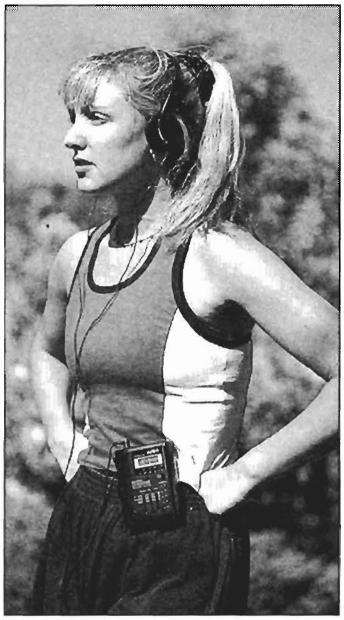
From: LePage, E. L. and Murray, N. M. Latent cochlear damage in personal stereo users: a study based on click- evoked otoacoustic emissions. Med J Aust. 1998 Dec 7-1998 Dec 21; 169(11-12) 588-592. (Editorial p.587)

Otoacoustic emissions and recreational hearing loss

Noise-induced hearing loss may now be detectable before it becomes clinically obvious



The risk of noise-induced hearing loss from amplified music was predicted by Carter et al in 1982, who noted that, in a study of 994 subjects aged 16 to 20 years, "...the accumulated exposure of some of them to noise is such that, if their recreational patterns remain the same, they are at risk of some noise-induced hearing loss by their mid 20s. Further empirical studies are necessary to determine whether these hearing losses will eventuate".[1] In this issue of the Journal (*page 588*), LePage and Murray use the relatively new technique of otoacoustic emission (OAE) analysis to investigate noise-induced hearing loss resulting from the use of personal stereo headsets.[2] Their results suggest that personal stereo use results in a decline in cochlear function analogous to rapid ageing of the cochlea, and comparable to hearing loss from industrial noise trauma. They also emphasise that, as OAE analysis can detect decline in cochlear function long before there is any clinically detectable hearing loss, this technique can potentially provide early warning of noise-induced hearing loss.

Otoacoustic emissions were first described by Kemp[3] in 1978. They are sounds thought to be generated by the cochlear outer hair cells in response to an external sound stimulus. Normal hearing threshold is achieved by a cochlear mechanism, thought to reside in the healthy outer hair cell, which magnifies the stimulus internally. When this mechanism loses the peak of its performance, OAEs diminish and hearing threshold is raised. As OAEs can be recorded in the outer ear, they may provide an objective, non-invasive and quantitative measure of hair-cell function.[4,5] High test–retest reliability has been demonstrated for individuals, although there is variability between subjects.[6]

Four types of OAEs have been described.[7,8] Spontaneous OAEs occur in 68% of infants younger than 18 months, but the incidence falls to 35% in adults under 50, and to 20% of adults over 50 years.[9] Transient-evoked OAEs (TEOAEs), a response to acoustic clicks delivered to the outer ear, are currently thought to be the most clinically useful OAEs, as they are detectable in 98% of people with normal hearing, regardless of age or sex, and the two ears of any individual produce similar TEOAEs.

Stimulus-frequency OAEs occur in 88%–100% of people with normal hearing, and resemble TEOAEs in behaviour. They represent fixed-place emissions corresponding to specific frequency sites along the organ of Corti, but their usefulness as a clinical test is limited by technical factors. Distortion-product OAEs (DPOAEs) also occur in 100% of people with normal hearing, and, while small in amplitude, can be used to intentionally test a specific frequency region of the cochlea. DPOAEs are technically difficult to measure, but will become an essential tool in the investigation of tonotopic outer hair cell function.

Probst et al have provided an extensive review of the technical details, experimental and clinical findings of otoacoustic emission analysis.[10]

Measurement of OAEs has become a useful audiologic and otoneurologic diagnostic test for neonatal screening,[4,9] otosclerosis,[11] sensorineural hearing loss,[4,5,12] Meniere's disease,[5] acoustic neuroma,[5] tinnitus,[13] ototoxicity,[7] and noise-induced hearing loss.[5,12,14] A limiting factor in this kind of ear testing is that eustachian tube dysfunction will reduce otoacoustic emission energy.[4] Thus, tympanometry is essential if no otoacoustic emission can be measured.

