The Influence of Selected Risk Factors on the Hearing Threshold Level of Noise Exposed Employees

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The aim of the study was to evaluate the combined effect of noise exposure and additional risk factors on permanent hearing threshold shift. Three additional risk factors were: exposure to organic solvents, smoking and elevated blood pressure.

The data on exposure and health status of employees were collected in 24 factories. The study group comprised of 3741 noise male exposed workers of: mean age 39 ± 8 years, mean tenure 16 ± 7 years and $L_{EX,8h} = 86 \pm 5$ dB. For each subject, hearing level was measured with pure tone audiometry, blood pressure and noise exposure were assessed from the records of local occupational health care and obligatory noise measurements performed by employers. Smoking and solvent exposure were assessed with questionnaire. The study group was divided into subgroups with respect to the considered risk factors. In the analysis, the distribution of hearing level of each subgroup was compared to the predicted one which the standard calculation method described in ISO 1999:1990. For each of the considered risk factors, the difference between measured and calculated hearing level distribution was used to establish, by the least square method, a noise dose related correction square function for the standard method. The considered risk factors: solvent exposure, smoking and elevated blood pressure combined with noise exposure, may increase degree of hearing loss.

Keywords: noise-induced hearing loss, risk factors.

1. Introduction

In 2003 the European union introduced the noise directive, 2003/10/EC *DIRECTIVE 2003/10/EC OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 6 February 2003, on the minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (noise),* to reduce the harmful effects of noise. In the directive there are lower exposure action levels, $L_{EX,8h} = 80$ dB and $L_{C\text{peak}} = 135$ dB, upper exposure action values $L_{EX,8h} = 85$ dB and $L_{C\text{peak}} = 137$ dB and limit values $L_{EX,8h} = 87$ dB and $L_{C\text{peak}} = 140$ dB. In addition to the exposure limit values of the directive sets, many new requirements that have to be taken into account in the risk assessment: any effects concerning the health and safety of workers belonging to particularly sensitive risk groups; effects on workers' health and safety resulting from interactions between noise and work-related ototoxic substances.

The noise-induced hearing loss is related to life-time exposure, i.e. the life-time *A*-weighted sound energy ISO 1999 (1990). Although the present exposure can be evaluated reliably according to ISO 9612, knowledge of the previous exposure periods is often missing.

There is evidence from epidemiological studies that exposure to organic solvent mixture is associated with an increased risk of hearing loss. Organic solvents have detrimental effects both on the peripheral and central parts of the auditory pathway (ŚLIWIŃSKA–KOWALSKA, 2007). Styrene exposure combined with noise produces worse pure tone audiometric thresholds. Workers exposed to styrene and noise have poorer mean thresholds at 2, 3, 4 and 6 kHz, at the same time they have poorer distortion product otoacoustic emissions (DPOAE) (PRASHER et al., 2009). Mixtures of styrene with other solvents, such as toluene and n-hexane, increases the risk for hearing loss (ŚLIWIŃSKA–KOWALSKA et al., 2003; JOHNSON, 2007). The interaction between solvents and noise on the risk of hearing loss is unclear.

There is a positive association between smoking and hearing loss. The joint influence of noise and smoking on hearing status was reported by (MIZOUE et al., 2003; DENGERINK et al., 1992). Earlier studies suggested that combined with noise exposure, causes more adverse effect on hearing. Smoking can accelerate noise-induced hearing loss, but it is not clear whether the joint effect of noise and smoking is synergistic (VIROKANNAS, ANTTONEN, 1995; STARCK et al., 1999) or additive (MIZOUE et al., 2003; YCHIDA et al., 2005).

The elevated blood pressure may affect the regeneration mechanisms of ear. In the case of elevated blood pressure, the investigations indicate excess risks of hypertension related to long-term noise exposure (JARUP et al., 2008). There are many studies on noise and heart diseases. The comparison of the study results (VIRKKUNEN et al., 2005) confirms a slight increase of cardiovascular disease risk in populations exposed to work noise. The comparison can lead to the conclusion that noise exposure can contribute to the prevalence of cardiovascular disease,

the evidence for a relation between noise exposure and ischemic heart disease is still inconclusive. Other study found that the female workers with low artery compliance or with high blood pressure might be suffering from hearing loss; those with noise-induced hearing loss might be suffering from hypertension if she is constantly exposed to loud industrial noise (NI *et al.*, 2007).

