Submission to the 2009 Senate Inquiry into Hearing Health in Australia

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Eric L. LePage[1], Ph.D. and Narelle M. Murray[2], Ph.D.

We are fully sympathetic to the pressing needs expressed in many submissions. However, we particularly address the terms of reference item d. "The adequacy of current hearing health and research programs, including education and awareness programs". There needs to be a clear separation of scientific issues at stake in dealing with any epidemic of hearing loss from technological issues and from cost issues. We question the current emphasis on commercially-driven rehabilitation technologies constraining how broadly-based scientific endeavour is integrated to further prevention activities. The outcome seems to be 'chasing the bolted horse' instead of dealing with causes of the trend.

Background

Until 1989 the risks for noise-induced hearing loss had been assessed entirely from estimating sound exposure and this method is still deemed audiological best practice – the higher the sound level, the longer the exposure, the higher the risk of acquiring a permanent loss, but only after a very long (but highly variable) lead time. This mathematical 'equal-energy' relationship describes the average response obtained from large numbers of studies over the last century. There are other empirical relationships, such as those describing the effects of age and gender, whose effects are effectively layered on top of the basic noise curve. Next, the particular industrial occupation is another major factor which is layered on top again – providing that the exposure is, more or less, periodic, such as for a fixed fraction of each day. However, in the last half century, another layer of uncertainty was introduced – the growth of leisure noise exposure. Unfortunately, this could not be assigned any periodic basis and could not be measured at all precisely. All these layers are now known to influence individual susceptibility to hearing loss. The problem is that by the time all these layers are stacked over one-another, together with any genetic predispositions, all statistical precision about who is at risk is lost.

By 1989, it seemed to us there was a more definitive approach to assessing risk, made possible by the progress in basic hearing science and the discovery of otoacoustic emissions (OAE). These are low-level signals measurable by inserting a probe into the ear canal. **Instead of juggling all these indirect risk factors, why not just directly estimate the ear damage in every person individually, with a quick, painless physical measurement?** This is what we set out to do. At that time the technique of clicked evoked otoacoustic emissions (CEOAE) was the most advanced option. In the NAL environment we had access to people with virtually every kind of background and our database grew. As was expected the emissions strengths dropped off with age. They also showed the expected variations with gender and noise exposure. The remarkable

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[1] 2009 Visiting Professor, The Univ. d'Auvergne, Clermont-Ferrand, France
2008 Visiting Lecturer, The Univ. of Aalborg, Denmark – a ten lecture course on the topic of this submission.
2007-8 Collaborator, Karolinska Institute, Stockholm, Sweden

[2] Editorial Assistant, Australian & New Zealand Journal of Audiology.

feature of the data was that unlike hearing level data, these data showed a pronounced dip in the middle of the age range suggesting many young people already had advanced damage in their inner ears. We spent three years trying to explain this dip as an artefact of the new kind of measurement. Every attempt resulted in the same conclusion – the dip was real. Research on OAE was intense at that time, with hundreds of publications each year. The bulk of publications demonstrated properties of the emissions, or used them for early detection of hearing problems in neonates and infants. Controversially, our result led us to question whether hearing thresholds were indeed the best measure of ear damage. Our evidence suggested that OAEs actually provide a much more sensitive approach to assessing the effects of noise.

By 1994 we had enough evidence to predict[1] that there would be a steady rise in hearing loss in the Australian community, evident within 10 years, and also that a blow-out would occur within 20 years. We tested the idea that the technique could provide not just confirmation of hearing loss, but early-warning before there were behavioural signs i.e. before changes in threshold. By 1998[2] we had shown, in a group of some 500 young people with normal hearing, many of whom used personal stereos, there was evidence strongly suggestive of hidden cochlear damage. It was very important that not only were we seeing a dip in the population data, we could see it in individual data – otherwise the technique would be no better than pure tone thresholds. Indeed, in many cases the estimated damage in the younger individuals was more pronounced than those a generation older. **This gave us some confidence that the technique could indeed be applied to assess the risk of any individual.** There were some uncertainties with the new approach because of the dynamic nature of the new data. We followed these until 2005 attempting to separate the long-term and short-term effects. We aimed for a "bio-assay" for both *individual susceptibility to hearing loss* and the *effectiveness of hearing protection devices*.

