

Molecules, managers or mentors: how can we minimise noise damage at the worksite?

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Good afternoon. Nice to see you all here and thank you very much to the organisers, James and Gordon and Leanne, for offering the invitation, and I think we all know how much work it is to put on something the size of this and have it come off so successfully.

As Jenny said, I'd like to talk to you this afternoon about molecules, managers and mentors. How can we minimise noise damage in the worksite? So it's with great pleasure that I have the opportunity to speak with you although I bring you bad news.

Like urban Australia, about one in 12 rural Australians has damaged their hearing at work—one in 12—for you rural people, that's one egg in a dozen. And almost all of that noise damage has occurred at work, so it's known as occupational noise induced hearing loss, or ONIHL, which I find to be a mouthful, so I'm going to refer to it, James, as noise damage, which is a lot easier to say.

So one in 12 adult Australians have damaged hearing from loud sound—similarly in rural areas and in urban areas. The hearing loss—and it's an important point—the hearing loss is slow, insidious and it's permanent. There are no drugs to help you recover. Once you've lost it, you've lost it. The good news is it's avoidable. The sad point is we're not avoiding it. Now, to put the magnitude of this problem in perspective, I want to point out a couple of things.

The National Occupational Health and Safety Commission of Australia rates the problem third out of a list of eight focus issues for Australian health—third. It points out that, in fact, in rural areas the problem is worse than it is in urban areas, to the extent that about 60 to 70 per cent of farmers have a measurable hearing loss—60 to 70 per cent—compared to only 27 per cent of those in the general Australian community. It's also very well known that mining and transport are particular problem areas. So much of it is concentrated largely in the rural sector.

Hearing loss has a very high social and economic impact on the individual and their family, but it also turns out that to we Australians, as a community, it has a very high economic cost overall, to the extent that a 2006 report from Access Economics, commissioned by the HEARing CRC in the east and the Victorian Deafness Foundation forum, found that the cost to Australia is of the order of a quarter of a billion dollars annually due to hearing loss generally. That's not all due to occupational noise induced hearing loss, but a large fraction of it

is due to occupational hearing loss and, therefore, it's avoidable. So we can save ourselves an awful lot of money if we address this problem seriously.

Not only did Access Economics do an economic assessment of the problem and the size of the problem economically, but last year, in 2010, the Australian Senate, in a special report, noted that there is no national coordinated occupational hearing loss prevention campaign. So even though this problem is widespread, focused to some extent in rural areas, the Senate—the Senate is telling us, after a very extensive study that I'll point out in a little while, that there is no prevention campaign. Not only that but, when they asked the National Acoustic Labs of Australia, NAL, largely centred in Chatswood, Sydney, about educational processes, the NAL found that there were no large-scale ongoing hearing health education programs across Australia—none.

It's also important to point out that over the years the legislation governing noise damage and noise in the workforce has improved enormously, but we have to realise that we cannot rely on legislation alone to solve this problem. This is particularly problematic in rural areas. Why? Because self-employment and family employment are large in rural areas—nobody sues themselves. So these sorts of legal constraints and regulations in the laws, such as noise abatement legislation, are not relevant if you're self-employed. And I'll talk a little bit about that later as well.

So clearly if this legislation encourages employers and worksites to look after their employees' hearing, one side of that is that employees are provided with the equipment and the instructions about how to reduce noise damage. The other side of the legislation is, if the employer does those sorts of things, they cannot be sued. So at one level it is getting—I'm not saying this in an accusing way—it is getting the employers, quite rightfully, off the hook. So just because you have noise legislation and employers take heed of that legislation and comply with it, it does not mean that the noise damage goes down, because that requires that the employees do as is required. So in the end, after all of that description of the problem, we come to the realisation that we need an education and awareness strategy to prevent this problem, because people have to take responsibility for their own hearing and their own hearing protection.

Now, there are studies of the problem and educational needs in rural areas, and I'll refer to it in a little bit. But the point is that education is the most important thing. And the good news is that pilot studies of these educational programs show that they are effective, for example, in helping young farmers to avoid occupational noise induced hearing loss. Now, that's one of the reasons or—the main reason that I'm here to talk today. I'm here to talk about what we used to call Decibel Danger when, some time ago, I presented this sort of information to high school students.