In properly screened hearing loss data, the two major causes exists: presbyacusis and noise exposure (ISO 1999, 1990). These two factors are strongly correlated and presbyacusis may even overrule the effect of noise in hearing loss. As a consequence, extremely large databases are needed, especially when combined effects are quantified. This is the major reason why no dose-relationships were created (ŚLIWIŃSKA–KOWALSKA *et al.*, 2003). Luckily, the ISO 1999 (1990) provides a mathematical model for presbyacusis. Applying this model to eliminate the effect of presbyacusis reduces the number of subjects substantially. The purpose of this study is to evaluate the safe limits for noise exposure in presence of selected risk factors, using mathematical modeling.

2. Material

The data consisting of totally 4668 subjects from seven types of industries. For each subject, the pure tone audiometry of 1, 2, 3, 4, 6 and 8 kHz was recorded by the local occupational health care unit. The noise exposure was measured according to the procedure equivalent to ISO 9612-1997. For each worker, the mean daily average noise exposure ($\overline{L_{EX,8h}}$) over all work periods was calculated using the measurement of all jobs (up to 8 jobs), when working for the current employer and the individual life-time noise exposures (L_{im}) are calculated under the assumption that workers were exposed to same noise exposure during work for previous employers. For L_{im} calculations, the following formulas were applied:

$$\overline{L_{EX,8h}} = 10 \lg \left(\frac{1}{\sum\limits_{i=1}^{N} T_i} \sum_{i=1}^{N} T_i \times 10^{0.1\,L_{EX_i}} \right) \tag{1}$$

and

$$L_{im} = \overline{L_{EX,8h}} + 10 \log(T), \tag{2}$$

where N is the total number of various time intervals/workplaces/jobs; L_{EXi} is the equivalent continuous A-weighted sound pressure level normalized to a nominal 8 hour working day in the time interval/workplace/job i, in dB; T_i is the duration of time interval i, in years; T is total time in years of noise exposure.

The following data were collected: age, gender, tenure, cigarette smoking (years and numbers), head injury, history of acoustic trauma, ear diseases and operations, dizziness, vertigo and tinnitus, blood pressure, weight and length and

Table 1. Mean ages, length of noise exposure, noise exposure levels and life-time noise exposure L_{im} of workers of the investigated risk subgroups (male subjects).

Subgroup exposed to	Age [years]	Exposure length [years]	Noise exposure level [dB]	L_{im} [dB]	Number of cases
	Mean ± SD				
Noise	38 ± 7	17 ± 7	85 ± 4	97 ± 5	928
Noise and solvents	35 ± 9	11 ± 8	87 ± 7	98 ± 5	304
Noise and smoking	39 ± 8	18 ± 7	88 ± 5	97 ± 8	606
Noise and elevated blood pressure	41 ± 7	18 ± 6	84 ± 4	97 ± 4	806
Noise and multiple risks	38 ± 6	15 ± 5	86 ± 5	97 ± 5	1097
All male subjects	39 ± 8	16 ± 7	86 ± 5	97 ± 5	3741

To investigate of the influence of combination of noise and each of selected risk factors on hearing, the first thing to do is to remove the effect of presbyacusis. The following subgroups were formed: workers exposed to noise and only one of selected risk factors – i.e. "single risk factor subgroups". For each risk factor the analysis was performed in the same way. Details of analysis described below considered only subjects exposed to noise or exposed to noise and solvents. The basic features of the chosen subgroups are presented in Table 1.

The removed of the effect of age ISO 1999 (1990) model was used. For each above-mentioned subgroup, the deciles (Q) of HL distribution of given by the standard ISO 1999 (1990) and measured, were found with respect to life-time noise exposure (L_{im}). To smooth the relationship between Q of HL distribution and L_{im} values, the distribution were found for each 5 dB wide intervals of L_{im}, moved with 1 dB step from the bottom to the upper range of the L_{im}. Simultaneously, the number of cases (n) and average values of the L_{im} within each position of the mowing 5 dB interval were found. In this way, relations between L_{im} and values of the observed and predicted HL for considered deciles of the HL distribution were obtained. The same procedure was repeated for HL at 1, 2, 3, 4 and 6 kHz.

