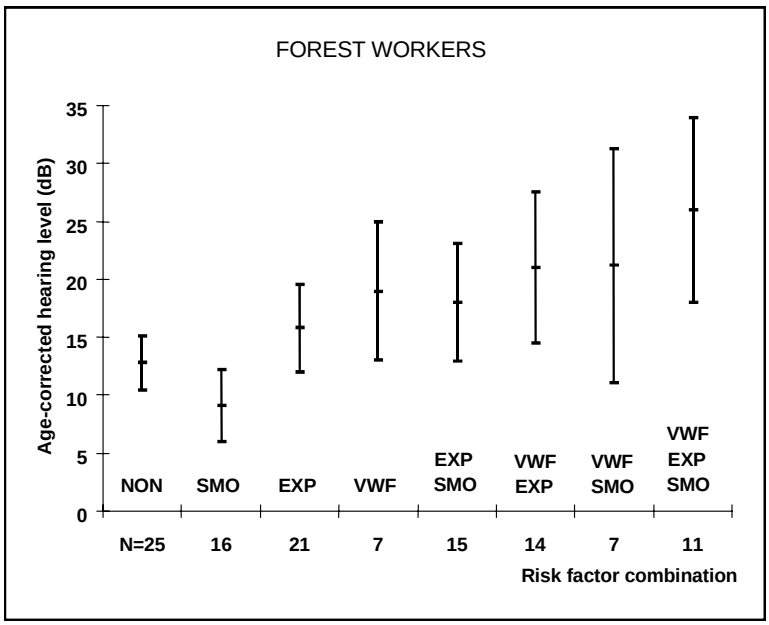


Fig. 9. Effects of different factors on NIHL at 4 kHz in workers after dividing the subjects into low-risk and high-risk groups based on median values. Abbreviations: E = noise exposure, Sm = Smoking habits, P = use of analgesics. 0 = below median, 1 = above median

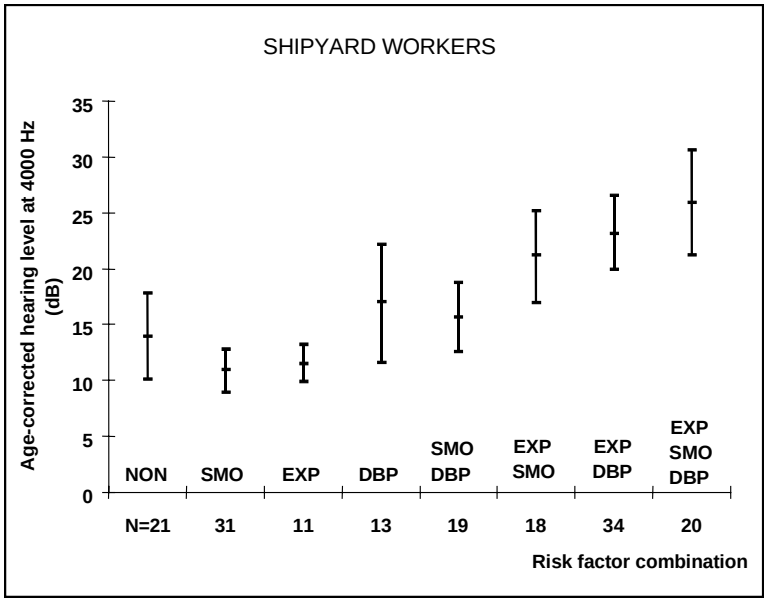
5.3. Smoking as a risk factor in sensory neural hearing loss among workers exposed to occupational noise (III)

The effect of smoking alone and also in combination with other risk factors was measured among forest and shipyard workers. Smoking without the presence of any other risk factors did not increase the risk for HL (Table 5). In linear regression analyses, aging was the most important single risk factor for HL. It explained about 26% of the variation of HL for forest workers and 48% for shipyard workers. The effect of age was greater than the effect of noise exposure, which explained about 10% of the variation of hearing loss among forest workers and 15% among shipyard workers. Diastolic blood pressure was a significant factor for hearing loss only among shipyard workers accounting for 7.5 % of the variation. Raynaud's phenomenon was a significant factor for hearing loss among forest workers.

In both groups of workers the measured hearing level showed greater variation than expected based on the ISO model. The individual hearing levels for smokers and non-smokers did not reveal any significant differences.



A) Forest workers



B) Shipyard workers

Figure 10. The effect of risk factors and their combinations on the measured and age-corrected hearing levels, mean and standard error of the mean.
Abbreviations: NON = no risk factor, SMO = smoking; EXP = exposure to noise; DPB = diastolic blood pressure; VWF = presence of Raynaud's phenomenon of occupational origin; N = Number of workers in various groups of risk factors and risk factor combinations.

