EUROPEAN COMMISSION

Directorate-General for Health & Consumers

Scientific Committee on Emerging and Newly Identified Health Risks

SCENIHR

Potential health risks of exposure to noise from personal music players and mobile phones including a music playing function

Scientific Committees
on consumer products
on emerging and newly identified health risks
on health and environmental risks

The SCENIHR adopted this opinion at the 26th plenary on 23 September 2008, after public consultation.

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Three independent non-food Scientific Committees provide the Commission with the scientific advice it needs when preparing policy and proposals relating to consumer safety, public health and the environment. The Committees also draw the Commission's attention to the new or emerging problems which may pose an actual or potential threat.

They are: the Scientific Committee on Consumer Products (SCCP), the Scientific Committee on Health and Environmental Risks (SCHER) and the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) and are made up of external experts.

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http://ec.europa.eu/health/ph_risk/risk_en.htm

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[1] Declared interest (see the minutes of the SCENIHR Plenary: http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_020.pdf ; http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_021.pdf)

[2] Declared interest (see the minutes of the SCENIHR Plenary: http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_021.pdf)

[3] Declared interest (see the minutes of the SCENIHR Plenary: http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_020.pdf)

[4] Declared interest (see the minutes of the SCENIHR Plenary: http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_020.pdf)

[5] Declared interest (see the minutes of the SCENIHR Plenary: http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_020.pdf ; http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_023.pdf)

[6] Declared interest (see the minutes of the SCENIHR Plenary: http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_mi_020.pdf)

ABSTRACT

Exposure to excessive noise is a major cause of hearing disorders worldwide. It is attributed to occupational noise. Besides noise at workplaces, which may contribute to 16% of the disabling hearing loss in adults, loud sounds at leisure times may reach excessive levels for instance in discos and personal music players (PMPs). It is estimated that over two decades the numbers of young people with social noise exposure has tripled (to around 19%) since the early 1980s, whilst occupational noise had decreased. The increase in unit sales of portable audio devices including MP3 has been phenomenal in the EU over the last four years. Estimated units sales ranged between 184-246 million for all portable audio devices and between 124-165 million for MP3 players.

Noise-induced hearing loss is a function of sound level and duration of exposure. In order to counteract noise-induced hearing loss more effectively, a European directive "Noise at Work Regulations" taking effect starting February 2006, established the minimal security level at the equivalent noise exposure limit to 80 dB(A) for an 8 hour working day (or 40 hour working week), assuming that below this level the risk to hearing is negligible. The 8-hour equivalent level ($L_{equ,\ 8h}$) is a widely used measure for the risk of hearing damage in industry, and can equally be applied to leisure noise exposures. The free-field equivalent sound pressure levels measured at maximum volume control setting of PMPs range around 80-115 dB(A) across different devices, and differences between different types of ear-phones may modify this level by up to 7-9 dB. The mean time of exposure ranges from below 1 hour to 14 hours a week.

Considering the daily (or weekly) time spent on listening to music through PMPs and typical volume control settings it has been estimated that the average, A-weighted, eight hour equivalent sound exposures levels (referred to "Noise at Work Regulations") from PMPs typically range from 75 to 85 dB(A). Such levels produce minimal risk of hearing impairment for the majority of PMP users. However, approximately 5% to 10% of the listeners are at high risk due to the levels patterns and duration of their listening preferences. The best estimate from the limited data we have available suggests that this maybe between 2.5 and 10 million people in EU. Those are the individuals listening to music over 1 hour a day at high volume control setting.

Excessive noise can damage several cell types in the ear and lead to tinnitus, temporary or permanent hearing loss (deafness). Published data indicate that excessive acute exposures to PMPs music at maximal or near maximal output volume can produce temporary and reversible hearing impairment (tinnitus and slight deafness). Major discrepancies exist between the results of the studies on permanent noise-induced hearing loss in PMP users, with both, positive and negative studies published. Tinnitus and hearing fatigue may occur more frequently in teenagers chronically exposed to music, including PMP users, than in non-users.

In addition to auditory effects harmful, lasting and irreversible non-auditory effects of excessive listening to PMP can be expected; they include cardiovascular effects, cognition as well as distraction and masking effects. However, there is not sufficient evidence to state that music from PMPs constitutes a risk for such effects.

In the face of an increasing population at risk of hearing loss and tinnitus due to i) increasing PMPs use and acceptance in the EU and ii) the possibility to use PMPs at high sound levels, there is a lack of data concerning:

a) the current PMP use pattern, duration, output level, choice of loud levels and exposure of users to other high level sound sources.

b) the contribution of loud sounds to hearing loss and tinnitus, as well as cognitive and attention deficits in children and young people.

c) long-term studies using more sensitive hearing impairment measures to assess the impact of PMPs on hearing and to identify the potential sub-groups more 'at risk' (e.g.

children, genetic sub-groups and environmental sub-groups such as those who commute to work or school in noisy surroundings).

d) biological basis of individual susceptibility to noise and the benefits from pharmacological treatment.

e) whether excessive voluntary PMP-listening leads to lasting and irreversible cognitive and attention deficits after the cessation of the noise.

Keywords : Health effects, Noise, Noise Induced Hearing Loss, Personal Music Players, SCENIHR

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EXECUTIVE SUMMARY

Exposure to excessive noise is a major cause of hearing disorders worldwide; 16% of the disabling hearing loss in adults is attributed to occupational noise, ranging from 7% to 21% in the various subregions. In order to better counteract noise induced hearing loss, a European directive taking effect starting February 2006, established the minimal security level at the equivalent noise exposure limit to 80 dB(A) (to take account of the responsiveness of the human ear to sound).

Outside the workplace, a high risk of hearing impairment arises from attending or participating in discos and rock concerts, using personal music players, exercising or attending noisy sports (hunting, sports shooting, speedway) or from exposures to military noise. The leisure noise sources including music devices usually generate sounds within a broad frequency and sound pressure level ranges. The equivalent sound levels in discos ranged between 104.3 and 112.4 dB(A), and between 80 and 115 dB(A) from personal music players. Sounds other than noise (such as music) can, at high acoustic levels, be as dangerous for hearing as industrial noise.

It is estimated that the numbers of young people with social noise exposure had tripled (to around 19%) since the early 1980s, whilst occupational noise had decreased.

Therefore the Commission requested the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) to assess:

1. Whether the exposure to noise from devices like personal music players and mobile phones with this function, at levels corresponding to current permissible noise emissions may cause quantifiable health risks, in particular hearing loss and/or hearing impairment to the user, and to specify the relevant outcomes;
2. In case health risks are identified, the SCENIHR is asked:
    a. to identify the level of noise emission safeguarding the health of citizens, taking into account the intensity, length and number of exposures to users of personal music players and mobile phones with the same function and
    b. to identify priority issues for further research.

Over the last few years, there is a trend for an increasing population risk due to PMPs, as their qualities improved and they have become used by an increasing proportion of the population. Indeed the increase in unit sales of portable audio devices including MP3 has been phenomenal in EU over the last four years (2004–2007). Estimated units sales could be in the range of 184–246 million for all portable audio devices and in the range of 124–165 million for MP3 players. Last year, the sales of mobile phones reached a similar number of units i.e ca. 200 million. However, so far the availability of the MP3 functionality is not widespread in these handsets (ca. ten percent). Its use is even more unknown. So, at present the major risk to hearing, if use is inappropriate, is through portable audio devices, and particularly MP3 players.

It should be mentioned that although the data for the portable audio market are accessible, there are no demographics easily available on these sales, nor any information on how many devices an individual may buy over a given time period, how long they last before being discarded and how long and in what situations they are used. Thus, it is hard to estimate the proportion of the population that has access to portable audio or to MP3 players, and how many use them on a daily basis. However, it may be estimated on rather conservative way that in EU a number of daily users of devices like personal music players and mobile phones with this function, are in the tens of millions.

As shown by many studies, noise-induced hearing loss (NIHL) is a function of sound level and duration of exposure. The amount of energy absorbed in the ear is physically the

product of sound level by exposure time. Using this simple rule, also known as the equal energy principle, it follows that a given increase of sound level associated with a proportional decrease in duration will amount to the same risks. All data indicate a large inter-individual variability in vulnerability to excessive sound exposures, some subjects being affected while others are not; up to now the factors underlying this variability are very poorly known.

Excessive noise can damage several cell types in the inner ear, but most affected are the outer hair cells. The sequence of these pathological events and their cause/effect relationships have been profoundly explored in animals showing good correlation between morphological signs of pathology and the functional (audiometric) measures. In humans NIHL accrues progressively and often unnoticed until it reaches a certain degree. Very high levels of noise exposure can lead to acute mechanical damage to inner and outer hair cells, but this form of damage is very rare. More commonly, there is a chronic damage that builds up slowly over time. Several factors can have detrimental effects to hearing, apart from noise exposure. These are exposures to several chemicals, ototoxic drugs and lowered levels of breathed oxygen which were found to increase NIHL. The study of the possible involvement of genetic factors has only recently started. Emerging evidence points to the implication of some genes and the exclusion of other candidate genes.

With the digital formats of sound currently available (e.g. MP3) for recording and reproduction, it is possible to reach high levels of sound output without distortion. The personal music players (PMPs) now play not only music, but provide podcasts of various broadcasts or lecture material, which is delivered largely through ear-bud type insert ear phones producing a range of maximum levels around 80-115 dB(A) across different devices. The difference in ear-phone type may increase the level by 7-9 dB with ear-bud type producing the highest levels in the ear canal. The actual sound level at the eardrum is then influenced by the insertion depth of the ear-bud in the ear canal. It is possible to obtain sound level of about 120 dB(A) in the worst case scenario.

In addition to the intensity level, another factor involved in the potential risk assessment is the time or duration of exposure at a particular level. Exposure to sound at the level exceeding 80 dB(A) is considered a potential risk if the exposure at that level continues for 8 hours a day, five days a week for tens of years. On the basis of equal energy, level and time of exposure may be traded with halving of time of exposure with every doubling in level (+3dB). The Noise at Work Regulations in the EU countries set the exposure level at 80dB for 8 hours per day for 5 days a week before action is taken. Using the equal energy basis it may be deduced that 80 dB(A) for 40 hours would be equivalent to 83 dB(A) for 20 hours and 89 dB(A) for 5 hours per week. Assuming that an average PMP user listens for 7 hours per week (1 hour/day), this would exceed the Noise at Work regulations if the sound level for the PMP exceeded 89 dB(A). The A-weighted, eight hour equivalent sound exposures levels from PMPs has been estimated in the literature to have a mean between 75 and 85 dB(A). However, there is a wide variation around those means. The type of music and environment only slightly influence exposure levels. Assuming that music as a cause of noise-induced hearing loss could be compared with industrial noise, such exposures produce minimal risk of hearing impairment for the majority of PMP users. However, a small proportion of users are at a higher risk due to the levels patterns and duration of their listening preferences. Considering the daily (or weekly) time spent on listening to music through personal music players and the typical volume control settings, approximately 5% to 10% of the listeners are at high risk of developing permanent hearing loss after 5 or more years of exposure. Those are the individuals listening to music over 1 hour a day at high volume control setting.

Literature data indicate that excessive acute exposures to PMPs music at maximal or near maximal output volume can produce reversible hearing impairment (temporary threshold shift) up to 30 dB at 4 kHz in some individuals after short time (one or more hours) of exposure. However, the risk of hearing loss and tinnitus is much smaller compared to pop concerts and discotheques music exposures.

There are major discrepancies between the results of the studies on permanent NIHL in PMP users. They could arise from different study designs and methodology. Most of these studies showed none or only small permanent effect of using PMP on hearing in the majority of users, if consequences were assessed with audiometric hearing thresholds, over a period of a few years, whilst participants in the research were still young. On the other hand there is a population study which indicates such a risk. In the third national health and nutrition examination survey of 1988-1994 in the USA it was found that, among children aged 6-19 years, 12.5% had noise-induced threshold shift (NITS) in one or both ears, with higher prevalence in boys (14.2%) compared to girls (10.1%), and in older children aged 12-19 (15.5%) compared to 6-11 year olds (8.5%). Moreover, among children meeting NITS criteria 14.6% had a noise notch for both ears. This warning study needs confirmation and no equivalent data exist on the European population.

In the face of an increasing population at risk of hearing loss and tinnitus due to i) increasing PMPs use and acceptance in the EU and ii) the possibility to use PMPs at high sound levels, there is a lack of data concerning:

a) the current PMP use pattern, duration, output level, choice of loud levels and exposure of users to other high level sound sources.

b) the contribution of loud sounds to hearing loss and tinnitus, as well as cognitive and attention deficits in children and young people.

c) long-term studies using more sensitive hearing impairment measures to assess the impact of PMPs on hearing and to identify the potential sub-groups more 'at risk' (e.g. children, genetic sub-groups and environmental sub-groups such as those who commute to work or school in noisy surroundings).

d) biological basis of individual susceptibility to noise and the benefits from pharmacological treatment.

e) whether excessive voluntary PMP-listening leads to lasting and irreversible cognitive and attention deficits after the cessation of the noise.

1. BACKGROUND

The health effects of exposure to noise have been known for a long time, in particular noise-induced hearing damage such as irreversible hearing loss and impairment. Hearing loss appears to accompany ageing, but noise induced hearing damage can be prevented to a large extent by reducing exposure time and levels. Measures to this effect have been introduced at the workplace.

Recently the attention of the Commission services has been drawn to the need to reassure itself that sufficient preventive measures are in place to prevent hearing damage among children and adolescents from the noise of personal music players and radio communication devices including such a facility[7].

The regulatory framework governing the safety of this equipment is as follows:

- The Radio and Telecommunications Terminal Equipment (R&TTE) Directive[8] 1999/5/EC governs the health and safety aspects of radio equipment, including mobile phones;
- The Low Voltage Directive (LVD) 2006/95/EC[9] governing the health and safety of electrical equipment within certain voltage ranges lists the standards referred to below for other types of equipment;
- The General Product Safety Directive[10] (GPSD) 2001/95/EC seeks to ensure that all consumer products are safe where this aspect is not further detailed in any other "specific" EU legislation (including personal music players).

The R&TTE and LV Directives make reference to European Harmonised Standard EN 60065:2002 "Audio, video and similar electronic apparatus - Safety requirements". This standard provides the technical detail to ensure the safety of users of personal music players with headphones or earphones. It requires compliance with maximum pressure level and maximum voltage outputs measured following the methods described in standards EN 50332-1:2000 and EN 50332-2:2003. None of the standards currently require any specific labelling in respect of noise emissions.

In 2005 the French authorities updated a national Order of 1998 aimed at preventing users of personal audio equipment from suffering long term hearing impairment[11]. In addition to the maximum pressure and voltage requirements prescribed in the two harmonised standards mentioned above the French national Order requires information and/or labelling for the end user. The revised Order entered into force on 1 May 2006 and its scope includes both personal audio equipment and mobile telephones.

The Commission considers it necessary to request the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) to assess whether the health of citizens is appropriately protected by the current requirements of the above-mentioned Community directives and European standards.

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7 Digital technologies have stimulated the distribution and use of a new generation of personal music players. The digital music players available on the market have maximum output noise levels of 90 to 120 dB(A). Furthermore, using software available on the internet enables to exceed these levels and reach values of 130 dB(A).

8 http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:31999L0005:EN:NOT

9 http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2006:374:0010:0019:EN:PDF

10 http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:32001L0095:EN:NOT

11 French Order of 8th November 2005 implementing Article L. 5232-1 of the Public Health Code

2. TERMS OF REFERENCE

The SCENIHR is asked to assess, in the light of current scientific data and knowledge:

1. Whether the exposure to noise from devices like personal music players and mobile phones with this function, at levels corresponding to current permissible noise emissions may cause quantifiable health risks, in particular hearing loss and/or hearing impairment to the user, and to specify the relevant outcomes;
2. In case health risks are identified, the SCENIHR is asked:
   a. to identify the level of noise emission safeguarding the health of citizens, taking into account the intensity, length and number of exposures to users of personal music players and mobile phones with the same function and
   b. to identify priority issues for further research.

3. SCIENTIFIC RATIONALE

3.1. Introduction

In a recent report WHO states (WHO 2002, Nelson 2005) "Worldwide, 16% of the disabling hearing loss in adults is attributed to occupational noise, ranging from 7% to 21% in the various subregions". For almost two decades, the level of 85 dB(A) was regarded as the critical intensity for the workplace; at exposures below 85 dB(A) the probability of hearing losses occurring with long-term exposure was then considered sufficiently limited (Welleschik 1979). Therefore, international standards recommended the equivalent sound pressure level ($L_{equ,\ 8h}$) of 85 dB(A) (A filter-weighted, 8-hour working day-weighted average) as the exposure limit for occupational noise (ISO 1999:1990; NIOSH revised criteria 1974). However, more recent studies showed that this standard did not guarantee the safety for the human auditory system. Therefore, the new EC Directive Noise at Work Regulations on the minimum health and safety requirements regarding exposure of workers to the risks arising from physical agents (noise) introduces lower exposure action value at $L_{equ,\ 8h}$ = 80 dB(A) (Directive 2003/10/EC).

Although early reviews (eg MRC 1986) concluded that leisure noise was unlikely to be a significant threat to hearing compared to occupational noise, they noted a need for more good data and research. Since then there have been huge changes in patterns of noise exposure. Smith et al. (2000) found that the numbers of young people with social noise exposure had tripled (to around 19%) since the early 1980s, whilst occupational noise had decreased.

There is a number of studies which documented that noise from environmental sources like traffic, aircraft, construction or neighbourhood, although sometimes very annoying, do not reach the equivalent levels that can be harmful to hearing. On the other hand, they can cause non-auditory effects. In the last years a pattern of environmental noise exposures has changed substantially; the leisure noise sources became of a main public concern as it was found that they can generate sounds across a broad frequency range and from high to low sound pressure levels. The equivalent sound levels in discos may range between 104.3 and 112.4 dB(A), and between 75 and 105 dB(A) from personal music players (Serra et al. 2005). The noise dose measures over 4 hours showed an L_{aeq} of 104.3 dB. The nightclubs' average sound level ranged between 93.2 to 109.7 dB(A). Therefore it may be concluded that sounds such as music can, at high acoustic levels, be as dangerous for hearing as industrial noise.

In the last decade, PMPs with improved qualities and suitable for playback at high sound levels became available and have been used by an increasing proportion of the population. Data shows that for the MP3 players and equivalent devices the unit sales in Europe, between 2004–2007, were estimated as about 124 million but could be as large as 165 million and for all portable audio could be in the range 184–246 million. On top of this there were about 161 million handset mobile phones sold in EU countries in 2007 only. It is estimated that today about 10-20% of these phones include a MP3 playback function. This results in an estimated additional number of 16 to 32 million PMP devices. It is expected that the fraction of mobile phones containing the PMP function will rapidly increase such that up to 75% of all phones sold by 2011 may provide this function. Notably, data are not very precise at present and it is not clear whether people who have access to PMP function actually use them on a regular basis.

The personal music players (PMPs) which now play not only music, but provide podcasts of various broadcasts or lecture material, which is delivered largely through ear-bud type insert ear phones producing a range of maximum levels around 88-113 dB(A) across different devices. In the worst case scenario, it is possible to obtain level of about 120 dB(A).

Taking into consideration the above mentioned data, the Commission requested the Scientific Committee on Emerging and Newly Identified Health Risks to assess whether the health of citizens is appropriately protected by current requirements of Community directives and European standards by formulating terms of reference.

3.2. Methodology

The Working Group has considered evidence derived from a wide variety of sources, including peer-reviewed scientific literature and published reports of institutional, professional, governmental and non-governmental organisations. In common with the usual practice of SCENIHR Working Groups, no reliance has been made on unpublished work or publicly available opinions that are not science based.

During the course of the deliberations of the Working Group, a Call for Information was issued by the Commission and the replies have all been considered.

As a general rule, scientific reports that are published in English language peer-reviewed scientific journals are considered primarily. This does not imply that all published articles are considered to be equally valid and relevant for health risk assessment. On the contrary, a main task is to evaluate and assess the articles and the scientific weight that is to be given to each of them. Only studies that are considered relevant for the task are commented upon in the opinion. Many more reports were considered than are cited in the reference list. However, only articles that contribute significantly to the update of the opinion are explicitly discussed, commented and cited. In some areas where the literature is particularly scarce, namely on market trends in sales of PMPs and mobile phones with MP3 function, data obtained from professional databases were obtained and analyzed for relevance and importance by experts.

Relevant research on the assessment of health risks related to listening to PMPs' can be divided into broad sectors such as epidemiologic studies and experimental studies in humans. Other studies, used frequently in other risk assessment procedures, such as experimental studies in animals and cell culture studies were considered occasionally, only when necessary to understand the mechanisms of potential noise induced hearing loss.

A health risk assessment evaluates the evidence within each of these sectors and then weighs together the evidence across the sectors to a combined assessment. This combined assessment should address the question of whether or not a hazard exists i.e., if there exists a causal relationship between exposure and some adverse health effect. The answer to this question is not necessarily a definitive yes or no, but may express the weight of the evidence for the existence of a hazard. If such a hazard is judged to be present, the risk assessment also estimates the magnitude of the effect and the shape of the dose-response function, used for characterizing the magnitude of the risk for various exposure levels and exposure patterns.

A full risk assessment also includes exposure assessment in the population and estimates of the impact of exposure on burden of disease. Epidemiological and experimental studies are subject to similar treatment in the evaluation process. It is of equal importance to evaluate positive and negative studies, i.e., studies indicating that the exposure to noise from devices like PMPs' and mobile phones with this function have an effect and studies not indicating the existence of such an effect. In the case of positive studies the evaluation focuses on alternatives to causation as explanation of the positive result: with what is the degree of certainty for ruling out the possibility that the observed positive result is produced by bias, e.g. confounding or selection bias, or chance. In the case of negative studies one assesses the certainty with which it can be ruled out that the lack of an observed effect is the result of (masking) bias, e.g. because of too small exposure contrasts or too crude exposure measurements; one also has to evaluate the possibility

that the lack of an observed effect is the result of chance, a possibility that is a particular problem in small studies with low statistical power.

Obviously, statistical significance is only one factor in this evaluation. Other characteristics of the study are also taken into account, such as the size of the database, the assessment of the participation rate, the level of exposure, and the quality of exposure assessment. The observed strength of association and the internal consistency of the results, including aspects such as dose-response relation are particularly important. Regarding experimental studies, additional important characteristics are the types of controls that have been used and the extent to which replication studies have been performed. It is worth noting that this process does not assess whether a specific study is unequivocally negative or positive or whether it is accepted or rejected. Rather, the assessment will result in a weight that is given to the findings of a study. In the final overall evaluation phase, the available evidence is integrated over various sectors of research.

3.3. Sound: Definitions and measurements

3.3.1. Definitions

In view of the clarity required for this document and as an aid to communications between disciplines and across national borders it is important to agree on definitions for scientific and technical purposes. It is also noted that the use of some words like e.g. 'noise' is not consistent between disciplines and therefore needs a definition.

Note that in the language of the electronic devices, noise is used for that part of the signal of statistical nature which is not carrying the intended information, as it is reflected for example in signal to noise ratios. Next to statistical noise there is non-statistical 'hum' on many signals to be tabulated. The signal on one line may actually contribute to the hum or noise on the other line specifying the 'cross talk'.

In the world of sound, however, noise has also a slightly different meaning in that it is any sound which is not desired by a certain observer. Therefore, in the context of the current mandate, the use of the word noise has to be carefully explained: While, on the basis of the above definitions, the use of the word 'noise' is reserved for those cases where the potentially affected person is not intentionally listening, this is not conclusive for the users of personal music players and the like. Because the sound pressure levels of earphone devices to an outside observer remain far below the limits of physiological effects, it is the sound of personal music players which is of concern to this mandate. This outside observer may nevertheless be distracted and annoyed and may rightfully, in the lexicographic meaning of the word call noise what is sound in his neighbours ears.

For the historic importance of noise protection in work environments like factories or the transportation industry the word 'noise protection' and 'noise-induced hearing loss and impairment' have been coined while a number of such terms e.g. in the context of professional musicians and their job do not really qualify the use of the word 'noise'. Therefore, and for the scope of comparability between the scientific literature in both cases, that of noise exposure and of sound exposure (which leads to very similar physiological effects at comparable levels) it has been decided for the purpose of this mandate to keep up with the established wording and to use the word 'noise' irrespective of whether the 'noise' exposure is wanted (e.g. when playing a personal music player) or not (e.g. in the typical workplace setting). Thus, 'noise' is used consistently in the context of all disease and malfunction patterns, while the word 'sound' is used consequently throughout this opinion to clarify that the concern is the voluntary listener of personal music players and not the observer of the listening situation.

3.3.2. Sound: Physical and technical background

Sound or Sound waves comprise a wave phenomenon. Sound waves are 'longitudinal' waves because sound waves consist of areas of higher and lower local pressure. The propagation of sound waves occurs in all media, i.e. in gases, liquids and solids as well as in more complex fluids like e.g. organisms and tissues.

Fundamentally, sound waves are characterized by their spectrum. A spectrum is the summation of individual frequencies (f) and amplitudes a certain signal has in the surrounding medium. In daily acoustic settings sound is a complex summation of many different sounds from different sources. Sound will not propagate through vacuum and its propagation is influenced by material properties like density and compression / shear strengths. Characteristic parameters of sound waves in a given situation derive from the fundamental wave equation which may be to challenging to evaluate for a given complex scenario.

The exposure to sound in a typical setting is determined by many factors which are not always easy to assess. For sound propagation, the geometry of the room, the surface materials and furnishings as well as its occupation, the materials and media surrounding the source and the listener play a determining role. Like for any other wave, the sound wave at a specific location depends on interference from different sources which depends on the relative phase reaching the location from different sources or after travelling different pathways. Thus, the distribution of energy and the energy absorption in sound exposure scenarios is not necessarily straightforward which leads to the many flavours of acoustics as subfields of physics and engineering, medicine and architecture. Well-known examples are the different acoustical characteristics of a furnished and unfurnished room, the sound-design of commercial products like cars and the engineering of anti-sound-reflection surfaces to be used in the prevention of sound propagation next to highways, railway lines, but also within sound-studios and in other architectural settings. Notably also details of the anatomy of the ear, the hair dress and clothing specific to one listener may affect the sound distribution before the sound reaches the sound sensitive cells in the inner ear of a specific observer.

To assess the exposure from different sources in a specific point, it is common use to analyse the different contributions by their frequency and to provide certain measurements related to sound (like power, amplitude etc) by their densities in the frequency spectrum. Depending on whether sound waves are harmonic ('tones', 'hum') or relate to uncorrelated events. Sound with an equal energy distribution across frequencies is called 'white noise', while most sources of sound exhibit dominating frequency bands originating from resonance phenomena. Typically, the above described complex interaction of sound waves with the particular environment and media (absorption, refraction, reflection and interference) leads to a changing spectrum of sound waves with progressing propagation or the modified position of an observer.

3.3.3. Units of noise exposure

3.3.3.1. Sound pressure level and dB SPL

One parameter of the acoustic (sound) wave which is generally used to assess sound exposure to humans is the sound pressure level expressed in μPa or Pa. Human ear' audible sound pressure levels range from 20 μPa (hearing threshold) till 20 Pa (pain threshold), resulting in the scale 1:10,000,000. Since using such a large scale is not practical, a logarithmic scale in decibels (dB) was introduced which is also in agreement with physiological and psychological hearing sensations.

dB of sound pressure level (dB SPL) is defined as: $20 \log_{10} p1/p0$ where p1 is actually measured sound pressure level of a given sound, and p0 is a reference value of 20μPa, which corresponds to the lowest hearing threshold of the young, healthy ear. In the logarithmic scale the range of human ear's audible sounds is from 0 dB SPL (hearing threshold) to 120-140 dB SPL (pain threshold) (see table 1 below).

Source / observing situation	Typical sound pressure level (db SPL)
Hearing threshold	0 dB
Leaves fluttering	20 dB
Whisper in an ear	30 dB
Normal speech conversation for a participant	60 dB
Cars/vehicles for a close observer	60-100 dB
Airplane taking-off for a close observer	120 dB
Pain threshold	120-140 dB

Table 1: The examples of sound pressure levels in relation to hearing threshold and pain threshold (in dB SPL)

3.3.3.2. Loudness level and filter A [dB(A)]

The human ear is not equally sensitive to sounds (tones) of the same sound pressure levels but different frequencies. This subjective or perceived magnitude of a sound by an individual is called its loudness. The loudness of a sound is not equal with its sound pressure level and differs for different frequencies. In order to assess loudness of a sound the isophonic curves are explored. Isophonic curves relate the characteristic of a given tone expressed in dB SPL to its subjective loudness level expressed in phones (see figure 1 below). As it could be seen in the figure below, the frequencies 3-4 kHz are the most sensitive within sound frequency range from 20 Hz to 20 kHz that can be heard by human ear. For frequencies lower than 3-4 kHz and higher sound frequencies, the ear becomes less sensitive.