The reduction in outer hair cell activity in patients with noise-induced hearing loss, measured by DPOAEs, is directly related to frequencies of the audiometric loss.[5] In one study, emissions were abnormal in 93.2% of ears with noise-induced hearing loss and in teenagers exposed to noise, and were found to be useful in the prediction of noise susceptibility.[12] In another, ears with a noise-induced impairment showed a significant reduction in the incidence of both spontaneous emissions and spectral peaks in evoked emissions that was not evident in ears with similar patterns of hearing loss caused by other factors.[14]

Are the listening habits of the younger generation potentially dangerous to hearing? Ising et al studied 681 students aged 10 to 19 years.[15] Although 50% of students listened to music for less than one hour per day, 10% listened for four or more hours. Among those aged 12 to 16 years, 10% chose to set the listening level at 110 dB(A). It was estimated that 7% were exposed to noise levels likely to damage the cochlea. They recommend that the sound levels for portable music players be limited to 90 dB(A).[15] Hearing loss has been documented in people who attended rock music concerts,[16] in employees of urban music clubs,[17] and one report indicates that exercise combined with exposure to music presents a greater risk to hearing than the music alone.[18] These authors conclude that "the results have implications related to contemporary lifestyle issues such as aerobics and the utilisation of personal music systems during physical exertion".

The risk of recreational noise-induced hearing loss is real, and our patients must be advised of this risk. LePage and Murray have demonstrated that early warning is now available in the form of the transient-evoked otoacoustic emission test.

John T Redhead
Director, Jean Littlejohn Deafness Investigation and Research Unit
Royal Victorian Eye and Ear Hospital, East Melbourne, VIC

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Latent cochlear damage in personal stereo users: a study based on click-evoked otoacoustic emissions

Eric L LePage and Narelle M Murray

Abstract

Objective: To assess the effects of use of personal stereo systems (PS) on hearing by means of the objective measure of transient-evoked otoacoustic emissions.

Participants and setting: People aged between 10 and 59 years who had otoacoustic emissions recorded by the National Acoustic Laboratories between 1989 and 1997 were eligible for inclusion. Recordings from participants with hereditary disorders or any form of aural disease (eg, otitis media, otosclerosis, fluctuant hearing loss, Meniere's syndrome, or exposure to ototoxic substances) were excluded.

Methods: Transient-evoked otoacoustic emission (TEOAE) records were obtained with a standard 260 repetitions of an 80dB train of clicks used for recording outer hair cell activity. The measure of otoacoustic emission strength was the Otodynamics ILO88 variable Waverepro%. For each participant, all the key factors relating to their hearing history were assessed from patient referral information or from demographic information obtained in writing at the time of recording either in the form of a detailed questionnaire or verbal assessment. Otoacoustic emission data were analysed according to age, industrial noise exposure and personal stereo use.

Results: Usable otoacoustic emission records were obtained from 1724 people (1066 males and 658 females). Otoacoustic emission strength declined with age, and was significantly lower in males than females, lower in people exposed to industrial noise than those not exposed, and significantly lower in users of personal stereo systems than non-users. People with both kinds of noise exposure had values which were significantly lower again, indicating an additive effect.

Conclusions: As only 39 people with PS exposure admitted any hearing problems, decline in otoacoustic emission strength forewarns premature hearing loss in personal stereo users.

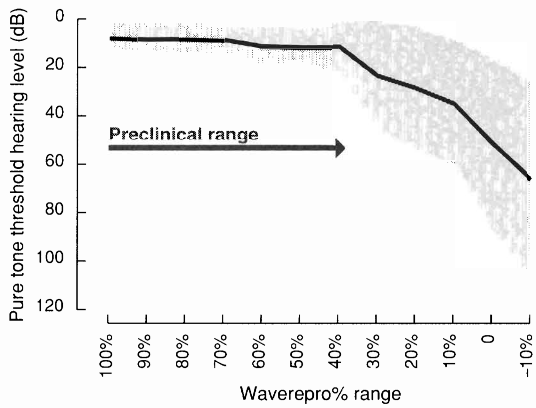
MJA 1998; 169: 588-592

The use of pure tone audiometry to assess the potentially harmful effects of amplified music on young people's hearing has failed to show any marked effect.[1-3] However, good grounds for concern remain as (i) the sound-pressure levels generated by live and recorded rock music are associated with premature hearing loss in industrial workers,[4,5] (ii) inexpensive stereo components have the capacity to generate high sound levels,[6] and (iii) the preferred listening levels of those listening to rock music through earphones in "Walkman"-style headsets is high — an average of 95dB(A) for females and 97dB(A) for males, with an overall range of 75–110dB(A)[7-16] ("A" signifies the standard "A-weighting" correction to indicate approximately equal loudness across the audible frequency range). One research group felt that "further research may reveal more sensitive measures of cochlear damage than pure tone audiometry".[3] One such measure may be otoacoustic emissions, the principle of which is described in Box 1.