Table 5. Individual risk factors explaining the variation in NIHL

Risk factor	**Forest workers (%)**	**Shipyard workers (%)**
Age	26***	48***
Noise exposure	10**	15**
Diastolic blood pressure	0.2 ns	7.5**
Smoking	1 ns	3.3*
Raynaud's phenomenon of occupational origin	4**	0 ns

*** $p < 0.001$
** $p < 0.01$
* $p < 0.05$
ns not significant

The results show that smoking alone does not cause deterioration in hearing. But smoking in combination with peripheral circulatory dysfunction and elevated diastolic blood pressure influences the hazardous effect of noise on hearing.

5.4. Age and Noise-Induced Hearing Loss (IV)

Age and Hearing Loss: Age correlated with the elevation of cholesterol ($r=0.539$, $p<0.001$), systolic blood pressure ($r=0.342$, $p<0.001$), diastolic blood pressure ($r=0.349$, $p<0,001$), use of analgesics ($r=0.184$, $p<0.001$), and use of hypertension drugs ($r=0.226$, $p<0.001$). Age also correlated with exposure to noise ($L_{ANI}=0.478$, $p<0.001$) and SNHL at all frequencies ($r=0.588$ at mean hearing levels of 0.5, 1, 2 and 4 kHz of both ears, $p<0.001$). Older persons had used hearing protectors less often than younger persons ($r=0.392$, $p<0.001$).

In logistic regression analysis contributing factors correlated with age-related hearing loss were searched. The model consisted of serum cholesterol, smoking, L_{ANI}, use of analgesics and systolic and diastolic blood pressure. From the model, serum cholesterol (odds ratio 3.2), use of analgesics (odds ratio 3.0), systolic blood pressure (odds ratio 1.1), and L_{ANI} (odds ratio 1.1), one could explain 78% of the variation of age-related HL. The results indicate that an increase of cholesterol level and use of analgesics are strongly associated with the older inner ear.

The age was divided into four 25-percentile classes and the mean hearing of both ears at 4 kHz was evaluated as a dependent variable in logistic

regression analysis (Table 6). In the different age groups cholesterol came out as one of the most important contributing factors for hearing loss. The odds ratio for cholesterol induced hearing loss was highest at the youngest age group. Analgesics were the leading cause of hearing loss in the age group ranging from 39 to 44 years. Noise exposure (L_{ANI}) was a significant factor for the oldest age group consisting of subjects with a wide exposure range.

Table 6. Hearing loss (HL) indicating mean threshold of right and left ear at 4 kHz, risk factors in logistic regression analysis and their odds ratio and explanation power when subjects were classified into 25 percentile groups based on their age.

Mean age	**Range years**	**HL, dB**	**Risk factor and odds ratio**	**Risk factor and odds ratio**	**Risk factor and odds ratio**	**Power (%)**
28	19-32	9.4	Cholesterol 7.2			83.3
36	33-38	13.7	Cholesterol 2.6			67.7
44	39-44	19.1	Analgesics 5.8	Cholesterol 1.8		65.4
49	45-62	31.5	Cholesterol 1.8	L_{ANI} 1.2	Blood press 1.1	80.2

Age related noise susceptibility: For comparison, pairs of subjects from the youngest 25-percentile and oldest 25-percentile groups were chosen. After matching the pairs with blood pressure, cholesterol, smoking, use of analgesics and exposure profiles, a comparison between the groups was made (Fig 11). Among the younger subjects exposure to noise caused a slight but insignificant increase in threshold values. In the oldest age group exposure to noise caused a highly significant ($p<0.001$) deterioration in the hearing threshold. The results support the idea that an elderly ear is more vulnerable to noise.

Interaction of Noise Exposure with Various Contributing Factors: The mean ages in the subjects when divided into nine groups, based on a combination of all defined contributing factors, were quite similar. Mean age in the high-exposure groups varied between 42 and 48 years, in the moderate-exposure groups between 38 and 41 years, and in the low-exposure groups between 35 and 43 years.