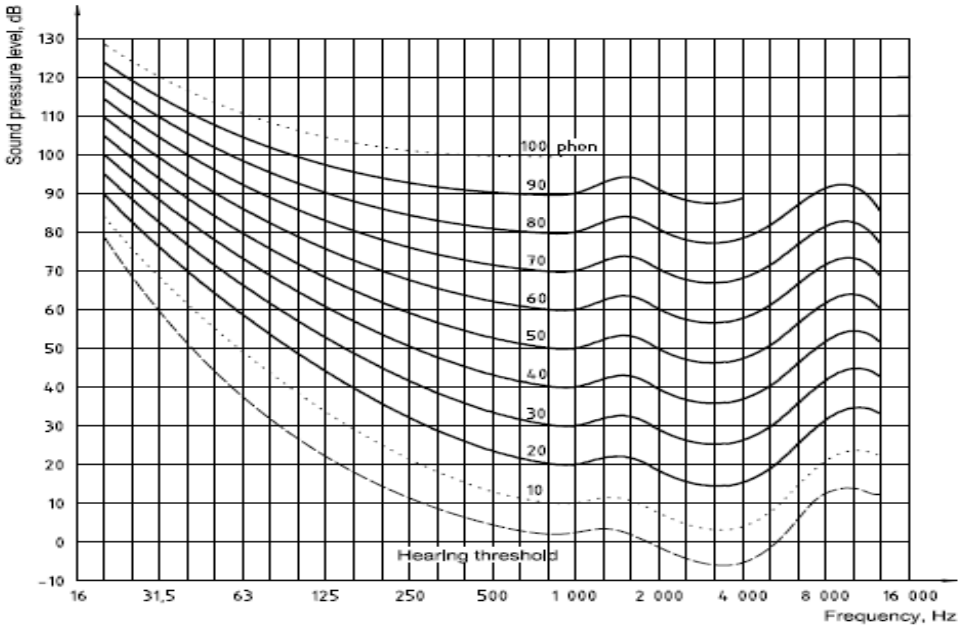

Figure 1[12]: Normal equal –loudness-level contours for pure tones under free-field listening conditions according to ISO 226:2003 (permission for publication kindly granted by ISO)

[12] It should be noted that free-field (binaural) thresholds given in the figure are different from headphone thresholds; the differences depend on the headphone type and frequency tested (BS EN 60645-1:2001*IEC 60645-1:2001; BS EN ISO 389-2:1997).

While sound pressure measurements should give a reading of the sound pressure in dB SPL, in the context of human hearing it is more practical to provide also a value which corresponds more closely to the hearing sensation or loudness in phones. The A, B, and C filters used currently in sound-level meters were aimed at mimicking isoloudness curves over frequency under different conditions of sound intensities, i.e. for sounds of low, medium, and high loudness levels, respectively (IEC 651 1979). The "A" network modifies the frequency response to follow approximately the equal loudness curve of 40 phons, while the "C" network approximately follows the equal loudness curve of 100 phons. A "B" network is also mentioned in some texts but it is no longer used in noise evaluations. The popularity of the A network has grown in the course of time. In current practice, the A- weighting curve filter is used to weight sound pressure levels as a function of frequency, approximately in accordance with the frequency response characteristics of the human auditory system for pure tones. This means that energy at low and high frequencies is de-emphasized in relation to energy in the mid-frequency range.

Correlation between noise effect hearing loss and sound exposure levels measured in A, B, or C weightings would not be very different. B (or even C) weightings provide a better correspondence between loudness and moderate (or high) acoustic levels, however A weighting differs only from B and C as underweighting frequencies below about 500 Hz. Since the human ear is much more resistant to noise-induced hearing loss (NIHL) at and by low frequencies A weighting is more in correspondence with NIHL risk.

It should be noted that the A-filter has been adopted so generally that sound pressure levels frequently quoted in audiology literature simply in dB are in fact A-weighted levels. Many older general purpose sound level meters are restricted solely to A-weighted sound pressure level measurements.

3.3.3.3. Decibel measures in audiometry

Different decibel measures are used in audiometry (evaluation of hearing sensitivity) than in sound pressure measurement. They depend on the reference value.

Pure-tone audiometric thresholds are expressed in dB HL (hearing level) and are referred to hearing thresholds of normal hearing young individuals. The differences between dB HL and dB SPL arise from isophonic curves. Their corresponding values are given in the table below.

Frequency [Hz]	dB SPL	dB HL
250	12	0
500	5	0
1000	2	0
2000	-2	0
4000	-5	0
8000	13	0

Table 2: Audiometric hearing thresholds of normal ears: conversion of dB SPL into dB HL (extracted from ISO, 2003)

Similarly to dB HL, the dB nHL (normal hearing level) values are referred to hearing thresholds of normal hearing individuals but they regard non-tonal sound stimuli (e.g. clicks).

3.3.4. Methodology of noise measurement

Sounds are usually identified by their frequency spectrum, which is also relevant to human perception because the ear analyses sounds in the cochlea by a spectral analysis.

The elemental component of a frequency spectrum is a sine wave or sinusoid with a specific frequency. All sound waves can be described as a linear superposition of sinusoids. Each sinusoid can be characterized by its frequency, its amplitude and the phase in relation to the zero-time mark. Sinusoids with the same frequency and amplitude superimpose either constructively by adding up to a sinusoid with double amplitude if the phase difference is zero and destructively by cancelling out if the phase difference is 180 degrees (or antiphase) resulting in no sound of that characteristic frequency at a given point.

Sound originating from speech and music can similarly be described by their spectrum. In general terms signals can be divided in signals with a tonal character and with a noisy character.

- Signals with a tonal character exhibit a spectrum made up of a basic frequency component (f0) with harmonics (components that have a frequency which is an integer multiple of the basis frequency (n*f0) and a related phase.
- Signals with a noisy character exhibit a spectrum which is more complex than a linear superposition of basic frequencies and their harmonics.

Sound measurements are done by determining the amplitude of the spectral components or by detecting the sound pressure through a physical device, e.g. a microphone. The total sound level of a signal is a root-sums-of-squares of the amplitude of all the spectral components.

Signal levels, including noisy signals and music, are measured by placing a calibrated sound meter (SPL meter) at the centre head location of a potential listener. This method is generally used to determine the risk for hearing loss in working conditions.

The method distinguishes between various possible measures:

- The averaged level, which is the average level of all frequency components over a certain time period
    - The level measurement can be recorded by filtering according to the A, B or C filter; dB (A)
- The peak level indicating the highest level recorded either of the total (weighted) signal or of specific components
- The 8-hour equivalent level ($L_{equ,\ 8h}$) which is a measure for the risk on hearing damage based on certain criteria

The method can also be used to determine the level of music in the open field. Due to the dependence of sound waves on the exact listening situation, as detailed in 3.2, it is clear that this type of measurement is not suitable to head phone use where only a small space between the head phones and the inner ear is exposed to sound waves.

Sound levels of signals presented through headphones are usually measured by artificial ears. Most common are two types, the occluded ear simulator (OES) and the 2 cc coupler. In audiometry and hearing aid specifications all measurements are measured using one of these two couplers. The design of the couplers is based on the resonance properties of the ear canal and the impedance of the tympanic membrane.

In the link of sound transfer from the open field to the ear, there is another transfer characteristic to be included and that is the baffle effect of head and torso. The head effects are usually determined by using a manikin, or as they are also called HATS, head and torso simulator. It consists of a torso and head in which artificial ears are included. The sound pressure is measure at the eardrum. If compared with the free field, this gives the head-related transfer function (HRTF).

It is obvious that HATS and the couplers are based on measurements, averaged over many torsos and ears of both genders taking a multitude of anatomic features into account. Sound levels in individual ears will always differ somewhat from these values. These have to do with the following features:

- Shape of the torso and clothing
- Hair style and head shape
- Shape and volume of the outer ear and ear canal
- Impedance of the tympanic membrane
- Distortion of the sound field caused by other listeners or objects in the room

For the purpose of estimating the risk of the use of individual music players we assume that the calculated sound levels based on the use of artificial heads and ears are good estimates of the real levels.

The risk for hearing damage depends on sound or noise level and exposure time. Criteria were originally developed using working conditions as a reference which are typically measured in the open field. If we want to assess the risk of PMPs we have to compare the levels produced by earplugs or headphones with the measurements done in free field. This implies we have to determine the HRTFs for the different PMPs.

The output level of a PMP is determined by using an artificial ear. It measures the actual sound pressure at the eardrum. To calculate the risk for hearing damage, the free field level has to be calculated by using the inverse HRTF.

3.3.5. Noise assessment

For long term (e.g. workplace) exposure, the level of 85 dB(A) was regarded as the critical intensity; at exposures below 85 dB(A) the hearing losses were significantly lower than for exposures exceeding this value (Welleschik 1979). International standards (ISO 1999:1990; NIOSH revised criteria, 1998) recommended the equivalent sound pressure level ($L_{equ,\ 8h}$) of 85 dB(A) (A filter-weighted, 8-hour working day-weighted average) as the exposure limit for occupational noise (ISO 1999:1990; NIOSH revised criteria, 1974). However, this limit did not guarantee the safety for the auditory system of workers. Therefore, the new EC Directive on the minimum health and safety requirements regarding exposure of workers to the risks arising from physical agents (noise) introduces lower exposure action value at $L_{equ,\ 8h}$ = 80 dB(A) (Directive 2003/10/EC).

Noise at Work Regulations (Directive 2003/10/EC, came into force in 2006) recommend three action levels for occupational settings depending on equivalent noise level for 8-hour working day. If these values are converted using the time-intensity trade-off of 3 dB increase for halving the time then the equivalent levels are shown, for example in a night club with sounds of 104 dB(A) 2 minutes of exposure is equivalent to 80 dB(A) $L_{equ,\ 8h}$. Thus, listening to a PMP player at 95 dB(A) for 15 minutes a day would equate to the first action level, under the assumption of this exposure repeated over a long period.

Action level	$L_{Aequ, 8h}$	Equivalent levels for time indicated (trade-off 3 dB)
First Action level (minimum) provide protection	80 dB(A)	83 dB(A)-4hr[13]; 86 dB(A)-2hr; 89 dB(A)-1hr; 92 dB(A)-30min[14]; 95 dB(A)-15min; 98 dB(A)-8min; 101 dB(A)-4min; 104 dB(A)-2min; 107 dB(A)-1min
Second Action level mandatory protection	85 dB(A)	88 dB(A)-4hr; 91 dB(A)-2hr; 94 dB(A)-1hr; 97 dB(A)-30min; 100 dB(A)-15min; 105 dB(A)-5min; 111 dB(A)-1min
Maximum Exposure limit value	87 dB(A)	90 dB(A)-4hr; 93 dB(A)-2hr; 96 dB(A)-1hr; 99 dB(A)-30min; 102 dB(A)-15min; 107 dB(A)-5min; 113 dB(A)-1min

Table 3: The examples of equivalent time-intensity levels referred to the action levels according to the Directive 2003/10/EC.

Although the above regulations and limits apply to the workplace, the fact that they rely on the exposure level and duration means that they can equally be applied to other situations where sound has a detrimental effect such as that from personal music players; whether use in workplace, or under leisure situations.

3.3.6. Conclusions

For the purposes of this mandate "noise" has been defined as any unwanted sound. The word "sound" is used consequently throughout this opinion to clarify that the concern is the voluntary listener of personal music players and not the observer of the listening situation. Noise exposures and sound exposures at high sound pressure level may result in similar damage to hearing.

The fundamental unit of noise exposure measurement is A-weighted decibel [dB(A)]. This unit corresponds well with the physiological sensitivity of human and it has been generally adopted in scientific literature.

Sound levels of signals presented through headphones are usually measured by artificial ears. In the link of sound transfer from the open field to the ear, the head and torso effects are usually determined by using a manikin.

The risk for hearing damage, as expressed in Noise at Work Regulations, depends on level and exposure time ("equal energy principle"). This regulation (Directive 2003/10/EC) came into force in 2006 and establishes a minimal action level of hearing protection to the limit of 80 dB(A) for an 8-hour working day, equivalent to 89 dB(A) for 1 hour, assuming that below this level the risk to hearing is negligible. The 8-hour equivalent level ($L_{equ, 8h}$) is a widely used measure for the risk of hearing damage.

[13] hr: Hours

[14] min: minutes

3.4. Hearing impairment

3.4.1. Definitions and evaluation

Hearing impairment may be defined to include as a reduction in hearing acuity or sensitivity, or presence of tinnitus. It relates primarily to the inability of the affected individual to hear sounds at certain levels. This is tested by presenting of pure tones at frequencies of 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz, 6 kHz and 8 kHz and is shown in steps of 5 dBHL on a chart known as an audiogram. The threshold of hearing is defined as 0dBHL on the basis of testing a number of young people. It is generally accepted that hearing thresholds lying between 0 dBHL and 20 dBHL across the frequency range tested may be deemed within "normal" limits[15]. Thus any threshold levels at any of the audiometric frequencies listed above may constitute a hearing loss at that frequency of a given amount raised above normal.

There are two types of hearing impairment, defined according to where the problem occurs:

- Conductive hearing impairment, which is a problem in the outer or middle ear. This type of hearing problem is often medically or surgically treatable, if there is access to the necessary services; childhood middle ear infection is the most common example;
- Sensorineural hearing impairment, which is usually due to a problem with the inner ear, and occasionally with the auditory nerve going from there to the brain.

  This type of hearing problem is usually permanent and requires rehabilitation, such as with a hearing aid. Common causes are ageing, excessive noise and ototoxic drugs etc.

World Health Organisation defines hearing impairment (www.who.int/pbd/deafness/en/) as below:

> Hearing impairment is a broad term used to describe the loss of hearing in one or both ears. There are different levels of hearing impairment:
>
> - Hearing impairment refers to complete or partial loss of the ability to hear from one or both ears. The level of impairment can often be usefully graded as mild, moderate, severe or profound;
> - Deafness refers to the complete loss of ability to hear from one or both ears.

[15] However, WHO proposed to set a limit of hearing impairment to 25 dB(A) (see Table 4)

Grade of impairment[16]	Corresponding audiometric ISO value[17]	Performance	Recommendations
0 - No impairment	25 dB or better (better ear)	No or very slight hearing problems. Able to hear whispers.	
1 - Slight impairment	26-40 dB (better ear)	Able to hear and repeat words spoken in normal voice at 1 metre.	Counselling. Hearing aids may be needed.
2 - Moderate impairment	41-60 dB (better ear)	Able to hear and repeat words spoken in raised voice at 1 metre.	Hearing aids usually recommended.
3 - Severe impairment	61-80 dB (better ear)	Able to hear some words when shouted into better ear.	Hearing aids needed. If no hearing aids available, lip-reading and signing should be taught.
4 - Profound impairment including deafness	81 dB or greater (better ear)	Unable to hear and understand even a shouted voice.	Hearing aids may help understanding words. Additional rehabilitation needed. Lip-reading and sometimes signing essential.

Table 4: World Health Organisation Grades of hearing impairment (WHO, 2008)

The WHO table (table 4) relates to the remediation after the acquisition of hearing loss and not to the purposes of protection to prevent noise damage. For the purposes of prevention it is important to consider any significant audiometric threshold shift as a sign of impairment. In order to prevent hearing impairment from occurring it is important to assess the sensitivity of hearing change as soon as possible. Consequently, changes in hearing sensitivity between 0 and 20 dB maybe important, especially in children and young people. Another area of concern is the lack of differentiation between hearing acuity or sensitivity which may be deemed "normal" for children, and young people and older adults as hearing is considered to be "normal" below 20 dB HL for all groups.

The above definitions of hearing impairment reflect one aspect of hearing that of an inability to hear sounds of certain level of intensity. Another factor of importance in hearing and understanding speech is the spectral and timing information. This is crucial for clarity of speech hearing. Timing information is also necessary for another hearing function which allows localisation of sounds in space. These aspects are not considered by the above definition of hearing impairment which considers only intensity information.

Hearing impairment may therefore arise despite a "normal audiometric "threshold which may be due to loss of timing information. In these instances a speech test may show a relatively poor score relative to what may be expected from the audiometric evaluation. This is normally seen in neural or central nervous system lesions.

Hearing impairment may also result from loss of frequency selectivity resulting in poor tuning or selective listening for sounds of interest relative to background sounds which may distract from the information of interest. This may be assessed using frequency tuning curves or the relative strength of the efferent auditory system in modulating incoming signals.

[16] Grades 2, 3 and 4 are classified as **disabling hearing impairment** (for children, it starts at 31 dB).

[17] The audiometric ISO values are averages of values at 500, 1000, 2000, 4000 Hz.

Another aspect of hearing impairment may result from a long-lasting buzzing or ringing known as tinnitus. This may result from over exposure to sounds of high intensity and be short lived or may remain a constant irritation for the listener. Tinnitus may also occur as a consequence of developing a hearing loss from any other cause.

3.4.2. Speech communication difficulties

The ability to understand speech can be described by mathematical models like the Speech Intelligibility Index SII (ANSI S3.5-1997, 1997) and Speech Transmission Index STI (Steeneken and Houtgast, 1980), as a function of the hearing loss and speech level. The STI approach shows that a normal-hearing person can understand speech (normal sentences, no contextual information) if about 30% of the information is present (STI of 0.33). Information can be inaudible due to either a masking noise, or speech being below the audibility threshold as determined in the pure-tone audiogram.

Verschuure and van Benthem (1992), using unmodulated speech noise (i.e, a noise with the same long-term spectrum as the speech), showed that the speech-in-noise threshold depends primarily on the high-frequency hearing loss. A 3 dB poorer threshold is considered clinically relevant and means that the communication distance has to be reduced by a factor 2. If we assume that a normal-hearing person can communicate at a party at a distance of about 1 m a high-frequency hearing loss of about 40 dB makes it impossible to do so; we have to come closer to a speaker and reduce the distance to 50 cm, close to the minimum distance socially acceptable. They also showed that hearing aids can only partly (about 2/3) compensate for that loss. This effect can be well described by the STI approach (Plomp et al. 1978; George et al. 2006). The poorer auditory discrimination causes an additional loss of auditory processing. High-frequency hearing loss, whether aided or unaided by hearing aids, will cause poorer speech understanding in a noisy environment.

Everyday environments consist of many different situations, usually with modulated noises in the background. Only when we are at a noisy party with many participants the noise becomes almost unmodulated. The effect on the speech-in-noise threshold of a six speaker babble is still somewhat better than the effect of an unmodulated noise. Tests as described above have been done using modulated speech noise by Smoorenburg et al. (Smoorenburg et al. 1982). They found that the higher the pure-tone hearing loss the poorer was the ability of a person to listen in the gaps of the modulated noise. This resulted in an extra effect of hearing loss on speech intelligibility in noise. For normal-hearing persons the beneficial effect of modulating noise was about 7 dB, for people with a hearing loss of about 60 dB, this advantage over unmodulated was almost nullified.

As to reverberation, Plomp and Duquesnoy (1980) showed that it has the same effect as a background noise. In a population of healthy elderly people he studied the effect of reverberation on the speech-in-noise threshold. He stated that for normal-hearing persons the maximum reverberation time can be quite long. A person with a relative small high-frequency hearing loss of about 40 dB will on average have a S/N ratio of about 0 dB, resulting in a maximum acceptable reverberation time of about 1.7 s. It means that this person can no longer understand speech in a big church or large meeting hall even if it is completely quiet. In case the loss is about 60 dB the person can only communicate well in well-furnished offices, but not in a large living with a modern design interior.

3.4.3. Tinnitus

Tinnitus may be defined as "a phantom perception of sound", which a person perceives as spontaneous auditory sensations, such as ringing, buzzing, or hissing in the absence of an external signal. The source of the tinnitus sound lies within, rather than outside, the auditory system. In almost all cases, persons with tinnitus have peripheral or central auditory nervous system involvement. Subjective tinnitus, tinnitus originating within the auditory system, is far more common than objective tinnitus.

Tinnitus is often associated with hearing impairment, ageing and noise (Davis, 1989). Estimates for the prevalence of tinnitus in the population need careful attention to detail concerning the wording used and how the response is obtained (either by postal survey, response to questionnaire in clinic, verbal response to clinician). It is useful to determine the prevalence of tinnitus that last for more that five minutes and is not only after loud sounds (Prolonged Spontaneous Tinnitus, PST), which was determined to be 10% by postal questionnaire and 16% by clinical interview (Davis, 1995). There was evidence that clinicians were not as rigorous as the patients in excluding tinnitus just after loud sounds. Davis et al 2007 have shown that whilst 17.7% of people said that they had ever experience such tinnitus about 4% have tinnitus most of the time and 0.4% have their quality of life substantially affected by tinnitus. An other study by Job et al. (2000) showed that in a representative sample of French youth of 18-24 years old, tinnitus was frequently experienced in 8% of the subjects. In young people Smith et al. (2000) and Lovell et al (1998) showed that 9.2% of young people aged 18-25 years reported PST with only 6.8% in those who had not had substantial social noise exposure equivalent to 80 dB(A) leq40 for 50 years. For those who had a greater social noise exposure then the prevalence of PST was 20%, a substantial increase over those who have less than this significant level of noise exposure.

Many theories exist and have been published about the underlying physiological mechanisms that cause subjective tinnitus (Baguley 2002). Generally, theories involve hyperactive hair cells or nerve fibers activated by a chemical imbalance across cell membranes or decoupling hair cell stereocilia. An alternate theory proposes that injury to cochlear integrity from any cause reduces the suppressive influence of the central nervous system, allowing increased neuronal activity higher in the auditory system.

Whatever the cellular mechanism is it appears that tinnitus can be caused by abnormal conditions in the cochlea, the cochlear nerve, the ascending auditory pathway, or the auditory cortex.

3.4.4. Age-associated hearing loss

Hearing ability deteriorates with increasing age in virtually all members of human populations. Numerous studies have quantified this phenomenon, to the extent that it is characterised in international standard ISO 7029 (ISO 7029, 2000). The standard models the distribution of hearing threshold levels in males and females separately in terms of deviations from a baseline set at the age of 18 years. The distributions are semi-normal, defined by mean values and standard deviations representing the upper and lower parts of the distribution. The mean (equal to median) values rise gradually with age at first then accelerate for older people. The standard deviations also increase with age, giving a wide spread for older people. Hearing deteriorates more with age in men than in women. The standard only specifies the distributions up to the age of 80 years, due to limitations on the source data.

For the purposes of the present study, which involves only young adults, ISO 7029 shows little change over the age range from 18-25 years. For example, at the median there is an increase of less than 1 dB at any frequency from age 18 to 25 years in either males or females.

There has been substantial debate about the baseline value used to represent hearing threshold level for 18-year-olds. The original standard implied that a value of 0 dB should be assumed based on numerous studies of highly screened populations of young adults. However, those studies involved participants who were not representative of the population at large and had thresholds that are slightly better than the whole population. More recent studies in the UK have shown that a baseline in the range 2-7 dB is more representative of the otologically normal UK population (Lutman and Davis 1994).

3.4.5. Conclusions

Hearing impairment may be defined as a reduction in hearing acuity or sensitivity, or tinnitus. Hearing loss is the inability of the affected individual to hear sounds at certain levels that can be measured with pure-tone audiometry. According to the WHO Grades of hearing loss no or very slight hearing problems exist when hearing threshold in the better ear is at or below 25 dB.

The reduction in hearing acuity results in the impairment of speech understanding. High-frequency hearing loss, which is typical for age and noise-related hearing impairments may result in worsening speech-in-noise comprehension. It is on average by 1.2 dB per 10 dB. The ability to understand speech may be also impaired despite a normal audiometric threshold due to loss of timing information.

Tinnitus defined as "a phantom perception of sound" is a common problem. It is usually originating within the auditory system and is caused by abnormal conditions in the cochlea, the cochlear nerve, the ascending auditory pathway, or the auditory cortex.

Hearing ability deteriorates with increasing age in virtually all people and this deterioration accelerates for older people. In young adults, up to the age of 40, this process is slow and leads to negligible levels of hearing impairment.

3.5. Noise-induced hearing loss and associated impairments

Noise-induced hearing loss (NIHL) accrues progressively and often unnoticed until it has reached a certain degree. The main site of impairment is the outer hair cells of the cochlea, where the damage is irreversible (Bamiou and Lutman 2007). Very high levels of noise exposure can lead to acute mechanical damage to inner and outer hair cells, but this form of damage is very rare. More commonly, there is chronic damage that builds up slowly over time. Since noise-induced hearing loss is irreversible, the main form of treatment is prevention.

3.5.1. Epidemiology of noise-induced hearing loss

Exposure to excessive noise is one major cause of hearing disorders worldwide. The Word Health Organization programme for Prevention of Deafness and Hearing Impairment (WHO 1997, Smith 1998) stated: "*Exposure to excessive noise is the major avoidable cause of permanent hearing impairment worldwide. Noise-induced hearing loss is the most prevalent irreversible industrial disease, and the biggest compensatable occupational hazard. More research is needed on basic mechanisms and means of prevention*". In a more recent report WHO states (WHO 2002, Nelson 2005) "*Worldwide, 16% of the disabling hearing loss in adults is attributed to occupational noise, ranging from 7% to 21% in the various subregions*". Surveys estimate that noise-induced hearing loss (NIHL) affects 10 to 15 million people in the USA (see Lynch and Kil 2005). In the UK (Palmer et al 2002b) showed that some about 180,000 people aged 35-64 years were estimated to have severe hearing difficulties attributable to noise at work and for tinnitus this increased to 350,000 people who were seriously affected. In France a survey by the ministry of employment (Sumer: surveillance médicale des risques professionnels 2003, Magaud-Camus 2005) indicates that approximately 7% of employed workers are exposed to excessive noise levels (more than 85 dB(A) for at least 20 hours per week) and about 25 % are exposed to hazardous noise exposures (more than 85 dB(A) but less than 20 hours per week); most exposed workers belong to industry (18%) and, to agriculture and house building (12%). Legally acceptable levels of noise exposure were defined many years ago taking into account the two main physical parameters of acoustic intensity and duration of exposure as used for reference above.

In order to better counteract NIHL a European directive that took effect in February 2006, lowered the first action level (provide protection) to 80 dB(A) (Directive 2003/10/EC, 2003). Acute acoustic trauma from firearms is the most frequent pathology

observed in the French army and unfortunately its prevalence increased by about 20 percent in 2006 (BS EN ISO 389-2:1997, 2006).

Although early reviews concluded that leisure noise was unlikely to be a significant threat to hearing compared to occupational noise (e.g. MRC 1986), they noted a need for more good data and research. Since then there have been significant changes in the patterns of noise exposure. Smith et al. (2000) found that the numbers of young people with social noise exposure had tripled (to around 19%) since the early 1980s, whilst occupational noise had decreased.

This increase of risk is consistent with a recent study by Niskar et al (2001), who estimated the prevalence of noise-induced hearing threshold shift among children aged 6-19 years in the third national health and nutrition examination survey of 1988-1994 in the USA. They found that 12.5% had noise-induced threshold shift (NITS) in one or both ears, with higher prevalence in boys (14.2%) compared to girls (10.1%), and in older children aged 12-19 (15.5%) compared to 6-11 year olds (8.5%). 6kHz was the most affected frequency (77.1%) compared to 4 kHz (23.8%) and 3 kHz (14.1%). A single frequency was involved in 88.4% of children. Among children meeting NITS criteria 14.6% had a noise notch for both ears. No equivalent data exist yet on the European population. In the earlier studies (Davis 1989, Davis 1995, Smith et al 2000) there was no evidence of such notches in 18-25 year old individuals in the UK.

3.5.2. Environmental noise exposure levels

In industry settings, the noise levels can average up to 90-125 dB in several areas of work. Outside the workplace, a high risk of hearing impairment arises from attending discos and rock concerts, exercising noisy sports (hunting, sports shooting, speedway) or from exposures to military noise. Children could be exposed to noisy toys as trumpets (92–125 dB SPL), whistles (107-129 dB SPL) and toy weapons (113- >135 dB SPL) (Plontke et al. 2004).

By their leisure activities individuals expose themselves to noise sources including personal music players which usually generate sounds across a broad frequency range and reaching high sound pressure levels. The equivalent sound levels in discos range from 104.3 to 112.4 dB(A), compared to 75 to 105 dB(A) from personal music players (Serra et al. 2005). The noise dose measures over 4 hours showed an L_{aeq} of 104.3 dB. The nightclubs' average sound level ranged between 93.2 to 109.7 dB(A). Sounds other than noise (such as music) can, at high acoustic levels, be as dangerous for hearing as industrial noise.

There seems to be a trend for increased distribution and use of PMPs, and their improved technical qualities allow for playback without distortion at high levels.

Environmental noise like traffic noise, aircraft noise, construction noise or neighbourhood noise, although sometimes very annoying, does not reach the equivalent levels that can be harmful to hearing. On the other hand, these sources of noise can cause non-auditory effects.

3.5.3. Exposure – effect relationship

As it was usefully summarised by Lutman et al. (2008), knowledge concerning the relationship between noise exposure and NIHL is based on cross-sectional studies of people exposed to noise, much of which was conducted several decades ago and which concentrated on people exposed continuously to high levels of noise that were more commonplace in the 1950's and 1960's. This knowledge is far from complete. Most studies have suffered from the lack of appropriate non-exposed control subjects and longitudinal studies are almost entirely lacking (Lutman and Davis 1996). Authoritative reports have involved large primary studies or have synthesised data from several large primary studies. The seminal study of Burns and Robinson (1970) has been influential in the UK and elsewhere. It formed the basis of the first edition of the international

standard ISO 1999 in 1975 and has been embodied in the National Physical Laboratory (NPL) tables that are still used widely for prediction of NIHL in populations exposed to noise. The later version of ISO 1999 in 1990 (ISO 1999:1990) synthesised data from studies in the US as well as from the studies of Burns and Robinson to derive formula for predicting NIHL. An advantage of ISO 1999 (ISO 1999:1990) is that it allows the user to insert different values to account for the effects of age-associated hearing loss. This facility has enabled ISO 1999 to keep up with developing the current understanding of the effects of age on hearing and the recognition that there are important socio-economic factors governing hearing acuity. This is an important achievement because the non-exposed controls used in many studies of NIHL have been drawn from different socio-economic groups than the exposed participants (e.g. office worker, researchers, university staff).

All of the above methods account for the combined effects of age and noise exposure by simple addition of the hearing losses from the two origins, or by a slight modification of simple addition. The modified addition incorporated in ISO 1999 (ISO 1999:1990) slightly reduces the resultant hearing loss compared to simple addition. However, this effect is negligible for combined hearing loss lower than 40 dB and for the present purposes can be ignored.