In this study, we examined the effects of personal stereo use on transient-evoked otoacoustic emissions (see Box 1) to investigate the possibility that this technique may indeed be a more sensitive method of early detection of ear damage resulting from sound amplification. To shed light on the significance of our findings, we compared people exposed to personal stereo use with those exposed to industrial noise — a recognised high risk form of exposure.

Hearing Loss Prevention Research, National Acoustic Laboratories, Sydney, NSW.
Eric L LePage, PhD, Senior Research Scientist; **Narelle M Murray,** MA(Aud), Senior Audiologist.
Reprints will not be available from the authors.
Correspondence: Dr E L LePage, Hearing Loss Prevention Research, National Acoustic Laboratories, 126 Greville Street, Chatswood, NSW 2067.
Email: Eric.LePage@nal.gov.au

1: Otoacoustic emissions



The cochleas of mammals contain two sets of hair cells — the inner hair cells (IHC) and the outer hair cells (OHC). The IHC are passive, sensory cells which directly stimulate the auditory nerve, while the OHC are active, muscle-like cells which act as a "cochlear amplifier". The primary reason people suffer a permanent hearing loss is a significant decline in the numbers or functionality of OHCs, leading to loss of internal amplification.

Otoacoustic emissions (OAEs) are the sounds that the activity of the OHCs generate in the ear canal,[17] which can be measured with a microphone and, with modern technology, separated from sounds entering the ear. Middle ear disorders affect OAEs, but are easily distinguishable from sensorineural hearing loss.

We used the Otodynamics ILO88 analyser (Otodynamics Ltd, Hertfordshire, England) in this study. A probe containing both a speaker (which delivers the stimulus) and a microphone (for recording the response) is sealed in the ear canal. We used the transient-evoked (or click-evoked) otoacoustic emission (TEOAE)[17,18] technique, which specifically targets the active OHC response and ignores the acoustic response of the external and middle ear. A standard 260 repetitions of an 80 dB train of clicks lasting 1 ms are delivered to the ear, and the otoacoustic emission response from the outer hair cells is detected by the microphone. The first 20 ms of the response following the click is averaged to improve the quality of the signal. Stability of probe placement is routinely monitored and displayed as a percentage with the record. Data collection for approximately one minute gives a clinically useful waveform. Alternate responses are summed into two arrays and at the end of recording the correlation coefficient between the arrays is calculated. This coefficient (range, -1.0 to 1.0) is normally expressed by Otodynamics as a percentage, denoted by the term Waverepro% (designated by some authors as Wholerepro%), which is a measure of strength of the emission.

Complete sets of OHCs (such as in neonates) produce emissions near 100%. As the ear ages or is progressively damaged, the Waverepro% decreases. Waverepro% may be compared across the population[19] provided a standard 80 dB peak stimulus is used.

The ability of TEOAE to measure net activity of any ear is of particular relevance to this report, which relates otoacoustic emissions to age and two specific risk factors. The relationship between Waverepro% and pure tone threshold hearing levels is illustrated,[20,21] showing that the Waverepro% needs to decline to below about 35% before hearing loss is detectable by audiometry. Hence, TEOAE may be able to detect accumulated ear damage in the preclinical range. If so, the technique may prove useful in detecting latent damage caused by noise and other factors.

Methods

The protocol we used for obtaining transient-evoked otoacoustic emission records is described in Box 1.

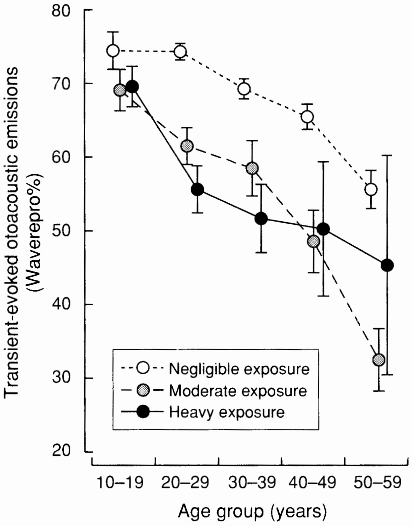
The data for this study were obtained from the records of some 2500 people tested as part of the National Acoustic Laboratories research program between 1989 and 1997. Information about participants' hearing histories was obtained from details supplied by referring clinics or otologists (12%), standardised questionnaires filled out (60%) or verbal evaluation (18%) at the time of recording, or from Australian Hearing's NALCAM (National Acoustic Laboratories Computer Aided Management) database (10%). Subjects were asked about hereditary hearing loss, exposure to industrial and leisure noise, prescription drug use (particularly antibiotics, diuretics and cytotoxic drugs), head injury and any hearing-related symptoms they were currently experiencing. Sound-exposure histories were obtained by asking subjects to estimate their average number of hours per week and their years of exposure to any noisy activity, such as industrial noise, sporting activity, firearm use, the music industry, personal stereo use and other exposure to amplified music.