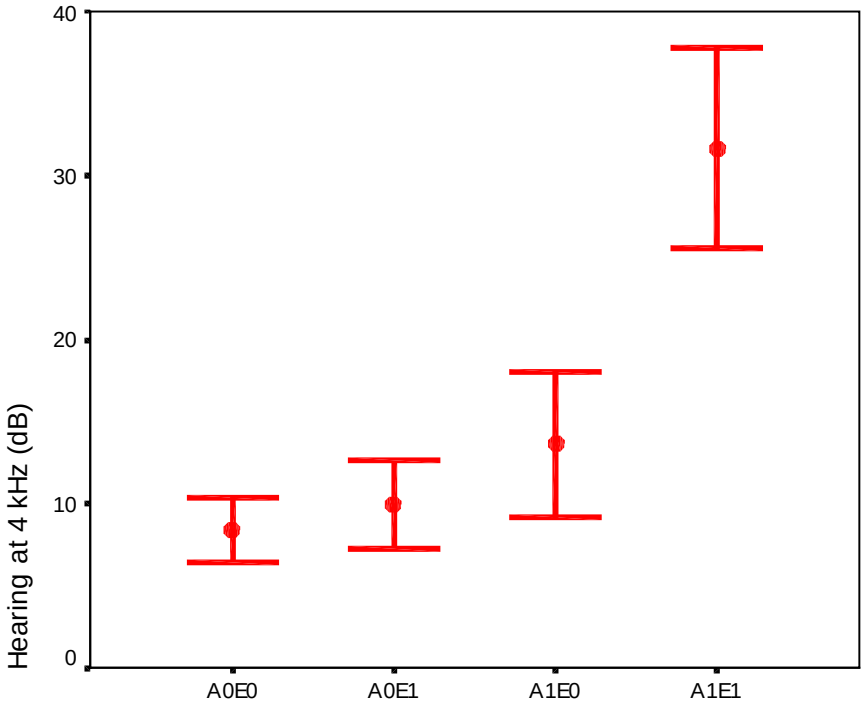


Figure 11. Effect of age (A) and exposure (E) on mean hearing of right and left ear. The lowest and highest percentiles of workers are shown with matched systolic blood pressure and cholesterol values. Means and standard deviations are shown. Abbreviations: A0 means lowest 25-percentile of age, A1 highest 25-percentile of age, E0 means lowest 25-percentile of life time exposure and E1 highest 25-percentile of life time exposure.

In subjects with zero or one contributing factors, the difference in hearing level between moderate- and low-exposure groups was not significant (Fig 12). The SNHL in the high exposure group differed significantly from that of the low-exposure group at 4 kHz ($p<0.05$).

In subjects with two contributing factors, the low- and high-exposure groups differed significantly from each other at 4 kHz ($p< 0.05$) and 8 kHz ($p<0.05$) (Fig 13).

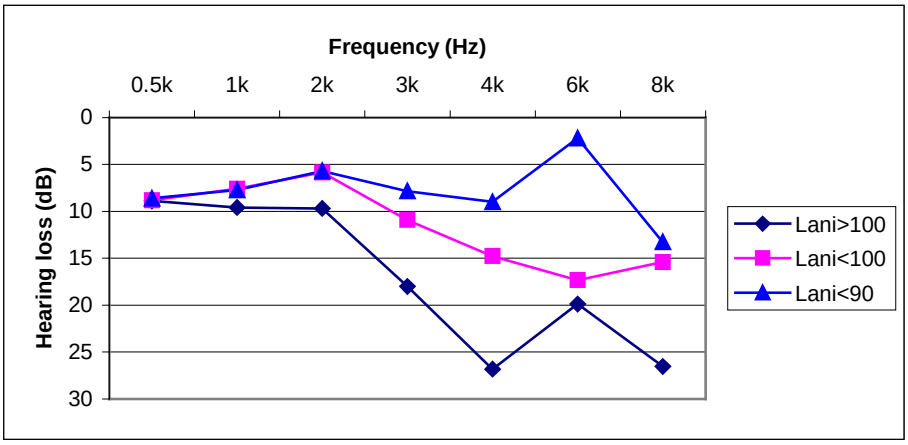


Figure 12. The mean hearing of workers with zero or one contributing factor; low exposure = $L_{ANI} < 90$ dB, moderate exposure = 90 dB $< L_{ANI} < 100$ dB, high exposure = $L_{ANI} > 100$ dB.