ISO 1999 allows prediction of the distribution of NIHL to be expected from any cumulative amount of noise exposure. This is combined with (in most cases simply added to) distribution of age-associated hearing loss appropriate to the population in question. This calculated distribution of NIHL allows estimation of the probability that a given magnitude of overall hearing loss will be exceeded. In the context of the present study, noise levels in the range from 80-95 dB(A) are of interests. Based on ISO 1999, the following table (Table 5) shows the extent of NIHL to be expected from a working lifetime of 45 years at daily continuous noise levels of 80, 85, 90 and 95 dB(A). The values are for NIHL at 4 kHz, which is the frequency predicted to give the greatest hearing loss. Values are given for the median and the 5th centile (value exceeded by 5% of population). These data constitute the noise-induced component of hearing loss alone. Note that hearing loss is minimal for exposures at 80 dB(A), even at the 5th centile, and increases at higher levels.

NIHL at 4 kHz in dB	***Daily noise level in dB(A)***			
	80	***85***	***90***	***95***
Median	1.7	6.6	14.9	26.5
5th centile	2.2	8.8	19.6	35.1

Table 5: NIHL predicted from ISO 1999 as a function of noise exposure level for 45 years (adapted from Lutman et al 2008)

Table 6 shows similar data for the much shorter exposure durations of 3 years, which is more relevant to the present opinion. Note that in that case, NIHL is less likely than after 45 years of exposure, as expected. However, the proportion is greater than simply dividing the amount of hearing loss *pro rata*. To a rough approximation, the magnitude of NIHL after 3 years is 43% of the NIHL after 45 years. The use of this model suggests a departure from the equal energy principle in the direction that more NIHL occurs in the early years of exposure and clearly suggests that preventive measures must be aimed at those who start noise exposure from PMPs when young. Note that at 4 kHz there is more damage earlier; at 1 kHz the damage is less and is later. Hence overall damage to the cochlear can be related to the equal energy principle. ISO 1999 accommodates this trend by assuming that the noise level has to exceed 90 dB(A) to affect hearing at 1 kHz and 93 dB(A) to affect hearing at 500 Hz, but 77/78 dB to affect hearing at 4 kHz.

NIHL at 4 kHz in dB	*Daily noise level in dB(A)*			
	80	***85***	***90***	***95***
Median	0.7	2.9	6.5	11.6
5th centile	1.0	3.8	8.4	15.0

Table 6: NIHL predicted from ISO 1999 as a function of noise level for 3 years (adapted from Lutman et al 2008)

Until now, we have limited evidence about what exactly makes some subjects more vulnerable than others, but it is well established, that anatomical details play a significant role among other factors.

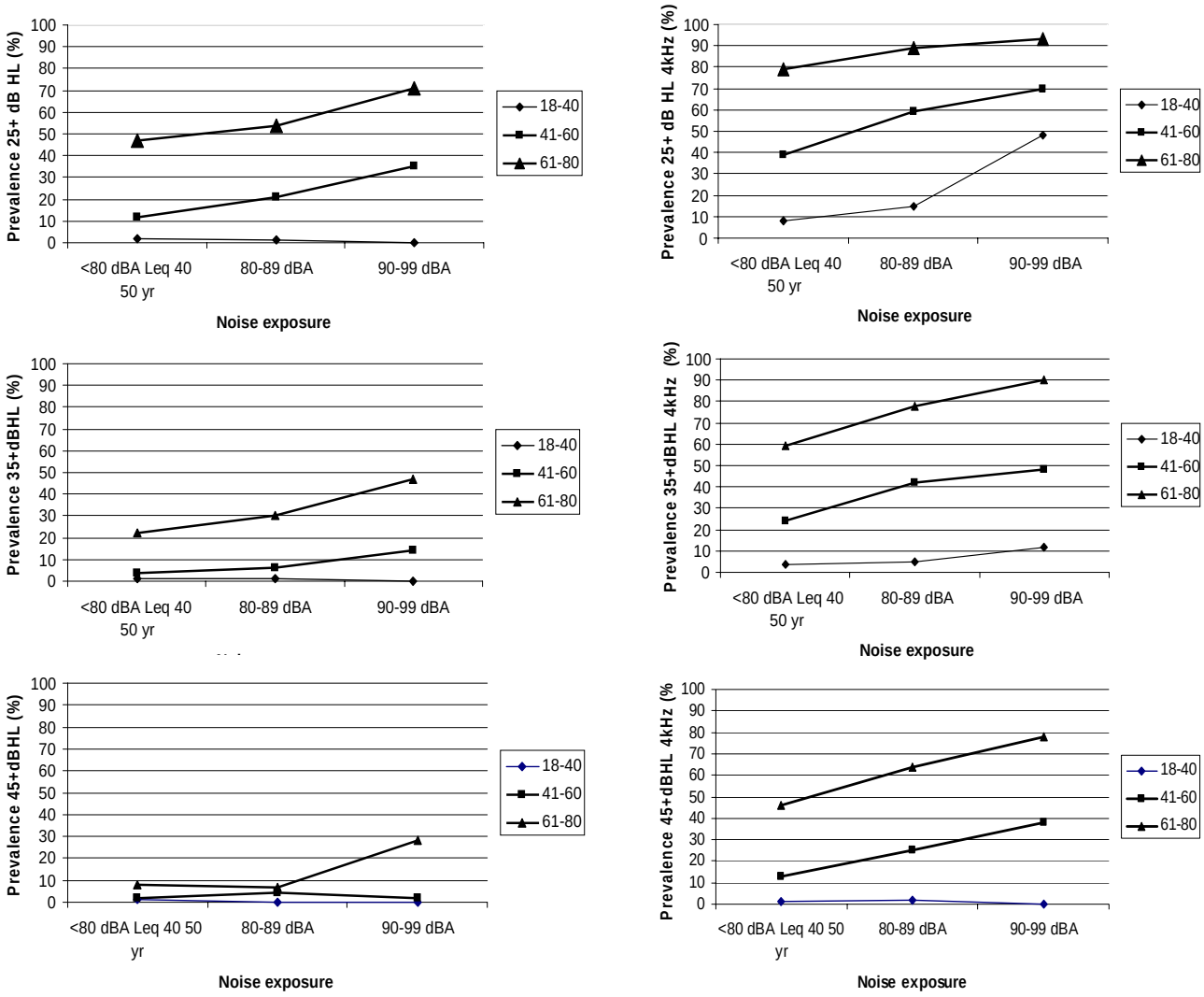

Figure 2: The prevalence of hearing impairment as a function of age group (18-40, 41-60 and 61-80) as a function of degree of hearing impairment (25, 35 and 45 dB HL+) and noise exposure (<80, 80-89 and 90-99 dB(A) L_{equ} 40 for 50 years equivalent) that was gained through occupational noise exposure for average hearing level (0.5, 1, 2 and 4 kHz) and for 4 kHz alone; data taken from Davis 1995 and presented in these figures especially to draw out main points for this work.

The relationship between age, noise exposure and prevalence is complex and takes many years to be manifest for a particular cohort. Data from the MRC National Study of Hearing (Davis 1989, Davis 1995) has been re-drawn for the working group to show the prevalence of hearing impairment for men only who have had a zero noise exposure or a measured amount of noise exposure from their occupations. Figure 2 shows the prevalence of three different degrees of hearing impairment as a function of age group (in roughly 20 years age bands). Noise immission has been allocated into three categories. The first is a level at which there would be no danger due to occupational noise, given the level, pattern and duration of occupational noise exposures (<80 dB(A) Leq 40 for 50 years or less). The second level is 80-89 dB(A) and the third 90-99 dB(A) on same scale equivalence. It is clear from the left panel of Figure 2 that shows the prevalence using the four frequency average threshold (0.5, 1, 2 and 4 kHz) that the 18-40 age group had no slope at all in terms of the prevalence over noise exposure groups. So there is no relationship at all between the prevalence and noise exposure until the age group of 40–60 when the data show a significant increase across noise exposure groups. This is similar for the oldest age group as well. It may be argued that the effects of earlier damage due to noise appear later, but they are evident at an earlier age in the 4 kHz region of the cochlear that are more susceptible to damage. The data for the 4 kHz threshold alone is shown in the right hand panel and shows that there is indeed a significant effect of noise immission for the youngest age groups at prevalence of at least 25 dBHL for <80 dB(A) L_{equ} 40 vs 80-89 dB(A) (χ^2 = 5.55, p<.0185, df=1), but not for the more disabling levels at 35 and 45 dBHL.

Clearly noise immission from occupational noise at the lowest levels of risk (80-89 dB(A) L_{equ} 40) affect prevalence of hearing impairment at 25 dBHL+, in younger people aged 18-40 but continue to have a larger impact in older people (for whom the noise has stopped on the whole). As there is no scientific evidence that social noise produces different NIHL levels compared to occupational noise, the model makes clear that at typical noise exposures it will take many years for the exposure to impact on the individual and to be measured in the population. However, 4 kHz seems an excellent frequency at which to measure initial effects.

3.5.4. Mechanisms of noise-induced hearing loss

3.5.4.1. Overview of pathophysiological effects of noise

At very high acoustic levels as in cases of bomb blast the traumatizing sound can induce mechanical breaks at different parts of the ear such as the eardrum, the ossicles joints and the basilar membrane, these effects being visible with simple optic microscopy. However, in the vast majority of cases acoustic trauma induces less visible damage to the inner ear. A loss of hair cells (the cochlear sensory cells which transform sound into biological processes) has long been the main or only morphological sign of pathology, detected by optic microscopy in specimen obtained after death. It can be observed only in cases of permanent hearing loss and not earlier than several days after acoustic trauma. It is always found in good correlation with the functional (audiometric) measures of hearing loss both for frequency extent and for amount of loss in decibels. The larger the frequency extent of the loss, the wider the loss of hair cells along the cochlea, and, for each sound frequency, the greater the loss in decibels the greater the number of lost hair cells. However, detection of (surviving) hair cells does not mean that they are functional. With the advent of electron microscopy many histological (tissue) and/or cytological (cell) alterations within the cochlea have been observed indicating several different pathological processes occurring more or less simultaneously in the cochlea in response to acoustic trauma. Many of these alterations are not specific for acoustic trauma but represent basic cellular pathological processes which occur in various other diseases of the ear. In early studies a major observed sign was the breaking or fusion of the cilia of hair cells which remains the most specific morphopathology for acoustic trauma. Among the several other alterations observed are: damage to cochlear vasculature associated with altered cochlear blood flow, loss of fibrocytes probably

associated with decreased endocochlear potential, rupture of attachments of stereocilia tips to the tectorial membrane, distension or rupture of tip links involved in transduction, damage to pillar cells, swelling or rupture of dendrites below inner hair cells. Excessive noise can induce damage to most cell types in the cochlea, but presently the sequence of these pathological events and their cause/effect relationships remain poorly known.

The histological examination of human temporal bones is a rare opportunity. In recent years only two such histological studies were performed from subjects with a known NIHL, which confirmed a loss of hair cell associated with a degeneration of neural cells with possible signs of alterations of the cochlear vasculature (Rask-Andersen et al. 2000, Nakamoto et al. 2005).

The development of cell and molecular biology provided new insights and investigation tools concerning various pathological cell processes. Among those pathologies associated with excess of free radicals and those involved in apoptosis (or "programmed cell death" proved very fruitful.

The damage of outer hair cells (OHC) impairs an active, non-linear, biomechanical cochlear feedback process along with a decreasing hearing sensitivity and frequency selectivity. Total OHC loss results in a hearing impairment of 50-70 dB (sensitivity threshold for persisting inner hair cells (IHC)), recruitment and a loss of otoacoustic emission (Hamernik et al. 1989, Gao et al. 1992). A decrease in frequency selectivity results in poor speech intelligibility particularly in noisy and/or reverberant environments.

Animal studies have shown that the damage to the cochlea corresponds well with the frequency of the noise (with ½ octave shift toward higher frequencies). However, human exposures to broadband occupational or environmental noise result uniformly in high frequency hearing threshold shift, particularly at the frequencies of 4-6 kHz, regardless of the noise spectrum. This phenomenon can be explained by the anatomical configuration of human external and middle ear and its nonlinear properties.

There are two functional consequences of noise exposure and cochlear lesion to hearing, namely temporary threshold shift (TTS) and permanent threshold shift (PTS) (Plontke et al. 2004).

The most essential parameters for TTS or PTS development include:

- Sound pressure level (SPL) of noise
- The rapidity with which sound levels increase (impulse noise vs continuous noise)
- Exposure time
- Vulnerability of the inner ear

In principle, short exposures to moderately high levels of non-impulse noise, producing reversible changes to the cochlea, result in the TTS; while long exposures (of 4 hours or more in animal experiments) to high levels of noise, producing irreversible changes to the cochlea, result in the PTS. Impulse noise is significantly more harmful than steady-state noise, because the impulses are of very high sound pressure levels (up to 190 dB SPL in the military), and the duration of impulses is too brief that the stapedial reflex (possibly protective contraction of middle ear muscles) has not enough time to conteract, this reflex offers anyhow very little or no protection at high frequencies. However, the relationship between exposure parameters is not as simple as described above. It has been shown that exposure to noise under similar, controlled conditions, in some subjects can result in TTS, while in others in PTS. This finding points to different inter-individual vulnerability of the internal ear.

The vulnerability of the inner ear depends on several environmental and intrinsic factors, like smoking, hypertension, lipids level, age, gender, eye colour and other parameters of anatomy and micro-anatomy some of which are controlled by genetic factors

3.5.4.2. Biological processes involved in noise effects

Many research studies have been performed over more than 50 years to understand physiological dysfunctions induced by excessive noise exposure. Over the last five years or so, new and promising data have uncovered several series of factors having a determinant role. The main results are presented below, schematically divided into four categories.

Acoustic factors

In some circumstances an acquired resistance to noise exposure can happen. Exposure to a previous non traumatizing sound may prevent acoustic trauma by a later noise exposure this is known as sound conditioning (Canlon et al. 1988). Liu et al. (2000) further extended earlier findings by showing that low-frequency conditioning sounds could protect from low and middle frequency noise damage. Niu and Canlon (2002) revealed an up-regulation of neurotransmitter release in cochlear efferents in the process of sound conditioning. Cochlear toughening refers to the increased resistance happening over repeated noise exposure in some conditions, in recent experiments Hamernik et al. (2003) further characterized acoustic parameters influencing this phenomenon.

The very long term effects of noise as possibly emerging only at an old age have received contradictory support from several epidemiologic studies (Ferrite and Santana 2005, Lee et al. 2005, Rosenhall 2003, Gates et al. 2000). Very recent experimental data (Kujawa and Liberman 2006) suggest that early noise exposure can render the inner ear more vulnerable to aging. Unnoticeable effects can also occur over years as indicated by small instabilities in cochlear functioning which were observed in students exposed to noise in their leisure-times (Rosanowski et al. 2006).

During the post noise exposure period the presence of loud sounds influences the amount of recovery. Very few studies were devoted to these influences the effective parameters of which are poorly known (Niu et al. 2004, Norena and Eggermont 2005). The beneficial effects of these post-trauma environmental sounds can be quite large and as they are easy to control in humans they have very high potential clinical implications. Epidemiologic data also point to similar significant effects in humans (Abbate et al. 2005).

Environmental factors other than acoustics

Exposure to several chemicals and lowered levels of breathed oxygen were found to increase NIHL. It was observed that chemical asphyxiants potentiated NIHL (Fechter et al. 2000) such as Hydrogen cyanide (Fechter et al. 2002), acrylonitrile (one of the 50 most commonly produced industrial chemicals) (Fechter et al. 2003). Hypoxia, the low oxygen breathing, was found to extend NIHL to all frequencies above those of the noise (Chen and Liu 2005). Smoking was also found a significant risk factor potentiating NIHL in epidemiologic surveys (Burr et al. 2005, Ferrite and Santana 2005, Wild et al. 2005).

Efferent and sympathetic innervations

The efferent and sympathetic innervations of the cochlea (a retrocontrol from the brain to the cochlea) seem to have almost no influence upon the normal functioning of the cochlea as their suppression does not lead to noticeable changes. However, they do influence cochlear reactivity in adverse conditions, and this has been particularly well observed with NIHL. A protective role of the efferent system upon NHIL was uncovered many years ago (Cody and Johnstone 1982). Over the last years significant progress was made regarding exposure parameters leading or not leading to protection (Rajan 2001, Rajan 2003). The predictive value of an efferent response to assess susceptibility to NHIL remains controversial (Maison et al. 2002, Luebke et Foster 2002, Wagner et al. 2005), its involvement in sound conditioning was shown by Niu and Canlon (2002). An influence of the sympathetic cochlear innervation on NIHL was uncovered several years ago (Borg 1982), and later studied. Some experiments (Horner et al. 2001, Giraudet et al. 2002)

further extended such observations and pointed to an interaction with the efferent innervation, they also showed modification of cochlear sensitivity to acoustic trauma by anaesthesia or even sedation.

Protective factors

Several newly tested drugs have been proven experimentally to provide protective or reparative properties with regard to NIHL. The pharmacological actions of the drugs are only partly known and many have several metabolic effects and it is difficult to know which of its metabolic properties is involved in NIHL. While recognizing this complexity it is fruitful both for presentation and reasoning to use main pharmacologic categories. Thus drugs are presented here below into five main categories.

Anti-inflammatory

Both steroidal and non steroidal anti-inflammatory drugs were found to provide protection against NIHL. Salicylate was found to facilitate recovery from acoustic trauma (Yu et al. 1999), in a later study salicylate in combination with trolox (an anti-NOoxidant) it was shown to decrease NIHL (Yamashita et al. 2005). Corticoids when combined with hyperbaric oxygenation were shown to provide rescue post-trauma in animal experiments (d'Aldin et al. 1999, Lamm and Arnold 1999), this was confirmed and extended by experiments last year in our group (Fakhry et al. 2007). A role of stress and corticosterone in protecting against NIHL was observed (Wang and Liberman 2002). Three recent studies indicate the beneficial action of dexamethasone on NIHL (Takemura et al. 2004, Tahera et al. 2006, Sendowski et al. 2006a) the last publication comes from a laboratory involved in the project.

Anti-oxidants

Over the last three years about twenty publications documented the protective effects of drugs with anti-oxidant properties upon NIHL. These drugs are further somehow differentiated by the authors with regards to their anti-ROS or anti-NOS properties, drugs of both classes were found effective. Approximately 12 different drugs were tested. Some were found repeatedly protective: - N-acetylcysteine (Ohinata et al. 2003, Duan et al. 2004, Kopke et al. 2005), - allopurinol (Franze et al. 2003, Cassandro et al. 2003), - ebselen (Pourbakht and Yamasoba 2003, Lynch et al. 2004), - edaravone (Takemoto et al. 2004, Tanaka et al. 2005) among these at least two are already clinically accepted drugs at least in some countries. Other drugs already clinically accepted as salicylate, vitamin c or vitamin e were also found protective.

Anti-apoptotics

Once NIHL cochlear damage has started as through inflammatory and/or oxidative or other processes apoptotic processes can be triggered and lead to sensory and neural cochlear cell disparition. Five different drugs were reported to be protective aginst NIHL : riluzole (Wang et al. 2002), a peptide inhibitor of c-Jun N-terminal kinase (Wang et al. 2003), calcineurin inhibitors (Minami et al. 2004), all-trans retinoic acid, an active metabolite of vitamin a (Ahn et al. 2005) and, Src-PTK inhibitors (Harris et al. 2005). The potential use of these drugs seems far away at present because of high dose levels needed and low bioavailability with clinical routes of administration.

Neurologic factors

Administration of several neurotrophic factors was found protective against NIHL: - ciliary neurotrophic factor (Zhou et al. 1999), - GDNF and/or NT3 (Yang et al. 2001, Chen et al. 2002), basic fibroblast growth factor (Zhai et al. 2002, Zhai et al. 2004). Modulators of neurotransmission were also found protective: noradrenergic related compounds (Horner

et al. 1998, Giraudet et al. 2002), and NMDA blocking agents (Chen et al. 2003, Diao et al. 2005, Ruel et al. 2005).

Miscellanious

Hypothermia (Henry 2003), prior heat acclimatation (Paz et al. 2004) and a heat shock protein inducer (Mikuriya et al. 2005) were reported to protect from NIHL, as were also ATP (Sugahara et al. 2004), NO inhibitors (Xiong et al. 2002, Ohinata et al. 2003) and a calcium pump activator (Liu et al. 2002). A special reference must be made to magnesium treatment which was found repeatedly protective (Scheibe et al. 2001, Scheibe et al 2002, Haupt et al. 2003, Attias et al. 2003 , Sendowski et al. 2006b).

3.5.5. Clinical evaluation of noise damage

3.5.5.1. Hearing loss

NIHL refers mostly to deafness, the inability to hear certain sounds, but this hearing loss is most often associated with considerable difficulties in auditory discrimination of simultaneous sounds, such as speech understanding in a noisy environment, which affects notably social interactions. In addition NIHL is often associated with tinnitus which may also be very damaging to a person's living, and sometimes also with hypersensitivity to loud sounds. Overall NIHL has a noxious socio-economic impact for both the affected person and the society in which he lives. The noise-induced auditory impairments are most often progressive and insidious, they begin at high frequencies with only slight disturbances hardly perceptible which usually disappear within some time after noise exposure and so they are almost always neglected. However, over time and repeated exposures these troubles progressively increase to become a patent nuisance, but then physiological damages to the ear are almost always irreversible and at present quasi incurable.

Subjective audiometry

NIHL is evident on the audiogram as mild or moderate bilateral sensory (cochlear) hearing loss, predominantly at high frequencies. The greatest hearing loss is commonly at 4 kHz, giving rise to the typical 4 kHz notch in the audiogram pattern (Alberti 1977). Although the notched audiogram is the most specific audiometric feature of NIHL, recent studies re-emphasized that caution should taken to this feature as it can be seen in ear pathologies of other causes and that a significant number of NIHL do not show a notched audiogram (Murai 1997, McBride and Williams 2001, Schmuzigert et al. 2006).

In line with previous studies several articles confirmed that very often in NIHL acoustic sensitivity at the very high frequencies (which are not measured in usual audiometry) shows deficiencies starting earlier than at other classical frequencies (Wang et al. 2000, Ahmed et al. 2001, Schmuzigert et al. 2006).

It was also confirmed that NIHL due to impulse sounds such as firearms shots produce on average more loss at high frequencies and may have longer-lasting effects (Schmuzigert et al. 2006, Tambs et al. 2006).

Although hearing losses appear stabilized over years it may continue to progress and affect lower frequencies (Gates et al. 2000, Brickner et al. 2005). The loss of sensitivity for quiet sounds is accompanied by a loss of frequency resolution which then affects speech recognition. Typically, people with NIHL complain of loss of perceived clarity of speech and greater difficulty than normal following speech in a background of noise.

Objective audiometry

Auditory-evoked potentials can be useful to monitor and/or ascertain NIHL. Past studies using late cortical potentials and brainstem potentials were forsaken in recent years (only

the new technique of steady state evoked potentials was tried (Hsu et al. 2003)) while all attention was given to early sensory cochlear responses known as otoacoustic emissions (OAEs).

Transient-evoked otoacoustic emissions (TEOAEs) allow a quick check of cochlear sensitivity and responsiveness to sound however because it uses a transient sound it lacks frequency specificity. TEOAEs were found to provide a coarse but reasonably good indication of NIHL quite often permitting to detect alterations occurring earlier than classical puretone subjective audiometry (Avan et al. 2000, Attias et al. 2001, Wang H, et al. 2004, Lapsley Miller et al. 2004, Konopka et al. 2005a, Konopka et al. 2005b, Jedrzejczak et al. 2005, Nottet et al. 2006, Job et al. 2002; Job et al. 2007).

Distortion-product otoacoustic emissions (DPOAEs) allow a frequency specific testing at least at middle and high frequencies at the price of being more time-consuming. Their frequency selectivity often but not always provide a good correspondence with the pure tone audiogram and may detect earlier alterations (Morant Ventura et al. 2000, Zhang et al. 2000, Sliwinska-Kowalska and Kotylo 2001, Han et al. 2003, Namysłowski et al. 2004, Balatsouras 2004, Zhang et al. 2004, Seixas et al. 2004, Avan and Bonfils 2005, Konopka et al. 2006, Sisto et al. 2007, Shupak et al. 2007).

3.5.5.2. Vestibular effect

It has been repeatedly observed that alterations of the vestibule (the other mechanoreceptor of the inner ear besides the cochlea participating in balance and posture) could show signs of dysfunction in several cases of NIHL. These vestibular responses to sound are also known as the Tullio phenomenon and can be objectively studied using the vestibule-collic reflex. This was confirmed by recent studies which also showed that there was no clear relation between the degrees of vestibular and of cochlear dysfunctions (Teszler et al. 2000, Golz et al. 2001, Wang et al. 2001, van der Laan 2001).

3.5.5.3. Noise-induced tinnitus

The prevalence of tinnitus (or ringing in the ear) in noise-exposed populations seems to be much higher than in general populations. It has been estimated at prevalence 37% for less than 10 years of exposure and 50% for 11-30 years of exposure to noise. Noise-induced tinnitus may be temporary or permanent. This can be the only indication of hearing damage in the early stage, which may then be accompanied by hearing loss with continued exposure. Recent studies confirmed that when associated with NIHL it is almost invariably of high pitch, with a tonal or narrow frequency-band timbre. It has been reported that the duration of tinnitus is not related to the amount of acoustic trauma (Nottet et al 2006). Tinnitus appears very early after an impulse sound trauma, as well as other very loud sound exposures, then it is often temporary. In opposition, in several continuous long-term noise exposures scenarios it often appears after years, but remains permanent.

Trials to alleviate tinnitus through physiological means re-emphasized the efficiency of electric stimulation of the cochlea (although potentially hazardous in the long term) and explored with uncertain success several drug treatments. Psychological therapies remain the most common option (Axelsson and Prasher 2000, Konopka et al. 2001, Kowalska and Sulkowski 2001, Markou et al. 2001, Mrena et al. 2002, Attias et al. 2003, Emmerich et al. 2002, Nicolas-Puel et al. 2002, Rosenhall 2003, Mrena et al. 2004, Bauer 2006, Holgers 2006, Nottet et al. 2006, König et al. 2006, Nicolas-Puel et al. 2006, Mrena et al. 2007). When tinnitus becomes permanent, wearing a hearing aid may also provide help.

3.5.6. Vulnerability factors

It remains a puzzle to observe a very large interindividual variability in susceptibility to NIHL. Whether and how much individual vulnerability is dependent upon external

conditions occurring at time of acoustic trauma or internal conditions linked to the genetics and physiological condition of the subject remains unknown. Significant progresses have been performed recently on these issues.

3.5.6.1. Environmental factors

Noise exposures in combination with several chemical and physical hazards, as well as ototoxic drugs may produce more hearing impairment than could be expected from noise-only exposure.

Chemicals

Chemicals are frequent contaminants in industry, some of them might be also common in general environment (heavy metals) or are used in everyday life (paints and lacquers). They are classified into three major groups: organic solvents, heavy metals, and asphyxiants.

Almost all studies about association of solvent fumes respiration with traumatizing sound exposure confirm their clear potentiation of NIHL, (Campo et al. 2001, Morata et al. 2002, Morata et al. 2003, Sliwinska-Kowalska 2003, Sliwinska-Kowalska et al. 2005, El-Shazly 2006).

Organic solvents

Ototoxic effects of organic aromatic solvents, such as toluene, styrene, xylene, trichloroethylene, benzene, n-hexane and their mixtures are well recognized. These chemicals are frequent air contaminants in industry, such as in paint and lacquer factories, dockyards, printing industry, yacht manufacturing, furniture making, plastics and fibers processing, rubber tires production and many other industrial activities. Exposure may also occur in domestic settings through processed wood products, plastics furnishing, paints and lacquers. Animal studies have shown that several organic solvents, as has been exemplified by styrene and toluene, damage the cochlea (predominantly the supporting and outer hair cells) in rats and the exposure produces mid-frequency hearing loss (Sliwinska-Kowalska et al. 2007). Alcohol exposure, although alone it does not produce hearing loss, increases significantly the degree of hearing impairment caused by styrene or toluene (Campo et al. 1998, Campo et al. 2000). Synergistic effects occur in rats exposed to both noise and solvents (Campo et al. 2001, Sliwinska-Kowalska et al. 2007). It means that hearing impairment is higher than the sum of hearing loss produced by solvent exposure and noise exposure alone. In combined exposures, the most important factor for inducing hearing impairment is potency of noise exposure (level, impulsiveness); concomitant exposure to organic solvents may induce impairment where the exposure to noise alone may have little effect.

The ototoxicity of organic solvents in occupationally exposed human individuals is more difficult to elucidate. This is because the concentration of chemicals is much lower than that used in animal studies, and the workers are usually exposed to a mixture of solvents at widely varying compositions and concentrations, disabling the assessment of the effect of a single substance (Sliwinska-Kowalska et al. 2001). However, investigations on humans confirm the findings in animals. It has been shown that organic solvents have detrimental effects not only on peripheral, but also on central part of the auditory pathway (Johnson et al. 2006, Fuente and McPherson B 2007). Thus, pure-tone audiogram might be insufficient to monitor this effect, and central auditory tests must be implemented. An additive or synergistic effect occurs in case of the combined exposure to noise and solvents, significantly increasing the odds ratio of developing hearing loss (Sliwinska-Kowalska et al. 2003, Sliwinska-Kowalska et al. 2004). The risk for hearing loss increases with the growing number of solvents in a mixture.

Heavy metals

Extensive use of heavy metals in industry adds to the environmental exposures to these substances. Heavy metals are not metabolised by the body and accumulate in the soft tissues or in the bones, causing toxic effects. They may enter the human body through food, water, air, or absorption through the skin when they come in contact with humans in residential and occupational settings as well as in the general environment. Commonly encountered toxic heavy metals include lead, mercury, cadmium and arsenic.

Lead

Most of the lead is used for batteries. The remainder is used for cable coverings, plumbing, ammunition, and fuel additives. It has been shown that the exposure to lead results in delayed wave I latency of ABR, implying cochlear dysfunction (Osman et al. 1999). But the findings on lead-induced hearing loss are inconsistent (Farahat et al. 1997, Forst et al. 1997, Baloh et al. 1979, Counter et al. 1997, Otto et al. 1985, Buchanan et al. 1999).

