

Consultation on Regulations for Noise-Induced Hearing Loss

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I am responding:

as an individual

on behalf of an organisation

Organisation:

Please highlight to describe who you represent:

Hearing Advocacy Group

Person with hearing loss

Employee

Small business (up to 10 staff)

Medium business (11 to 50 staff)

Large business (over 50 staff)

Business representative organisation/industry group

Trade Union

Training Organisation

Consultant

Other (please describe), Hearing Researcher and Educator

Hearing service provider

Audiology vendor

This submission questions the **fundamental basis** for the proposed introduction of these new regulations. There is low scientific validity to the proposal to retrospectively assign a percentage to the amount of hearing loss from noise exposure. There is no appropriate discriminative diagnostic test to enable the contribution from noise to be separately identified from all other causes, in most cases. Additionally, there has never been any consultation on the concept of part charges and apportionment of noise and age-related hearing loss and thus the evidence to support the introduction of this into New Zealand has not been scrutinised and debated. It is essential to have the open, robust discussion on this issue and the evaluation of the evidence base for the proposal. Indeed, over the last 12mths, ACC has led a process to look at the assessment of NIHL and to develop a workbook for ENT consultants making these decisions. The results of this process have not yet been published and discussed. Furthermore the view that the increasing number of claims to ACC reflects an underlying increase in the incidence of NIHL has not been established and thus the basis for the introduction of these regulations is not yet well founded. There is a substantial amount of ACC and Department of Labour supported research to understand more about the epidemiology, prevention and assessment of NIHL in New Zealand currently underway and nearing completion, which may shed light on the epidemiology and current prevention strategies for NIHL in New Zealand and any changes in ACC regulations should wait until completion and scrutiny of this research.

1. *There is no accurate method to separate the injury and age component for an individual*

The idea of separating the injury-related (noise mostly) component from any other cause may on the face of it appear practical and simple but it is not a fair or equitable approach because there is no established formula or diagnostic test that provides definitive proof of the injury contribution **for any individual**. An examination of the research literature reveals serious doubts about the accuracy of methods available to assign a proportion of the hearing loss to noise-induced injury and to a contribution of rehabilitation. This can only be briefly described here.

1.1 Hearing loss is the manifestation of injury to the auditory system, which may be in the outer, middle or inner ears or may occur because of trauma or degenerative changes in the brain. Outer and middle ear disease can often be observed clinically by ear examination and can generally be diagnosed by specialist clinical tests.

1.2 Inner ear disease and the injury, such as that which occurs with noise, cannot be seen, however. This is because the inner ear is located within the hard bone of the skull and there is currently no imaging technique that has been developed that enables the soft tissues contained within it to be observed physically. The best available window into the inner ear is a combination of physiological and behavioural tests (hearing tests) which can help to determine which cells are affected and their location in the ear, but not the cause of the injury. Aetiology can be speculated by the combination of tests, clinical examination and history but separating and accurately apportioning the contribution from multiple causes, such as noise and age is not possible. This can only be estimated subjectively from a combination of population-based tables, clinical history, and audiometric profile. There is a large literature on attempts to develop diagnostic procedures (eg otoacoustic emissions) or to use evidence of the rate of change of hearing loss and different algorithms to describe the audiometric profile but these have been unsuccessful or prone to considerable inter-operator variation and error ^{1,2}.

1.3 **Noise exposure** causes hearing loss through an acute or gradual process depending on the type of exposure. The noise targets the sensory cells and the nerve fibres, and most likely other structures in the ear causing their slow degeneration and death. If the noise exposure an individual experiences, such as through work, is reasonably constant over time then the level of hearing loss increases steadily over a 10 year period to reach a maximum or asymptote.