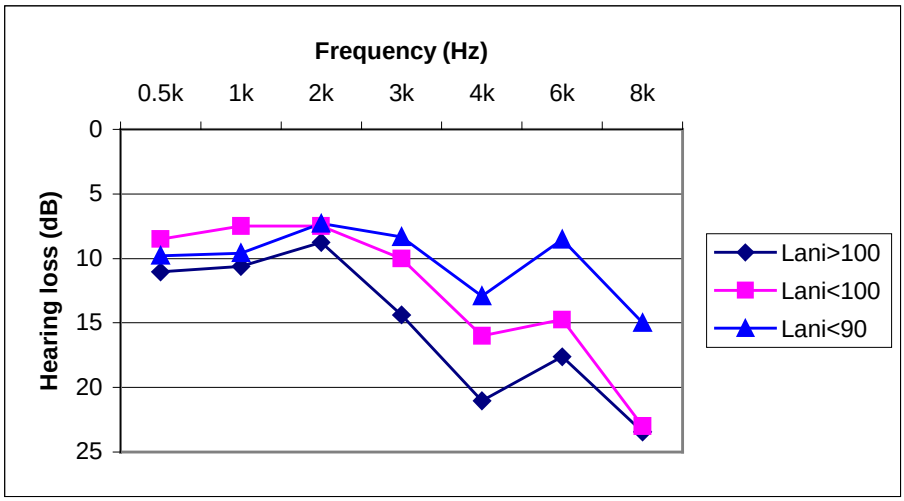


Figure 13. The mean hearing of workers with 2 contributing factors, low exposure = $L_{ANI} < 90$ dB, moderate exposure 90 dB $< L_{ANI} < 100$ dB, high exposure = $L_{ANI} > 100$ dB.

In subjects with more than two contributing factors, no significant differences were found between any of the exposure groups (Fig 14).

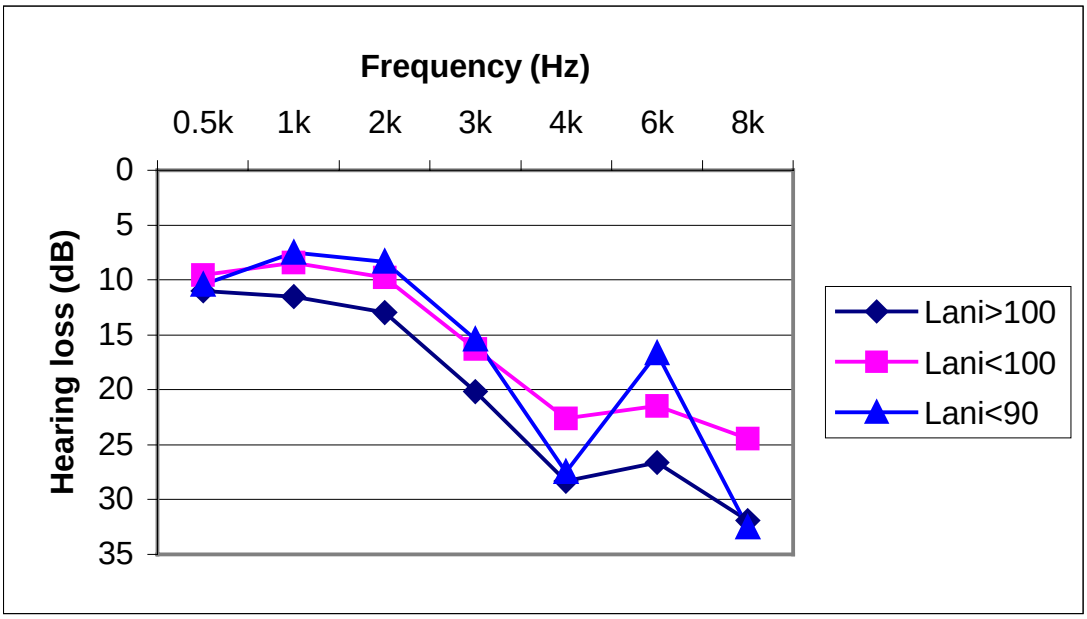


Figure 14. The mean hearing of workers with more than 2 risk factors, low exposure = L_{ANI} < 90 dB, moderate exposure = 90 dB < L_{ANI} < 100 dB, high exposure = L_{ANI} > 100 dB.

5.5. Hereditary hearing loss – the role of environmental factors (V)

The mean age at onset of HL in a family was 22 years. The mean deterioration of hearing was 1 dB per year at 2 kHz frequency. When compared with their relatives, affected family members had a history of more frequent installation of tympanostomy tubes in the ear (right and left, $p<0.001$), more vertigo ($p<0.001$), and a greater incidence of tinnitus ($p<0.001$). The affected family had a history of more frequent use of analgesics ($p<0.01$) and analgesics of NSAID type ($p<0.01$). In the logistic regression analysis the use of analgesics ($p<0.05$) and especiallythe use of analgesics of NSAID type correlated with hearing loss ($p<0.01$). Exposure to military noise correlated with hearing loss but the correlation was biased by gender (male). Exposure to occupational or leisure-time noise, music, concerts, discotheques and portable players, did not emerge as a risk factor for hearing loss.