The HL values of the observed deciles differ from the predicted ones and these differences are related to L_{im} and (Q). To describe the difference between the observed and predicted distribution in examined subgroups, the second degree polynomial functions were fitted. In the fitting, a weight $w = n^{0.5}$ was applied to each point, where the observed and predicted HL distributions were found. The difference function $DF_s(L_{im}, Q)$ for noise and solvent exposed subgroup and noise exposed subgroup were calculated and functions are expressed by the following equation:

$$DF_s(L_{im}, Q) = \text{const}_s + A_s \cdot L_{im} + B_s \cdot (100 - Q)^2$$
$$+ C_s \cdot L_{im} \cdot (100 - Q) + D_s \cdot L_{im} + E_s \cdot (100 - Q), \qquad (3)$$

where: Q are the deciles corresponding to the ISO 1999 convention of fractiles, equal to (10; 20; ...; 90), in %; L_{im} – is the life-time noise exposure, in dB, Const, A_s, B_s, C_s, D_s; E_s and Const – are parameters depending on frequency and risk subgroups (noise exposed, noise and chemical exposed).

It was assumed that the combined exposure to noise and solvents in one of the considered subgroups leads to difference between the founded CF_s functions. The CF_{chem} function relates the difference between founded DF_s functions with life-time exposure to noise and deciles of the HL distribution (L_{im}, Q):

$$CF_{\text{chem.}}(L_{im}, Q) = DF_{\text{noise \& chem}}(L_{im}, Q) - DF_{\text{noise}}(L_{im}, Q). \qquad (4)$$

In the same form as the one presented by Eq. (3) with modified parameters, the function CF_{chem} was found with no weighted fitting procedure. The function CF_{chem} describes the deviation of the observed HL distribution of subjects exposed to combined exposure to noise and chemical agents from the standard ISO 1999 model prediction. This means that all the deciles Q of the HL distribution calculated with standard model for considered L_{im} should be corrected by adding penalties equal to values of the CF_{chem} function at the same L_{im} and Q. As the predictions of the HL for different subjects depend on L_{im} and subjects' age, the total distributions of predicted HL were build by summarizing the predicted HL distribution of each subject. Total distribution calculated with standard ISO 1999 and ISO corrected models were compared with the observed HL distribution in examined subgroups.

The correction functions were used to calculate the HL predicted distribution in the whole study group – including subjects exposed to more than one risk factor. The appropriate penalties were applied for ISO 1999 model prediction with respect to the subjects' risk factors. Distribution of measured HL and predicted according to ISO 1999 model and ISO model with corrections were compared with statistical tests.

4. Data analysis

The calculation and data analysis were performed with (StatSoft, Inc. (2004). STATISTICA (data analysis software system), version 6. www.statsoft.com). The functions were founded with the least square method. Observed and predicted HL distribution were compared using the Kolmogorov Smirnov test and non-parametric Mann Whitney U test.

5. Results

In spite of the observed life-time noise exposure depending differences between the measured and ISO 1999 predicted hearing threshold level (HL) at all frequencies 1, 2, 3, 4 and 6 kHz, the correction functions for noise and chemical exposure CF_{chem} were found for 2, 3, 4 and 6 kHz. Due to the fact that the

hearing losses are distributed in quite narrow range at first three investigated frequencies, especially at 1 kHz, only 86% of cases have HL smaller than 20 dB, the fitting procedure could not be applied at 1 kHz. At 2 and 3 kHz, the correction functions were founded with fitting procedure applied for small deciles Q of HL distribution.

Table 2 presents the coefficients of fitted correction functions at 2, 3, 4 and 6 kHz with intervals of the L_{im} and Q, where fitting procedure was performed. This was caused by the very uncertain results for HL predictions and measurements of values les than 10 dB. Due to these limitations the correction functions at frequencies 2 and 3 should be considered just as an approximation for deciles above 50 at frequencies 2 and 3 kHz; the same might be told on deciles above $Q = 70$ at frequencies 4 and 6 kHz.

The predicted HL for noise and solvent exposed group were calculated at 3, 4 and 6 kHz. Figures 1a and 1b show comparison between the measured and predicted distribution of hearing losses at 4 and 6 kHz as a function of life time exposure. The drawn deciles are 10, 50 and 90. It may be assumed that calculation with standard ISO 1999 method describe noise-induced hearing loss due to noise exposure, while ISO with corrections shows hearing loss caused by combining exposure to noise and chemical agents.

The medians and widths of the measured HL distribution at 3 and 6 kHz (marked with points and whiskers showing deciles obtained from the data, Fig. 1b), remain quite stable trough the whole range of L_{im} life-time exposures. This is due to the fact that the subjects in the upper end, exposure over 100 dB, they were relatively young and had the highest daily exposures of all populations, majority of them were working as painters in shipyards. There are small elevations of the measured HL distribution visible at the lower end of L_{im} ranges (probably because of underestimation of L_{im}, non-occupational noise exposure may be a significant in the case of low L_{im} values).