Recent research

Dr. Lynne Marshall from the submarine base at Groton, CT took our claim seriously. Early this year she and her team published a significant study specifically testing our claims of early warning, also testing a rival theory[3]. The findings rejected the alternate theory and supported ours. Americans have a strong preference for the distortion product method (DPOAE). Common to both investigations, however, was the interpretation that the strength of the OAE signal is a direct measure of the **redundancy (excess) of outer hair cell numbers needed for normal hearing – the lower the emission strength, the fewer the outer hair cells and by implication the higher the risk of imminent hearing loss.** Both studies point to a subtle but important distinction – the difference between *early detection* of hearing loss and *early warning* or status of increased risk. A good analogy might be the arrival of a tsunami, versus the warning of the likelihood of one considerably in advance.

For decades researchers have tried, using mathematical models, to connect the concepts of temporary hearing loss (due to trauma) and permanent hearing loss, but now there is substantial progress. However, the language is mostly in terms of molecular biology, e.g. cell death pathways, apoptosis and anti-oxidants. By contrast, most submissions to this Inquiry, including the 2006 Access Economics Report, give scant attention to this major activity.

Our own research has continued to try to grapple with basic issues of not just our own doubts but considerable skepticism. In recent times we have harked back to our original research on cochlear mechanics prior to the introduction of the "cochlear amplifier" hypothesis. The notion

of the cochlear amplifier makes intuitive sense – that when the internal amplifier fails, it can be replaced by an external amplifier. **However, our latest research in France suggests that the hair cells may be doing much more than just amplification of sounds entering the ear** – very low frequency components of OAE may be very important[4-5], components filtered from our early data built into the protocol in the commercial equipment. These components are being used to characterise the internal operations of the cochlea – active regulation of the hearing sensitivity according to several local and central influences. Importantly, these operations are eliminated from the signal which goes to the brain – audiometry gives no measure of them. This regulation process strongly depends upon potassium currents in the cochlea[6]. It undoubtedly incorporates **the voluntary control over how we listen (not just how we hear) which deafened sufferers might once have taken for granted**. The efficiency of this regulation is challenged any time the ear must handle loud sound – be it an iPod, rock concert, deep coal mine or hearing aid.

In 2006 we published a special edition[5] referring to hundreds of scientific articles on the 'extended' internal workings of the ear. For many people they account for why hearing aids, for all their 'latest advances', fall far short of duplicating the internal operation of the ear, leaving no doubt as to why they are so frequently worn 'in the top drawer'. There was no doubt in our minds that our priority was prevention – by investigating how to get at the regulatory properties of the inner ear using emissions.

Prevention

Over the last decade there has been increasing worldwide use of the term 'epidemic' in relation to hearing loss. Highly relevant is the NHANES studies in the USA and European Commission (SCENIHR) report adopted on 23 September 2008[7] both of which we view as support for the notion that our 1994 predictions of a blow-out was sound.

Drawing an analogy with the global warming debate is interesting. Both phenomena coincidentally display long lead times (decades). In turn there are inherent delays to achieve general recognition that there really exists an exponential trend needing correction. It seems to be human to ignore early warning information because it is not proof. Only a blow-out will typically generate action, when the consequences are so obvious they bite into personal finances and national economics. The claims for help for the afflicted occupy pride of place over the silence emanating from the individuals who don't know yet that they have a problem. As the costs of rehabilitation escalate, predictably reasons will be found to justify at least a fraction of total support funds towards prevention. **It behoves funding agencies to ensure that such work is well directed and cost-effective.** It is significant that Prof. Richard Kopke is a co-author on the Marshall et al paper. He is known foremost in the search for pharmacological protection for noise-induced hearing loss.