5.6 Management of a sophisticated hearing conservation program (VI)

The following Multiple Input - Multiple Output (MIMO) model was used. There are three input blocks in the system: Work noise exposure, other exposure(e.g., smoking), and other noise exposure. The output

blocks are audiogram, high frequency audiogram, tinnitus and otoacoustic emission. The input-output relationships are defined by control blocks, which are individual susceptibility and hereditary factors.

Work noise: Unlimited number of exposure periods can be fed with the data on the possible impulsiveness, usage HPD and frequency content of noise. The program calculates the total occupational exposure to noise, taking into account the protection efficiency of hearing protectors with the usage rate. In case of missing exposure data, the program will come up with an estimate from results in the register of hygienic measurements. The attenuation of HPDs is evaluated using the HML-check method of the standard EN 458 (EN 458-1993).

Other exposure: Smoking, the use of analgesics and exposure to solvents are questioned by periods. Smokers are divided into non-smokers, smokers and heavy smokers. A similar division is made for the use of analgesics. Exposure to solvents is a Yes or No response.

Other noise exposure: The recorded non-occupational noise exposures includes music, shooting, use of power tools and exposure during military service. Information about these exposures is collected periodically. The form used for collecting information concerning military service exposure is also used in collecting the exposure data of professional soldiers. Music exposure is questioned in detail. Rock music playing, classical music playing, use of portable stereos and visits to discos and rock concerts are asked separately. For each activity, an evaluation of the sound pressure level and the use of hearing protectors are queried. On the same form there are questions about the use of power tools and noise in public events and shooting, and about leisure-time exposure to solvents.

Control parameters: Hereditary factors are asked on a separate sheet for those who have an early hearing loss or relatives with early hearing loss. These include questions about family history and any other symptom that might explain early hearing loss. Skin sensitivity to sunlight is also questioned as a factor, which might explain higher vulnerability to noise. The individual susceptibility factors includes blood pressure and cholesterol levels, and are collected as snapshots. In addition to the factors mentioned above, other factors that may explain hearing loss are collected, such as some inner ear diseases, head trauma and different infections.

NoiseScan provides two models for the hearing level prediction,the ISO 1999-1990 model and NoiseScan model. The ISO 1999 model takes as input the work noise level outside the hearing protector (ISO 1999-1990). Based on this figure the ISO 1999 model gives the distribution of hearing

loss. NoiseScan provides the prediction based on population A of ISO 1999. NoiseScan prints the latest audiogram on top of these statistical curves. Importantly, NoiseScan evaluates on other possible sources of exposure, i.e. free time noise and military noise, on the same screen.

Unlike the ISO 1999 model, the NoiseScan model takes into account known factors behind the large variation of NIHL. For this purpose the NoiseScan model utilizes the individual risk factors as input. Unlike the ISO 1999 model, NoiseScan models use the total noise exposure to ear as the input parameter.

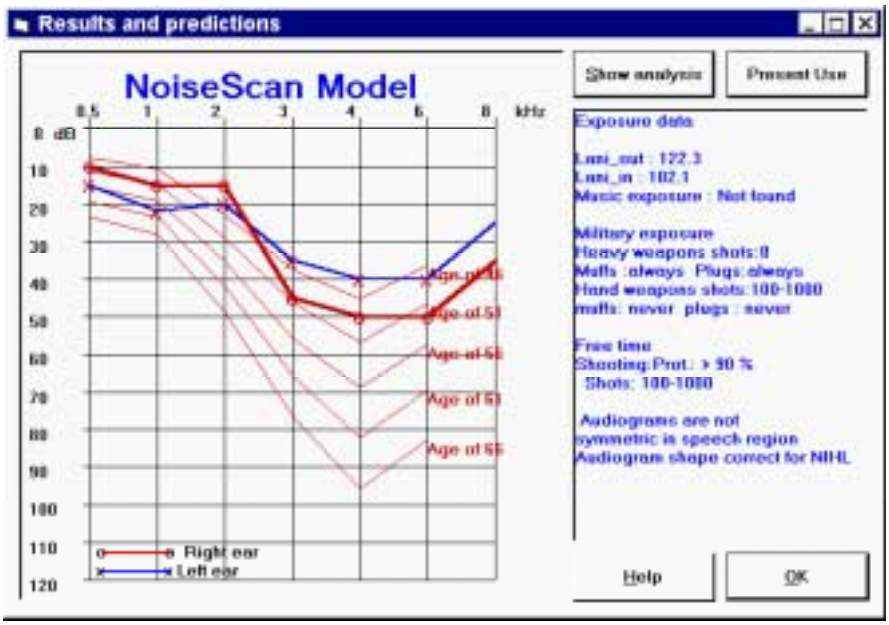


Figure 15. The NoiseScan model. NoiseScan displays the latest audiogram for right and left ears and predicts the development of NIHL in five years intervals. Exposure data is summarized and the comparison of shape form of audiograms is made.