There are very few studies exploring the effects of combined lead and noise exposure. Elevated hearing thresholds have not been reported for lead and noise combined exposure (Wu et al. 2000).

Mercury

Mercury is found in dental amalgams, aquatic sediments, thermometers, vaccine preservatives, to quote a few examples. It is present in the atmosphere, and also in shark-, sword-, tuna-fish and other fish species. First, mercury intoxication was reported in 1953 among persons living in the vicinity of Minamata, Japan, where mercury-containing effluent flowing from a chemical manufacturing plant into the local bay contaminated shellfish. Hearing impairment and deafness were reported among other neurological symptoms of the "Minamata disease".

Mercury affects hearing, with central conduction time delay (ABR I-V, III-V), but cochlear function may be unaffected (Counter et al. 1998a and b, Rice and Gilbert 1992; Murata et al. 1999).

Cadmium

Cadmium is used e.g. in nickel-cadmium batteries, PVC plastics, and paint pigments. Cadmium causes dose-dependent hearing loss in rats; wave I was delayed, implying cochlear dysfunction. Zinc-enriched diet reduced the ototoxic effect of cadmium, while noise exposure shows a synergistic effect at 4 and 6kHz (De Abreu and Suzuki 2002).

Arsenic

Arsenic is released into the environment by the smelting process of copper, zinc, and lead, as well as in the manufacture of chemicals and glass. Arsenic overexposure results in disorders in the Organ of Corti beginning at the apex with the greatest hearing losses in the lower frequencies (at 125, 250, and 500 Hz). Arsenic produces also balance disturbances.

Asphyxiants

Carbon monoxide (CO) and hydrogen cyanide (HCN) bind hemoglobin heme, thereby preventing oxygen transportation. The CO intoxication (e.g. in gas stove accidents) results in hearing impairment, dizziness and headache. Dizziness and headache were also noted in the prolonged intoxication with HCN and SO_2. These gases are common air pollutants; thus, HCN and SO_2 exposures affect the majority of individuals. CO and HCN potentiate damaging effect of noise to hearing in animals. The effects of combined exposure to noise and asphyxiants in human are not fully recognized.

Vibration

Vibration-induced hearing loss may be developed in patients after temporal bone surgery or in subjects working with vibrating tools. In such cases, co-exposure to noise and vibration can increase hearing threshold shift compared to noise-only exposure.

Recent studies concerning association of body vibration with sound trauma brought contradictory and inconclusive results (Palmer et al. 2002a, Silva et al. 2005).

3.5.6.2. Ototoxic drugs

Several drugs used in contemporary medicine can damage hearing. Ototoxic effect depends on the dose, way of application and the type of medicine. Although these drugs can damage hearing at different levels of the auditory pathway, majority of them exert mainly cochlear ototoxic effect and they are competitive with noise in damaging hair cells.

The main groups of drugs that can cause hearing loss are:

- antibiotics (aminoglycosides, macrolides)
- antineoplastic drugs (cisplatinum, carboplatinum)
- loop diuretics (furosemide, ethacrinic acid)
- non-steroid anti-inflammatory drugs (acetyl salicylate acid)
- antimalaric drugs.

The most commonly used drugs that have been reported in the literature to result in hearing damage are aminoglycosides and anti-neoplasmatic drugs. Aminoglycosides are used parenterally in treating severe bacterial infections. After prolonged treatment with such aminoglycosides like gentymycin, kanamycin, amikacin, hearing loss at high frequencies, tinnitus and vestibular disorders were noted. The changes in hearing are irreversible. Prior exposure to noise (and vibration) increases the risk of hearing impairment due to aminoglycosides. The ototoxic effect depends on genetically determined susceptibility; it increases with high concentration of ferrum ions in the blood, and low protein diet. Anti-oxidant substances (like Vitamins A, C and E) have been shown to be protective.

It has been shown that cancer chemotherapy with cis-platinum produces hearing loss in up to 31% of patients. As in noise-induced hearing loss and aminoglycoside-induced hearing loss, these chemotherapeutics affect mainly hair cells of the basic turn in the cochlea and result in high-frequency (above 2 kHz) hearing impairment. Noise exposure at the time of chemotherapy significantly increases the risk of hearing damage.

3.5.6.3. Genetics

The advance of genetic research and associated tools triggered a series of explorations of the human genes possibly involved in NIHL, first evidences point to some candidate genes and seem to exclude other genes (Fortunato et al. 2004, Heinonen-Guzejev et al. 2005, Yang et al. 2005, Yang et al. 2006, Van Laer et al. 2006, Yang et al. 2006, Sliwińska-Kowalska et al. 2006, Konings et al. 2007, Van Eyken et al. 2007). The mechanisms of acoustic trauma involve both metabolic stress and micromechanical damage to the outer hair cells, predominantly to their stereocilia. Thus, good candidate genes are those encoding oxidative stress enzymes, mitochondrial proteins, and proteins involved in K^+ recycling pathway. The importance of oxidative stress genes has been shown in knockout mice, including $SOD1^{-/-}$ (Ohlemiller et al. 1999), $GPX1^{-/-}$ (Ohlemiller et al. 2000), and $PMCA2^{-/-}$ mice (Kozel et al. 2002), all of which were more sensitive to noise than their wild-type littermates. However, these results have not been confirmed in humans (Carlsson et al. 2005). A more recent study suggests a possible role of potassium recycling pathway genes in the susceptibility to NIHL in human workers (Van Laer et al. 2006).

Some of the differences in susceptibility to NIHL have been attributed to various other genetically dependent factors, like eye colour (blue-eyed more susceptible), and pigmentation (African-Americans showed a somewhat better average in hearing threshold levels than Caucasians), gender (women less susceptible than men), age, etc (Henderson et al. 1993, Pyykkö et al. 2007). Also short stature has been recently recognized as a risk factor for developing sensorineural hearing impairment (Barrenäs et al. 2005).

3.5.6.4. Other factors

The gender of an individual has been often considered as a possible influencing factor with men appearing somewhat more affected than women. The difference however seems minimal if present (Müller 1989).

A predominance of left ear vulnerability as compared with right ear has been confirmed (Nageris et al. 2007) but the difference is small and shows mostly on average data.

Cardio-vascular alteration was often studied as a possible factor influencing NIHL but data are contradictory and the subject remains a matter of debate. Recent studies tend to confirm that alterations of blood pressure can be related with NIHL but it remains unknown whether it might be a cause or a simultaneous effect (Souto Souza et al. 2001, Toppila et al. 2001, Narlawar et al. 2006, Ni et al. 2007). A more detailed presentation is provided further in this report.

Evidence that smoking increases the risk of NIHL were provided long ago, all recent studies on this matter confirmed this assertion (Mizoue et al. 2003, Ferrite and Santana 2005, Uchida et al. 2005, Burr et al. 2005, Wild et al. 2005, García Callejo et al. 2006, Pouryaghoub et al. 2007).

Vitamin deficiencies were previously suspected to influence NIHL. Two recent studies brought evidence for the involvement of vitamin B12 (Quaranta et al. 2004, Gok et al. 2004).

The cochlear efferent innervation has long been known to be involved in NIHL. Recent studies further showed that assessment of cochlear efferent functioning did not clearly relate with NIHL (Veuillet et al. 2001, Shupak et al. 2007, Wagner et al. 2005).

The production of heat shock proteins constitute a physiological response to stress, first evidence for their implication in NIHL was recently provided (Yang et al. 2004, Yuan et al. 2005).

3.5.7. Therapies

Many therapies have been tried in the past with at best limited positive results. Recent progress in cell biology has provided a wealth of new molecules with possible therapeutic potential and several animal experiments have provided positive and promising results (as presented in another section of this report). Clinical trials over the last years have followed these progresses and brought preliminary results.

Magnesium treatment, repeatedly found beneficial in the past, has been confirmed as efficient (Attias et al. 2004). Hyperbaric oxygenation was also confirmed as having protective effects (Winiarski et al. 2005) although some adverse effects have also been reported depending on conditions of administration. Steroid administration a classical clinical treatment for NIHL was again recently reported as beneficial (Nakaya et al. 2002, Winiarski et al. 2005).

Many new drugs with anti-oxidant properties were found protective in animal experiments, the published clinical trials however do not yet provide fully convincing evidence (Kaygusuz et al. 2001, Gok et al. 2004, Kramer et al. 2006). New drugs with anti-apoptotic properties were also shown beneficial in animal experiments, NIHL clinical therapy is limited at present to one positive report (Suckfuell et al. 2007).

3.5.8. Conclusions

Exposure to excessive noise is one major cause of hearing disorders worldwide. Some data suggest an increased risk due to increase in use of audio leisure activities. There seems to be a trend for increased risk due to PMPs, as their qualities improved and they have become used by a largely increasing proportion of the population. The noise-induced hearing impairments have received much attention in the past decades mostly because of hazards of industrial noise exposures. Based upon many scientific studies the International Standard Organization has published recommendations for health safety. A most used ISO reference for risk assessment is that an exposure to a sound level of 85 dB (A) for 8 hours a day, 5 days per week will induce in 5% of the exposed population a hearing loss of about 4 dB after 3 years and 9 dB after 45 years. These losses being considered as quasi negligible this sound level-duration was considered as a safe limit above which preventive actions should be taken. The ISO recommendations also express that, as shown by many studies, the noise-induced hearing loss is the product of sound level by duration of exposure, and follows the equal energy principle stating that a decrease of sound level if associated with a proportional increase in duration (for example a halving in sound level associated with a doubling of exposure duration) induce similar risks. All data indicate a large inter-individual variability in vulnerability to excessive sound exposures, some subjects being affected while others are not; up to now the factors underlying this variability are very poorly known.

In the last decade many new and promising data were obtained concerning the biology of pathological processes responsible for hearing impairments due to excessive sound exposures. Excessive noise can induce damage to most cell types in the inner ear, but presently the sequence of these pathological events and their cause/effect relationships remain poorly known. Several environmental factors can have detrimental effects, such as exposure to several chemicals and lowered levels of breathed oxygen which were found to increase NIHL. The study on involvement of genetic factors has only recently started and first evidences point to some possible genes and seem to exclude others. Following the development of molecular biology many new drugs were found to have protective effects against NIHL in fundamental experiments, these constitute new and very promising perspectives to prevent and cure NIHL in the future in humans. Although some few studies have started to assess some new drug treatments in humans much further research is needed over coming years before definite clinical applications can be considered.

3.6. Technical aspects of personal music players

3.6.1. General characteristics

Personal music players have a very wide field of application ranging from professional tools at the workplace to the leisurely consumer and to children who use these devices as toys. They are portable digital music players that play music as audio files, such as MP3. In addition, most of these devices allow to store video, pictures, and to receive radio and TV programs (podcasting). Earphones and external speakers are the typical output devices delivering sound to the listener. Personal music players (PMPs) are widely used in conjunction with several headphones of different styles (insert, supra-aural, vertical, and circumaural).

To identify the risk levels of PMPs one has to realize that the chain of music reproduction is organised in stages which are more or less independent of each other but together affect the output signal level. Personal music players reproduce music from a recording. The sound of a signal has usually been recorded through a microphone and the oscillations resulting from the pressure changes are stored as a sampled wave form. Different procedures and algorithms are used for storing of analogue or digital

representations of the wave forms which are offering low or high data compression and provide the means for reconstruction of the original waveform during play-back. The sound level is not significantly affected by the compression algorithm (if any) and by the degree of compression used in a particular recording. Often recordings are made by mixing samples from a number of microphones.

The dynamic range of a recording is generally chosen to fit the technical characteristics of the storage device (e.g. music cassette tape) and depending on the tone engineer and the style of the music. Thus, the full dynamic range of a 'life music' situation is typically compressed to a variable degree into the dynamic range available for recording and play back.

Using the appropriate algorithm, the recorded signal is re-synthesized into a waveform, typically, in the form of an analogue electronic signal, by the player. Manufacturers of players usually specify the electronic characteristics of the output (e.g. maximum voltage, impedance) of their equipment.

To produce sound the player is connected to a transducer: headphones, earphones or earbuds. Important for our purposes is how effective the transducer is in transforming voltage into sound energy.

As said above, two matters are important. The electronic coupling of the transducer to the player may affect the output of the player. This is technically a matter of input and output impedances and can be described as a coupling factor. The transducer produces a certain amount of sound energy for a given electronic energy (voltage times current drain) delivered to the input. This is the sensitivity of the transducer.

The earphones or earbuds are inserted in the ear. The place in the ear canal also determines the effectiveness of the transfer from transducer to the tympanic membrane. It seems evident that a circum-aural headphone has to produce more energy than an earbud inserted in the ear canal, simply because of the larger volume of air that has to be excited.

3.6.2. Sound output

The volume of the sound emitted by PMPs varies from manufacturer to manufacturer, and is difficult to estimate.

With the currently available digital formats (e.g. MP3) of sound recording and reproduction, it is possible to reach high levels of sound output without distortion. The personal music players now play not only music, but provide podcasts of various broadcasts or lecture material, which is delivered largely through ear-bud type insert ear phones producing a range of maximum levels around 80-115 dB(A) across different devices. Sound pressure levels change with the insertion depth of the ear-bud in the ear canal, the maximum output provided by the particular device and ear-bud combination and the type of music.

Fligor and Cox (2004) tested some devices by different manufacturers and style of headphones. They found that free-field equivalent sound pressure levels measured at maximum volume control setting ranged from 91 dB(A) to 121 dB(A). Moreover, they estimated an influence of 7-9 dB with an ear-bud type producing the highest levels in the ear canal.

3.6.3. Conclusions

With the currently available digital formats (e.g. MP3) of sound recording and reproduction, it is possible to reach high levels of sound output without distortion (around 80-115 dB(A) of maximum levels across different devices) and the difference in ear-phone type may increase that level. These levels change with the insertion depth of the

ear-bud in the ear canal, the volume setting of the device, the maximum output provided by the particular device and ear-bud combination and lastly the type of music. In the worst case scenario, it is possible to estimate maximum levels of about 120 dB(A).

3.7. PMP usage in the population

In the last decade PMPs have become used by an increasing portion of the population. The maximal levels of noise produced by the new generation devices are very high and obviously cause an increased risk of hearing impairment. The factors involved in the potential risk of hearing loss are noise intensity and duration of exposure at a particular level. Listening environment, type of headphones / earphones as well as type of music may play additional roles. This chapter describes the habits of listening to music through PMPs on a regular basis by teenagers, as well as the availability of PMPs. The data provided in this chapter are crucial for a risk assessment.

3.7.1. Listening levels

Several studies are accessible in the literature assessing PMP in terms of maximum sound pressure level measured in dB SPL or dB(A). Already Katz et al. in 1982 warned that stereo earphones could deliver acoustic levels up to 120 dB(A) (Katz et al. 1982). Later in 1985 Lee et al. measured portable headphone cassette radios peak outputs of 90 to 104 dB (Lee et al. 1985).

Rice et al. examined over 60 users of personal cassette players (PCP). They were asked to set the volume control of a PCP to the level at which they would normally listen to different types of music. The ranges of measured sound pressure levels were between 60 and 108 dB(A). Mean free-field equivalent continuous A-weighted sound pressure level (unobstructed field L_{Aeq}) was close to 85 dB L_{Aeq}, 25% of the sample experienced levels of at least 90 dB (A), and 5% had levels above 100 dB(A) (Rice et al. 1987a).

In the study by Wong et al. the equivalent measured music noise levels were of 56 to 116 dB(A) among 394 PMP users (Wong et al. 1990).

In the study by Turunen-Rise et al. published 16 years ago, A-weighted maximum and equivalent sound pressure levels (SPLs) were measured on KEMAR (Knowles Electronics Manikin for Acoustic Research) system on five different PCPs while playing selected types of music and using different gain (volume) settings. The types of music included pop music, classical music and light classical music. The transformed A-weighted field equivalent SPLs were on average from 75 to 85 dB(A) only (Turunen-Rise 1991).

In the study by Ising et al. sound levels of music played from mini-cassette players via headphones were measured in a nonrepresentative group of 681 pupils whose ages were between 10 to 19 years. They set music levels measure as free field corrected short time Leq, between 60 dB(A) and 110 dB(A) (Ising et al. 1994).

In a study of social noise in a population sample of representative 18-25 year olds in the UK Smith et al showed that the level of preferred listening on PMP was 74 dB(A) which was 72.6% in the low noise category and 80.3% in those with significant social noise exposure. In the tail of distribution it was found that 6.9% adjusted to levels in excess of 90 dB(A). If adjusting for enjoyment then the average level of adjustment in a quiet room was 85 dB(A) with a mean of 92 dB(A) in those who had significant social noise exposure (Smith et al. 2000).

The availability of portable high-performance digital players, with an increased dynamic range, has facilitated the listening to music at high levels due to reduced distortion at these levels. Some measurements point to the fact that very high acoustic levels could be reached (125-127 dB(A)) with a $L_{equ, 1h}$ of 110 dB(A) (Loth et al. 1992). The study by Fligor and Cox published in 2004 indicated that for several different styles of headphones/CD players, free-field equivalent sound pressure levels measured at

maximum volume control setting ranged from 91 dB(A) to 121 dB(A). Output levels varied across manufacturers and style of headphone, although generally the smaller the headphone, the higher the sound level for a given volume control setting. Specifically, in one manufacturer, insert earphones increased output level by 7-9 dB, relative to the output from stock headphones included in the purchase of the CD player (Fligor and Cox, 2004).

Based on measured sound pressure levels across systems and the noise dose model recommended by National Institute for Occupational Safety and Health for protecting the occupational worker, a maximum permissible noise dose would typically be reached within 1 hr of listening with the volume control set to 70% of maximum gain using supra-aural headphones. Using headphones that resulted in boosting the output level (e.g., insert earphones used in this study) would significantly decrease the maximum safe volume control setting; this effect was unpredictable from one manufacturer to another. According to Fligor and Cox (2004), in the interest to protect the hearing of the majority of consumers, reasonable guidelines would include a recommendation to limit headphone use to 1 hr or less per day if using supra-aural style headphones at a gain control setting of 60% of maximum.

3.7.2. Listening habits

This chapter addresses the habits of music listening regarding the average sound pressure level of music and time spent on listening to music through PMPs in an everyday life. In some of these investigations, individuals were either queried about subjective level to which they set their volume and time spent on listening to music through PMP (how loud/how long) or asked to set the level of an equivalent device in the laboratory setting. In some other studies these measurements were performed in real world situations, which is a more preferable approach rather than extrapolating the data from laboratory studies. Several studies are accessible in the literature assessing PMP sounds in terms of equivalent sound pressure levels and permissible dose of noise. These studies used various study designs and methods of measuring maximum output levels of headphones (artificial ear vs KEMAR system). Because there is no standard for recreational noise, all of them referred equivalent sound pressure levels from music players to the occupational noise standards (ISO 1999:1990 standard).

In the study by Catalano and Levin (1985) 154 public college students in New York City were studied via a self-administered questionnaire regarding the volume setting used and weekly exposure in hours to these units. The values were referred to the "A" weighted scale for permissible noise dose according to OSHA criteria (OSHA 1983). Of all students who used such radios 31.4% (41.2% males vs 29.2% females) equalled or exceeded the maximum allowable dose, with the predominance of males. The mean weekly exposure time of females was 8 hours (+/- 10 hours/week) while of men it was almost 14 hours (+/- 10 hours/week). Of the total "at risk" group 50% exceeded the risk criteria by more than 100%. The authors concluded that portable radios with headphones may be capable of causing permanent hearing loss in a large proportion of radio users (Catalano and Levin 1985).

Rice et al. by referring the levels of sound to the listening times, 5% of the sample were listening in a manner causing that habitual use would constitute a damage risk to hearing. The authors conclude that there could be some damage to hearing from PCP devices if habitually used over long period time (Rice et al. 1987a, 1987b). The shortcoming of this study is that the authors used the admissible daily level of noise of 90 dB L_{Aeq} recommended in 80s, while currently it is 80 dB L_{Aeq}. Re-evaluating the data it would increase the population at risk to over 10%.

In another early study Wong et al. assessed the prevalence of use of personal Cassette Players among youths in a residential community in Hong Kong. They interviewed 487 individuals aged 15-24 years, with 394 (81%) reported using PCP regularly (i.e. for 3

days or more in a week for at least 6 months). The mean duration of PCP use was 2.8 years, and the mean listening time was 4.5 hours per week (Wong et al. 1990).

In the study by Ising et al. nearly 50% of the total group listened to music less than one hour per day, and only less than 10% listened for four or more hours (Ising et al. 1994). Estimating the expected hearing loss (HL) based on ISO 1999 standards, about 10% of the total group were predicted to have a HL more than 10 dB at 4 kHz after 5 years of using personal music devices (Ising et al. 1994). The music habits were evaluated in 569 pre-teens and teenagers by the same group of authors (Ising et al. 1995). They concluded that taking into account only portable music players, one can expect that even after 5 years of music approximately 5% of the total group would have a hearing loss of 20 dB. Based on their assumptions, the authors suggest that decreasing output level of PMP devices to 90 dB(A) would minimise the risk of hearing loss (Ising et al. 1995). In a study by Job et al (2000) it is reported that 39% of young subjects from 18 to 24 years old went to discos more than twice a month and 17% listened to their personal stereos (cassette players, CD) more than one hour a day.

A recent report by Bohlin and Erlandsson (2007) examined risk behaviour and loud music exposure in young 310 adolescents aged 15-20 years. They observed that although women judged risk situations as generally more dangerous than men but they nevertheless behave in the same way. Adolescents reporting tinnitus judged loud music as more risky than those with no symptoms and they did not listen to loud music as with those with occasional tinnitus. They suggest that hearing protection should acknowledge and make use of theories on risk behaviour especially due to the existence of a relationship between adolescents' risk taking in noisy environments and other types of risk taking.

3.7.3. Listening environments

Rice et al. examined over 60 users of personal cassette players (PCP). They were listening to various music and speech against quiet and noisy backgrounds, in the field and laboratory conditions. There was no significant difference between listening levels for the different pieces of music, but there was a significant increase in the mean listening level from 80.7 dB to 85.1 dB (approximately by 4 dB) in the presence of background noise (Rice et al. 1987a).

Skrainar et al. 1987 found that users of PMP in occupational noise set levels of 70 to 98 dB(A) with an average of about 87 dB(A) which did not significantly add risk (Skrainar et al. 1987).

In the study by Williams (2005) measurement was made in a sample of 55 individuals who were using a PCP as part of their daily activity, when commuting to work, in what could be considered noisy backgrounds (the worst-case conditions). The average, A-weighted, eight hour equivalent continuous sound exposure level from PMP in the worst case condition was determined to be 79.8 dB (80.6 dB in males and 75.3 dB in females) (Williams, 2005). These sound exposures do not indicate a significant increase in the risk of potential sound injury from PMP alone for the majority of the user population. However, there is a subpopulation of individuals who set the volume control of PMPs very high and/or listen to music through PMPs for long hours a day. In the cited study of Williams (Williams 2005), 25% of individuals exceeded the limit 85 dB(A) of noise exposure, while as much as 40% exceeded the level of 80 dB(A).

Recently published study by Hodgetts et al. (2007) indicated that preferred listening levels were higher with earbud earphones than with over the ear headphones but in a group of 38 users in quiet or noisy environments levels settings ranged from 70 to 90 dB(A) only (Hodgetts et al. 2007).

3.7.4. Market trends and availability of portable audio equipment

The market surveillance information was obtained for this report from GfK (http://www.gfk.com/) by MRC Hearing and Communication Group on 14th March 2008. The data comprised all the Domain Product Groups relating to audited unit sales for portable audio equipment for the countries Germany, UK, France, Italy, Spain, Netherlands, Belgium, Austria, Switzerland and Sweden. The coverage period was for 2004-2007 except for CD players which had data from 2001-2007. The overall coverage of sales channels was highest in the UK, but very similar market trends were observed for Europe. The market coverage for UK for MP3 players averaged at 91%, with an average of 80% overall the selected European countries (which total estimated population of 357,239,716 in 2007 or about 71% of EU population).

In order to extend these data to the EU countries as a whole two methods were used. The simple method was to multiply the data up on population basis pro rata. In addition to that the data were weighted according to internet access penetration in EU countries (http://epp.eurostat.ec.europa.eu/ , data accessed on April 28 2008). These weighted data could then be used as a broad range with the multiplied up pro rata data to give a position for Europe.

Figure 3 shows estimated units sales in EU of all portable audio devices and MP3 devices as a function of estimate procedure over the period 2004–2007. The estimates have been weighted by each country's take up of the internet or on a pro rata population basis. In addition there has either been allowance for the audited data coverage or not (on an annual basis where available, otherwise on an average basis for that medium).

This figure shows that for all portable audio devices the unit sales were in the range 184-246 million units, while for MP3 devices the units sales were estimated as about 124 million but could be as large as 165 million. This equates at the top end to units sales equivalent to 1:2 of the population. So, it is assumed that some people buy more than one device in that time period.

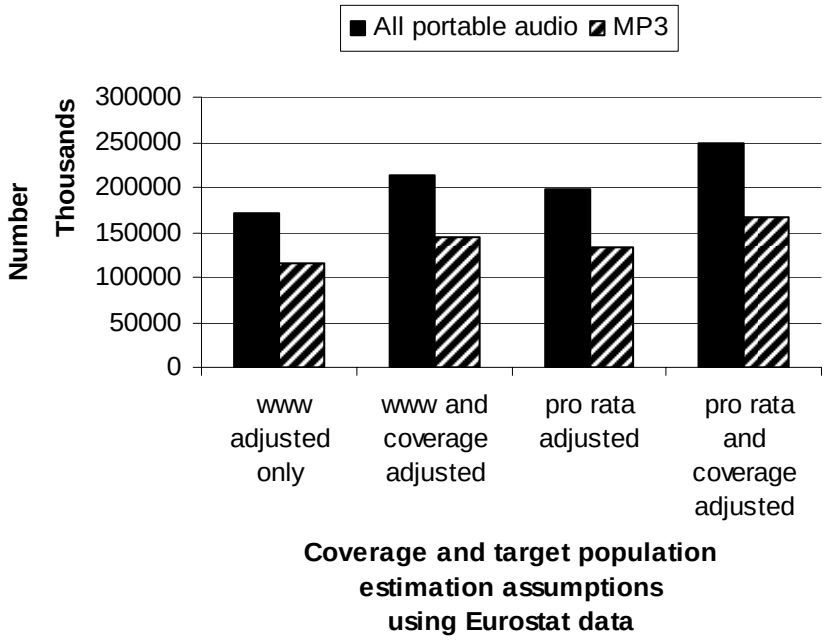

Figure 3: Estimated units sales in EU of all portable audio devices and MP3 devices as a function of estimate procedure over the period 2004–2007, adjusted for the EU population estimated by EuroStat.

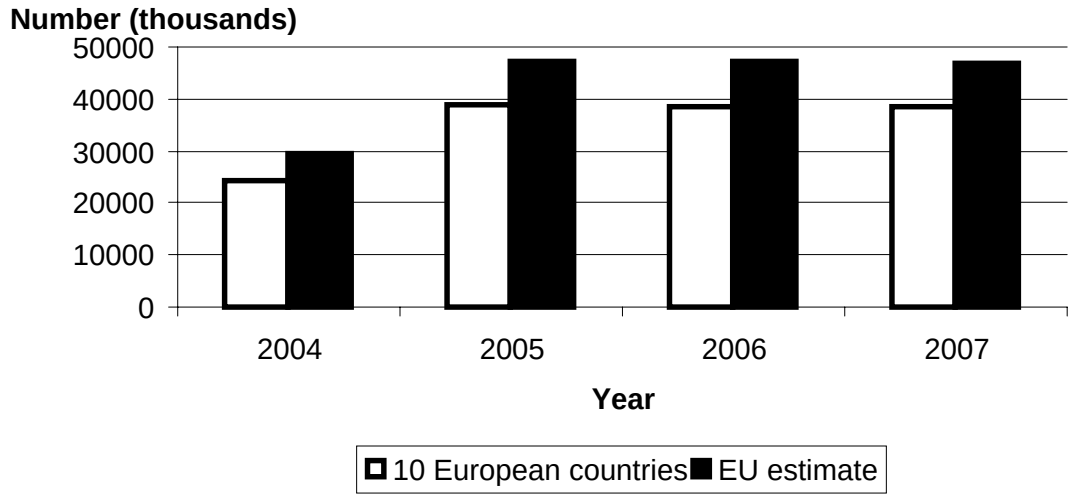

Figure 4: The number of unit sales (in thousands) for all portable audio devices for the ten countries and for the EU, adjusted according to the internet users in each country as estimated by EuroStat.

The overall number of audio devices sold in the ten countries and in the EU as a whole is shown in Fig 4. This shows the overall increase in sales in 2005 that has been maintained in the following two years, with more than 50 million devices being sold per year by 2007. Figure 5 shows that the PMP player sales were responsible for that increase. The percentage of MP3 players sold as proportion of all portable audio devices has increased dramatically from over 20% to over 80% in the last three years (from 2004 to 2007), and in 2007 it consisted of over 30 million unit sales in ten European countries[18] (vs of 10 million for CD players at its peak in 2004) (fig. 5).