At 3 kHz, there are no differences between both the predicted HL distribution up to $L_{im} = 96$ dB. Above this value, the differences of 10% deciles start to increase up to 15 dB. Differences between medians appear for $L_{im} > 100$ dB.

Comparison of HL predicted with both methods shows constant shift at 4 and 6 kHz, when the Lim life time exposure is small, below 96 dB.

At 4 kHz (Fig. 1a), the median corrected due to chemical exposure, is shifted ∼5 dB upwards and the 10% decile about ∼10 dB. This shift is due to the pure ototoxic effect of the solvents. When the life-time exposure exceeds 95–96 dB, the distribution becomes wider and the median value of HL increases; at life-time exposure of 104–105 dB the median HL is ∼20 dB higher than predicted by the ISO model and the difference of 10% deciles at 104–105 dB is ∼30 dB.

At 6 kHz (Fig. 1b) there is a constant ∼10 dB shift between medians of both predicted HL, the shift is independent of the L_{im} value. Similar shift can be seen for 10% deciles when the L_{im} is below 96 dB; with increasing L_{im} the shift increases up to ∼25 dB.

Table 2. Coefficients of correction functions at different frequencies.

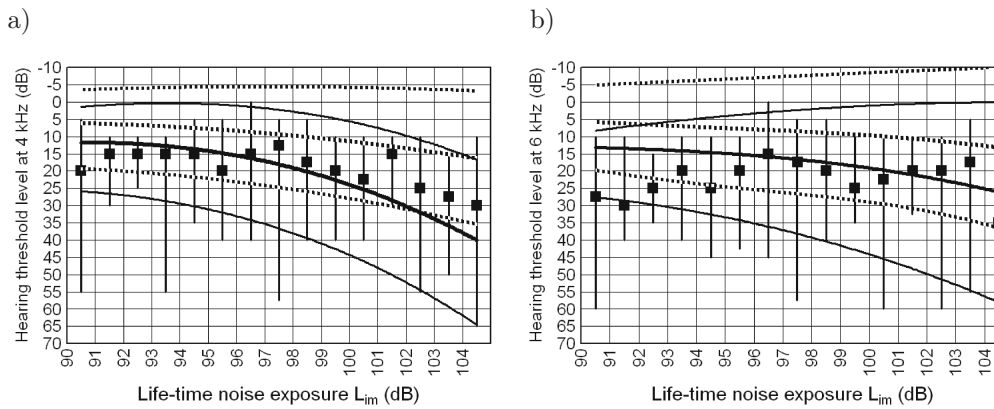| Correction function	Const	$A \cdot L_{im}$	$B \cdot (100 - Q)$	$C \cdot L_{im}^2$	$DL_{im}(100 - Q)$	$E(100 - Q)^2$
at 2 kHz in the ranges: L_{im} (90–105) dB; Q (50–10)						
chemical exposure	553.67	−10.360	−1.663	0.0480	0.0165	0.0015
smoking	454.11	−8.901	−0.815	0.0453	0.0031	0.0043
elevated blood pressure	−86.36	1.923	0.201	−0.0091	−0.0066	0.0038
at 3 kHz in the ranges: L_{im} (90–105) dB; Q (60–10)						
chemical exposure	1182.38	−24.170	−1.545	0.1241	0.0118	0.0040
smoking	920.06	−18.838	−0.978	0.0974	0.0055	0.0047
elevated blood pressure	173.27	−3.432	−0.106	0.0180	−0.0034	0.0046
at 4 kHz in the ranges: L_{im} (90–105) dB; Q (70–10)						
chemical exposure	753.77	−15.134	−1.000	0.0762	0.0105	0.0004
smoking	1081.97	−22.407	−0.700	0.1160	0.0075	0.0004
elevated blood pressure	35.28	−0.300	−0.289	−0.0002	0.0024	0.0012
at 6 kHz in the ranges: L_{im} (90–105) dB; Q (60–10)						
chemical exposure	384.00	−6.453	−2.136	0.0268	0.0202	0.0018
smoking	701.43	−14.516	−0.417	0.0756	0.0030	0.0011
elevated blood pressure	−74.79	1.540	0.383	−0.0073	−0.0055	0.0014

a)

b)