Finally, NoiseScan calculates a prediction of the progress of the NIHL based on the assumption that the present exposure with the present use of hearing protectors continues. NoiseScan will also simulate the full-time use of protectors.

DISCUSSION

Most of the studies on NIHL are made using a relatively small number of subjects (50 to 500). Larger studies (Bauer et al. 1991, Neuberger et al. 1992) have not contributed significant new information on the effect of noise on hearing and associated risk factors for vulnerability to noise induced hearing impairment. There are several reasons for this. Firstly, the control of the data is laborious in a large database. The present study demonstrates how time consuming, costly and multidisciplinary the approach should be to achieve accurate control over the database. For example, noise exposure requires thorough analysis of the whole work history and leisure time exposure. The use of HPDs must be evaluated by using questionnaires and measurements of the true attenuation of HPDs. Realistically, in spite of all efforts, archived data is subject to error and, in estimation of life time exposure, may contribute as much as 10 dB variability. At present adequately careful data collection methods are not performed routinely because they are time-consuming and no standard procedure exists to combine free time noise and occupational noise. Secondly, the measurements of audiograms must be done using a clinical audiometer in a properly isolated booth and not with a screening audiometer as is currently practiced. Thirdly, the confounding factors are difficult to control, as noise vulnerability seems to be determined significantly by genetic factors, of which very little is known.

Careful data acquisition tends to limit the size ofthe database. With only a relatively small number of subjects, the present study could confirm the effect of smoking, elevated blood pressure, cholesterol and analgesics in the etiology of NIHL. When these confounding factors were included in the model explaining NIHL and aging of the cochlea, the role played by age was found to be smaller than what is usually estimated (ISO 1999-1990, NIOSH 1974). The difference between the results on aging in the present study and the previous estimations maybe due to the observation that most risk factors are inherently age related, and ifthis factor is not considered in the analysis, age is credited for their contribution.

In the present study a database was designed in such a way that the known risk factors of NIHL could be easily collected. This required that the interface between the computer program and the user must satisfy several demands. The interface must be user friendly (i.e. the content of the questions and input data must be easy to understand) and it must self-control the input errors. It must be self-explanatory, and when needed, provide help in inputting required data or using required interfaces. The advantage of the graphical display is that the item can be quickly pointed to by one hand, and interest can be focused constantly on the item

displayed on the screen. The objects on the screen can be self-explanatory, and accordingly, easy to understand. Clearly, the time required to fill out the forms is an essential factor. This leads to a compromise between accuracy and filling out time. A simple example of the compromises needed is the evaluation of music exposure. As there is no regular exposure, the question about weekly exposure time may be difficult. The logarithmic relation between exposure time and influence is reflected in a doubling of the exposure time increasing the exposure by 3 dB. Thus, it is reasonable to question only the exposures occurring on a weekly basis. This causes a relatively small errorwhen compared to the inaccuracy of the sound level estimates. A different approach has been adopted for medical factors. A general question concerning a group of factors was first made in the present study. If the answer was positive, detailed questions are asked. This works well in situations where the symptoms are relatively rare.

Due to proper design and compromise described above, the present program was found to fulfill the requirement for accuracy and user friendliness, and is currently being used in several industries.

In the present study, the effects of aging are less than that previously estimated (ISO 1999-1990), primarily because many of the risk factors in concern are age-related. The statistical models and databases of these previous models, did not include these factors. The contributionof these factors to age were not previously adequately assessed. If this explanation accurately explains the difference between the previous studies and the present study, omitting these age-related risk factors, should eliminate the difference between the studies. If these factors are omitted, the current results correlated well with the ISO 1999-model. Today, the role of risk factors and age seem to be greater than the role of exposure at exposure levels of 90 – 97 dB of this study. This finding suggests that an extreme accuracy may not be needed in the evaluation of noise exposure. However, accuracy may deteriorate significantly if the measurements are performed with IEC 651 class 2 equipment by non-qualified personnel. This can result in an error of over 10 dB (Thiery et al. 1991). In a carefully designed measurement, where the accuracy of the instrument is ±1 dB, the given sound pressure level is accurate as mean value, but there might be individual variation due to working methods or the wear of tools that may worsen the measurement result (Pekkarinen et al. 1988). In an epidemiological study this will show up as decreased power of the established model and may even cause a significant underestimation of the importance of noise exposure on hearing loss.