Now, I am not a numbers man. I know a lot about maths and scientific statistics and whatever but I'm really not a numbers man and I don't really want to dwell on statistics for the rest of this talk. So I want to point out to you some very key documents if you're interested to go and look at some of these things. The first one is the Senate Inquiry, the report on this matter, in 2010—very recent. They did a wonderful, wonderful job of scouring Australia, listening to what people had to say about hearing loss in Australia, and including occupational hearing loss. So I suggest—I encourage you to download this document and have a read through it. It is exhausting and exhaustive, but it's worth it to get an overview of the problem.

I also encourage you to access this document that the Senate also accessed and used. It was an Access Economics report commissioned by the Deafness Foundation of Victoria and also the HEARing CRC, Cooperative Research Centre. Access Economics was commissioned to investigate the economic impact of hearing problems in Australia. So if you really want the numbers of how this is impacting on us economically, I suggest you download and read through the "Listen Hear!" report from Access Economics.

There's other information available, particularly for rural and remote health, and I would encourage you to download and have a look at this particular study to get an idea from a rural focus. It's called "Improving Hearing Health for Farming Families" by a group of people that I won't go into in detail, but including my old friend Warwick Williams sitting here from National Acoustics Laboratory. This is a very interesting document describing some pilot studies and how things can be improved. If you're really into numbers and you want to see what the levels are and what the legislation is like, you need to download this particular document called "Work-Related Noise Induced Hearing Loss in Australia" by the Australian government and occupational health people. Again, it's very exhaustive.

One thing that you can see here is age across the bottom axis versus incidence of compensation cases and you will notice that, as people get older, more and more compensation cases come towards the courts. But, of course, you accumulate hearing loss so the compensation cases go up, but then people start dying. But the other important thing from this point is that females are damaged much less than males. Males are at much greater risk of this sort of problem than females. I'll get to—at the end of the talk I'd like to touch again on this compensation claim issue as an indicator.

So the point I want to get to is—now getting into the main meat of the talk rather than that as the introduction is that I want to tell you something about a groundbreaking education course that I was involved in in Western Australia called Decibel Danger. The good news—it was fantastic. The bad news is that it was 30 years ago and its funding was cut. So I don't want to dwell on my past, although some people know I do. What I'd like to do is to tell you a little bit about Decibel Danger as a focus to what we need in future. So I think there's a lot to be learnt about from the old Decibel Danger program that haunted Western Australian high schools.

It goes like this. Essentially, when we chatted to these children, a team of us went out to different high schools all across Western Australia in a program that integrated a perspective from a lot of different people. I talked on the care of the ear, for example, as physiologist. We had GPs going around with the school. We had noise abatement officers. We had audiologists talking to the children. This was in a one or two-day intensive session with the students. But, extremely importantly, we had hearing-impaired people, either from Better Hearing Australia so they were hearing people who were losing their hearing, or they were profoundly deaf people.

So the important point there that was absolutely fundamental to this process of educating young children to understand the worth of their hearing and how to avoid hearing loss, is that the children—you could tell them an awful lot of things about how the ear worked and talk to them about the molecules inside the ear and how things work and how they're damaged. You could also tell them an awful lot about noise abatement and measurement of sound levels and how we have to control things in the workplace, so you would tell them about the management of noise at the workplace.

But, in the end, the thing that drove these children's learning was not scare tactics or lots of scientific data. The thing that really drove their learning process was the bond that they emotionally made with the mentors they saw in front of them. In particular, the Western Australian equivalent of Libby Harricks who was Ursula Holtz, an older lady who had a hearing impairment and was very active in promoting hearing impairment issues in Western Australia and a profoundly deaf lady by the name of Norma Levitski from the Deaf Society. The children were mesmerised by these two ladies explaining to them what it was to lose hearing and the problems that would occur if they lost it.

So the point I want to make is I want to touch this morning on the molecules. I want to go over some of the issues the children were introduced to. So I want to talk to you a little bit about the molecules involved in hearing and deafness. I want to touch a little bit about the management of the worksite to give us some insight of how we can avoid it using management techniques. But I think it's very important to emphasise that, in educating young children and also older farmers or miners, people need an emotional bond to get the message. They need mentors. So I want to talk about molecules, managers and mentors to understand the process.

So I'm going to start off then with the molecules, which is very much my domain as an auditory physiologist. I'll point out to you that I'm Italian, my father was Italian, so I talk rapidly and I apologise to the people in Sydney at the moment who are doing the captioning; I'll try to slow down—it's genetic. But also I teach this sort of stuff to medical students. Apparently I lectured James when dinosaurs ruled the earth in his medical course. But I normally teach this in days and weeks and months to audiology students and medical students and science students. So I'm going to give you a lightning tour of the ear but I hope I won't lose you and apologies if I go a bit too fast.