Participants' data were included in the analysis only if an acceptable pair of recordings were obtained for both left and right ears in the same recording session. Participants' records were excluded if the recording stability values (see Box 1) were less than 80%, if participants failed otoscopic inspection, or if they had a clear inherited factor or any form of aural disease (eg, otitis media, otosclerosis, fluctuant hearing loss, Menières syndrome, or exposure to ototoxic substances). No form of noise or music exposure was considered grounds for exclusion.

For the 4.3% of participants who provided repeat records over the nine years of recording, we included only the pair of records with the highest emission strength. Participants were aged 10 years to less than 60 years on the day of recording.

On the basis of the sound exposure histories recorded in the database, participants who reported personal stereo (PS) use were divided into three categories: PS = 0 (negligible, < 1 hour per week), PS = 1 (moderate, 1 hour to < 6 hours per week), and PS = 2 (heavy, ⩾ 6 hours per week). Similarly, subjects were classified into two industrial noise (IND) categories according to whether they had ever worked in noisy industry: IND = 0 ("no"), and IND = 1 ("yes"). These classifications did not exclude other noise exposure factors.

2: Changes in otoacoustic emissions with three levels of personal stereo (PS) use and with age

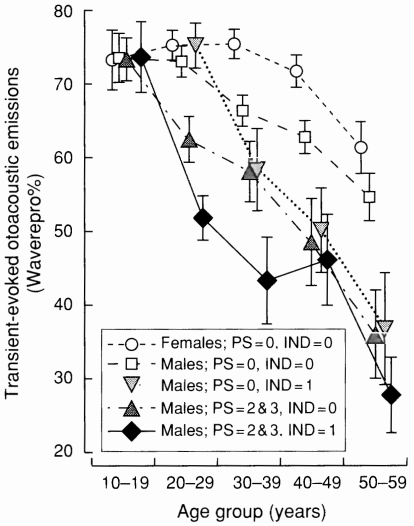


A decrease in transient-evoked otoacoustic emissions indicates greater accumulated cochlear damage. Data points represent mean values and error bars indicate ± 1 standard error.

Sample sizes for the three personal stereo use groups

Personal stereo use	Age group (years)					
	10–19	20–29	30–39	40–49	50–59	Total
Negligible (PS = 0)	107	348	312	257	145	1169
Moderate (PS = 1)	53	138	60	63	41	355
Heavy (PS = 2)	61	86	37	13	3	200
Total	221	572	409	333	189	1724

3: Changes in otoacoustic emissions with personal stereo use, industrial noise exposure and with age



The bottom four curves compare the effects of different kinds of exposure versus age for males only. Females with negligible personal stereo use and no industrial exposure are shown for reference. Data points represent mean values and error bars indicate ± 1 standard error.

Sample sizes for males in the four mutually exclusive groups of personal stereo (PS) use and industrial noise exposure

Noise exposure	Age group (years)					
	10–19	20–29	30–39	40–49	50–59	Total
Negligible PS, no industrial (PS 0, IND 0)	47	138	150	126	79	540
PS only (PS 1&2, IND 0)	61	88	40	30	21	240
Industrial only (PS 0, IND 1)	13	44	25	21	10	113
PS and industrial (PS 1&2, IND 1)	4	93	28	30	18	173
Total	125	363	243	207	128	1066

Corresponding sample sizes for females in the PS 0, IND 0 group were: 10–19 y, 45; 20–29 y, 158; 30–39 y, 136; 40–49 y, 107; 50–59 y, 55. Total, 501.