In the present study there was no correlation between noise-exposure and HL among affected family members, as the levels of occupational and free time noise-exposures were quite low, seldom exceeding 90 dB.

Nevertheless, subjects who had passed military service demonstrated worse hearing, though this association could be gender-biased, as females were not attending the military service. An association between analgesics and hearing loss was found. The strongest association was found among non-steroid anti-inflammatory analgesics, which in other studies, have been linked to the deterioration of hearing (Pyykkö et al. 1989). Thus, these factors (non-steroid anti-inflammatory analgesics) that promote hearing loss in noise exposed populations aggravates hearing loss in populations with genetic factors that predispose to hearing loss in the absence of noise exposure.

In a pedigree with hereditary hearing impairment, subjects with impairment demonstrated glue ears, vertigo, and/or tinnitus, more frequently compared to their normal hearing relatives. Possibly the gene mutation(s) involved in present hearing disorder contributes to the immune response of the middle ear. A similar degeneration that causes hearing loss in the cochlea may also causepathology in other parts of the inner ear and the observed vestibular symptoms. This may explain the presence of vertigo in the affected family members.

As the genetic testing is not yet in use and the results for those genes known to be involved in non-syndromic hearing loss are not comprehensive, a list of symptoms indicating possible hereditary origins of hearing loss is included into NoiseScan (Fig 16).

The IHCP can be useful only when NIHL can be predicted with reasonable accuracy. Accuracy can improve by taking into account the characteristics of noise and using additional information retrieved from early indicators. The following criteria will lead to a successful IHCP:

- it must be capable of predicting the hearing loss more accurately than statistical models
- it must be able to find the people with a high risk of NIHL at an early stage
- it must be easy to use in spite of the many input parameters required
- it must be able to rank the sources of exposure

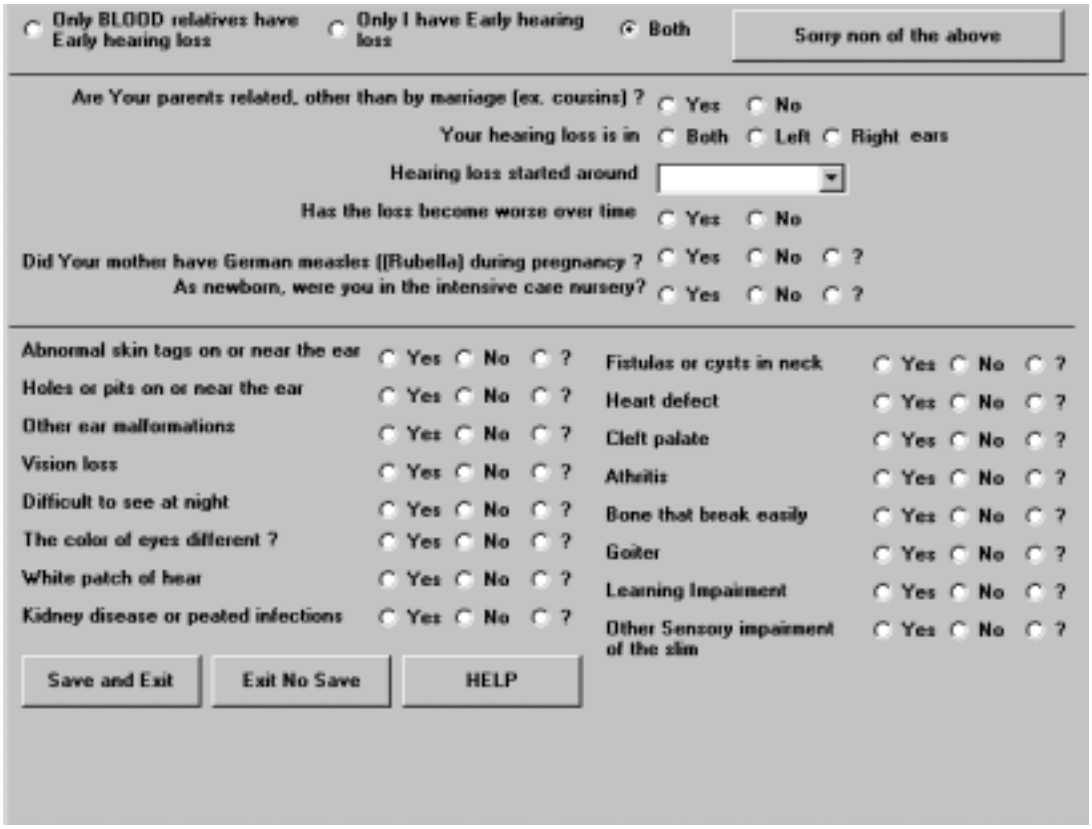


Figure 16. Questions indicating possible genetic HL in NoiseScan.