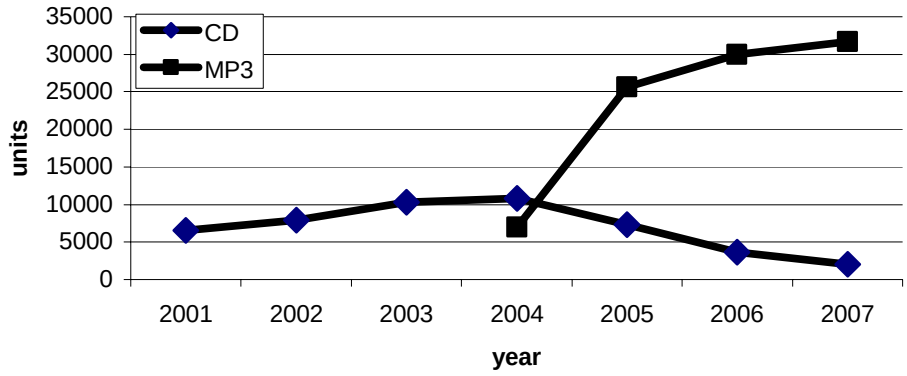

Figure 5: Number of unit sales (thousands) in ten European countries for CD and MP3 devices between 2001 and 2007.

[18] Austria, Belgium, Germany, UK, France, Italy, Spain, Netherlands, Sweden and Switzerland

The sales data, not adjusted for coverage, for MP3 players and for all audio devices are shown in Figure 6. These data show the large increase in unit sales for the MP3 players over the 4 year period from audited figures and from those estimated for the EU countries.

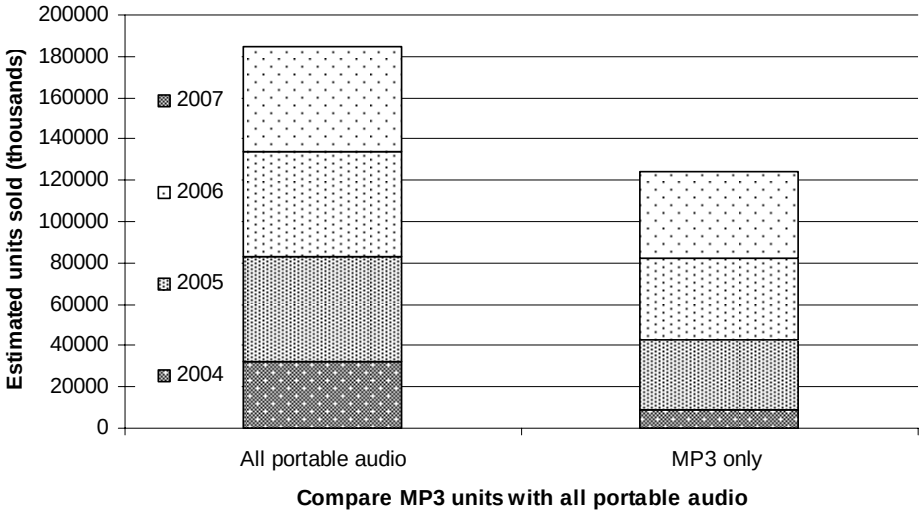

Figure 6: Cumulative numbers of MP3 players and all portable audio equipment (not adjusted for coverage of audit) in EU (web users adjusted).

Figure 7 shows the proportion of portable audio devices sold in 2004-2007, which indicates that about 67% of all portable audio devices sold were MP3 players, with 17% being CD players.

Figure 8 shows the percentage of each portable audio device category sold in 2007 and the estimated numbers sold in that year. It shows that the percentage of MP3 players was about 83% in 2007 compared to 67% over the four year time period. Clearly this proportion of MP3 players will increase over time.

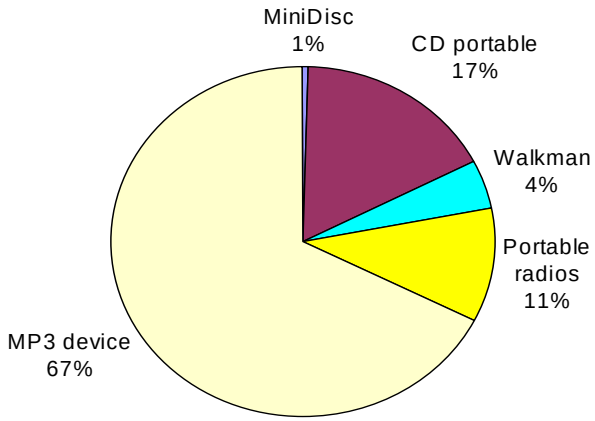

Figure 7: Percentage of each portable audio device sold in 2004-2007

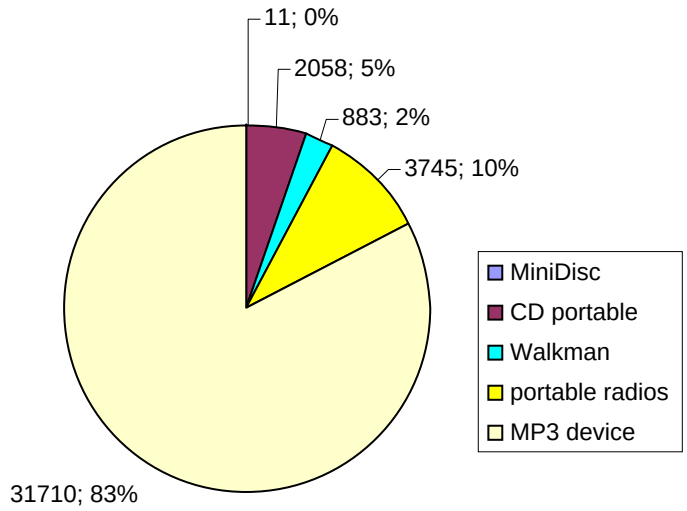

Figure 8: Percentage of each portable audio device sold in 2007 and the estimated number of units sold in EU.

In addition to the data on portable audio the increasing importance of the use of mobile telephone equipment, with high quality audio facilities (very similar if not the same as MP3 players) was examined. These data are very sensitive to market variations and there are no data in terms of the exact availability and use of the mobile phones (Gartner Inc.).

The data suggest that in Q4 of 2007 there were 55 million mobile phone handsets sold in 'Western Europe', which on a pro rata basis (about 28% units sales in 2006 and 2007 worldwide) gives a 2007 sales of 192 million. This is equivalent to about 161 million in EU countries. It is estimated that presently about 10-20% of these phones may have smart features such as MP3 players. This will increase rapidly so that up to 75% of all phones sold by 2011 may have features similar to the MP3 players available now on the market. So in rough terms an estimate might currently be that 16 to 32 million devices (and hence probably close to that in terms of people) have access to these sorts of emerging "hi technology" features. Data are not very precise at present and it is not clear at all whether people who have access to these features actually use them.

Although the data for the portable audio market are accessible, there are no demographics easily available on these sales, nor any information on how many devices an individual may buy over a given time period, how long they last before being discarded and how long and in what situations they are used. Thus, it is hard to estimate the proportion of the population that has access to portable audio or to MP3 players, and how many use them on daily basis. However, it may be estimated in a rather conservative way that in EU a number of daily users of devices like personal music players and mobile phones with this function could be very high and in the range of 50-100 million.

3.7.5. Conclusions

The levels of exposure to sounds from PMP range widely from 80 dB(A) to 115 dB(A) among PMP users, while mean weekly exposure time ranges from below 1 to 14 hours, and is typically longer for men than for women. The type of music and environment may influence exposure levels.

There is a literature evidence that, the average, A-weighted, eight hour equivalent sound exposures levels from PMPs range between 75 to 85 dB(A). Assuming that music as a cause of noise-induced hearing loss could be compared with industrial noise, such exposures produce minimal risk of hearing impairment for the majority of PMP users. However, a small proportion of users are at a higher risk due to the levels patterns and duration of their listening preferences. Considering the daily (or weekly) time spent on listening to music through personal music players and the typical volume control settings, approximately 5% to 10% of the young listeners are at high risk of developing permanent hearing loss after 5 or more years of exposure.

The increase in unit sales of portable audio devices mainly MP3 has been phenomenal in EU over the last four years. Estimated units sales could be in the range 184-246 million for all portable audio devices and in the range 124-165 million for the MP3. The increase overall in sales was noted in 2005 and has been maintained in the following years with more than 50 million devices being sold per year by 2007.

The yearly sales of mobile phones reach a similar number of units. However, at present the availability of the MP3 functionality is not widespread in these handsets (e.g. may be ten percent). Its use is even more unknown. So, at present the major risks to hearing, if use is inappropriate is through portable audio devices, and particularly through the very large numbers of people using MP3 players.

3.8. Effects of sound from PMP on hearing

Noise pollution remains the most frequent environmental hazard accounting for hearing loss. Over the last years an increasing exposure to noise has been noted outside workplace, during recreational/leisure activities. The latter affects not only adults, but also children and adolescents (Plontke et al. 2004). Personal music players were widely introduced to the market in 1980s first as cassette players, and in 1990s as CD players. In the 21st Century, MP3 and currently i-Pods have become very popular. Most PMP users are teenagers and children. Although the equivalent levels of exposure to noise from using these devices on regular basis seem to be substantially lower than e.g. from discos or rock concerts (Serra et al. 2005) they continue to be a concern in the mainstream media. The question is whether or not loud sounds from personal music players including mobile phones with a music playing function could raise a potential risk to hearing loss? This chapter describes the changes in prevalence of hearing loss in young people that could be attributed to increasing environmental noise exposures, as well as the influence of listening to the music through PMPs on hearing threshold shift, either temporary or permanent.

3.8.1. Changes in prevalence of hearing loss in young people

Majority of epidemiological studies on the hearing, including very recent ones, failed to prove an increased prevalence of hearing impairment in teenagers and young adults over the last decades (Strauss et al. 1977, Carter et al. 1982, Persson et al. 1993, Axelsson et al. 1994, Augustsson and Engstrand 2006, Rabinowitz et al. 2006) or did not find a correlation between leisure time activities and hearing impairment (Axelsson et al. 1981, Axelsson 1994, Mostafapour et al. 1998, Tambs et al. 2003). It has been shown that from 5% to almost 20% of young individuals have audiometric "notches" at 4-6 kHz consistent with noise exposure, but this rate has remained constant over the last 20 years (Wong et al. 1990, Meyer-Bisch 1996, Niskar et al. 2001, Axelsson et al. 1981, Axelsson et al. 1994, Peng et al. 2007, Rabinowitz et al. 2006). These studies were performed in German, Australian, Swedish and American populations where the PMP were very common.

Three cohort studies reported increasing prevalence of hearing loss in young individuals over the last 30 years, i.e. during the period when PMP have been extensively used since the 1980s. Montgomery and Fujikawa reported in 1992 that over a decade second

graders with hearing loss has increased by 2.8 times, and eighth graders had an increase of 4 times (Montgomery and Fujikawa, 1992). Danish children starting school in 1977, 1987 and 1997 were evaluated for hearing ability by a review of 1,605 school health records (Gissel et al. 2002). Higher prevalence of impaired hearing ability in children who started school in 1987 and 1997 compared to those who started school in 1977 was found; in addition at the end of school year group 1977 hearing had become as poor as that of year group 1987. Reduced hearing was typically at high frequencies, indicating to noise-induced hearing impairment (Gissel et al. 2002).

Boys and girls (aged 14–17 years) were examined during a four-year period. Audiological, psychosocial, and sound measurements were performed yearly to determine the hearing threshold level (HTL) of participants in the 250–16.000 Hz range, their participation in recreational activities, and the sound levels at discos and through personal music player use. A tendency of the mean HTL to increase in both genders during the study was observed, especially at 14.000 Hz and 16.000 Hz. Boys had a higher mean HTL than girls. The participation in musical activities increased yearly, "attendance at discos" being the favourite musical activity for both groups. In general, boys were more exposed to high sound levels than girls. In this 4-year longitudinal study it was concluded that the exposure to high sound levels during leisure activities (but not necessarily from PMP) could be a cause of permanent hearing damage among young people with "tender ears" (Biassoni et al. 2005).

Although epidemiological literature data does not support the view that there is wide-spread hearing loss caused by exposure to amplified music in young people under the age of 21 years, some authors stress that if the recreational pattern remains the same, there could be some risk of noise-induced hearing loss by the age of mid-twenties (Carter et al. 1982). Slight alterations of hearing function have been detected as possible early signs of ear impairment before deficits were detected with classical audiometry by frequency selectivity and high definition audiometry (West and Evans 1990, Meyer-Bisch 1996), otoacoustic emissions (LePage and Murray 1998) and very high frequency audiometry (Peng et al. 2007). However no follow-up data is available for these studies.

3.8.2. Hearing threshold shift

In the population with increased risk of hearing impairment from personal music players either temporary hearing threshold shift (TTS) or permanent hearing threshold shift (PTS) may develop. TTS may result from short (few hours) exposures at the levels near the maximum output of the music device. PTS may result from repeated exposures (over years) to moderate sound levels exceeding allowable dose of noise.

3.8.2.1. Temporary threshold shift (TTS)

Only few studies have been published on temporary hearing threshold shift due to music sounds from personal music players. They were mainly laboratory investigations on the healthy volunteers overexposed to sounds.

In a pilot study by Lee et al. 16 volunteers listened to headphone sets for 3 hours at their usual maximum level. Six of them showed TTS of 10 dB at one or more audiometric frequencies, and one volunteer showed a TTS of approximately 30 dB at 4 kHz. These shifts returned to normal within 24 hours in all individuals. The average post-test music sound level ranged from 94 to 104 dB SPL (Lee et al. 1985).

In the study by Turunen-Rise et al. the TTS was measured in 6 volunteers with normal hearing (3 males and 3 females aged 23-40 years). They were exposed for 1h to pop music (that generated the highest noise level among all types of music tested) at the gain setting of 8 on the scale. Corresponding field levels of music sound ranged from 85 to 95 dB(A). A small notch at 4 kHz (2-3 dB on average, no more than 15 dB) was observed after exposure to pop music with moderate use of percussion instruments, while significantly more TTS was observed with the pop music extensively using percussion instruments. The highest exposures produced greatest TTS. Hearing

impairment recovered completely within 20-40 min. for the majority of subjects (Turunen-Rise et al. 1991).

In the study by Loth et al (1992), 12 volunteers listened to two recordings (classical music and hard rock) at an acoustic level complying with safety regulation. TTS at 4 and 6 kHz were measures just after exposure and it was, on the average 5 dB. No difference between frequencies and type of music was found.

3.8.2.2. Permanent threshold shift (PTS)

Permanent threshold shift that could be related to music players was investigated by several authors in the field studies designs. Both, positive and negative findings were published.

The first study evaluating the potentially harmful effects of amplified music on young people's hearing was published in early 80's of 20th Century, and failed to prove hearing impairment (Carter et al. 1982).

In their study Wong et al. (1990) assessed the prevalence of use of personal cassette players among youths in a residential community in Hong Kong. They interviewed 487 individuals aged 15-24 years, with 394 (81%) reported using PCP regularly (i.e. for 3 days or more in a week for at least 6 months). The mean duration of PCP use was 2.8 years, and the mean listening time was 4.5 hours per week. Among the 78 PCP users and 25 non-users examined with pure-tone audiometry, no significant difference in the mean hearing threshold was observed for the frequencies tested (250–8000 Hz). However in the studied group, only four subjects were habitually exposed to sound levels higher than 85 dB(A). One was exposed to 116 dB(A) and was found to have a 4000 Hz dip on his audiogram. The authors conclude that despite the high prevalence of PCP use, most youths used their PCP at relatively safe sound levels with low risk of hearing loss (Wong et al. 1990).

Similar results were obtained by Kawada et al in 1990 and West et al. (1990). In the first study no significant differences between mean hearing acuity between PMP users and non-users were found in 155 medical students aged between 22-29 years. However, the mean hearing acuity at 4 kHz showed a tendency of being lower in users than non-users, and higher percent of individuals with hearing impairment was found in the user group as compared to the control. The authors suggest that 10% of the young generation is at risk for permanent hearing loss from use of portable music players (Kawada et al. 1990). In the second paper, sixty subjects in the 15-23 age range were examined. While the most exposed groups did not show significantly greater averaged thresholds, there was a significantly increased prevalence of notches in the 3.5-6 kHz audiograms of the older age group (West, 1990).

The concern about safety of PMPs arose after publication of the study by Meyer-Bisch (1996). The investigation involved young people 14-18 years who were just discovering music played loudly. Three subgroups were defined for users of personal cassette players: occasional, 2-7 h/week and >7 h/week. High definition audiometry (449 frequencies over the spectrum from 125 to 16000 Hz) was applied and early warning indicator (EWI) was calculated from the arithmetic average of the thresholds at 3, 4 and 6 kHz. The groups were matched according to disco attendance, PCP use and concert attendance. PCP users were defined as those who never or only occasionally go to rock/variety concerts or discotheques, but who use a PCP for at least 2 h/week. In total 249 individuals from 14-30 years of age (mean 17.7) were assessed including 54 individuals (21.7%) with the use of a PMPs for at least 8 h/week (majority of them were 15-16 years old subgroup). A statistically significant increase of average hearing thresholds was found in people using PMPs > 7 h/week (54 subjects) as compared to those using PCP 2–7 h/week (195 subjects) and as compared to their matched control. The differences were significant at the range of frequencies from 2 to 12 kHz. From the same study of Meyer-Bisch, it could be concluded that only the individuals with high rate of using PMPs (more than 7 h/week) develop significant permanent threshold shift. On

the other hand, subjective auditory suffering signs (tinnitus, hearing fatigue) were found to be three times more frequent in the PMPs listeners group than in PMPs non-users, suggesting that may be pure-tone audiometry is not the most sensitive method of discovering subclinical or early damage to the cochlea.

More sensitive approach to detect subtle changes in hearing due to exposure to noise is to assess otoacoustic emissions (OAEs). OAEs are thought to be affected early, before clinical signs appear (LePage and Murray 1998, Mansfield et al. 1999, Rosanowski 2006, Shupak et al. 2007), and are simpler to record, thus offering a possible monitoring and educational tool.

In 1998 LePage and Murray applied transient-evoked otoacoustic emission (TEOAE) to assess the effects of personal stereo systems on hearing. Usable records were obtained from 1724 people (1066 males and 658 females) aged between 10 and 59. The level of TEOAE was significantly lower in PMPs users than non-users, although only small proportion (39 people) of PMPs users admitted any hearing problems. For the teenage range (10-19 years) there was no significant difference between non-users, those who used PMPs below 1 hour per week and those who wear PMPs from 1 hour to 6 hours a week. However, for the group of people aged 20-29 and more both users groups was significantly different from non-users group. The authors conclude that the decline in otoacoustic emission strength forewarns premature hearing loss in PMPs. They also suggest that hearing impairment from PMPs music occurs only in the late-teenage and early-adult period (LePage and Murray, 1998). While interpreting the data of LePage and Murray, one should conclude that preclinical phase of hearing loss and relatively short length of time of exposure prior to hearing evaluation (early teen-age) are probably two factors justifying why some previous studies failed to observe any effect of music on hearing.

The other methods of early detection of NIHL applied to occupational noise-exposed population include extended high frequency audiometry and Bekesy audiometry. The studies that used these tools in PMP users were positive. Dieroff et al (Dieroff et al. 1991, Peng et al. 2007) examined 181 persons aged between 16 and 18. The group of individuals using PMP often revealed a significant change in hearing threshold at the audiometric frequencies above 8 kHz; it was also true for the disco attending population. Early elevation in thresholds was also better detected by high resolution Bekesy tracking than by conventional fixed-frequency audiometry in the group of sixty subjects in the 15-23 age range examined by West et al. (West et al. 1990).

More recently published studies are again equivocal. In a prospective auditory testing of fifty college volunteers with retrospective history of exposure to recreational noise, no difference in pure-tone threshold, speech reception threshold or speech discrimination was found among subjects when segregated by noise exposure (Mostafapour et al. 1998). No significant effects of frequent use of personal music players or regular attendance at disco and rock concerts was also demonstrated in the Norway survey of 51,975 adult participants performed in 1995-1997 (Tambs et al. 2003). Similar results were obtained in a subcohort of 358 young (18 year old) adults with a history of otitis media; use of PMPs had no effect on hearing (de Beer et al. 2003). On the other hand, based on audiometric testing of 120 personal music players users and 30 normal-hearing young adults it was revealed that hearing thresholds in the 3 to 8 kHz frequency range were significantly poorer in the group using PMPs (Peng et al. 2007). Also, in some of the PMPs users, the hearing thresholds were worse with high-frequency audiometry even if their hearing thresholds in conventional frequency audiometry were normal (Peng et al. 2007).

3.8.3. Speech comprehension impairment

In the only study on fifty college volunteers with retrospective history of exposure to recreational noise, no differences in speech reception threshold and speech discrimination

was found among individuals when segregated by noise exposure (Mostafapour et al. 1998).

3.8.4. Tinnitus

There are numerous reports of high levels of music-induced tinnitus in young people (Holgers et al. 2005, Chung et al. 2005, Axelsson et al. 2000, Davis et al. 1998, Widén and Erlandsson 2004, Rosanowski 2006). However, only three studies compared the rate of subjective complaints of hearing problems and tinnitus in PMPs users. Two studies were positive showing that these signs were more frequent in walkman users (Becher 1996, Meyer-Bisch 1996). In the study by Meyer-Bisch auditory suffering (AS) was assessed using two subjective parameters – presence of tinnitus (even temporary) and/or hearing fatigue. Auditory suffering was two times significantly more frequent in PCP users (2–7 h/week) than in matched control group. Such difference was not confirmed in those using PCP > 7 h/week, although in PCP group twice as many individuals had some complaints relative to the control group (Meyer-Bisch 1996). In the more recent investigation no correlation between the exposure to PMP and self-reported hearing loss and/or incidence of tinnitus was found (Williams 2005).

3.8.5. Risk associated with pop concerts and discotheques

The data from these studies are presented here since the acoustic levels of exposure are quite similar to those that could be achieved with PMP. However, the sounds being delivered in free field are subject to many more fluctuations as exact position of the ears from the sound sources changes whereas for PMPs no such fluctuations of position from sound sources occur.

In 1977 and 1978 Axelsson and Lindgren published a review of previous studies (5 reports from 1967 to 1974) which indicated that on a total of 160 pop musicians examined only 5 were found to have a hearing loss. They also reported their own observations on 83 pop musicians exposed on average for about 9 years 18 hours a week to levels of up to 115 dB(A); small hearing losses were observed in 13-30% of the subjects depending on the definition of hearing loss; the authors concluded that the risk of NIHL was very small. These authors also indicated that after two hours of pop music pop musicians exhibited TTS for levels starting at 98 dB(A) whereas normal listeners started to have TTS for a level of 92 dB(A), this difference seeming only partly explainable by the original slight elevation of hearing of pop musicians (Axelsson and Lindgren 1978b). Irion (1981) described one case of acute bilateral hearing loss while attending a pop concert followed by almost complete recovery within a few days, this exceptional vulnerability was attributed to genetic predisposition.

Two epidemiologic surveys were reported by Babisch and Ising (1989), one on 204 the other on 3133 young people, showed that those with some hearing loss indicated on average more time spent in discotheques. Such a relation was later confirmed by Dieroff et al. in 1991, within a group of 181 persons (Dieroff et al. 1991). Those who went more than three times a month to discos showed on average a slightly greater loss at very high frequencies. In 1992 Drake-Lee measured TTS in a group of four pop musicians after a concert in which levels could be up to 135 dB(A), with less TTS for those who wore ear defenders. In 1996 Liebel et al. observed TTS of up to 10 dB on average after 2 hours of attendance to a discotheque for two hours at an average level of 105 dB(A). Meyer-Bisch (1996) states that although 211 discotheques patrons did not show audiometric damage, people having gone to rock concerts at least twice a month exhibited some hearing losses. Metternich and Brusis (1999) examined 24 patients consulting after musical acoustic trauma, in two thirds of the patients the hearing loss occurred after a one-time exposure to a pop concert, in the other third the loss occurred after repeated attendances to discotheques or parties, five patients reported tinnitus. In a study on 46 employees in discotheques with at least 89 dB(A) average acoustic level, Lee (1999) observed a higher prevalence of hearing loss and tinnitus as compared to a control group. Sadhra et al. (2002) report on 14 students working in entertainment

venues exposed to more than 90 dB(A) and up to peak levels of 124 dB(A), small but significant TTS was observed. In a study by Bray et al. (2004) on 23 dance music disk jockeys three exhibited dip losses of NIHL type in their audiogram and sixteen reported TTS and tinnitus after job sessions. Among eighty-eight young adults with normal hearing and no tinnitus Rosanowski et al. (2006) indicated about 20% reporting tinnitus after visiting a disco and about 50% reporting a transient hearing loss. Schmuziger et al. (2006) examined 42 non professional pop/rock musicians exposed for at least five years and compared with a control group of 20 non exposed matched subjects, on average a small but significant hearing loss was found in pop/rock musicians, eleven of the musicians were hypersensitive to loud sounds and seven reported tinnitus. Stormer and Stenklev (2007) reviewed seven publications on pop musicians emphasising prevalence of permanent hearing loss, tinnitus hyperacusis and increased resistance to loud music. Finally Schmuziger et al. (2007) indicate in 16 non professional rock/pop musicians a TTS after rehearsal of 90 minutes at a mean acoustic level of 103 dB(A), the TTS affected usual audiometric frequencies while surprisingly very high frequencies were not affected.

Meecham and Hume (2001) questioned 545 students attending night clubs and showed a significant association between attendance at night clubs and duration of post-exposure tinnitus. Non-attendees were significantly less likely to get spontaneous tinnitus.

Overall the data concerning NIHL, associated with pop concerts and discotheques, presents some analogies with those presented above for PMPs. The range of acoustic levels of exposure is can go higher, however the duration and number of times of exposures is smaller. Short term studies clearly demonstrate reversible hearing losses after exposures. The studies started about 30 years ago so that rather long-term data are now available and there is no clear evidence that prevalence of NIHL linked to pop concerts has increased significantly over the last 30 years.

3.8.6. Risk associated with classical orchestral music

In classical orchestras sound levels are on average considerably less than in pop concerts but in some music pieces may also be quite high for long durations and thus musicians appear to be also at some risk of NIHL. A few studies only have dealt with assessment of this risk. Westmore and Everdsen (1981) found slight hearing losses of the notched NIHL type in about a third of 68 musicians, acoustic recordings during rehearsals revealed levels in excess of 90 dB(A) in only 4 hours out of a total of 14 hours. Johnson et al. (1986) tested 60 orchestral musicians in comparison with matched non musicians and they found no difference in hearing sensitivity even at very high frequencies. In contrast Ostri et al. (1989) tested 95 orchestral musicians and found hearing losses in 58% of them in the form of a NIHL type notched audiogram. McBride et al. (1992) did not find clear evidence for NIHL in 36 musicians. Assessment of hearing in 62 choir singers indicated some hearing losses mostly at low frequencies in contrast with the usual pattern of NIHL. From audiometric testing of 140 classical orchestral musicians Kahari et al. (2001) did not find clear evidence for NIHL related hearing losses. Laitinen et al. (2003) measured sound levels for a variety of instrument players and playing conditions, during performances individual exposure levels could be 95 dB(A) while at rehearsals levels could be 100 dB(A).

These studies indicate that for classical musicians involved in classical music acoustic levels of exposure exceed occasionally risk thresholds, but there is no undisputable evidence for an associated NIHL.

3.8.7. Conclusions

It seems that the majority of young users of personal listening devices are at low risk for a substantial NIHL.

The risk of permanent sensorineural hearing loss arises from repeated, regular daily exposures to high sound levels.

Excessive acute exposures to PMPs music at maximal or near maximal output volume can produce reversible hearing impairment (temporary threshold shift) up to 30 dB at 4 kHz in some individuals after short time (one or more hours) of exposure. However, the risk of hearing loss and tinnitus is much smaller compared to pop concerts and discotheques music exposures.

There are major discrepancies between the results of the studies on permanent NIHL in PMP users. They could arise from different study designs and methodology. Most of these studies showed none or only small permanent effect of using PMP on hearing in the majority of users, if short term consequences were assessed with audiometric hearing threshold. A lack of long-term studies and with using more sensitive hearing outcomes, like for example otoacoustic emissions makes it difficult to conclude whether the exposure to PMP music in teenage may influences hearing in older age.

Overall the data concerning NIHL, associated with pop concerts and discotheques, presents some analogies with those presented above for PMPs. The range of acoustic levels of exposure can go higher, however the duration and number of times of exposures is smaller. Short term studies clearly demonstrate reversible hearing losses after exposures.

Studies for classical musicians indicate that their level of exposure occasionally exceed risk threshold, but there is no undisputable evidence for an associated NIHL.

3.9. Non-auditory effects

The non-auditory effects of noise on children and adolescents basically fall into two categories. (1) At the psychological level, seen as changes in reading, memory, attention, school achievement, and motivation and (2) other effects, mainly those who show up at biological or physiological level.

3.9.1. Psychological effects

Pertaining to the psychological effects on cognition and attention, there is no reported research on noise from PMPs. However there are reliable findings of the noise effects from other noise sources on cognition and attention in children and young adults. Thus, to consider possible outcomes of PMP-use it is worthwhile to briefly summarize relevant research, coming mainly from studies of aircraft and road-traffic noise.

3.9.1.1. Reading and memory

The best documented impact of noise on children's performance is research showing negative effects on reading acquisition. Close to twenty studies have found indications of negative relations between chronic noise exposure and delayed reading acquisition in young children (Evans and Lepore 1993). There are no contradictory findings and the few null results are likely due to methodological problems, such as comparing children across school districts who have different reading curricula (Cohen et al. 1986).

There are fewer studies of other cognitive processes and noise among children relative to reading. However, noise effects on memory have been the focus of a handful of studies. The most ubiquitous memory effects occur in chronic noise, particularly when complex, semantic materials are probed (Hygge 2003). Several studies of both chronic (Evans et al. 1995, Haines et al. 2001a, Hygge et al. 2002) and acute noise (Boman 2004, Boman et al. 2005, Hygge 2003, Hygge et al. 2003) have found adverse impacts of aircraft or road traffic noise exposure on long term memory for complex, difficult material. Stansfeld et al. (2005) replicated these effects on long term memory for chronic aircraft noise.