1.4 **Most importantly, the level of hearing loss for identical noise exposure varies greatly across individuals (Figure 1). For example, according to the ISO1999-1990³ the median hearing loss at a frequency of 4kHz following exposure to 100dBA for 8hrs each day for 40 years is 41 dB with a range (10 and 90 percentiles) of 26dB.** This variation is likely due to a variety of biological and behavioural factors⁴. There are genetic variations that lead to greater or lesser susceptibility to noise; females are less susceptible and there may be very large individual differences in the effectiveness of hearing protection and possibly interaction with drugs or ototoxic chemicals. **There is no test to predict**

¹ McBride, D, Williams, S Characteristics of the audiometric notch as a clinical sign of noise exposure. *Scand Audiol* 2001;30:106– 111

² Nondahl, D M, et al., Notched Audiograms and Noise Exposure History in Older Adults, *Ear & Hearing* 2009;30:696–703

³ ISO 1999-1990. Acoustics – Determination of occupational noise exposure and estimation of noise-induced hearing impairment

⁴ Pykko I., et al., Individual susceptibility to noise-induced hearing loss. *Audiological Medicine*. 2007; 5: 41_53

whether an individual is more or less sensitive to injury from noise despite promising avenues in animal studies⁵.

1.5 **The Aging process** is a major contributor to the development of hearing loss (presbycusis). The effect increases with age, accelerating as we pass about 50-60 yrs of age. The hearing loss from age predominately affects the inner ear where it mostly is associated with the loss of sensory cells and nerve fibres in the (basal) part of the inner ear that detects high frequency sounds⁶, overlapping with the same region mostly affected by noise.

1.6 Unlike the pattern of the loss from noise, the age-related hearing loss increases steadily with time. **But same as the hearing impairment from noise, there is considerable variability in the level of hearing loss among individuals at any specific age (Figure 1). For example, according to the ISO1999-1990⁷ the median hearing loss at a frequency of 4kHz (the same frequency region affected by noise) at 60 years of age is 28dB with a range (10 and 90 percentiles) of 48dB** There are well established genetic factors that contribute to this variability and other contributors such as cardiovascular disease, diabetes, and smoking, possibly. Socioeconomic factors also influence both normal thresholds and the level of hearing loss with age^{8,9}. The prevalence of hearing loss with age increases from about 22% at 50yrs to about 66% at 70 yrs¹⁰. Cognitive decline may also contribute to the hearing loss but in much later years.

1.7 **Other diseases** affect the inner ear and lead to hearing loss. For example Meniere's disease which affects the balance and hearing organs, disorders of the bony capsule of the inner ear and genetic abnormalities leading to progressive decline of hearing function. The prevalence of these conditions is very low. For example hereditary progressive hearing loss affects approximately 0.2%¹¹ and Meniere's disease less than 0.1% of the population¹². This contrasts with the various estimates for age (8-12%) and noise (0.7-3%).

⁵ Maison, SF and Liberman, MC. Predicting Vulnerability to Acoustic Injury with a Noninvasive Assay of Olivocochlear Reflex Strength *The Journal of Neuroscience*, June 15, 2000, 20(12):4701-4707

⁶ Schuknecht HF, Pathology of the Ear. 1974. Harvard University Press.

⁷ ISO 1999-1990. Acoustics – Determination of occupational noise exposure and estimation of noise-induced hearing impairment

⁸ Helvik AS et al., Socioeconomic inequalities in hearing loss in a healthy population sample: The HUNT Study. *Am J Public Health*, 2009, 99: 1376-1378

⁹ Hasson, D et al., Prevalence and characteristics of hearing problems in a working and non-working Swedish population, *J Epidemiol Community Health* 2010;64:453-460

¹⁰ Cruickshanks, KJ., et al. Prevalence of hearing loss in older adults in Beaver Dam, Wisconsin. The Epidemiology of Hearing Loss Study. *Am J Epidemiol*. 1998;148:879-86.