But I think it's important to understand what goes on in hearing to get an empowerment to understand what you've got and why you can't afford to lose it. Now, so here we go. Soundwaves, pressure fluctuations in the air, normally travel down, go past this high-tech reflector, the pinna, that gives us directional hearing. It travels down the ear canal and, eventually, the pressure causes the eardrum to wobble. The eardrum wobbles and then conveys the vibration through the middle ear bones that exist inside this air-filled middle ear cave.

Now, there are an important number of points to get from that. There's a lot of mythology that accompanies noise induced hearing loss. People think that, if they have a loud sound, there'll be bleeding from the ears if there's damage. The answer is no. The vibrations we are talking about that are damaging are atomic. I know because I've measured it in animals. The vibrations of these structures are atomic. You will not cause any ruptures of things. You will not get bleeding. You will not get any pain. So you will not have any warning that it is occurring. It's a bit like gum disease, okay. It's a bit like tooth decay but there's no pain to warn you and there are certainly no dentists around.

Now, there's another bit of mythology that goes along with this noise abatement issue and noise protection and that is that people seem to think that ... There are middle ear muscles inside the middle ear attached to the stirrup bone over here and also to the hammer bone over here. And the idea—we know that when this loud sound goes in these muscles contract, tense up, and people who know a little bit about hearing but not a lot have the mythology that this contraction or tightening of the muscles protects them from loud sound in some way. It's a myth. First off, the muscles fatigue after five minutes. So in an industrial environment, they have long given up before you get to morning tea. Not only that, they're slow to contract—a fraction of a second—so with loud impulse noises like a hammer, the damaging vibrations have gone through before the muscles contract. So the muscles in the middle ear offer no protection at all against normal noise induced hearing loss.

There's another bit of myth, and that is that ears toughen somehow or other on the worksite. The more you expose your ears to noise, the tougher they get—like they get calloused. Somehow or other these's a bit of machismo associated with these hearing structures. Well, I can tell you there are lots of advantages to lots of testosterone but it's got nothing to do with hearing. There is no machismo in the ear. All of these noises we expose ourselves to come from post-industrial age before evolution came to a grinding halt, okay. All of these things were there before we introduced industrial noise. You have no protection against these industrial noises—none. And don't believe it for one minute if somebody tells you that you do. So it's important that we get past that point.

But, anyway, once we get to the vibrating stirrup bone—no protection so far—that vibrating stirrup bone pulses backwards and forwards on the fluid inside the snail-shell structure here, the inner ear. The hearing part of the inner ear is known as the cochlear, the snail shell in Latin, and you can see why it's called a cochlear or a snail shell. The whole process spirals around and around. You can see a nice diagram of it here or, if you look down on the spiral, you can see—in fact, you can see all the nerve fibres going into the central core going to the brain. But the point is that when the sound goes into the inner ear, that is where all the action really takes place and you detect the sound, but that is the site of almost all of the noise damage. And that is why people don't understand noise damage, because they don't normally know much about this structure. This is my field.

Now, I want to point out as we go through this, there are cases of industrial noise damage that do create damage in the middle ear, but those are enormous pressure bursts from explosions or direct mechanical trauma to the head. Don't confuse those types of noise damage with the ongoing death of the cells inside the inner ear. Those sorts of pressure traumas are normally called baro- or pressure trauma. There are parts of the legislation that govern those once-off explosive events, but they are not the main source of damage in the worksite. Most of us don't undergo industrial accidents with explosions or mechanical trauma to the head.

The normal industrial noise damage that we're talking about here is a slow, insidious suicide of the cells that detect the vibration in the inner ear. It literally is a suicide. The cell kills itself, and I'll talk a little bit about the process in a little while. But the point is that, when the stirrup bone pulses backwards and forwards on this structure, there is a ribbon-like trampoline that spirals around here. You can see it in this little diagram here. There's the inner ear cut through so you can see it in cross section. You've got a little trampoline here. This is not air. This is salt water all around here. And the vibration travels along and that process is what's known as a travelling wave.