There are valid concerns about how to translate any demonstration of significant ear damage in young people into behavioural change. After all, even the demonstration of a mild hearing loss doesn't necessarily carry over to a change in attitude. Why should early-warning technology change that? The reason is that a mild hearing loss is extremely subtle. The individual's relatives are frequently the first to realise it, but by then it is too late. Another reason early-warning technology should be adopted is that it is not just quick and cheap, it has the capacity to single out an individual for special education. There is no question that education of the young should be a key priority. But the long-lead time says that that the weighting of

education cost should be targeted at children from pre-school, but adults need more than advice. They need the kind of risk assessment we built into the NAL-OAE software. Genetic predispositions to hearing loss are widely accepted, but whether any individual is potentially affected needs to be demonstrated by early-warning for best management.

A strong advocate for our approach was the late Dr. Ken Rowe who championed the potential value of incorporating biophysical measures of cochlear performance to quantify the differences in learning capacities of school children. He strongly believed in a rate-limiting peripheral factor responsible for the progress of slow-learners, and saw possibilities in the fact that CEOAEs give a direct measure of the speed of cochlear response.

The main reason early-warning technology should be adopted is that traditional approaches are inherently flawed as outlined at the beginning. Sound levels project damage through a model based on average data, when 'hearing damage' itself is nothing more than a vague notion. As earphone exposure may be a major factor in any modern epidemic, it is worth noting that this factor was the most significant of all forms of noise in our early data. It was no doubt interesting to measure the sound levels under iPod earphones, but it is virtually impossible to adequately monitor the total effect of music exposure which does not conform to simple time patterns such as daily exposure. Yet it is being revealed that while sound levels may be important, the complete story needs a physiological account of recovery from the trauma. **Modern prevention research simply cannot be carried out from the same outlook which focuses only on the sound source. It is simply incomplete and not cost-effective.** It has been proven many times that pure tone audiometry is impractical to use for screening in the event of an epidemic; it is too slow, two expensive, too uncertain – and too late.

Rehabilitation

The 'vocabulary' of most hearing professional help is almost entirely in terms of the supply of some assistive listening device to increase the signal-to-noise ratio, with or without amplification – but predominantly with – and increasingly expensive. Loss of speech reception is such a serious consequence that retaining it by any means is justifiable, even if the noise-exposure it delivers damages what remains of cochlear function. In our view residual numbers of outer hair cells may play a vital part in maintaining some level of internal regulation, meaning that there are much broader issues at stake, e.g. the progressive decline in that residual capacity is clearly related to the period of acclimatisation after first introduction of the aid with its multiple costs for post-fitting adjustment. It is therefore remarkable that rehabilitative effort, for so long, has resisted the suggestion that outcomes might be improved by specifically studying the physiological effects of the loud sound they deliver to the cochlea. Refreshingly, there is a new initiative in this respect from the Massachusetts Institute of Technology[8].

We need to be forgiven for continuing to think that there is a large gulf between these two huge areas of hearing science as they apply to noise-induced hearing loss. In a crisis, such a standoff seems untenable. We submit that failing to bridge this gap, preferring to stay with traditional assessment schemes based only upon sound levels and hearing aid solutions will keep Australia forever locked into the vicious cycle which this Inquiry is called to address.

The urgency of responses to the trend world-wide suggests that is high time to make more serious attempts at crossover. There are serious science issues to be dealt with relevant to this Inquiry. To this end we submit that it is reasonable for the Australian government to titrate its funding of rehabilitation research, by stipulating that a non-negligible fraction must go towards multi-disciplinary effort on prevention, integrating behavioural with objective approaches, genetics, molecular biology, biophysics.

Summary

We published in the Medical Journal of Australia in 1998 evidence that OAEs provide early warning of hidden damage -- a study which has now been duplicated. This capability has multiple applications. It originally flagged a blow-out in premature hearing loss, back in 1994. The finding almost seems to have been used to promote a widespread proliferation of rehabilitation activities at the expense of prevention. There now seems to be growing international recognition of such a trend. It means that the technique is potentially applicable to any situation testing changes to the cochlear amplifier which requires the earliest indication of effectiveness, e.g. clinical interventions, prevention programs, pharmacological treatments and gene-therapies. Latest research has been directed at subtle inner-workings of the ear, explaining many of the difficulties we encountered. It is clear this avenue of research has enormous scope, not just for prevention but a whole new raft of strategies for rehabilitation. Far from being washed up, the real potential of the OAE approach is only just starting to be realised.

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