Statistical analysis

We applied analysis of variance with multiple linear regression in which the independent variables were sex, age (grouped by decade: 10–19, 20–29, 30–39, 40–49 and 50–59 years), PS use category, and industrial noise category. The dependent variable was the mean of values for both ears of otoacoustic emission strength (TEOAE Waverepro%; see Box 1). The multiple linear regression was performed using Statistica software.[22]

Results

Records from 1724 people (1066 males and 658 females) were included in the analysis.

Box 2 shows the relationship between age and otoacoustic emission strength for all subjects who reported being negligible, moderate or heavy users of PS systems (regardless of other noise exposure) and the corresponding sample sizes for these three groups. Considering each age range in turn, for the teenage range (10–19 years) there was no significant difference between any of the three PS use groups. For people aged 20–29 years and 30–39 years the PS = 0 (negligible exposure) group was significantly different from both the PS = 1 and the PS = 2 groups ($P < 0.01$). For people in both the 40–49-years and 50–59-years ranges the PS = 0 group was significantly different from the PS = 1 group and also from the PS = 1 and PS = 2 groups combined ($P < 0.01$). For all the adult age ranges, PS users had significantly lower values of the Waverepro% than non-users, with the lowest values in heavy users.

Box 3 compares, firstly, females and males with no industrial noise exposure and negligible PS use (the top two curves with open symbols) and shows that, for the three oldest age groups, the values of emission strength for males were significantly lower than those for females ($P < 0.01$). Secondly, the bottom three curves with filled symbols show the effect of PS use, industrial noise exposure and both forms of exposure for males only (we did not include females in this com-

4: Multiple linear regression model showing additive effects relative to the reference condition for each of the risk categories listed

Variable	Reference condition	Added risk	Effect	Standard error	Confidence interval	P
Intercept			86.8	2.6	81.7 to 91.9	< 0.001
Sex	Female	Male	-5.4	1.3	-7.9 to -2.9	< 0.001
Age (years)	10–19	20–29	-3.5	2.0	-7.4 to 0.4	0.083
		30–39	-8.4	2.1	-12.5 to -4.3	< 0.001
		40–49	-12.9	2.2	-17.2 to -8.6	< 0.001
		50–59	-24.1	2.5	-29.0 to -19.2	< 0.001
Personal stereo use	Negligible	Moderate	-10.7	1.6	-13.7 to -7.6	< 0.001
		Heavy	-12.3	2.0	-16.2 to -8.4	< 0.001
Industrial noise exposure	None	Any	-7.6	1.7	-10.8 to -4.3	< 0.001

parison as very few women [33] had had industrial noise exposure compared with men [286]). Thus, the four lowest curves in Box 3 compare four mutually exclusive noise exposure groups for males only. The corresponding sample sizes for males in these exposure categories are also shown in Box 3. Considering each age range in turn, there was no significant difference between four noise exposure groups at 10–19 years of age. However, for participants aged 20–29 years, all noise exposure groups were significantly different from each other ($P < 0.01$), with the exception of groups IND = 1 (industrial noise exposure only) and PS = 0, IND = 0 (negligible PS use and no industrial noise exposure). In the 30–39-years group the only significant difference was between the PS = 0, IND = 0 group and the group with both PS and industrial noise exposure ($P < 0.01$). For both the 40–49-years and the 50–59-years groups, the PS = 0, IND = 0 group was significantly different from the group with PS use only, and also the group with both PS and industrial noise exposure ($P < 0.01$). The mean values for the industrial noise exposure only group were the same as for the PS exposure only group but the sample size was small.

Box 4 shows the results of the multiple linear regression. For each of the selection conditions tested, the slopes (the "Effect" column) representing the rates of decline of Waverepro% are highly significant. This Table indicates that Waverepro% for males was about 5% lower than for females. Waverepro% for moderate PS users was about 11% lower than for non-users, while for heavy PS users this value was 1.6% lower still; Waverepro% for respondents indicating industrial noise exposure was also 8% lower than for non-exposed people. The age dependence was a decline in Waverepro% of 0.49% per year, or 4.9% per decade.