In the experimental acute noise studies by Boman (2004), Hygge (2003), Hygge et al. (2003) worse (approx. 15-20 %) long-term learning and memory in children was induced

by exposure to aircraft and road-traffic noise and speech noise at 66 dB(A) L_{equ} during 15 min exposure time while reading a text and tested for memory of the text an hour later or a week later. For aircraft noise there was impaired memory also from 15 min exposure to 55 dB(A) L_{equ}.

For chronic aircraft noise exposure the Munich study (Hygge et al. 2002) and the RANCH study (Stansfeld et al. 2005) indicated that children exposed to chronic aircraft noise showed cognitive deficits compared to children not having been exposed to chronic aircraft noise. It was also found that the children at the old airport in Munich, who got rid of aircraft noise, improved their cognitive performance. Thus, there was some reversibility in the negative effects of noise on cognition when the noise ceased. To what extent this recovery is dependent upon the age of the children in question (11-12 years) and the accompanying continuing growth in cognitive development, we do not know.

Thus, short time exposure (15 min) to noise with average levels of 65 dB(A), impairs memory and learning. Long-time chronic exposure to, at least aircraft noise, indicate that there will be statistically significant impairments of memory and language skills when the noise levels increase from around or below 55 to above 60 dB(A) L_{equ}.

3.9.1.2. Attention and distraction

Use of music is sometimes employed to distract from a noisy working environment, and sometimes this is beneficial. One reason for this to happen is that the more boring, repetitive and simple a task is, the more will it benefit, both in quality and quantity, from being performed in noise (Kryter 1994). On the other hand, the more complex and difficult the task is, the more it is prone to be hampered by excessive sounds.

When the noise is preferred music from PMP one would in addition expect more of a perceived comfort. Further, when the music from the PMP also masks distracting sounds in the environment, devoid of relevant information or warning characteristics, it will most likely be a subjective advantage to listen to the PMP rather than to shut it off. On the other hand, the more cognitively demanding the task is, the more it is dependent upon speech communication, and the more there are of potential warning sounds in the close environment, the more to the disadvantage of the task performance and the security of the listener the PMP-listening is.

With regard to attention, there is always a risk that the sound of the music listened to from the PMP will acoustically mask warning sounds e.g. from approaching cars, street crossings or reversing trucks. Even if the music is not in a physical sense masking the warning sound, the focused attention on the music will from time to time make the listener inattentive to other sounds, some of which my be warning sounds.

3.9.1.3. School performance

There are a several cross-sectional studies that have reported a covariation between high noise levels (from aircraft or road traffic) and low grades or low levels of school achievement (Cohen et al. 1981, Cohen et al. 1986, Green et al. 1982, Evans and Maxwell 1997, Haines et al. 2001a, Haines et al. 2001b, Haines et al. 2001c, Haines et al. 2002, Maser et al. 1978, Stansfeld et al. 2005). However, cross-sectional studies suffer from two possible short-comings. The first is the differential socio-demographic composition of the noise dose groups, which may favour children in quiet middle-class housing and living areas. Adjusting statistically for the social class effects may not be sufficient to control for this. The second is the possible confound between being exposed to noise both while learning and when tested for what is learnt. Noise at testing may lower the test scores without learning being effected, but the effects of noise on learning and performing can not be disentangled. Thus, cross-sectional studies are not the best platform for a strong inference on cause-effect relationships.

3.9.1.4. Motivation

One laboratory study (Glass 1977) and several field studies (Bullinger et al. 1999, Cohen et al. 1986, Evans et al. 1995, Maxwell and Evans 2000) have found that children chronically exposed to noise are less motivated when placed in achievement situations where task performance is contingent upon persistence. Cohen et al. (1986) also found that a second index of motivation, abrogation of choice, was affected by chronic noise exposure. Children chronically exposed to noise, following a set of experimental procedures in quiet conditions, were more apt to relinquish choice over a reward to an experimenter, in comparison to their well matched quiet counterparts. Haines et al. (2001a) could not replicate the effects of aircraft noise on puzzle persistence in elementary school children although they administered the task in small groups rather than individually.

Perceived control is at the heart of the theorising about noise after-effects. When the noise exposed person perceives that (s)he has control over the noise exposure or noise source, the motivational after effects vanish. Thus, we can not really expect that the persons that freely expose themselves to music from PMPs will lose any motivation just because of that.

3.9.1.5. Lasting after effects on cognition from listening to PMPs

No directly relevant study of lasting after effects (effects that last also after the cessation of noise exposrue) of listenting to PMP on memory, learning, attention or other facets of cognition has been located in the international research literature. Studies of lasting cognitive effects from involuntary exposure to chronic aircraft and road traffic noise (Hygge et al. 2002, Stansfeld et al. 2005) have indicated impaired memory and learning with an increased noise level. It is questionable though whether those studies validly can be stretched to make any inference about voluntary, non-chronic exposure to music. And even if the studies of chronic noise and cognition in some ways are applicable to PMP-listening, they can not state in any detail how long (years) the chronic noise must be present to result in impaired cognition, and whether this cognitive impairment will be permanent or not. For instance, in a study around the Munich airport (Hygge et al. 2002) children chronically exposed to noise at the old airport, and lagging behind their silent control group on memory and language performance, recovered from their deficits within 18 months after the airport was closed down.

Thus, there does not seem to be sufficient research on PMPs to conclude anything about long lasting effects on cognition, and the available evidence from research on other noise sources is not detailed enough to give any strong indications about exposure duration and permanence of cognitive deficits.

3.9.2. Other Effects

The obvious beneficial effect of listening to PMPs is indulging in a preferred activity, which is also the intended outcome. As long as this activity does not interfere with intended or required task performance, there should be no need to restrict listening to PMPs.

3.9.2.1. Sleep

Although there is not much of relevant research, the little research there is point to children having somewhat better sleep than adults. Lukas (1972) stated that children are not as easily awakened by noises adults are. Öhrström et al. (2006) compared children aged 9-12 years with their parents in a road traffic study and reported that for parents there was a significant exposure-effect between noise and several self reported sleep parameters, but this relationship was less marked for children.

3.9.2.2. Cardiovascular and other physiological effects

Twelve studies found some association between increased blood pressure and noise-induced hearing loss (Pyykkö et al. 1981, Lang et al. 1986, Pyykkö et al. 1987, Verbeek et al. 1987, Milković-Kraus 1990, Talbott et al. 1990, Solerte et al. 1991, Starck et al. 1999, Souto Souza et al. 2001, Toppila et al. 2001, Narlawar et al. 2006, Ni et al. 2007). In contrast eleven other studies did not find such an association (Lees RE, Roberts JH. 1979, Willson et al. 1979, Ickes and Nader 1982, Kent et al. 1986, Gold et al. 1989, Kontosić et al. 1990, Tarter and Robins 1990, Hirai et al. 1991, Garcia and Garcia 1993, Zamarro et al. 1992, Barberino 1995). Overall both groups of positive and negative studies are quite comparable in sampling and other methodologies. It must be noted however that the positive findings report moderate average differences sometimes restricted within studies to sub-groups such as only the more exposed or the youngers or those who also smoked showing altered blood pressure. The question of causality remains open, the cardiovascular differences having been simply observed as concommittant. Two studies (Tomanek 1975, Dengerink et al. 1982) produced experimental temporary threshold shifts which were found related with altered cardiovascular parameters, however physiological processes underlying temporary and permanent threshold shifts are known to be notably different. A recent extensive review by Babisch (2006) dealing specifically with exposure to road or aircraft noise on blood pressure, hypertension and ischaemic heart disease concludes that there is no clear evidence of increased blood pressure. Whereas for aircraft noise (but not road noise exposure) most recent studies (Babisch 2006) indicate some significant relationship, finally concerning ischeamic heart disease more recent studies also suggest a trend towards increased risk as compared with previous studies.

3.9.3. Conclusions

Exposing oneself to music from a PMP is a matter of a personal choice of leisure activity. Harmful lasting and irreversible non-auditory effects of excessive listening to PMP can be expected in three areas: (1) Cardiovascular effects, (2) cognition and (3) distraction and masking effects. Cardiovascular effects, in particular increases in blood pressure, build up and accumulate over time, when there is not enough silent time in between noise exposures to recover. However, we do not have sufficient evidence to state that music from PMPs constitutes a risk for hypertension and ischaemic heart disease in children and young adults.

Effects on cognition (memory and learning) of excessive sound exposure has been shown from acute noise exposure and from chronic noise exposure. Noise exposure for 15 min to 66 dB(A), and for aircraft noise down to 55 dB(A) has been shown to cause impaired learning and memory of a text. We have no study stating that the same is true also for music, but we also have no reason to believe that music should be substantially less harmful to cognition that aircraft noise, road traffic noise or speech noise. Thus, listening to music from PMP while at the same time trying to read a text and learn from it, will hamper memory and learning. This learning impairment has been shown at fairly short (15 min) exposure times and at sound levels that are moderate (55-65 dB(A)).

Prolonged exposure to chronic aircraft noise has been shown to impair cognition in children, but there is also one indication that children may recover from the noise induced cognitive deficit when the noise exposure stops.

We do not as yet have a sufficient scientific basis to assume that excessive voluntary PMP-listening leads to lasting and irreversible cognitive and attention deficits after the cessation of the noise.

4. OPINION

The SCENIHR was asked to assess, in the light of current scientific data and knowledge:

1. Whether the exposure to noise[19] from devices like personal music players and mobile phones with this function, at levels corresponding to current permissible noise emissions may cause quantifiable health risks, in particular hearing loss and/or hearing impairment to the user, and to specify the relevant outcomes;
2. In case health risks are identified, the SCENIHR is asked:
    a. to identify the level of noise emission safeguarding the health of citizens, taking into account the intensity, length and number of exposures to users of personal music players and mobile phones with the same function and
    b. to identify priority issues for further research.

Background

The increase in unit sales of portable music players (PMP) including MP3 playback function has been phenomenal since their introduction in the EU around four years ago. Estimated units sales range between 184-246 million for all portable audio devices and range between 124-165 million for MP3 players. There was a marked increase in overall portable audio devices sales in 2005 and sales were maintained in the following years with more than 50 million devices being sold per year by 2007. Mobile phones are sold by similar numbers of units every year. However, at present the availability of the MP3 functionality is not widespread in these handsets (maybe ten percent) while their frequency of use remains as yet unknown.

Although the data for the portable audio market are accessible, there are no demographics easily available on these sales, nor any information on how many devices an individual may buy over a given time period, how long they last before being discarded and how long and in what situations they are used. Thus, it is hard to estimate the proportion of the population that has access to portable audio or to MP3 players, and how many use them on daily basis. However, it may be estimated on rather conservative way that in EU the number of users of devices like personal music players and mobile phones with this function, are in tens of millions daily.

The digital formats of sound recording and reproduction currently available (e.g. MP3) make it possible to reach high levels of sound output with virtually no distortion, that could possibly cause a risk to human hearing originating from the inappropriate use of portable music players.

As shown by many studies, noise-induced hearing loss (NIHL) is a function of sound level and duration of exposure. The fundamental unit of noise exposure measurement is A-weighted decibel [dB(A)]. This unit corresponds well with the physiological sensitivity of human ear and it has been generally adopted in scientific literature.

Noise at Work Regulations (Directive 2003/10/EC, came into force in 2006) establish a minimal action level of hearing protection to the limit of 80 dB(A) for an 8 hour working day (or 40 hour working week) assuming that below this level the risk to hearing is negligible. The exposure to sound at the level exceeding 80 dB(A) is considered a risk if it continues at that level for 8 hours a day, five days a week for tens of years. The 8-hour equivalent level ($L_{equ,\ 8h}$) is a widely used measure for the risk of hearing damage in industry. On the basis of equal energy, level and time of exposure may be traded with

[19] Please note that throughout this opinion, the term 'noise' is used consistently in the context of all disease and malfunction patterns, while the word 'sound' is used consequently to clarify that the concern is the voluntary listener of personal music players and not the observer of the listening situation. For details see paragraph 3.3.1

halving of time of exposure with every doubling in level (+3dB). Using the equal energy basis it may be deduced that the exposure to 80 dB(A) for 40 hours would be equivalent to the exposure to 83 dB(A) for 20 hours and 89 dB(A) for 5 hours per week. However, because the model was built on the basis of tens of years of exposure, such calculation for exposures of short length should be interpreted with caution.

Although all the above regulations and limits apply to the workplace, the fact that they rely on the exposure level and duration means that they can equally be applied to other situations where sound has a detrimental effect such as that from personal music players (PMPs); whether use in workplace, or under leisure situations.

The free-field equivalent sound pressure levels measured at maximum volume control setting of PMPs range around 80-115 dB(A), across different devices. Differences between different types of ear-phones may modify the level by up to 7-9 dB. In the worst case scenario, it is possible to estimate maximum levels of about 120 dB(A). The hazard to hearing from listening to the music at such levels might be extremely high, as it is considered that levels exceeding 80 dB(A) may pose a risk.

Question 1:

SCENIHR is asked whether the exposure to noise from devices like personal music players and mobile phones with this function, at levels corresponding to current permissible noise emissions may cause quantifiable health risks, in particular hearing loss and/or hearing impairment to the user, and to specify the relevant outcome.

Answer:

It is estimated that the number of young people with social noise exposure has tripled (to around 19%) since the early 1980s, whilst occupational noise has decreased. It should be recognised that exposure to different types of noise and sounds can have cumulative effects in hearing impairment.

There is evidence in the scientific literature that the levels of exposure to sounds from using PMP on regular basis range widely from 60 dB(A) to 120 dB(A) among PMP users, but a vast majority of listeners use it at a level below 80-85 dB(A). The type of music and environment may influence exposure levels. The mean weekly exposure time spent on listening to music ranges from below 1 to 14 hours, and is typically longer for men than for women. It has been estimated that the average, A-weighted, eight hour equivalent sound exposures levels (referred to "Noise at Work Regulations") from PMPs range from 75 to 85 dB(A), producing minimal risk of hearing impairment for the majority of PMP users.

However, a certain proportion of users are at a higher risk due to the levels, patterns and duration of their listening preferences. Considering the daily (or weekly) time spent on listening to music through personal music players and the typical volume control settings, approximately 5% to 10% of the listeners are at risk of developing permanent hearing loss after 5 or more years of exposure – the best estimate available on the limited data (which may be an underestimate based on unpublished information) suggests that this may be between 2.5 and 10 million people in EU. Those are the individuals listening to music over 1 hour a day at high volume control setting.

Literature data indicate that the consequences of prolonged exposure to loud sounds from the PMPs may possibly result in:

- TTS: Temporary (hearing) threshold shift
- PTS: Permanent (hearing) threshold shift
- Tinnitus: Ringing in the Ears
- Poor Speech Communication in Noisy Conditions

- Change in behaviour with the environment (pedestrian/driving behaviour while listening, acoustic isolation during use)
- Non-auditory effects

It has been shown that acute exposure to music listened through PMPs at the level between 94-104 dB of sound pressure level (dB SPL) leads to around 10dB of temporary threshold shift, but in sensitive individuals may cause up to 30dB shift. Although the data are very limited they confirm that reversible hearing impairment may occur in some individuals. Clearly 30 dB temporary threshold shift could affect performance on listening-sensitive tasks such as driving or communicating.

There are major discrepancies between the results of the studies on permanent noise-induced hearing loss (NIHL) in PMP users. Both, positive and negative studies have been published. The discrepancies could arise from different study designs and methodology. Most of these studies showed none or only small permanent effect of using PMP on hearing in the majority of users, if short term consequences were assessed with audiometric hearing threshold. It is difficult to conclude with the available data whether the exposure to PMP music in teenage may influence hearing in older age. This is due to a lack of long-term studies using sensitive measures of hearing impairment.

A few studies indicate that tinnitus and hearing fatigue occur more frequently in teenagers exposed to music, including PMP users, than in those unexposed.

Harmful, lasting and irreversible non-auditory effects of excessive listening to PMP can be expected in three areas: (1) cardiovascular effects, (2) cognition and (3) distraction and masking effects. It was shown that noise exposure to 66 dB(A) for 15 min, and down to 55 dB(A) for aircraft noise may cause impaired learning and memory of a text, and we assume the same to be true for music from PMPs.

Prolonged exposure to chronic aircraft noise has been shown to impair cognition in children, but there is also one indication that children may recover from the noise induced cognitive deficit when the noise exposure stops. To what extent this recovery is dependent upon the age of the children in question and the accompanying general growth in cognitive development, we do not know. Thus, we can not say with any precision across age groups how long a noise induced cognitive deficit will last when the noise exposure has ceased.

As regards the physiological non-auditory effects of listening to PMPs, increased blood pressure and ischemic heart disease are of principal relevance. However, at present, there is not sufficient evidence to state that music from PMPs constitutes a risk for hypertension and ischaemic heart disease in children and young adults.

Question 2:

In case health risks are identified, the SCENIHR is asked:

a. to identify the level of noise emission safeguarding the health of citizens, taking into account the intensity, length and number of exposures to users of personal music players and mobile phones with the same function and

b. to identify priority issues for further research

Answer 2a, level of noise emission safeguarding the health of citizens:

Based on workplace studies, the probability of acquiring a hearing loss is negligible at sound levels below 80 dB(A), and this level might be regarded as safe, no matter how long (daily or weekly) the exposure to sounds from PMPs. It still remains uncertain whether this threshold can be applied to young children.

For higher levels (above 80 dB(A)), the safety of the sound exposure levels for hearing is determined by the time (hours a day) spent on listening to music through the PMPs. With

caution, this allowable time can be calculated by using the equal energy rule and the 3 dB exchange rate as described in the background. Assuming that an average PMP user listens for 7 hours per week (1 hour/day), this would exceed the Noise at Work Regulations if the sound level for the PMP exceeded 89 dB(A). However, since these devices have been introduced in the market only very recently, there is inevitably insufficient population data on hearing impairment. In assessing the likelihood of hearing loss, consideration of other environmental sources of high level sound emissions need to be taken into account.

As for non-auditory effects of sound exposure from PMPs, no level of noise emission safeguarding the health could be established so far.

Answer 2b, priority issues for further research:

In the face of an increasing population at risk of hearing loss and tinnitus due to i) increasing PMPs use and acceptance in the EU and ii) the possibility to use PMPs at high sound levels, there is a lack of data concerning:

a) the current PMP use pattern, duration, output level, choice of loud levels and exposure of users to other high level sound sources.

b) the contribution of loud sounds to hearing loss and tinnitus, as well as cognitive and attention deficits in children and young people.

c) long-term studies using more sensitive hearing impairment measures to assess the impact of PMPs on hearing and to identify the potential sub-groups more 'at risk' (e.g. children, genetic sub-groups and environmental sub-groups such as those who commute to work or school in noisy surroundings).

d) the biological basis of individual susceptibility to noise and the benefits from pharmacological treatment.

e) whether excessive voluntary PMP-listening leads to lasting and irreversible cognitive and attention deficits after the cessation of the noise.

5. COMMENTS RECEIVED FROM THE PUBLIC CONSULTATION

Information about the public consultation has been broadly communicated to national authorities, international organisations, and other stakeholders. The relevant web site was opened for comments on 27 June 2008 and the deadline for submission was 27 August 2008. In total, 9 contributions were received from which 7 were from organisations and 2 from individuals. Of the organisations, 1 was non governmental, 2 public authorities, 2 academic institutions and 2 business organisations.

In evaluating the responses from the consultation, submitted material has only been considered for revision of the opinion if:

1. it is directly referring to the content of the report and relating to the issues that the report addresses,
2. it contains specific comments and suggestions on the scientific basis of the opinion,
3. it refers to peer-reviewed literature published in English, the working language of the SCENIHR and the working group,
4. it has the potential to add to the preliminary opinion of SCENIHR.

Each submission has been carefully considered by the Working Group. Overall, the comments were of good quality. The report has been revised to take account of relevant comments and the literature has been updated with relevant publications. The Opinion, however, remained essentially unchanged, but was, in certain respects, clarified by the amendments to the scientific rationale.

6. MINORITY OPINION

None.

7. LIST OF ABBREVIATIONS

dB(A)	A-weighted decibel
dB HL	Hearing level decibel
dB nHL	Normal hearing level decibel
dB SPL	Decibel of sound pressure level
DPOAEs	Distortion-product otoacoustic emissions
EWI	Early warning indicator
HATS	Head and torso simulator
HRTF	Head-related transfer function
HTL	Hearing threshold level
IHC	Inner hair cells
ISO	International standard organization
KEMAR	Knowles electronics manikin for acoustic research
NIHL	Noise-induced hearing loss
NITS	Noise induced threshold shift
OAEs	Otoacoustic emissions
OHC	Outer hair cells
OES	occluded ear simulator
PCP	Personal cassette players
PMP	Personal music player
PST	Prolonged spontaneous tinnitus
PTS	Permanent threshold shift
SII	Speech intelligibility index
STI	Speech transmission index
TEOAEs	Transient-evoked otoacoustic emissions
TTS	Temporary threshold shift
WHO	World Health Organisation

8. REFERENCES

Abbate C, Concetto G, Fortunato M, Brecciaroli R, Tringali MA, Beninato G, et al. Influence of environmental factors on the evolution of industrial noise-induced hearing loss. Environ Monit Assess 2005; 107(1-3):351-61.

Ahmed HO, Dennis JH, Badran O, Ismail M, Ballal SG, Ashoor A, Jerwood D. High-frequency (10-18 kHz) hearing thresholds: reliability, and effects of age and occupational noise exposure. Occup Med (Lond) 2001; 51(4):245-58.

Ahn JH, Kang HH, Kim YJ, Chung JW Anti-apoptotic role of retinoic acid in the inner ear of noise-exposed mice. Biochem Biophys Res Commun 2005; 335(2):485-90.

Alberti PW. Noise and the ear. In: Scott-Brown's Otolaryngology, Volume 2, Adult Audiology, Stephens D (ed.). Oxford: Butterworth-Heinemann, 1977: 2/11/1:-34.

Attias J, Horovitz G, El-Hatib N, Nageris B. Detection and Clinical Diagnosis of Noise-Induced Hearing Loss by Otoacoustic Emissions. Noise Health 2001; 3(12):19-31.

Attias J, Reshef I, Shemesh Z, Salomon G. Support for the central theory of tinnitus generation: a military epidemiological study. Int J Audiol 2002; 41(5):301-7.

Attias J, Bresloff I, Haupt H, Scheibe F, Ising H. Preventing noise induced otoacoustic emission loss by increasing magnesium (Mg2+) intake in guinea-pigs. J Basic Clin Physiol Pharmacol 2003; 14(2):119-36.

Attias J, Sapir S, Bresloff I, Reshef-Haran I, Ising H. Reduction in noise-induced temporary threshold shift in humans following oral magnesium intake. Clin Otolaryngol Allied Sci 2004; 29(6):635-41.

Augustsson I, Engstrand I. Hearing ability according to screening at conscription; comparison with earlier reports and with previous screening results for individuals without known ear disease. Int J Pediatr Otorhinolaryngol 2006; 70(5):909-13.

Avan P, Wit HP, Guitton M, Mom T, Bonfils P. On the spectral periodicity of transient-evoked otoacoustic emissions from normal and damaged cochleas. J Acoust Soc Am 2000; 108:1117-27.

Avan P, Bonfils P. Distortion-product otoacoustic emission spectra and high-resolution audiometry in noise-induced hearing loss. Hear Res 2005; 209(1-2):68-75.

Axelsson A, Lindgren F. Does pop music cause hearing damage? Audiology. 1977; 16(5):432-7.

Axelsson A, Lindgren F. Hearing in pop musicians. Acta Otolaryngol 1978a; 85(3-4):225-31.

Axelsson A, Lindgren F. Temporary threshold shift after exposure to pop music. Scand Audiol 1978b; 7(3):127-35.

Axelsson A, Jerson T, Lindberg U, Lindgren F. Early noise-induced hearing loss in teenage boys. Scand Audiol 1981; 10(2):91-6.

Axelsson A, Rosenhall U, Zachau G. Hearing in 18-year-old Swedish males. Scand Audiol 1994; 23(2):129-34.

Axelsson A, Prasher D. Tinnitus induced by occupational and leisure noise. Noise Health 2000; 2(8):47-54.

Babisch W, Ising H. The effect of music in discothèques on hearing ability. Soz Praventivmed 1989; 34(5):239-42.

Babisch W Transportation noise and cardiovascular risk : updated review and synthesis of epidemiological studies indicate that the evidence has increased. Noise and Health 2006; 8:1-29.

Baguley DM. Mechanisms of tinnitus. Br Med Bull 2002; 63:195-212.

Balatsouras DG. The evaluation of noise-induced hearing loss with distortion product otoacoustic emissions. Med Sci Monit 2004; 10(5):CR218-22.

Balow RW, Spivey GH, Brown CP, Morgan D, Campion DS, Browdy BL, et al. Subclinical effects of chronic increased lead absorption—a prospective study. II Results of baseline neurologic testing. J Occup Med 1979; 21(7):490-6.

Bamiou D, Lutman ME. Interaction of NIHL and ageing: epidemiological aspects. In: Luxon L, Prasher D (eds). Noise and its effects. Chichester: John Wiley, 2007; 64-84.

Barrenäs ML, Bratthall A, Dahlgren J. The association between short stature and sensorineural hearing loss. Hear Res 2005; 205(1-2):123-30.

Basta D. Erratum to „Noise-induced changes of neuronal spontaneous activity in mice inferior colliculus brain slices". Neurosci Lett 2005; 374(1):74-79.

Bauer CA, Brozoski TJ. Effect of gabapentin on the sensation and impact of tinnitus. Laryngoscope 2006; 116(5):675-81.

Becher S, Struwe F, Schwenzer C, Weber K. Risk of hearing loss caused by high volume music--presenting an educational concept for preventing hearing loss in adolescents. Gesundheitswesen. 1996; 58(2):91-5 [Article in German].

Biassoni EC, Serra MR, Richtert U, Joekes S, Yacci MR, Carignani JA, et al. Recreational noise exposure and its effects on the hearing of adolescents. Part II: development of hearing disorders. Int J Audiol 2005; 44(2):74-85.

Boeda B, El-Amraoui A, Bahloul A, Goodyear R, Daviet L, Blanchard S, et al. Myosin VIIa, harmonin and cadherin 23, three Usher I gene products that cooperate to shape the sensory hair cell bundle. EMBO 2002; 21:6689-99.

Bogoch II, House RA, Kudla I. Perceptions about hearing protection and noise-induced hearing loss of attendees of rock concerts. Can J Public Health 2005; 96(1):69-72.

Bohlin MC, and Erlandsson SI. Risk behaviour and noise exposure among adolescents. Noise Health 2007; 9(36):55-63.

Boman E. The effects of noise and gender on pupil's episodic, and semantic memory. Scandinavian Journal of Psychology 2004; 45:407-16.

Boman E, Enmarker I, Hygge S. Strength of noise effects on memory as a function of noise source and age. Noise & Health 2005; 7:11-26.

Borg E. Protective value of sympathectomy of the ear in noise. Acta Physiol Scand 1982; 115(2):281-2.

Bray A, Szymański M, Mills R. Noise induced hearing loss in dance music disc jockeys and an examination of sound levels in nightclubs. J Laryngol Otol 2004; 118(2):123-8.

Brickner D, Carel R. Annual rate of development of noise induced hearing loss in exposed workers Harefuah 2005; 144(10):692-5, 751.

BS EN 60645-1:2001* IEC 60645-1:2001. Audiometers. Pure-tone audiometers.

BS EN ISO 389-2:1997. Acoustics. Reference zero for the calibration of audiometric equipment. Reference equivalent threshold sound pressure levels for pure tones and insert earphones. Bulletin épidémiologique des armées, juin 2006.

Buchanan LH. Distortion Product Oto-acoustic Emissions in Andean Children and Adults with Chronic Lead Intoxication. Acta Oto-Laryngologica 1999; 119(6):652-658.

Bullinger M, Hygge S, Evans GW, Meis M, von Mackensen S. The psychological costs of aircraft noise among children. Zentralblatt für Hygiene und Umweltmedizin 1999; 202:127-38.

Burns W, Robinson DW. Hearing and noise in industry. London: HMSO, 1970.

Burr H, Lund SP, Sperling BB, Kristensen TS, Poulsen OM. Smoking and height as risk factors for prevalence and 5-year incidence of hearing loss. A questionnaire-based follow-up study of employees in Denmark aged 18-59 years exposed and unexposed to noise. Int J Audiol 2005; 44(9):531-9.

Caban AJ, Lee DJ, Gómez-Marín O, Lam BL, Zheng DD. Prevalence of concurrent hearing and visual impairment in US adults: The National Health Interview Survey, 1997-2002. Am J Public Health 2005; 95(11):1940-2.

Campo P, Lataye R, Loquet G, Bonnet P. Styrene-induced hearing loss: a membrane insult. Hear Res 2001; 154(1-2):170-80.

Campo P, Lataye R. Noise and solvent, alcohol and solvent: two dangerous interactions on auditory function. Noise Health 2000; 3(9):49-57.

Campo P, Lataye R, Cossec B, Villette V, Roure M, Barthelemy C. Combined effects of simultaneous exposure to toluene and ethanol on auditory function in rats. Neurotoxicol Teratol 1998; 20(3):321-32.

Canlon B, E. Borg and Å. Flock , Protection against noise trauma by pre-exposure to a low level acoustic stimulus. Hear Res 1998; 34:197–200.

Carlsson P, Borg E, Grip L, Dahl N, Bondeson ML. Variability in noise susceptibility in a Swedish population: The role of 35delG mutation in the Connexin 26 (*GJB2*) gene. Audiol Med 2004; 2(2):123-30.

Carlsson PI, Van Laer L, Borg E, Bondeson ML, Thys M, Fransen E, et al. The influence of genetic variation in oxidative stress genes on human noise susceptibility. Hear Res 2005; 202:87-96.

Carter NL, Waugh RL, Keen K, Murray N, Bulteau VG. Amplified music and young people's hearing. Review and report of Australian findings. Med J Aust 1982; 2(3):125-8.