Fig. 1. Deciles 10, 50, 90 of the HL distribution as a function of L_{im} at 4 and 6 kHz; measured (points) and predicted according to ISO 1999 (dashed lines) and ISO corrected model (solid lines).
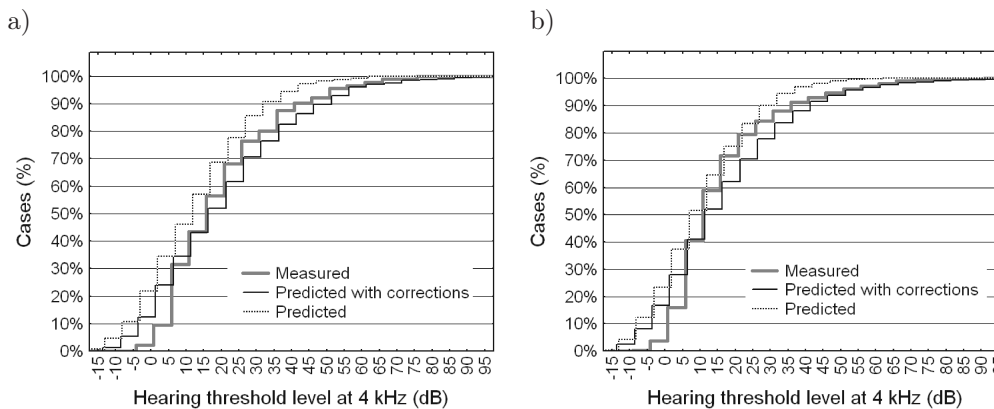
a)

b)

Fig. 2. Cumulative distribution of HL, measured and calculated according to ISO 1999 model and ISO with corrections: a) noise and solvent exposed, b) whole study group.

Figure 2a presents cumulative HL distribution in noise and solvent-exposed subgroup (only exposure to solvent) and (Fig. 2b) presents HL distribution in whole study group. It was found that predicted HL distributions are closer to the measured ones especially for HL greater than 35 dB.

The results showed that elevated blood pressure increased the hearing loss but the increase seems to be independent of the noise exposure. For smoking there seems to exist an effect combined with noise. The effect seems to start when L_{im} exceed 96 dB.

6. Discussion

The 2003/10/EC noise directive sets the lower action limit value to $L_{EX,8h} = 80$ dB. Under this value no hearing loss can occur at speech frequencies (0.5–

2 kHz) according to ISO 1999, but for high frequencies it is possible. In our study, the combined effect seems to start when the life time exposure $L_{im} > 96$ dB. This is equivalent to 20 years of daily exposure to $\overline{L_{EX,8h}} = 83$ dB(A). From this point of view the lower action limit value seems to be appropriate. In this study we could not set a limit value for ototoxic exposure, because the accuracy of the chemical exposure is a more complicated issue and there is no good metrics for the life-time exposure. This is why we contented to divide the subjects into exposed and non-exposed.

The impact was highest at 4 kHz, not observable at 1 kHz and small at 2 kHz. In literature, styrene causes a high frequency hearing loss in the region of 2–6 kHz whereas mixtures affect the mid-frequencies, 1–2 kHz. This indicates that different solvent exposures may have different impact to the number of NIHL case.

ŚLIWIŃSKA–KOWALSKA *et al.* (2007) showed that the mean hearing thresholds at frequencies of 2–4 kHz were poorer for workers exposed to solvents plus noise than for the solvent-only group. Our result confirms this. In addition, we are able to show that the combined effect starts to use when the life time exceeds 96 dB.

Our results are also in good agreement with the results of the animal studies made by CAMPO and MAGUIN (2007). Their result is that daily exposure limit 80 dB is the threshold value below which no combined effect with styrene exist. Thus when a combined exposure to solvents and noise exist, the hearing protection should always start when daily exposure exceeds 80 dB. Smoking and elevated blood pressure combined with noise exposure may increase the degree of hearing loss. The influence of smoking combined with noise exposure on hearing loss depend on L_{im} while in the case of elevated blood pressure and noise the influence is independent from L_{im}.

7. Conclusions

Exposure to solvents has adverse effect on the hearing status. It enlarges hearing threshold levels by 5–10 dB at 4 and 6 kHz even for life-time noise exposure $L_{im} < 96$ dB. Exposure to chemicals combined with noise exposure of higher levels causes greater hearing damages in comparison with the ones caused by noise. The damages increase with increasing life-time noise exposure level, combined exposure enlarged medians of HL distribution by 10–30 dB at $L_{im} = 105$ dB. The effect is greater for sensitive persons. The combined effects between noise and solvents are negligible below the lower action limit value $L_{EX,8h} = 80$ dB set by the 2003/10/EC noise directive. The influence of smoking combined with noise exposure on hearing loss depends on L_{im}, while in the case of elevated blood pressure and noise the influence is independent of L_{im}.

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