¹¹ Sakihara Y., et al., Prevalence of hereditary hearing impairment in adults. *Scand Audiol*, 1999, 28, 29-46

¹² Stahle J., et al., Incidence of Menieres disease. *Arch Otolaryngol.*, 1978, 104, 99-102

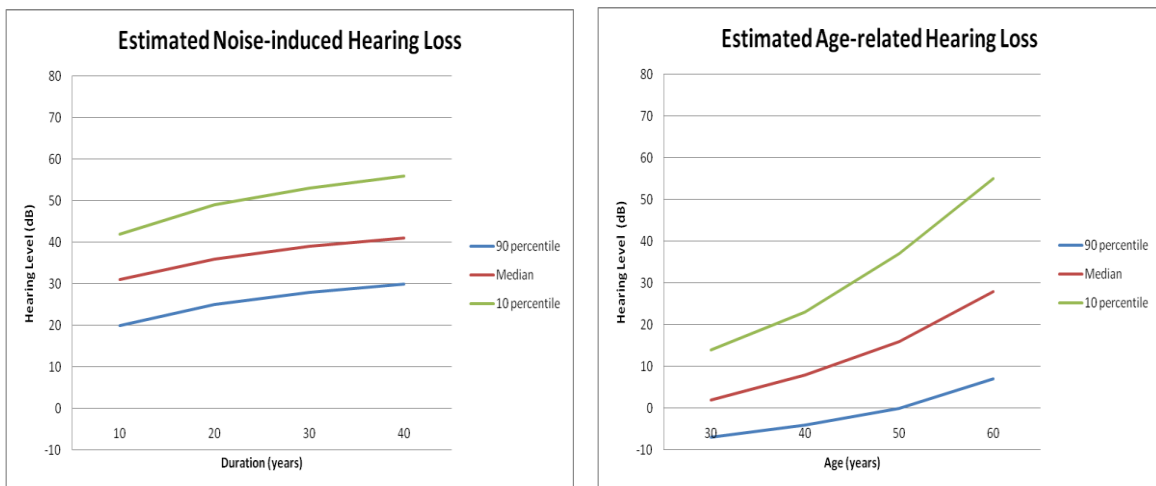


Figure 1. Estimated Median and 10th and 90th percentiles of hearing loss from age (screened population) and noise exposure (100dBA 8hrs/day) for the population according to ISO1999-1990⁷ showing the very large variance across the population.

1.8 There is an assumption that the effects of noise and aging are additive rather than interactive or synergistic. Thus the hearing loss is mostly regarded as the sum of the age-related and the noise-induced components. It has been suggested that the noise and age in humans may be additive up to 60 years of age¹³ but not beyond that. The ISO 1999-1990 standard allows for the nonlinear interaction of noise and aging which is more consequential above about 40dB of hearing loss. However these assumptions have never been fully tested.

1.9 Animal studies shed some light on this and particularly indicate that the additive relationship may differ whether the ears are more or less susceptible to noise¹⁴. Recent studies show that the exposure of young animals to noise makes them very susceptible to the effects of aging¹⁵. In other words, animals exposed to noise when young and had small hearing losses as a consequence developed substantially greater hearing losses than their non-noise exposed cousins as they aged. Furthermore animal models where the animals age more quickly show greater susceptibility to noise than those strains with a different genetic background which do not age.

¹³ Rosenhall U. The influence of ageing on noise-induced hearing loss. *Noise Health*. 2003; 5: 47_53.

¹⁴ Ohlemiller, KK. Contributions of mouse models to understanding of age- and noise-related hearing loss. *Brain Res* 2006;1091:89-102

¹⁵ Kujawa SG and Liberman MC, Acceleration of Age-Related Hearing Loss by Early Noise Exposure: Evidence of a Misspent Youth. *The Journal of Neuroscience*, 2006, 26:2115-2123

1.10 **Whilst all these interactions have yet to be proven in humans the data clearly point to a more complex interactive relationship between noise and age that cannot be explained solely by an additive and linear association.**

1.11 The use of population data to describe the effects of age or noise on the individual must to be treated with considerable caution. **Because there is no clinical discriminative test, clinical history and tables based on population data are used to assess the individual proportion of hearing loss with age and noise** (despite the fact that it is clearly stated in ISO 1999-990 Standard that these population data cannot be applied to individuals¹⁶). Whilst scales and tables, such as the NAL scale may be used to estimate **the degree of handicap** associated with a particular level of hearing loss these cannot accurately define the contribution of any particular cause for an individual because there is such a large variance in the range of hearing loss with age and noise it is not possible, without serial monitoring, to know where any individual may sit within the range (Figure 1).