Cassandro E, Sequino L, Mondola P, Attanasio G, Barbara M, Filipo R. Effect of superoxide dismutase and allopurinol on impulse noise-exposed guinea pigs--electrophysiological and biochemical study. Acta Otolaryngol 2003; 123(7):802-7.

Catalano PJ, Levin SM. Noise-induced hearing loss and portable radios with headphones. Int J Pediatr Otorhinolaryngol 1985; 9(1):59-67.

Chen GD, Liu Y. Mechanisms of noise-induced hearing loss potentiation by hypoxia. Hear Res 2005; 200(1-2):1-9.

Chen Q, Guo WW, Wu Y, Liu H, Zhai SQ, Wang JZ, et al. Adenovirus-mediated NT3 gene transfer protects spiral ganglion neurons from degeneration after noise trauma Sheng Li Xue Bao 2002; 54(3):263-6.

Chen Z, Duan M, Lee H, Ruan R, Ulfendahl M. Pharmacokinetics of caroverine in the inner ear and its effects on cochlear function after systemic and local administrations in Guinea pigs. Audiol Neurootol 2003; 8(1):49-56.

Chung JH, Des Roches CM, Meunier J, Eavey RD. Evaluation of noise-induced hearing loss in young people using a web-based survey technique. Pediatrics 2005; 115(4):861-7. [see also comment in: Pediatrics 2006; 117(1):248-9; author reply 249]

Cody AR, Johnstone BM Temporary threshold shift modified by binaural acoustic stimulation. Hear Res 1982; 6(2):199-205.

Cohen S, Krantz D, Evans GW, Stokols D, Kelly S. Aircraft noise and children: Longitudinal and cross-sectional evidence on adaption to noise and the effectiveness of noise abatement. Journal of Personality and Social Psychology 1981; 40:331-45.

Cohen S, Evans GW, Stokols D, Krantz DS. Behavior, health, and environmental stress. N.Y.: Plenum, 1986.

Counter SA, Vahter M, Laurell G, Buchanan LH, Ortega F, Skerfving S. High lead exposure and auditory sensory-neural function in Andean children. Environmental Health Perspectives 1997, 105:522-526.

Counter SA, Buchanan LH, Laurell G, Ortega F. Blood mercury and auditory neuro-sensory responses in children and adults in the Nambija gold mining area of Ecuador. Neurotoxicology 1998a; 19:185-196.

Counter SA, Buchanan LH, Laurell G, Ortega F. Field screening of blood lead levels in remote Andean villages. Neurotoxicology 1998b; 19:871-878.

d'Aldin C, Cherny L, Devriere F, Dancer A. Treatment of acoustic trauma. Ann N Y Acad Sci 1999; 884:328-44.

Davis AC, Lovell EA, Smith PA, Ferguson MA. The contribution of social noise to tinnitus in young people - a preliminary report. Noise Health 1998; 1(1):40-46.

Davis RR, Newlander JK, Ling X, Cortopassi GA, Krieg EF, Erway LC. Genetic basis for susceptibility to noise-induced hearing loss in mice. Hear Res 2001; 155(1-2):82-90.

de Abreu MT, Suzuki FA. Audiometric evaluation of noise and cadmium occupationally exposed workers. Brazilian Journal of Otorhinolaryngology 2002; 68(6):488-494.

de Beer BA, Graamans K, Snik AF, Ingels K, Zielhuis GA. Hearing deficits in young adults who had a history of otitis media in childhood: use of personal stereos had no effect on hearing. Pediatrics 2003; 111(4 Pt 1):e304-8.

Dengerink JE, Dengerink HA, Chermak GD. Personality and vascular responses as predictors of temporary threshold shifts after noise exposure. Ear Hear 1982; 3(4):196-201.

Diao M, Zhang Y, Liu H, Han H, Gao W Observation on the protective effect of MK-801 against hearing loss in acoustic trauma Lin Chuang Er Bi Yan Hou Ke Za Zhi 2005; 19(1):27-30.

Dieroff HG, Schuhmann G, Meissner W, Bartsch R. Experiences with high-frequency hearing tests in the selection of personnel for noise occupations]. Laryngorhinootologie 1991; 70(11):594-8 [Article in German].

Directive 2003/10/EC of 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) (17th individual Directive within the meaning of Article 16(1) of Directive 89/391/EEC). Official Journal of the European Communities. No L42/38, 15.2.2003, 2000.

Drake-Lee AB. Beyond music: auditory temporary threshold shift in rock musicians after a heavy metal concert. J R Soc Med 1992; 85(10):617-9.

Duan M, Qiu J, Laurell G, Olofsson A, Counter SA, Borg E. Dose and time-dependent protection of the antioxidant N-L-acetylcysteine against impulse noise trauma. Hear Res 2004; 192(1-2):1-9.

El-Shazly A. Toxic solvents in car paints increase the risk of hearing loss associated with occupational exposure to moderate noise intensity. B-ENT 2006; 2(1):1-5.

Emmerich E, Richter F, Hagner H, Giessler F, Gehrlein S, Dieroff HG. Effects of discotheque music on audiometric results and central acoustic evoked neuromagnetic responses. Int Tinnitus J 2002; 8(1):13-9.

Erway LC, Shiau Y-W, Davis RR, Krieg EF. Genetics of age-related hearing loss in mice. III. Susceptibility of inbred and F1 hybrid strains to noise-induced hearing loss. Hear Res 1996; 93:181-187.

Evans GW, Hygge S, Bullinger M. Chronic noise and psychological stress. Psychological Science 1995; 6:333-8.

Evans GW, Lepore SJ. Nonauditory effects of noise on children: A critical review. Children's Environments 1993; 10:31-51.

Evans GW, Maxwell L. Chronic exposure and reading deficits: The mediating effects of language acquisition. Environment and Behaviour 1997; 29(5):638-56.

Fakhry N., Rostain JC, Cazals Y. Hyperbaric oxygenation with corticoid in experimental acoustic trauma. Hear Res 2007; 230(1-2):88-92.

Farahat TM, Abdel-Rasoul GM, EL-Assy AR, Kandil SH, Kabil MK. Hearing thresholds of workrs in a printing facility. Environ Res 1997; 73(1-2):189-92.

Fechter LD, Cheng GD, Rao D. Characterising conditions that favour potentiation of noise induced hearing loss by chemical asphyxiants. Noise Health 2000; 3(9):11-21.

Fechter LD, Chen GD, Johnson DL. Potentiation of noise-induced hearing loss by low concentrations of hydrogen cyanide in rats. Toxicol Sci 2002; 66(1):131-8.

Fechter LD, Klis SF, Shirwany NA, Moore TG, Rao DB. Acrylonitrile produces transient cochlear function loss and potentiates permanent noise-induced hearing loss. Toxicol Sci 2003; 75(1):117-23.

Fechter LD. Promotion of noise-induced hearing loss by chemical contaminants. J Toxicol Environ Health A 2004; 67(8-10):727-40.

Ferrite S, Santana V. Joint effects of smoking, noise exposure and age on hearing loss. Occup Med (London) 2005; 55(1):48-53.

Fligor BJ, Cox C, Output levels of commercially available portable compact disc players and the potential risk to hearing. Ear Hear 2004; 25:513-527.

Florentine M, Hunter W, Robinson M, Ballou M, Buus S. On the behavioural characteristic of loud-music listening. Ear Hear 1998; 19(6):420-8.

Forst L, Persky V, Freels S, Williams R, Conroy L. Lead exposure in ironworkers. Am J Ind Med 1997; 32(5):540-3.

Forst LS, Freels S, Persky V. Occupational lead exposure and hearing loss. J Occup Environ Med 1997; 39(7):658-60.

Fortunato G, Marciano E, Zarrilli F, Mazzaccara C, Intrieri M, Calcagno G, et al. Paraoxonase and superoxide dismutase gene polymorphisms and noise-induced hearing loss. Clin Chem 2004; 50(11):2012-8.

Franze A, Sequino L, Saulino C, Attanasio G, Marciano E. Effect over time of allopurinol on noise-induced hearing loss in guinea pigs. Int J Audiol 2003; 42(4):227-34.

Fuente A, McPherson B. Central auditory damage induced by solvent exposure. Int J Occup Saf Ergon 2007; 13(4):391-7.

Gao W, Ding D, Zheng X. A comparison of changes in the stereocilia between temporary and permanent hearing losses in acoustic trauma. Hear Res 1992; 62:27-41.

Garcia AM, Garcia A. Occupational noise as a cardiovascular risk factor. Schriftenr Ver Wasser Boden Lufthyg 1993; 88:212-22.

García Callejo FJ, García Callejo F, Conill Tobías N, Ramírez Sabio JB. Effect of smoking withdrawal on hearing loss induced by occupational exposure to noise. A preliminary study. Acta Otorrinolaringol Esp 2006; 57(9):432-4.

Gartner, Inc. (NYSE: IT). http://www.gartner.com/DisplayDocument?ref=g_search&id=550312&subref=simplesearch and the slightly more detailed report on http://www.gartner.com/it/page.jsp?id=612207 [accessed on 29th March 2008]

Gates GA, Schmid P, Kujawa SG, Nam B, D'Agostino R Longitudinal threshold changes in older men with audiometric notches. Hear Res 2000; 141(1-2):220-8.

George EL, Festen JM, Houtgast T. Factors affecting masking release for speech in modulated noise for normal-hearing and hearing-impaired listeners. J Acoust Soc Am 2006; 120(4):2295-311.

Giraudet F, Horner KC, Cazals Y. Similar half-octave TTS protection of the cochlea by xylazine/ketamine or sympathectomy. Hear Res 2002; 174(1-2):239-48.

Gissel S, Mortensen JT, Juul S. Evaluation of hearing ability in Danish children at the time of school start and at the end of school. Int J Adolesc Med Health 2002; 14(1):43-9.

Glass DC. Behavior patterns, stress, and coronary heart disease. Hillsdale, N.J.: Erlbaum, 1977.

Gok U, Halifeoglu I, Canatan H, Yildiz M, Gursu MF, Gur B. Comparative analysis of serum homocysteine, folic acid and Vitamin B12 levels in patients with noise-induced hearing loss. Auris Nasus Larynx 2004; 31(1):19-22.

Golz A, Westerman ST, Westerman LM, Goldenberg D, Netzer A, Wiedmyer T, Fradis M, Joachims HZ. The effects of noise on the vestibular system. Am J Otolaryngol 2001; 22(3):190-6.

Gold S, Haran I, Attias J, Shapira I, Shahar A. Biochemical and cardiovascular measures in subjects with noise-induced hearing loss. J Occup Med. 1989; 31(11):933-7.

Green K, Pasternack B, Shore R. Effects of aircraft noise on reading ability of school age children. Archives of Environmental Health 1982; 37:24-31.

Haines MM, Stansfeld SA, Brentnall S, Head J, Berry B, Jiggins M, et al. The West London schools study: the effects of chronic aircraft noise exposure on child health. Psychological Medicine 2001a; 31:1385-96.

Haines MM, Stansfeld SA, Job SFR, Berglund B, Head J. A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. International Journal of Epidemiology 2001b; 30:839-45.

Haines MM, Stansfeld SA, Job SFR, Berglund B, Head J. Chronic aircraft noise exposure, stress responses, mental health and cognitive performance in school children. Psychological Medicine 2001c; 31:265-77.

Haines MM, Stansfeld SA, Head J, Job RFS. Multi-level modeling of aircraft noise on performance tests in schools around Heathrow London airport. International Journal of Epidemiology and Community Health 2002; 56:139-44.

Hamernik RP, Patterson JH, Turrentine GA, Ahroon WA. The quantitative relation between sensory cell loss and hearing thresholds. Hear Res 1989; 38:199-212.

Hamernik RP, Qiu W, Davis B. Cochlear toughening, protection, and potentiation of noise-induced trauma by non-Gaussian noise. J Acoust Soc Am 2003; 113(2):969-76.

Han J, Li F, Zhao C, Zhang Z, Ni D. Study on distortion product otoacoustic emissions and expanded high frequency audiometry in noise exposure workers Lin Chuang Er Bi Yan Hou Ke Za Zhi 2003; 17(1):16-9.

Hannaford PC, Simpson JA, Bisset AF, Davis A, McKerrow W, Mills R. The prevalence of ear, nose and throat problems in the community: results from a national cross-sectional postal survey in Scotland. Fam Pract 2005; 22(3):227-33.

Hendrickx JJ, Huyghe JR, Demeester K, Topsakal V, Van Eyken E, Fransen E, et al. Familial aggregation of tinnitus: a European multicentre study. B-ENT 2007; 3(Sup7):51-60.

Harris KC, Hu B, Hangauer D, Henderson D. Prevention of noise-induced hearing loss with Src-PTK inhibitors. Hear Res 2005; 208(1-2):14-25.

Holgers KM, Pettersson B. Noise exposure and subjective hearing symptoms among school children in Sweden. Noise Health 2005; 7(27):27-37.

Haupt H, Scheibe F, Mazurek B. Therapeutic efficacy of magnesium in acoustic trauma in the guinea pig. ORL J Otorhinolaryngol Relat Spec 2003; 65(3):134-9.

Hearing assessment of classical orchestral musicians. Scand Audiol 2001; 30(1):13-23.

Heinonen-Guzejev M, Vuorinen HS, Mussalo-Rauhamaa H, Heikkilä K, Koskenvuo M, Kaprio J. Genetic component of noise sensitivity. Twin Res Hum Genet 2005; 8(3):245-9.

Henderson D, Subramaniam M, Boettcher FA. Individual susceptibility to noise-induced hearing loss: an old topic revisited. Ear & Hear 1993; 14 (3):152-168.

Henry KR. Hyperthermia exacerbates and hypothermia protects from noise-induced threshold elevation of the cochlear nerve envelope response in the C57BL/6J mouse. Hear Res 2003; 179(1-2):88-96.

Hirai A, Takata M, Mikawa M, Yasumoto K, Iida H, Sasayama S, Kagamimori S. Prolonged exposure to industrial noise causes hearing loss but not high blood pressure: a study of 2124 factory laborers in Japan. J Hypertens 1991; 9(11):1069-73.

Hodgetts WE, Rieger JM, Szarko R. The effect of listening environment and earphone style on preferred listening levels of normal hearing adults using an MP3 player. Ear and Hearing 2007; 28:290-297.

Holgers KM, Juul J. The suffering of tinnitus in childhood and adolescence. Int J Audiol 2006; 45(5):267-72.

Horner KC, Higueret D, Cazals Y. Efferent-mediated protection of the cochlear base from acoustic overexposure by low doses of lithium. Eur J Neurosci 1998; 10(4):1524-7.

Horner KC, Giraudet F, Lucciano M, Cazals Y. Sympathectomy improves the ear's resistance to acoustic trauma--could stress render the ear more sensitive? Eur J Neurosci 2001; 13(2):405-8.

Hsu WC, Wu HP, Liu TC. Objective assessment of auditory thresholds in noise-induced hearing loss using steady-state evoked potentials. Clin Otolaryngol Allied Sci 2003; 28(3):195-8.

Hygge S, Evans GW, Bullinger M. A prospective study of some effects of aircraft noise on cognitive performance in school children. Psychological Science 2002; 13:469-74.

Hygge S. Classroom experiments on the effects of different noise sources and sound levels on long term recall and recognition in children. Applied Cognitive Psychology 2003; 17:895-914.

Hygge S, Boman E, Enmarker I. The effects of road traffic noise and meaningful irrelevant speechon different memory systems. Scandinavian Journal of Psychology 2003; 44:13-21.

Ickes WK, Nader C. Noise-induced hearing loss and stress-prone behavior. Ear Hear 1982; 3(4):191-5.

IEC 651:1979. Sound level meters. Geneva: International Electrotechnical Commission; 1979.

IEC 61672-3:2006. Electroacoustics. Sound level meters, Part 3: Periodic tests. Geneva: International Electrotechnical Commission; 2006.

Irion H. Acute bilateral hearing loss during a pop concert: consideration for differential diagnosis. Adv Otorhinolaryngol 1981; 27:121-9.

Ising H, Hanel J, Pilgramm M, Babisch W, Lindthammer A. Risk of hearing loss caused by listening to music with head phones HNO 1994; 42(12):764-8. [Article in German]

Ising H, Babisch W, Handel J, Kruppa B, Pilgramm M. Empirical studies of music listening habits of adolescents. Optimizing sound threhold limits for cassette players and discoteques. HNO 1995; 43(4):244-9. [Article in German]

ISO 1999:1990 Acoustics. Determination of occupational noise exposure and estimation of noise-induced hearing impairment. Geneva: International Committee for Standardisation; 1990.

ISO 7029 Acoustics. Statistical distribution of hearing thresholds as a function of age. Geneva: International Organization for Standardisation; 2000.

ISO 226:2003 Acoustics. Normal equal-loudness-level contours. Edition: 2, Geneva: International Committee for Standardisation; 2003.

Jedrzejczak WW, Blinowska KJ, Konopka W. Time-frequency analysis of transiently evoked otoacoustic emissions of subjects exposed to noise. Hear Res 2005; 205(1-2):249-55.

Job A, Raynal M, et al. Hearing status of French youth aged from 18 to 24 years in 1997: a cross-sectional epidemiological study in the selection centres of the army in Vincennes and Lyon. Rev Epid Sante Pub 2000; 48(3):227-37.

Job A and Nottet JB. DPOAEs in young normal-hearing subjects with histories of otitis media: evidence of sub-clinical pairments. Hear Res 2002; 167(1-2):28-32.

Job A, Raynal M, et al. Susceptibility of tinnitus revealed at 2 kHz range by bilateral lower DPOAEs in normal hearing subjects with noise exposure. Audiol Neurootol 2007; 2(3):137-44.

Johnson AC, Morata TC, Lindblad AC, Nylén PR, Svensson EB, Krieg E, et al. Audiological findings in workers exposed to styrene alone or in concert with noise. Noise Health 2006; 8(30):45-57.

Johnson DW, Sherman RE, Aldridge J, Lorraine A. Extended high frequency hearing sensitivity. A normative threshold study in musicians. Ann Otol Rhinol Laryngol 1986; 95(2 Pt 1):196-202.

Johnson KR, Erway LC, Cook SA, Willott JF, Zheng QY. A major gene affecting age-related hearing loss in C57BL/6J mice. Hear Res 1997; 114 (1-2):83-92.

Juman S, Karmody CS, Simeon D. Hearing loss in steelband musicians. Otolaryngol Head Neck Surg 2004; 131(4):461-5.

Kähäri KR, Axelsson A, Hellström PA, Zachau G. Hearing assessment of classical orchestral musicians. Scand Audiol 2001; 30(1):13-23.

Katz AE, Gerstman HL, Sanderson RG Buchanan R Stereo earphones and hearing loss. N Engl J Med 1982; 307:1460-1461.

Kawada T, Koyama H, Suzuki S. Decrease of hearing acuity from use of portable headphones. Nippon Koshu Eisei Zasshi 1990; 37(1):39-43 [Article in Japanese].

Kaygusuz I, Oztürk A, Ustündağ B, Yalçin S. Role of free oxygen radicals in noise-related hearing impairment. Hear Res 2001; 162(1-2):43-7.

Kent SJ, von Gierke HE, Tolan GD. Analysis of the potential association between noise-induced hearing loss and cardiovascular disease in USAF aircrew members. Aviat Space Environ Med 1986; 57(4):348-61.

König O, Schaette R, Kempter R, Gross M. Course of hearing loss and occurrence of tinnitus. Hear Res 2006; 221(1-2):59-64.

Konings A, Van Laer L, Pawelczyk M, Carlsson PI, Bondeson ML, Rajkowska E, et al. Association between variations in CAT and noise-induced hearing loss in two independent noise-exposed populations. Hum Mol Genet 2007; 16(15):1872-83.

Konopka W, Zalewski P, Olszewski J, Olszewska-Ziaber A, Pietkiewicz P. Tinnitus suppression by electrical promontory stimulation (EPS) in patients with sensorineural hearing loss. Auris Nasus Larynx 2001; 28(1):35-40.

Konopka W, Olszewski J, Pietkiewicz P, Mielczarek M. Impulse noise influence on hearing. Pol Merkur Lekarski 2005a; 19(111):296-7.

Konopka W, Pawlaczyk-Luszczynska M, Sliwinska-Kowalska M, Grzanka A, Zalewski P. Effects of impulse noise on transiently evoked otoacoustic emission in soldiers. Int J Audiol 2005b; 44(1):3-7.

Konopka W, Olszewski J, Pietkiewicz P, Mielczarek M. Distortion product otoacoustic emissions before and after one year exposure to impulse noise. Otolaryngol Pol 2006; 60(2):243-7.

Kontosić I, Vukelić M, Grubisić-Greblo H. Noise as a risk factor for arterial hypertension in sailors. Arh Hig Rada Toksikol 1990; 41(2):187-99.

Kopke R, Bielefeld E, Liu J, Zheng J, Jackson R, Henderson D, et al. Prevention of impulse noise-induced hearing loss with antioxidants. Acta Otolaryngol 2005; 125(3):235-43.

Kowalska S, Sułkowski W. Tinnitus in noise-induced hearing impairment. Med Pr 2001; 52(5):305-13.

Kozel PJ, Davis RR, Krieg EF, Shull GE, Erway LC. Deficiency in plasma membrane calcium ATPase isoform 2 increases susceptibility to noise-induced hearing loss in mice. Hear Res 2002; 164(1-2):231-9.

Krähenbühl D, Arnold W, Fried R, Chüden H. Hearing loss caused by a walkman radio?. Laryngol Rhinol Otol (Stuttg) 1987; 66(5):286-9 [Article in German].

Kramer S, Dreisbach L, Lockwood J, Baldwin K, Kopke R, Scranton S, et al. Efficacy of the antioxidant N-acetylcysteine (NAC) in protecting ears exposed to loud music. J Am Acad Audiol 2006; 17(4):265-78.

Kryter KD. The Handbook of Hearing and the Effects of Noise: Physiology, Psychology, and Public Health. San Diego: Academic Press; 1994.

Kujawa SG, Liberman MC. Acceleration of age-related hearing loss by early noise exposure: evidence of a misspent youth. J Neurosci 2006; 26(7):2115-23.

Lacerda A, Leroux T, Morata T. Ototoxic effects of carbon monoxide exposure: a review. Pro Fono 2005; 17(3):403-12.

Lahoz Zamarro MT, Abenia Ingalaturre JM, Vallés Varela H, Herrero Blasco T. Interaction between hypertriglyceridemia and noise in human hearing. Acta Otorrinolaringol Esp 1992; 43(6):422-6.

Laitinen HM, Toppila EM, Olkinuora PS, Kuisma K. Sound exposure among the Finnish National Opera personnel. Appl Occup Environ Hyg 2003; 18(3):177-82.

Lamm K, Arnold W. Successful treatment of noise-induced cochlear ischemia, hypoxia, and hearing loss. Ann N Y Acad Sci 1999; 884:233-48.

Lang T, Fouriaud C, Degoulet P. Occupational exposure to noise, hearing loss and arterial hypertension. Rev Epidemiol Sante Publique 1986; 34(4-5):318-23.

Lapsley Miller JA, Marshall L, Heller LM. A longitudinal study of changes in evoked otoacoustic emissions and pure-tone thresholds as measured in a hearing conservation program. Int J Audiol 2004; 43(6):307-22.

Lee FS, Matthews LJ, Dubno JR, Mills JH. Longitudinal study of pure-tone thresholds in older persons. Ear Hear 2005; 26:1–11.

Lee LT. A study of the noise hazard to employees in local discotheques. Singapore Med J 1999; 40(9):571-4.

Lee PC, Senders CW, Gantz BJ, Otto SR. Transient sensorineural hearing loss after overuse of portable headphone cassette radios. Otolaryngol Head Neck Surg 1985; 93(5):622-5.

Lees RE, Roberts JH. Noise-induced hearing loss and blood pressure. Can Med Assoc J 1979; 120(9):1082-4.

LePage EL, Murray N. Latent cochlear damage in personal stereo users: a study based on click-evoked otoacoustic emissions. MJA 1998; 169:588-592.

Liebel J, Delb W, Andes C, Koch A. Detection of hearing loss in patrons of a discoteque using TEOAE and DPOAE. Laryngorhinootologie 1996; 75(5):259-64.

Liu J, Yu N, Han D, Yang W, Li X. Protective effect of indirect activator of calcium pump on noise-induced hearing loss. Zhonghua Er Bi Yan Hou Ke Za Zhi 2002; 37(6):425-7.

Liu YG, He YJ, Li DD, Zheng SX, Niu CM. Effects of sound preconditioning on hearing loss from low or middle-frequency noise exposure. Space Med Med Eng (Beijing) 2000; 13(5):313-7.

Loth D, Avan P, Menguy C, Teyssou M. Secondary auditory risks from listening to portable digital compact disc players. Bull Acad Natl Med 1992; 176(8):1245-52 [Article in French].

Luebke AE, Foster PK. Variation in inter-animal susceptibility to noise damage is associated with alpha 9 acetylcholine receptor subunit expression level. J Neurosci 2002; 22(10):4241-7.

Lutman ME, Davis AC. The distribution of hearing threshold levels in the general population aged 18-30 years. Audiology 1994; 33(6):327-50.

Lutman ME, Davis AC. Distributions of hearing threshold levels in populations exposed to noise. In: Scientific Basis of Noise-Induced Hearing Loss, Axelsson A, Borchgrevink H, Hamernik RP, Helstrom P, Henderson D, Salvi RJ, eds. New York: Thieme Medical Publishers, 1996: 378-396.

Lutman ME, Davis AC, Ferguson MA. Epidemiological evidence for the effectiveness of the Noise at Work Regulations. This report and the work it describes were funded by the Health and Safety Executive. Its contents, including any opinions and/or conclusions expressed, are those of the authors alone and do not necessarily reflect HSE policy (in press).

Lukas JS. Effects of aircraft noise on human sleep. American Industrial Hygiene Association Journal 1972; 33:298-303.

Lynch ED, Gu R, Pierce C, Kil J. Ebselen-mediated protection from single and repeated noise exposure in rat. Laryngoscope 2004; 114(2):333-7.

Lynch ED, Kil J. Compounds for the prevention and treatment of noise-induced hearing loss. Drug Discov Today 2005; 10(19):1291-8.

Magaud-Camus I, Floury MC, Vinck L and Waltisperger D. Le bruit au travail en 2003 : une nuisance qui touche trois salariés sur dix. Direction de l'animation, de la recherche, des études et des statistiques DARES, 2005; 25(3):1-6 [Article in French].

Maison SF, Luebke AE, Liberman MC, Zuo J. Efferent protection from acoustic injury is mediated via alpha9 nicotinic acetylcholine receptors on outer hair cells. J Neurosci 2002; 22(24):10838-46.

Makitie AA, Pirvola U, Pyykko I, Sakakibara H, Riihimaki V, Ylikoski J. The ototoxic interaction of styrene and noise. Hear Res 2003; 179(1-2):9-20.

Mansfield JD, Baghurst PA, Newton VE. Otoacoustic emissions in 28 young adults exposed to amplified music. Br J Audiol 1999; 33(4):211-22.

Markou K, Lalaki P, Barbetakis N, Tsalighopoulos MG, Daniilidis I. The efficacy of medication on tinnitus due to acute acoustic trauma. Scand Audiol Suppl. 2001; (52):180-4.

Maser A, Sorensen P, Kryter KD, Lukas J. Effects of intrusive sound on classroom behavior data from a successful lawsuit. Western Psychological Association. San Francisco 1978.

Maxwell LM, Evans GW. The effects of noise on preschool children's prereading skills. Journal of Environmental Psychology 2000; 20:91-7.

McBride D, Gill F, Proops D, Harrington M, Gardiner K, Attwell C. Noise and the classical musician. BMJ 1992; 305(6868):1561-3.

McBride DI, Williams S. Audiometric notch as a sign of noise induced hearing loss. Occup Environ Med 2001; 58(1):46-51.

Meecham EA, Hume KI. Tinnitus, attendance at night-clubs and social drug taking in students. Noise Health 2001; 3(10):53-62.

Metternich FU, Brusis T. Acute hearing loss and tinnitus caused by amplified recreational music. Laryngorhinootologie 1999; 78(11):614-9.

Meyer-Bisch C. Epidemiological evaluation of hearing damage related to strongly amplified music (personal cassette players, discotheques, rock concerts) – high definition audiometric survey on 1364 subjects. Audiology 1996; 35(3):121-42.

Meyer-Falcke A, Lanzendörfer A, Jansen G. Predictors for noise sensitivity: how to use them for a prognostic test. Schriftenr Ver Wasser Boden Lufthyg 1993; 88:223-37.

Mikuriya T, Sugahara K, Takemoto T, Tanaka K, Takeno K, Shimogori H, et al. Geranylgeranylacetone, a heat shock protein inducer, prevents acoustic injury in the guinea pig. Brain Res 2005; 1065(1-2):107-14.

Milković-Kraus S. Noise-induced hearing loss and blood pressure. Int Arch Occup Environ Health 1990; 62(3):259-60.

Minami SB, Yamashita D, Schacht J, Miller JM Calcineurin activation contributes to noise-induced hearing loss. J Neurosci Res 2004; 78(3):383-92.

Mizoue T, Miyamoto T, Shimizu T. Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers. Occup Environ Med 2003; 60(1):56-9.

Montgomery J, Fujikawa S. Hearing thresholds of students in the second, eighth and twelfth grades. Language, Speech and Hearing Services in School 1992; 23:61-63.

Morant Ventura A, Mata Peñuela JJ, Orts Alborch M, Postigo Madueño A, Marco Algarra J. The acoustic distortion products application in noise pathology. An Otorrinolaringol Ibero Am 2000; 27(4):341-52.

Morata TC. Chemical exposure as a risk factor for hearing loss. J Occup Environ Med 2003; 45(7):676-82.

Morata TC, Johnson AC, Nylen P, Svensson EB, Cheng J, Krieg EF, et al. Audiometric findings in workers exposed to low levels of styrene and noise. J Occup Environ Med 2002; 44(9):806-14.