Accuracy can be improved by increasing the accuracy of total exposure evaluation and by increasing the number of parameters used in the model. Neither of these will increase the accuracy infinitely. The accuracy of total exposure evaluation is limited by the measurement accuracy (ISO 9612-1997), and a lack of measurements of leisure-time noise. In addition to energy contents, other characteristics of noise, like impulsiveness, should also be considered in the risk evaluation. The number of parameters cannot be increased infinitely. Again the number of unknown or poorly defined factors like interaction of noise exposure with different solvents and the combination of different risk factors makes the estimation of any model difficult. These factors should be considered when assessing an ideal model for IHCP.

The Risk factors and their interactionswe have included explained 36% of the permanent threshold shift in the present study (II). In earlier studies about 50% of the permanent threshold shift could be explained among paper mill workers (Nieminen et al. 2000) and 40% among forest workers (Starck et al. 1995). The resultant lack of explanation in the present study may be due to several factors. In this study the groups were heterogeneous, which leads to greater variation. The highly impulsive noise in the shipyard also decreased the power of the model. The leisure-time exposure to noise was not taken into account in this model. Many of the forest workers had hunted in their free time, which caused an

additional exposure to shooting noise. Also the methodological accuracy of the audiometry testing made in the connection of annual health examinations may have contributed variability. However, a previous study showed that neither free time exposure nor time domain noise characteristics could predict permanent threshold shiftvery well in this heterogeneous material (Toppila et al. 1997). A drop in the power of the model emphasizes the need to take into account the above-mentioned factors. In the present study the interaction of occupational noise and leisure-time noise was not observed since there is no accepted method to combine shooting noise with steady-state noise. The risk assessment of impulse noise lacks a generally accepted method. The methodological work to resolve both of these issues is underway and will be tested soon on the present database. Another concern is validation of the NoiseScan program and model in connection with an international database, as dietary habits, different social backgrounds and genetic factors may demand different solutions in an IHCP.

CONCLUSIONS

1. To enhance our knowledge of the individual development of NIHL, several factors linked to SNHL must be collected systematically. Such a large number of factors require the use of an organized database and systematic data collection program, much like the one created in the present study. Noise exposure data includes the occupational, leisure-time and military service noise for the subject's entire lifetime. If any gaps are present in the exposure data, they can be augmented with "educated guesses" contained in the program. The program collects data on confounding factors such as cholesterol, blood pressure, organic solvents, tobacco smoking and use of analgesics, among others. Furthermore, symptoms or diseases that can cause hearing loss not related to noise are also analyzed.

2. NIHL is confounded by the elevation of cholesterol, elevation of blood pressure, smoking and regular use of analgesics. These factors could explain a 35% variation of NIHL. These factors also correlate with exposure and aging and could account for 2 dB of the hearing loss. Impulsive noise in shipyard workers caused excessive hearing loss of 12 dB at 4 kHz when compared to steady state noise. Smoking in combination with other confounding factors increased the level of hearing loss.

3. The effect of age on hearing loss was less than assumed in the current noise models. The difference in estimation is due to the fact that most confounding factors for hearing loss are closely related to age. Elderly workers are more susceptible to noise trauma than younger workers. When the noise exposure level is less than 100 dB, the role of age and confounding factors dominates the effect of exposure. These risk factors seemto have an additive effect with each other and even with noise.

4. In one large pedigree with hereditary hearing loss, the mean deterioration of hearing was 2 dB per year. The use of analgesics and exposure to military noise correlated with hearing loss. The noise exposure at work showed no interaction with the hearing loss in this family.

5. IHCP focuses on prediction of the development of NIHL at the individual level. For this purpose IHCP relies upon input of all factors contributing to NIHL and all factors indirectly contributing

to hearing lossthat are not related to noise. Algorithms combining work noise and leisure-time noise should be included in the IHCP as actual use of HPDs. Such a program should, for research purposes, also have OAE and high frequency audiometer data as input. The prediction of NIHL should be based on algorithms working in a nonlinear manner, as knowledge-based algorithms.

The ultimate goal for IHCP is to prevent hearing loss in industry and if possible, to extend the prevention to leisure-time activities. For these purposes the IHCP should include all relevant variables affecting the mechanism leading to hearing loss. With the present approach it is possible to improve and build an expert system against NIHL that can be used in preventing predictable hearing loss. Hearing loss that is due toother factors or incidents must be approached with other strategies**.** Much more research and knowledge is needed to prevent and treat the genetic factors behind hearing loss. For such a large task, a European network of laboratories is currently being developed.

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