Now, you're all familiar with travelling waves. You see it most days of the week. Here are some travelling waves coming in on the surface of water with air above it. They're known as interfacial waves. So a lot of you are familiar with that type of travelling wave structure. The same thing travels along the structures inside the inner ear. And I might point out that many of you may not have noticed that you get the same process in the sky. This has got nothing to do with hearing; I just like the pictures. Next time you're wandering around, look up. You will see these travelling interfacial waves between the hot air and the cold air, and it's known as a

mackerel sky because it looks like the side of a fish. Sometimes the waves are travelling left to right and back to front, so you get two waves crossing, and the sky breaks into dots. So that's got nothing to do with hearing; I just like the pictures; but next time you're out there, look up and if you see that sort of sky remember travelling waves in the inner ear.

Now, the point about this is that the travelling wave inside the cochlear or the inner ear is extraordinary. I spent a large part of my research career studying this vibration inside guinea pig ears after doing the microsurgery to get inside of this thing. And the point is that the vibrations, when the stirrup bone here—by the way, the snail-shell structure has been unravelled to simplify the picture—as the stirrup bone vibrates backwards and forwards with sound, the ripple, or travelling wave, travels away from this end of the inner ear known as the base, out towards the apex, that's the point of the snail shell, in this travelling wave structure. As it travels, it grows, and what happens is—this is a diagram for a very particular note, like a particular whistle point put into the ear. It causes vibration at a very specific site along the ribbon, or the basilar membrane, so that high frequencies vibrate this end, low frequencies vibrate this end most, and all the frequencies are spread out along this ribbon beautifully like a player piano.

That's an important point in noise induced hearing loss, because that explains why some frequencies go and some don't. Depending on what you're exposed to, the vibration becomes too large in this area and those structures there are damaged and those cells die so that, when you are undergoing a noise induced hearing loss, you may not notice initially because some frequencies are going but some aren't. That's one of the reasons it's insidious. It's slowly accumulating but it can affect only some frequencies initially.

Now, this vibration is extraordinary for another reason. It's atomic. It's incredibly small, and that's why it was so difficult to understand this process for many years until we developed the experimental procedures to understand it. Not only is it very, very small but the vibration occurs over an incredible range of sound level. The softest sound produces atomic movements but, as we go to louder and louder sounds, the vibration does not grow proportionally. That's extraordinary. So you can increase the sound level from a whisper to a jet engine, which is a million times more sound, and yet the vibration only grows 100 times. That's the secret to how we manage to squeeze so much sound level information into our ear without it being blown apart.

Why do I mention that? Because that is what you lose if you damage the structures. Not only do you lose sensitivity, you will lose frequency selectivity—you won't be able to discriminate different pitches, but you also lose the ability to squeeze a million to one range of sound loudness into a 100 to one range of vibration. That's an extraordinarily valuable thing to lose. Now, here's another issue I want to raise. Because sound that we listen to can be over a million to one range, hearing is dogged by too many zeros and Australia, as many of you know, has a terror of Nooleus. Okay. The joke was funny about 3 o'clock in the morning! But there are too many zeroes in hearing, so what I want to explain is the decibel scale that might help you understand the legislation a bit better.

First off, if we say one unit of vibration or sound is the softest sound we can hear, then it's easy to understand you could have 10 times that vibration, 100 times that vibration, 1000 times as much sound, 10,000, 100,000, 1,000,000 times more sound vibration in the air. That's easy to understand. But there are too many zeroes. So we simply write down the number of zeroes as a code, like the Richter scale for earthquakes which, unfortunately, is rather untimely to mention that. But no zeroes there, one zero there, two zeroes there, three zeroes, and so you can see we can summarise the range of sound level by just having zero to six. That's a little bit too coarse. So instead of having zero, one, two three, we have zero 20, 40, 60, 80. That is the decibel scale. That's all there is to it. So if I say to you that a sound is 40 decibels louder than it should be, I mean it's 100 times louder than it should be. Or if I say you have lost 60 decibels of your hearing, it means you've lost 1000 times your sensitivity. See? So that's how the decibel scale works.

So you now understand, when you look at a scale like this—this little bar chart here that summarises the sorts of sound levels we're exposed to—a very quiet room may only be 20 or 30 decibels, only about 10 to 30 times the softest sound we could hear. You can go up to the average home, depending on how average your home is, it may only be 60 decibels or so, which is relatively quiet. So you can go through this whole range. The point I made earlier on is it's only when you get to firearms and artillery fire that you get to enormous sound levels louder than a jumbo jet. It's only at the extremely large sound levels at 130 or 140 or 150 decibels do you actually rupture structure. That isn't the normal noise induced hearing loss.