Discussion

Our findings suggest that there is a strong trend for the strength of otoacoustic emissions to decline with protracted use of PS headsets, and that the size of this decline is proportional to the amount of exposure. Although the separation of PS users into moderate and heavy categories was based on self-report, the multiple regression showed an effect between the moderate and heavy users. However, the design of our study did not exclude the effects of other forms of noise to which people who tend to use PS systems may also be exposed. If PS exposure is associated with other lifestyle factors, our analysis would not have differentiated between them. Other factors, such as leisure and other non-occupational noise exposure (eg, power tools, car racing, concerts) and forms of injury to which males are more exposed (eg, head injury, barotrauma), may have accounted for the significant differences between the sexes in people with neither industrial nor PS exposure. We believe all such factors contribute to the high level of variance of the otoacoustic emission levels.[23]

Further, for males exposed to both PS use and industrial noise, the otoacoustic emission strength was significantly lower ($P < 0.001$) in all age ranges other than the 10–19-years and 40–49-years range. Remarkably, for the 30–39-years range there was no significant difference between the group exposed to industrial noise and the PS user group, both of which were significantly different from the non-exposed groups ($P < 0.001$). Yet the young adult PS users (20–29 years) had otoacoustic emission strengths significantly lower than non-users ($P < 0.001$), suggesting that the decline occurs in the late-teenage and early-adult period — a decade earlier than the expected industrial effect.

The multiple regression analysis showed that, in our sample, the apparent rate of decline in otoacoustic emission strength among young adults was greater for PS users who were also exposed to industrial noise. However, it is worth noting that this group included a subgroup of 26 deep coal miners whose mean values were a whole standard deviation lower than males with no industrial noise exposure.

Based on existing guidelines for occupational noise level limits in all Australian States and Territories of an eight-hour equivalent continuous A-weighted sound pressure level of 85 dB, it is surprising that PS use for less than six hours weekly at typical sound levels of 95 dB results in such a high level of damage accumulation. This leads to speculation that there may be other factors involved with sound delivered through earphones compared with free-field sound, such as more efficient delivery of high-frequency sound coupled with the high dynamic range of modern PS units. The popularity in recent years of units offering "additional bass boost" is consistent with the notion that users may be endeavouring to enhance the sense of sound envelopment which occurs at higher levels. Our findings strongly support the previous assertions by Waugh and Murray[24] of increased risk of ear damage from PS use, particularly if personal stereos are used in other environments in which users tend to raise the listening level to mask out background noise (such as on public transport or while engaging in aerobic exercise), which may lead to generalised inner-ear problems.[25]

A 1996 study by Meyer-Bisch appears to be the only one to have succeeded in showing a significant difference between actual hearing levels of PS users and those of a control group.[26] Our findings illustrate why most previous studies of PS exposure have failed to observe any effect — the preclinical phase of hearing loss[23] is extended, and PS units have not been around for long enough for critical levels of damage to be apparent in most users. Indeed, as only 39 PS users in our sample reported any hearing difficulties, the primary significance of our study is that transient-evoked (or click-evoked) otoacoustic emission measurement offers early warning for hearing loss.

Our findings on the effects of PS use and industrial noise exposure, both separately and together, illustrate an additive effect long held to be a basic property of noise-induced hearing loss.[23] In our study this effect may be partly the result of a small association between PS use and industrial exposure — PS use was higher in the group who had industrial exposure.

The technique of measuring evoked otoacoustic emissions has direct application beyond the screening of neonates to programs for hearing loss prevention. It sheds light on the nature of presbycusis, or the normal hearing loss during ageing. Thus, by comparing the rates of decline shown in Box 4 with the relationship between Waverepro% and audiometric hearing level illustrated in Box 1, it is possible to linearly estimate the remaining period of normal hearing. Beginning with Waverepro% values above 80%, as in normal neonates, and assuming a linear model, a decline in Waverepro% of up to 5% per decade should result in no hearing problems for life, while a decline of 7% per decade results in normal presbycusis (about seven decades of normal hearing). However, a decline of 20% per decade would give only 2.5 decades of normal hearing. The fact that many young people in our sample appear to have been subjected to such accelerated hearing loss suggests that it is not unreasonable to predict a rise in the number of young adults with premature hearing impairment.[27]

In summary, our findings highlight three important points related to hearing health:

- otoacoustic emissions may offer new precision in determining an individual's risk of hearing loss;
- the use of PS headsets, even in typical moderate use, is associated with rapid ageing of the cochlea comparable with industrial noise trauma;
- as personal stereos are here to stay, the essential message for preventing premature hearing loss in users is that listening times and volumes should be moderate, and that users should be aware of the potentiating effect of noisy background conditions which both add directly to the noise dose and encourage them raise the PS volume.

Acknowledgements

We thank audiologists from Australian Hearing's centres, A Butler of the NSW Department of Health and R Deller for assistance with data collection. We also thank Professor John Kaldor and Matthew Law for assistance with the statistical analysis.

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(Received 7 Jul 1997, accepted 1 Oct 1998)