Moselhi M, El-Sadik YM, El-Dakhakhny A. A six-year follow up study for evaluation of the 85 dB(A) safe criterion for noise exposure. Am Ind Hyg Assoc J 1979; 40(5):424-6.

Mostafapour SP, Lahargoue K, Gates GA. Noise-induced hearing loss in young adults: the role of personal listening devices and other sources of leisure noise. Laryngoscope 1998; 108(12):1832-9.

Mrena R, Savolainen S, Kuokkanen JT, Ylikoski J. Characteristics of tinnitus induced by acute acoustic trauma: a long-term follow-up. Audiol Neurootol 2002; 7(2):122-30.

Mrena R, Savolainen S, Pirvola U, Ylikoski J. Characteristics of acute acoustical trauma in the Finnish Defence Forces. Int J Audiol 2004; 43(3):177-81.

Mrena R, Ylikoski M, Mäkitie A, Pirvola U, Ylikoski J. Occupational noise-induced hearing loss reports and tinnitus in Finland. Acta Otolaryngol 2007; 127(7):729-35.

Müller W, Richartz G. Can the thesis of higher noise resistance in working women be maintained? Z Gesamte Hyg 1989; 35(8):505-7.

Murai K. Investigation of the 4,000-Hertz dip by detailed audiometry. Ann Otol Rhinol Laryngol 1997; 106(5):408-13.

Murata K, Weihe P, Araki S, Budtz-Jørgensen E, Grandjean P. Evoked potentials in Faroese children prenatally exposed to methylmercury. Neurotoxicol Teratol 1999; 21:471-2.

Nageris BI, Raveh E, Zilberberg M, Attias J. Asymmetry in noise-induced hearing loss: relevance of acoustic reflex and left or right handedness. Otol Neurotol 2007; 28(4):434-7.

Nakamoto Y, Iino Y, Kodera K. Temporal bone histopathology of noise-induced hearing loss. Nippon Jibiinkoka Gakkai Kaiho 2005; 108(2):172-81.

Nakaya M, Morita I, Okuno H, Takeda K, Horiuchi M. Clinical investigation of acute sensorineural hearing impairment due to rifles. Nippon Jibiinkoka Gakkai Kaiho 2002; 105(1):22-8.

Namysłowski G, Morawski K, Trybalska G, Urbaniec P. The latencies of the 2f1-f2 DPOAE measured using phase gradient method in young adults and in workers chronically exposed to noise. Otolaryngol Pol 2004; 58(1):131-8.

Narlawar UW, Surjuse BG, Thakre SS. Hypertension and hearing impairment in workers of iron and steel industry. Indian J Physiol Pharmacol 2006; 50(1):60-6.

National Institute for Occupational Safety and Health (NIOSH). Occupational noise and hearing 1968-72. NIOSH Pub 74-116; 1974.

Nelson DI, Nelson RY, Concha-Barrientos M, Fingerhut M. The global burden of occupational noise-induced hearing loss. Am J Ind Med 2005; 48(6):446-58.

Ni CH, Chen ZY, Zhou Y, Zhou JW, Pan JJ, Liu N, et al. Associations of blood pressure and arterial compliance with occupational noise exposure in female workers of textile mill. Chin Med J (Engl). 2007; 120(15):1309-13.

Nicolas-Puel C, Faulconbridge RL, Guitton M, Puel JL, Mondain M, Uziel A. Characteristics of tinnitus and etiology of associated hearing loss: a study of 123 patients. Int Tinnitus J 2002; 8(1):37-44.

Nicolas-Puel C, Akbaraly T, Lloyd R, Berr C, Uziel A, Rebillard G, et al. Characteristics of tinnitus in a population of 555 patients: specificities of tinnitus induced by noise trauma. Int Tinnitus J 2006; 12(1):64-70.

Niskar AS, Kieszak SM, Holmes A, Esteban E, Rubin C, Brody DJ. Prevalence of hearing loss among children 6 to 19 years of age: the Third National Health and Nutrition Examination Survey. JAMA 1998; 279(14):1071-5.

Niskar AS, Kieszak SM, Holmes AE, Esteban E, Rubin C, Brody DJ. Estimated prevalence of noise-induced hearing threshold shifts among children 6 to 19 years of age: the Third National Health and Nutrition Examination Survey, 1988-1994, United States. Pediatrics 2001; 108(1):40-3 [also see comment in: Pediatrics 2002; 109(5):987-8]

Niu X, Canlon B. Activation of tyrosine hydroxylase in the lateral efferent terminals by sound conditioning. Hear Res 2002; 174(1-2):124-32.

Niu X, Tahera Y, Canlon B. Protection against acoustic trauma by forward and backward sound conditioning. Audiol Neurotol 2004; 9:265-273.

Noben-Trauth K, Zheng QY, Johnson KR. Association of cadherin 23 with polygenic inheritance and genetic modification of sensorineural hearing loss. Nat Genet 2003; 35(1):21-23.

Norena AJ, Eggermont JJ. Enriched acoustic environment after noise trauma reduces hearing loss and prevents cortical map reorganization. J Neurosci 2005; 25(3):699-705.

Nottet JB, Moulin A, Brossard N, Suc B, Job A. Otoacoustic emissions and persistent tinnitus after acute acoustic trauma. Laryngoscope 2006; 116(6):970-5.

Ohinata Y, Miller JM, Schacht J. Protection from noise-induced lipid peroxidation and hair cell loss in the cochlea. Brain Res. 2003; 966(2):265-73.

Ohlemiller KK, Mc Fadden SL, Ding DL, Flood DG, Reaume AG, Hoffman EK, et al. Targeted deletion of the cytosolic Cu/Zn-superoxide dismutase gene (Sod1) increases susceptibility to noise-induced hearing loss. Audiol Neurootol 1999; 4(5):237-46.

Ohlemiller KK, Mc Fadden SL, Ding DL, Lear PM, Ho YS. Targeted mutation of the gene for cellular glutathione peroxidase (Gpx1) increases noise-induced hearing loss in mice. J Assoc Res Otolaryngol 2000; 1(3):243-54.

Öhrström E, Hadzibajramovic E, Holmes M, Svensson H. Effects of road traffic noise on sleep: Studies on children and adults. Journal of Environmental Psychology 2006; 26:116-26.

OSHA, Dept. of Labour. Occupational noise exposure; hearing conservation amendment. Federal Register 1983; 48 (46):9738-85.

Osman K, Pawlas K, Schütz A, Gazdzik M, Sokal JA, Vahter M. Lead exposure and hearing deficit in children in Katowice, Poland. Environ Res 1999; 80:1-8.

Ostri B, Eller N, Dahlin E, Skylv G. Hearing impairment in orchestral musicians. Scand Audiol 1989; 18(4):243-9.

Palmer KT, Griffin MJ, Syddall HE, Pannett B, Cooper C, Coggon D. Raynaud's phenomenon, vibration induced white finger, and difficulties in hearing. Occup Environ Med 2002a; 59(9):640-2.

Palmer KT, Griffin MJ, Syddall HE, Davis A, Pannett B, Coggon D. Occupational exposure to noise and the attributable burden of hearing difficulties in Great Britain. Occup Environ Med 2002b; 59(9):634-9.

Paz Z, Freeman S, Horowitz M, Sohmer H. Prior heat acclimation confers protection against noise-induced hearing loss. Audiol Neurootol 2004; 9(6):363-9.

Peng JH, Tao ZZ, Huang ZW. Risk of damage to hearing from personal listening devices in young adults. J Otolaryngol 2007; 36(3):181-5.

Persson BO, Svedberg A, Göthe CJ. Longitudinal changes in hearing ability among Swedish conscripts. Scand Audiol 1993; 22(2):141-3.

Plomp R, Duquesnoy AJ. Room acoustics for the aged. J Acoust Soc Am 1980; 68(6):1616-21.

Plontke S, Zenner Tübingen HP. Current aspects of hearing loss from occupational and leisure noise. In: Schultz-Coulon HJ, editors. Environmental and Occupational Health Disorders. Videel OHG, Germany 2004; 233-325.

Pourbakht A, Yamasoba T Ebselen attenuates cochlear damage caused by acoustic trauma. Hear Res 2003; 181(1-2):100-8.

Pouryaghoub G, Mehrdad R, Mohammadi S. Interaction of smoking and occupational noise exposure on hearing loss: a cross-sectional study. BMC Public Health 2007; 7:137.

Pyykkö I, Starck J, Färkkilä M, Hoikkala M, Korhonen O, Nurminen M. Hand-arm vibration in the aetiology of hearing loss in lumberjacks. Br J Ind Med 1981; 38(3):281-9.

Pyykkö I, Pekkarinen J, Starck J. Sensory-neural hearing loss during combined noise and vibration exposure. An analysis of risk factors. Int Arch Occup Environ Health 1987; 59(5):439-54.

Pyykkö I, Toppila E, Zou J, Kentala E. Individual susceptibility to noise-induced hearing los. Audiol Med 2007; 5:41-53.

Quaranta A, Scaringi A, Bartoli R, Margarito MA, Quaranta N. The effects of 'supra-physiological' vitamin B12 administration on temporary threshold shift. Int J Audiol 2004; 43(3):162-5.

Rabinowitz PM, Slade MD, Galusha D, Dixon-Ernst C, Cullen MR. Trends in the prevalence of hearing loss among young adults entering an industrial workforce 1985 to 2004. Ear Hear 2006; 27(4):369-75.

Rajan R. Cochlear outer-hair-cell efferents and complex-sound-induced hearing loss: protective and opposing effects. J Neurophysiol 2001; 86(6):3073-6.

Rajan R. Crossed and uncrossed olivocochlear pathways exacerbate temporary shifts in hearing sensitivity after narrow band sound trauma in normal ears of animals with unilateral hearing impairment. Audiol Neurootol 2003; 8(5):250-62.

Rask-Andersen H, Ekvall L, Scholtz A, Schrott-Fischer A. Structural/audiometric correlations in a human inner ear with noise-induced hearing loss. Hear Res 2000; 141(1-2):129-39.

Rice CFG, Breslin M, Roper RG. Sound levels from personal cassette players. Br J Audiol 1987a; 21:273-278.

Rice CG, Rossi G, Olina M. Damage risk from personal cassette players. Br J Audiol 1987b; 21:279-88.

Rice DC, Gilbert SG. Exposure to methyl mercury from birth to adulthood impairs high-frequency hearing in monkeys. Toxicol Appl Pharmacol 1992; 115(1):6–10.

Robinson DW. Noise exposure and hearing: A new look at the experimental data. Health and Safety Executive Contract Research Report No. 1., 1987.

Rosanowski F, Eysholdt U, Hoppe U. Influence of leisure-time noise on outer hair cell activity in medical students. Int Arch Occup Environ Health 2006; 80(1):25-31.

Rosenhall U. The influence of ageing on noise-induced hearing loss. Noise Health 2003; 5(20):47-53.

Ruel J, Wang J, Pujol R, Hameg A, Dib M, Puel JL Neuroprotective effect of riluzole in acute noise-induced hearing loss. Neuroreport 2005; 16(10):1087-90.

Sadhra S, Jackson CA, Ryder T, Brown MJ. Noise exposure and hearing loss among student employees working in university entertainment venues. Ann Occup Hyg 2002; 46(5):455-63.

Santana VS, Barberino JL. Occupational noise exposure and hypertension. Rev Saude Publica 1995; 29(6):478-87.

Scheibe F, Haupt H, Mazurek B, Konig O. Therapeutic effect of magnesium on noise-induced hearing loss. Noise Health 2001; 3(11):79-84.

Scheibe F, Haupt H, Ising H, Cherny L. Therapeutic effect of parenteral magnesium on noise-induced hearing loss in the guinea pig. Magnes Res 2002; 15(1-2):27-36.

Schmuziger N, Patscheke J, Probst R. An assessment of threshold shifts in nonprofessional pop/rock musicians using conventional and extended high-frequency audiometry. Ear Hear 2007; 28(5):643-8.

Schmuziger N, Patscheke J, Probst R. Hearing in nonprofessional pop/rock musicians. Ear Hear 2006; 27(4):321-30.

Schmuzigert N, Fostiropoulos K, Probst R. Long-term assessment of auditory changes resulting from a single noise exposure associated with non-occupational activities. Int J Audiol 2006; 45(1):46-54.

Seixas NS, Kujawa SG, Norton S, Sheppard L, Neitzel R, Slee A. Predictors of hearing threshold levels and distortion product otoacoustic emissions among noise exposed young adults. Occup Environ Med 2004; 61(11):899-907.

Sendowski I, Abaamrane L, Raffin F, Cros A, Clarencon D. Therapeutic efficacy of intra-cochlear administration of methylprednisolone after acoustic trauma caused by gunshot noise in guinea pigs. Hear Res 2006a; 221(1-2):119-27.

Sendowski I, Raffin F, Braillon-Cros A. Therapeutic efficacy of magnesium after acoustic trauma caused by gunshot noise in guinea pigs. Acta Otolaryngol 2006b; 126(2):122-9.

Serra MR, Biassoni EC, Richter U, Minoldo G, Abraham S, Carignani JA, et al. Recreational noise exposure and its effects on the hearing of adolescents. Part I: An interdisciplinary long term study. Int J Audiol 2005; 44:65-73.

Shupak A, Tal D, Sharoni Z, Oren M, Ravid A, Pratt H. Otoacoustic emissions in early noise-induced hearing loss. Otol Neurotol 2007; 28(6):745-52.

Siemens J, Kazmierczak P, Reynolds A, Sticker M, Littlewood-Evans A, Muller U. The Usher syndrome proteins cadherin 23 and harmonin form a complex by means of PDZ-domain interactions. Proc Natl Acad Sci USA 2002; 99:14946-51.

Silva LF, Mendes R. Combined exposure to noise and vibration and its effects on workers' hearing. Rev Saude Publica 2005; 39(1):9-17.

Sisto R, Chelotti S, Moriconi L, Pellegrini S, Citroni A, Monechi V, et al. Otoacoustic emission sensitivity to low levels of noise-induced hearing loss. J Acoust Soc Am 2007; 122(1):387-401.

Skrainar SF, Royster LH, Berger EH, Pearson RG. The contribution of personal radios to the noise exposure of employees at one industrial facility. Am Ind Hyg Assoc J 1987; 48(4):390-5.

Sliwinska-Kowalska M, Prasher D, Rodrigues CA, Zamysłowska-Szmytke E, Campo P, Henderson D et al. Ototoxicity of organic solvents - from scientific evidence to health policy. Int J Occup Med Environ Health 2007; 20(2):215-22.

Sliwinska-Kowalska M, Dudarewicz A, Kotylo P, Zamyslowska-Szmytke E, Pawlaczyk-Luszczynska M, Gajda-Szadkowska A. Individual susceptibility to noise-induced hearing loss: choosing an optimal method of retrospective classification of workers into noise-susceptible and noise-resistant groups. Int J Occup Med Environ Health 2006; 19(4):235-45.

Sliwinska-Kowalska M, Zamyslowska-Szmytke E, Szymczak W, Kotylo P, Fiszer M, Wesolowski W, et al. Exacerbation of noise-induced hearing loss by co-exposure to workplace chemicals. Environ Toxicol Pharmacol 2005; 19:547-553.

Sliwinska-Kowalska M, Zamyslowska-Szmytke E, Szymczak W, Kotylo P, Fiszer M, Wesolowski W, et al. Effects of coexposure to noise and mixture of organic solvents on hearing in dockyard workers. J Occup Environ Med 2004; 46(1):30-8.

Sliwinska-Kowalska M, Zamyslowska-Szmytke E, Szymczak W, Kotylo P, Fiszer M, Wesolowski W, et al. Ototoxic effects of occupational exposure to styrene and co-exposure to styrene and noise. J Occup Environ Med 2003; 45(1):15-24.

Sliwinska-Kowalska M, Zamyslowska-Szmytke E, Szymczak W, Kotylo P, Fiszer M, Dudarewicz A, et al. Hearing loss among workers exposed to moderate concentrations of solvents. Scand J Work Environ Health 2001; 27(5):335-42.

Sliwinska-Kowalska M, Kotylo P. Otoacoustic emissions in industrial hearing loss assessment. Noise Health 2001; 3(12):75-84.

Smith AW. The World Health Organisation and the prevention of deafness and hearing impairment caused by noise. Noise Health 1998; 1(1):6-12.

Smith PA, Davis A, Ferguson M, Lutman ME. The prevalence and type of social noise exposure in young adults in England. Noise Health 2000; 2(6):41-56.

Smoorenburg GF, de Laat JA, Plomp R. The effect of noise-induced hearing loss on the intelligibility of speech in noise. Scand Audiol Suppl 1982; 16:123-33.

Solerte SB, Fioravanti M, Ferrari E, Vittadini G, Battaglia A, Candura F. Changes in the blood rheology and an audiometric deficit in a group of patients exposed to occupational noise pollution. G Ital Med Lav 1991; 13(1-6):55-60.

Souto Souza NS, Carvalho FM, de Cássia Pereira Fernandes R. Arterial hypertension among oil-drilling workers exposed to noise. Cad Saude Publica 2001; 17(6):1481-8.

Stansfeld SA, Berglund B, Clark C, Lopez-Barrio I, Fischer P, Öhrström E, et al. Aircraft and road traffic noise and children's cognition and health: A cross-sectional study. Lancet 2005; 365:1942-9.

Starck J, Toppila E, Pyykkö I. Smoking as a risk factor in sensory neural hearing loss among workers exposed to occupational noise. Acta Otolaryngol 1999; 119(3):302-5.

Steeneken HJM and Houtgast T. A physical method for measuring speech transmission quality. Journal of Acoustical Society of America 1980; 67:318-326.

Steurer M, Simak S, Denk DM, Kautzky M. Does choir singing cause noise-induced hearing loss? Audiology 1998; 37(1):38-51.

Størmer CC, Stenklev NC. Rock music and hearing disorders. Tidsskr Nor Laegeforen 2007; 127(7):874-7.

Strauss P, Quante M, Strahl M, Averhage H, Bitzer M. Is hearing of the students damaged by environmental noise in their leisure time? (author's transl). Laryngol Rhinol Otol (Stuttg) 1977; 56(10):868-71 [Article in German].

Suckfuell M, Canis M, Strieth S, Scherer H, Haisch A. Intratympanic treatment of acute acoustic trauma with a cell-permeable JNK ligand: a prospective randomized phase I/II study. Acta Otolaryngol 2007; 127(9):938-42.

Sugahara K, Shimogori H, Okuda T, Takemoto T, Hashimoto M, Yamashita H. Cochlear administration of adenosine triphosphate facilitates recovery from acoustic trauma (temporary threshold shift). ORL J Otorhinolaryngol Relat Spec 2004; 66(2):80-4.

Sumer 2005-25.3 - Le bruit au travail en 2003 : Une nuisance qui touche trois salariés sur dix. http://www.travail.gouv.fr/article.php3?id_article=1441

Tahera Y, Meltser I, Johansson P, Bian Z, Stierna P, Hansson AC, Canlon B. NF-kappaB mediated glucocorticoid response in the inner ear after acoustic trauma. J Neurosci Res 2006; 83(6):1066-76.

Takemoto T, Sugahara K, Okuda T, Shimogori H, Yamashita H. The clinical free radical scavenger, edaravone, protects cochlear hair cells from acoustic trauma. Eur J Pharmacol 2004; 487(1-3):113-6.

Takemura K, Komeda M, Yagi M, Himeno C, Izumikawa M, Doi T, et al. Direct inner ear infusion of dexamethasone attenuates noise-induced trauma in guinea pig. Hear Res 2004; 196(1-2):58-68.

Talbott EO, Findlay RC, Kuller LH, Lenkner LA, Matthews KA, Day RD, et al. Noise-induced hearing loss: a possible marker for high blood pressure in older noise-exposed populations. J Occup Med 1990; 32(8):690-7.

Tambs K, Hoffman HJ, Borchgrevink HM, Holmen J, Samuelsen SO. Hearing loss induced by noise, ear infections, and head injuries: results from the Nord-Trøndelag Hearing Loss Study. Int J Audiol 2003; 42(2):89-105.

Tambs K, Hoffman HJ, Borchgrevink HM, Holmen J, Engdahl B. Hearing loss induced by occupational and impulse noise: results on threshold shifts by frequencies, age and gender from the Nord-Trøndelag Hearing Loss Study. Int J Audiol 2006; 45(5):309-17.

Tanaka K, Takemoto T, Sugahara K, Okuda T, Mikuriya T, Takeno K, et al. Post-exposure administration of edaravone attenuates noise-induced hearing loss. Eur J Pharmacol 2005; 522(1-3):116-21.

Tarter SK, Robins TG. Chronic noise exposure, high-frequency hearing loss, and hypertension among automotive assembly workers. J Occup Med 1990; 32(8):685-9.

Teszler CB, Ben-David J, Podoshin L, Sabo E. Sonovestibular symptoms evaluated by computed dynamic posturography. Int Tinnitus J 2000; 6(2):140-53.

Tománek R.The effect of the parasympathetic autonomic nervous system on auditory fatigue. Physiol Bohemoslov 1975; 24(3):283-6.

Toppila E, Pyykkö I, Starck J. Age and noise-induced hearing loss. Scand Audiol 2001; 30(4):236-44.

Turunen-Rise I, Flottorp G, Tvete O. Personal cassette players ("Walkman"). Do they cause noise-induced hearing loss? Scand Audiol 1991; 20:239-244.

Uchida Y, Nakashimat T, Ando F, Niino N, Shimokata H. Is there a relevant effect of noise and smoking on hearing? A population-based aging study. Int J Audiol 2005; 44(2):86-91.

Widén SE, Erlandsson SI. Self-reported tinnitus and noise sensitivity among adolescents in Sweden. Noise Health 2004; 7(25):29-40.

Van der Laan FL. Noise exposure and its effect on the labyrinth, Part I Int Tinnitus J 2001; 7(2):97-100.

Van Eyken E, Van Laer L, Fransen E, Topsakal V, Hendrickx JJ, Demeester K, et al. The Contribution of GJB2 (Connexin 26) 35delG to Age-Related Hearing Impairment and Noise-Induced Hearing Loss. Otol Neurotol 2007; 28(7):970-5.

Van Laer L, Carlsson PI, Ottschytsch N, Bondeson ML, Konings A, Vandevelde A, et al. The contribution of genes involved in potassium-recycling in the inner ear to noise-induced hearing loss. Hum Mutat 2006; 27(8):786-95.

Verbeek JH, van Dijk FJ, de Vries FF. Non-auditory effects of noise in industry. IV. A field study on industrial noise and blood pressure. Int Arch Occup Environ Health 1987; 59(1):51-4.

Veuillet E, Martin V, Suc B, Vesson JF, Morgon A, Collet L. Otoacoustic emissions and medial olivocochlear suppression during auditory recovery from acoustic trauma in humans. Acta Otolaryngol 2001; 121(2):278-83.

Wagner W, Heppelmann G, Kuehn M, Tisch M, Vonthein R, Zenner HP. Olivocochlear activity and temporary threshold shift-susceptibility in humans. Laryngoscope 2005; 115(11):2021-8.

Walter O, Wienke A. Hearing defects after a rock concert. OLG Koblenz, Urteil vom 13. 9. 2001-5 Laryngorhinootologie 2002; 81(10):739-40.

Wang H, Jiang Z, Duan C, Wang Z, Jiang Z, Feng B, et al. Study on the working noise in BYPC and the effects caused by working noise on the workers' vestibular and auditory function. Lin Chuang Er Bi Yan Hou Ke Za Zhi 2001; 15(4):176-8.

Wang H, Jiang C, Zhou F, Wu P, Zhang S, Liu W, et al. A clinical study about occupational noise-induced hearing loss measured and diagnosed with transient evoked otoacoustic emissions. Lin Chuang Er Bi Yan Hou Ke Za Zhi 2004; 18(4):209-11.

Wang J, Dib M, Lenoir M, Vago P, Eybalin M, Hameg A, et al. Riluzole rescues cochlear sensory cells from acoustic trauma in the guinea-pig. Neuroscience 2002; 111(3):635-48.

Wang J, Van De Water TR, Bonny C, de Ribaupierre F, Puel JL, Zine A. A peptide inhibitor of c-Jun N-terminal kinase protects against both aminoglycoside and acoustic trauma-induced auditory hair cell death and hearing loss. J Neurosci 2003; 23(24):8596-607.

Wang Y, Yang B, Li Y, Hou L, Hu Y, Han Y. Application of extended high frequency audiometry in the early diagnosis of noise--induced hearing loss. Zhonghua Er Bi Yan Hou Ke Za Zhi 2000; 35(1):26-8.

Wang Y, Liberman MC. Restraint stress and protection from acoustic injury in mice. Hear Res 2002; 165(1-2):96-102.

Welleschik B. The effect of the noise level on the occupational hearing loss. Observations carried cut in 25,544 industrial workers (author's transl). Laryngol Rhinol Otol (Stuttg) 1979; 58(11):832-41 [Article in German].

West PD, Evans EF. Early detection of hearing damage in young listeners resulting from exposure to amplified music. Br J Audiol 1990; 24(2):89-103.

Westmore GA, Eversden ID. Noise-induced hearing loss and orchestral musicians. Arch Otolaryngol 1981; 107(12):761-4.

World Health Organisation. Prevention of noise-induced hearing loss: report of an informal consultation held at the World Health Organization, Geneva, on 28-30 October 1997.

World Health Organization. Occupational noise : assessing the burden of disease from work-related hearing impairment at national and local levels. Environmental burden of disease Series 2004, Number 9, Geneva.

World Health Organization Grades of hearing impairment: http://www.who.int/pbd/deafness/hearing_impairment_grades/en/index.html [accessed on 5 June 2008]

Wild DC, Brewster MJ, Banerjee AR. Noise-induced hearing loss is exacerbated by long-term smoking. Clin Otolaryngol 2005; 30(6):517-20.

Williams W. Noise exposure levels from personal stereo use. Int J Audiol 2005; 44(4):231-6.

Willson GN, Chung DY, Gannon RP, Roberts M, Mason K. Is a healthier person less susceptible to noise-induced hearing loss? J Occup Med 1979; 21(9):627-30.

Winiarski M, Kantor I, Smereka J, Jurkiewicz D. Effectiveness of pharmacologic therapy combined with hyperbaric oxygen in sensorineural hearing loss following acute acoustic trauma. Preliminary report Pol Merkur Lekarski 2005; 19(111):348-50.

Wong TW, Van Hasselt CA, Tang LS, Yiu PC. The use of personal cassette players among youths and its effects on hearing. Public health 1990; 104(5): 327-30.

Xiong M, Su J, Wang J, He Q, Deng H, You J. The experimental study of the anti-damage effect of iminoethyl-lysine on noise-induced cochlea damage in guinea pig. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi 2002; 20(5):356-8.

Yamashita D, Jiang HY, Le Prell CG, Schacht J, Miller JM. Post-exposure treatment attenuates noise-induced hearing loss. Neuroscience 2005; 134(2):633-42.

Yang M, Zheng J, Yang Q, Yao H, Chen Y, Tan H, et al. Frequency-specific association of antibodies against heat shock proteins 60 and 70 with noise-induced hearing loss in Chinese workers. Cell Stress Chaperones 2004; 9(2):207-13.

Yang M, Tan H, Zheng JR, Jiang CZ. Relationship between GSTM1 and GSTT1 gene polymorphisms and noise induced hearing loss in Chinese workers. Wei Sheng Yan Jiu 2005; 34(6):647-9.

Yang M, Tan H, Zheng JR, Wang F, Jiang C, He M, et al. Association of cadherin CDH23 gene polymorphisms with noise induced hearing loss in Chinese workers. Wei Sheng Yan Jiu 2006; 35(1):19-22.

Yang W, Hu B, Guo W, Hu Y, Wang P, Jiang S. Synergetic protective effects of glial cell line-derived neurotrophic factor combined with neurotrophin-3 in F-actin on hair cell after noise trauma. Zhonghua Er Bi Yan Hou Ke Za Zhi 2001; 36(5):342-5.

Yu N, Li X, Hu B. The effects of salicylate on noise-induced hearing loss in the guinea pig. Zhonghua Er Bi Yan Hou Ke Za Zhi 1999; 34(6):344-6.

Yuan J, Yang M, Yao H, Zheng J, Yang Q, Chen S, et al. Plasma antibodies to heat shock protein 60 and heat shock protein 70 are associated with increased risk of electrocardiograph abnormalities in automobile workers exposed to noise. Cell Stress Chaperones 2005; 10(2):126-35.

Zhai SQ, Cheng JC, Wang JL, Yang WY, Gu R, Jiang SC. Protective effect of basic fibroblast growth factor on auditory hair cells after noise exposure. Acta Otolaryngol 2002; 122(4):370-3.

Zhai SQ, Wang DJ, Wang JL, Han DY, Yang WY. Basic fibroblast growth factor protects auditory neurons and hair cells from glutamate neurotoxicity and noise exposure. Acta Otolaryngol 2004; 124(2):124-9.

Zhang QR, Lei ZX, Shi JH, Wang KX, Huang J, Wu HF, et al. Detection of distortion-product otoacoustic emissions in well-drilling workers. Lin Chuang Er Bi Yan Hou Ke Za Zhi 2000; 14(2):78-80.

Zhang Y, Zhang X, Zhu W, Zheng X, Deng X. Distortion product of otoacoustic emissions as a sensitive indicator of hearing loss in pilots. Aviat Space Environ Med 2004; 75(1):46-8.

Zhou Y, Zhai S, Yang W The protective effects of ciliary neurotrophic factor on inner ear damage induced by intensive impulse noise. Zhonghua Er Bi Yan Hou Ke Za Zhi 1999; 34(3):150-3.

Zuskin E, Schachter EN, Kolcić I, Polasek O, Mustajbegović J, Arumugam U. Health problems in musicians--a review. Acta Dermatovenerol Croat 2005; 13(4):247-51.