But the law has to cover that one-off event. So there is part of the legislation that says that nobody will be exposed in the worksite to a sound louder than 140, which is that peak sound level. So one part of the legislation says you must not go above 40 decibels because then you will rupture things with a barotrauma. But there's another part of the noise abatement legislation that covers this grey area here. As it turns out, above 85 decibels, there's a region here of sounds that don't rupture things directly but cause the cells inside the ear to commit suicide slowly, and that's what we're going to talk about now.

So what are these cells that commit suicide? Well, I've told you a little bit about this vibration that travels along the cochlear spiral like a travelling wave, a ripple in a pond, and it peaks at certain distances along there, depending on its frequency. But the brain needs to know that that vibration is going on, so you have to have specialised cells sitting on top of the trampoline, as shown here, which detect the vibration. They're called hair cells, for the simple reasons that—there's one of the cells there in cartoon form, and it's got these little protrusions at the top that look like hair. They're not hair; they're actually the cell membrane poked through like the fingers of a glove but they look like hairs under the microscope.

Those hair cells come in two types. They're called inner hair cells and they're in a very long row with one row of inner hair cells along the whole spiral, and outer hair cells in three rows. Why are they called inner and outer? It's because it's a spiral structure. The inner ones are on the inside of the spiral and the outer ones are on the outside of the spiral. Why do I tell you about these things? Because they are the key to how we hear and they are the things that die with loud sound. Let's have a quick look.

Inner hair cells, as it turns out—let's have a quick close up of them. They're the single row of inner hair cells that have the hair bundles poking out the top, and when the vibration of the trampoline underneath happens, the hairs get pushed backwards and forwards. And what happens is it opens up little molecular trapdoors at the top that allow the salt inside here to flow through the cell as an electric current. By the way, the same thing happens in the petrol tank of your car, the float of the petrol tank. As the float goes up and down with the petrol level, it opens and closes a resistance that allows more or less current to flow. These things are equivalent, if you like, to a float level inside a tank, a petrol tank.

But when that electrical signal occurs inside these cells, it controls the release of a special chemical that stimulates the nerves. There are about 10,000 of these nerves in each one of your ears—10,000 of them—about 20 of them attached to each one of these hair cells. Now, an interesting point to note—interesting in terms of hearing loss, noise damage—is that these inner hair cells here have 95 per cent of all the nerves—95 per cent. That means our brain—we—hear through these cells. These cells, the outer hair cells, do more or less the same thing, but they're not connected to the brain very much, and this was a great puzzle to us for many, many years. How come we've got more of the outer hair cells and yet they're not connected to the brain?

We couldn't understand for many, many years in auditory research what was going on until there were some clues. There was a whole sequence of clues that told us what was going on. The first clue came from the invention of a brand new antibiotic to save children with pneumonia. It was called Kanamycin. It was given in high doses to children who were dying of pneumonia to save them. But what happened was, when they recovered from the pneumonia, people realised that they now were deaf. Now, this caused—you can imagine the quandary going on there. Very soon after, some of the children who'd died on Kanamycin treatment donated their ears. People looked inside the ear and found that these were the cells that had died from the Kanamycin overdose. So we knew that they were crucial to hearing but we didn't know why because they weren't connected to the brain.

Then another series of clues came through from the research labs across the world and in ours, and one of them was that almost every one of you has—at least one of your ears is whistling. And if we could put a small microphone inside your ear, we could hear a little whistle, like a whistling kettle, coming out of one or both of your ears. Just about everybody has got a whistling ear—quite soft but it's there. That shouldn't happen if the ear is just being pushed around by the sound. It was a telltale sign that the ear was moving in some way. Another telltale sign was that, if you made a clicking sound, echoes came back and the echoes were far too big for it just to be a passive process. Something was kicking back.

Then another breakthrough came at our lab over at UWA there where we perfected the surgery and the technical process to do the surgery to get inside this structure and drop a minute speck of radioactive metal to

sit on the vibrating membrane and measure the radioactivity coming off it. And we would measure then atomic movements. What we discovered was extraordinary—that the vibration was 1000 times more than anybody had measured previously. To cut a long story short, what happened was every time previous researchers had done the surgery to get inside the structure, they had produced noise induced hearing loss and only ever measured from a deaf animal. But we managed to get the surgery so sensitive and delicate that we measured that the vibration was 1000 times more than it should have been. Now the problem was how come the vibration is so good? But, as we watched the vibration in the animal experiments, over an hour or two the vibration faded away. The animals went deaf and we didn't know why. But at least now we knew that the vibration was excellent and that the vibration was being damaged.