1.12 An outcome associated with the use of median population data to assess the individual that highlights the inaccuracy of this method is that for some individuals there may be more actual hearing loss than can be accounted for by the sum of the estimated age and noise contributions. This residual component is then considered to be idiopathic or unexplained hearing loss. Dobie (2008) comes to the reasonable conclusion that given that other inner ear conditions are of such low prevalence (see 1.9) it is highly probable that the residual or idiopathic hearing loss reflects advanced aging and/or greater susceptibility to noise.

2. *Incidence of Hearing Loss*

The proposed changes seem to be predicated on an assumption that the number and hence cost of claims to ACC will continue to rise at the same rate and that this reflects the increasing incidence of NIHL. This has not been established. In fact because of this concern ACC and the Health Research Council established and funded several research projects at the University of Auckland and Massey University to understand the epidemiology of hearing loss and its prevention in New Zealand. These studies are due to report at the end of 2010 and any changes to the way ACC assesses and funds noise-induced hearing loss should wait until the results of their own commissioned research are reported.

¹⁶ ISO 1999-1990. Acoustics – Determination of occupational noise exposure and estimation of noise-induced hearing impairment

2.1 The prevalence of NIHL is not at all well established. Estimates vary from 7% to about 50% of the hearing-impaired population throughout the world. Our own estimates of prevalence from models is that the proportion is in the vicinity of 16% (about 80,000 people in NZ) which is more in agreement with other modelling studies (eg Dobie, 2006, 10% in the USA¹⁷) and the WHO (<http://www.who.int/pbd/deafness/en/>) which estimates it to be between 7 and 21%. Our estimates of incidence are about 2300 new cases each year (in 2006). Our modelling indicates that the incidence (the rate of new cases) is declining and will continue to decline, if for no other reason than the participation rates in noisy industries are declining. In contrast claims to ACC have increased substantially over the last decade and are now at about 4600 each year. This may represent historical claims and, with time, the number of claimants should naturally decline in line with the projected decreasing incidence. It is therefore very questionable whether the rate of claims to ACC is a true reflection of the extent of the current problem and this should be further examined before such drastic changes are made to the assessment processes.

2.2 *Noise-induced hearing loss is essentially a preventable condition.* Turn down the noise and the potential for injury is reduced. A combined approach from ACC and Department of Labour towards addressing the **prevention** of noise-induced hearing loss will have a more significant impact on claims and the liability to ACC as the extent of the condition declines. Furthermore, such an approach would be aligned with the ACC's Strategic Priorities. The international evidence supports the view that attention to the principles of injury prevention with a concerted effort to eliminate the hazard and minimise the harm can reduce the impact of noise on hearing. Preliminary research by Dr Ian Laird at Massey University indicates that there is relatively poor compliance with the New Zealand OSH regulations around noise in the workplace. Ensuring compliance with the noise regulations is the way to reduce the incidence of NIHL.

3. Summary and conclusions

In summary, the proposal to assign a percentage for the injury related component of the hearing loss which can then be used to calculate the ACC contribution does not have a robust scientific basis. There is no appropriate discriminative diagnostic test to enable the contribution from noise to be separately identified from all other causes, in most cases. There is so much variability in the individual response to noise exposure and in the aging effect on hearing that making judgements on the proportion of injury and age components of

¹⁷ Dobie, R. A. The burdens of age-related and occupational noise-induced hearing loss in the United States. *Ear and Hearing*, 2008, 29, 565-577.

hearing loss for any individual is prone to considerable error. This makes it unfair and inequitable across individuals and would open ACC to challenge which would likely be difficult to defend.

It is disturbing that there has been no consultation on the introduction of apportionment of noise and age-related hearing loss that has allowed the scientific background to be examined and evaluated. Furthermore it seems illogical to introduce this while a considerable amount of ACC and Department of Labour supported research to understand more about the epidemiology, prevention and assessment of NIHL in New Zealand is underway and nearing completion.

I recommend that the proposal to introduce these regulations for apportionment and part charges be deferred until the scientific validity, fairness and equity of the process is robustly reviewed.

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