Here you can see a close up of the top of the hair cells. Something was going on and it probably had something to do with these outer hair cells but we didn't know what until some colleagues of ours did an extraordinary thing from Baltimore in the United States. They released some of these outer hair cells in a dish by using a little bit of enzyme like Bio-ad, separated the cells, put them on the end of a wire and passed electric current. And what they discovered was the outer hair cells, although they didn't talk to the brain directly, twitched every time they got an electrical signal.

Suddenly the penny dropped for all of us. The outer hair cells didn't talk to the brain directly. What they did was kick every time the wave came past so that, as this vibration wave, the travelling wave along the membrane came past, they all jumped up and down in time with the electrical signal that was coming through their hairs. So what you had was exactly the same as a child on a swing. Every time the child moves in one direction, they kick their legs to assist. Suddenly we understood what the outer hair cells did. They didn't talk to the brain directly. What they did was they acted as a built-in hearing aid so that any time a soft sound came in, the vibration was amplified by these built-in hearing aids and that good vibration was passed on to the inner hair cells to tell the brain what was going on.

So we now understood. We had two processes, quite different, going on inside the ear. We had the simple process where the vibration bent the hair bundles on the inner hair cells, caused the salt to flow, caused an electrical signal, made them release a chemical that stimulated nerves to tell the brain what was happening. But, at the same time, the outer hair cells were also causing an electrical signal. But they didn't release chemicals to tell the nerves what to do. All they did was kick in time and help the vibration. Finally we understood what noise damage was. These outer hair cells are being killed off by the sound in the factory or on the farm. And what happens first off is, as they slowly die, the vibration inside your ear gets the internal hearing aids turned off and the vibration gets less and less and less until, eventually, you've got a terrible vibration pattern inside your ear. The inner hair cells and brain know that something is going on but the vibration pattern is totally abnormal.

Now, we did a lot of—I did a lot of—research as well on how it was that this damage accrued; how it built up and recovered over time. Here's a ridiculous picture of me doing some experiments back in the mid-'90s or so, where I wanted to find out how hearing loss came on and how it recovered. And I used to wear this thing on my head for about a week at a time, making a very, very loud sound into my ear, watching my hearing get worse over days, and then watching how it recovered. And what I discovered with those sorts of experiments—which spooked an awful lot of shop assistants. I can tell you, when you walk in with a headband on looking a bit like Rambo with a loud whistle coming from your rear, you had great service, I might say, because they want you out of the shop as fast as possible.

But we discovered what was going on at the top of the hair cells. The temporary hearing loss you get after loud sound is because, as you over-wiggle the outer hair cells, the trapdoors at the top twist out of shape and don't work for a while. But if you leave them for a while, the trapdoors snap back into shape and things start working again. That caused temporary hearing loss. The good thing about those experiments was it also told us the mathematical link that the more outer hair cell help you lose, we can tell you how many decibels you'll lose. So we had a beautiful mathematical description that described, if we lose 50 per cent of the outer hair cell help, how much of a decibel hearing loss will you have. And it all started to fit in.

What we didn't discover, and only has been discovered more recently, is the link between the temporary hearing loss that we've all experienced and the permanent cell death. It turns out that—this is research that's not so much from our lab but comes from world wide and also, I might add, from the people over in Melbourne and Sydney working on this as part of the HEARing CRC. What has since been discovered since we

did those sorts of experiments is that, inside every one of your cells—not just hair cells but every cell in your body—is a very complex interplay of chemical messengers—signals—chemical message going left, right and centre. They're all interacting in a very complex dance inside the cell.

Now, we don't exactly know how it happens and how it's triggered, but we do know that, if the loud sound is there, on the farm or in the mine or in a factory, for too long, the chemical messages are triggered into having these cells kill themselves. You trigger a process known as apoptosis. Many cells in your body have this process going on. That's how you get the gap between your fingers. When you're in utero, before you're born, the tissue is joined together, but the chemical suicide goes on, and so the cells that join your fingers together die off to leave you with gaps. So apoptosis is not necessarily a bad thing. It's a bad thing when you trigger the process inside the inner ear and they kill themselves. That's what loud sound is doing very slowly.

So what you end up with is, instead of—here's a beautiful picture looking down on the inner hair cells that talk to the brain and the outer hair cells as the built-in hearing aids. That's the normal look—different haircuts, different hairstyles. After loud industrial noise, you don't notice it but what happens is the cells start killing themselves with this process of apoptosis and you're left with scar tissue. So you start to lose a percentage of your outer hair cells. You lose a percentage of your built-in hearing aid, and your hearing gets worse and worse and worse.

If it goes on for too long, not only will you lose your outer hair cells but the inner hair cells also start to go. So not only do you have bad vibration—you don't have built-in hearing aids—you no longer have a way to tell the brain that there's any vibration at all. And not only that, the nerves connected to these inner hair cells die. They also undergo apoptosis. And so you end up—here's that cochlear spiral, the inner ear spiral, going from high frequency down to low frequency. You might notice—that light area here is the basilar membrane trampoline I was talking about before, the ribbon. Note there are the rows of hair cells shown as these little stripes—three rows of outer hair cell, one row of inner hair cell. Look what happens when there's loud industrial noise for too long. The hair cells have committed suicide. There's nothing left. If they go, there's nothing to keep the neurons, the nerves, around. They also die off.

The problem there is that, if you do that, you've got no communication between the vibration and the brain any more. I don't care how many hearing aids you can buy, how many cochlear implants you can get, you're not going to hear anything. You cannot let it get to this stage. What that means is that you end up with—normal hearing on one of these charts goes from low frequencies down here to high frequencies up here. You have soft sounds at the top, and zero decibels is the very soft sound as I explained before, to very loud sounds a million times is 120 decibels, and that's how you represent your hearing ability. So you've got a very soft medium-frequency rustle of leaves there, a screaming high-frequency jet engine there, a low-frequency rumble of a—loud low-frequency truck up there. So you get a picture of the auditory world.

What happens with industrial noise damage is that, first off, the hair cells in this frequency region go first, and you end up with a notch in your hearing at these medium frequencies here. It's known as a noise notch. That's the first sign that something is going wrong. But if you keep the noise up for too long, the notch gets deeper and deeper and deeper and you end up with what we call a ski-slope audiogram where you've got no high frequencies. What does that mean for anybody who has it? Well, here's a phrase. Without any high frequencies—"E-Ls E-Ls on the E-or." Here it is with only the high frequencies—*(in a whisper)* "She sells sea shells on the sea shore." She sells sea shells on the sea shore. Most of the information is at the high frequency. If you lose that, all you will hear is "E-L E-L on the E-or." Now, you try following a conversation at the family dinner table under those circumstances. It doesn't work.

So what you lose with occupational noise induced hearing loss is you lose a lot. You lose the sensitivity. You need to amplify sounds to hear it at all. You lose your ability to discriminate pitch. You lose your ability to cope with a very loud range of sound level, from whispers to jet engines. You lose timing discrimination, so you can't tell left from right—where is the sound coming from? You, therefore, start to lose your ability to discriminate speech in the background noise. You can't follow conversations at parties or at the workplace or at the dinner table. You start to lose your self-confidence. You start to lose social interactions because you don't want to go out, okay. You lose an awful lot of things. You get aggravation, frustration and an awful lot of other things.

You can provide amplification. You can get sophisticated amplification. But, in the end, to get anywhere near correcting this, you need very sophisticated hearing aids that are microcomputers inside your ear. That's why

they cost a lot of money. They have to be fitted properly. You need good diagnosis, good devices, good price, good fitting, good professional back-up, attitude, counselling. With all of that, why would you let it happen in the first place? Why would you let it happen? We can't afford young people to allow it—we can't afford to have it happen.

So how can we avoid it? Well, I've told you a lot about the molecules. We need management. Now, a lot of you are very good managers and much better managers than I will ever be. But, in the workplace, I will just make this point that many of you would know about, but I'll just make the point very, very briefly—that when you are managing occupational noise induced hearing loss at the worksite, you need to follow what's known as the hierarchy of control, and I can very, very briefly go through it without the jargon. You need elimination—and I'm going to translate this into rural speak. "Do you have to use that noisy thing around here?" Substitution: "Oh, for gawd sake, go and use the new one will you; it's a lot quieter." Engineering controls—isolation engineering controls, source engineering controls, controls in transmission—that translates to, "And if you're going to use it, stick it in the back of the shed, put the muffler back on and, for gawd sake, put the box over the top." That's what that translates into. Administrative controls—"Don't go anywhere near it and if you do"—and the last one, hearing protection—"If you do, put some earplugs in." That's translated into something we can understand.

You need a workplace culture to do that. Don't start with hearing protection. Hearing protection is one aspect, but it's not everything. You need to get good advice on what hearing protection works and you certainly need to choose the right ear protection for the right situation because if people don't want to use it, what's the reason for giving it to them? Let them and the authorities tell you what the best thing is for the situation.

Now, I'm going to end off with a parable of the carpenters. This is not the brother/sister singing duo. This is my sister who married the son of a carpenter. Her husband became a carpenter, and they gave birth to a son who was a carpenter—apparently it's genetic—three carpenters in the family—and every one of those three have given themselves a ski-slope audiogram, every one of them, and that's with me in the family, all right. Now, that's why we need to understand how can somebody who is told it's a problem not look after their ears? I can't say much more—just to point you in the right direction if you're not familiar with this at all.

When you go to educate people and understand why they're not doing it, you need to have a psychological theory—a behavioural or management theory—behind you. And a very good one to start with is known as the theory of planned behaviour. Every minute of the working day, people are making risk assessments and making decisions on the run. What's going through their head? In the theory of planned behaviour—I'll summarise it in block diagram—everybody has a belief about the behaviour. "I think ear protection works." They believe that it works, but they have an attitude to that. "But I'm not like everybody else. I've got tough ears and I don't need it." Okay. Or they have norm beliefs—"All carpenters don't wear ear protection. I'm a good carpenter. I'll get tough ears. I won't wear ear protection." Or control beliefs—"I know that I can go down to the store and get some ear muffs. I believe that, but I'm not going to wear them because I look stupid in them."

So all of those three aspects combine to give the intention to either behave in a certain way or not behave in a certain way, and even when you've got an intention, things can go wrong. "I was going to put my ear muffs on but Trevor's taken off with the jeep and all the gear is in the back." Okay. So we have to understand how people make decisions that way. So what that means is we have to design education programs that suit the psychology of people. It's not going to be a one-size-fits-all that educates children or adults in the workplace.

And on that point, I just want to finish with this few points. There's a lot of kerfuffle going on about MP3 players and preaching the gospel of MP3 temperance. Hallelujah, it's the MP3. It's this new funky music that's causing all the problems. It's true that these MP3 players are a dangerous item and people need to be educated in their use, but I think we need to be very, very careful that we don't overdo it and make these kids just tune out to any education whatsoever. It's got to be a very balance education program that isn't looking for scapegoats. I think that's extremely important in whatever we do.

Now, finally, are things getting better or worse? Well, the data is hard to get because a lot of people aren't tested, especially in rural areas where you don't have the support services and the screening processes. So you really have to keep an eye on things in rural sectors because you're not getting the data for the feedback. But

be very, very careful of compensation claims as an indicator. Why? This was pointed out by the Senate of Australia in their report.

Look, here's the compensation claims in 1998 across Australia to 2003—6000, 4000, 4000, 3900, 3800—it looks as if things are getting better, doesn't it? Well, that drop there was because they changed the regulations and you have to have twice as much hearing loss to get a compo case. So that's not really cause for joy. But also, look, this drop here is also caused by other aspects including casualisation of the workforce. Who's to blame for my hearing loss? If the regulations are getting better and employers are getting better at following the regulations there will be less compensation claims but it won't mean that people's hearing is getting any better. It's just that the employee can no longer sue.

So it all focuses in that we have to have a functioning education program to make people look after their own ears. Now, we have to do it at primary school and high school level to make them understand, but we also have to run adult education. We clearly have to start cleaning up this mess very early and we need mentors at a very young age and also on the worksite to help these people get the message. Decibel Danger was extremely useful for me at least in understanding what the dynamics of the education process was, how well children respond, and adults as well respond, to an understanding of the science, the molecules, of the management—they understand that there's complex issues involved—and the mentors. But they need people to look up to, models in the working world, to say it's okay to behave in this way that protects hearing.

Last point. Please go and download and read through these documents. They are a mine of information to tell you how bad things have got and that we need to start acting on, and it also tells you that we haven't done enough already. I'll leave you with that and thank you very much to the organisers and for your time this afternoon.

