Noise-induced hearing loss: a modern epidemic?

ABSTRACT

Hearing loss is an increasingly common problem in the UK, and noise-induced hearing loss is the second most common acquired cause. There is a greater burden on the younger population, as recreational noise exposure is an important contributor. New damaging hearing behaviours have emerged including loud music exposure at concerts, nightclubs and via personal music players. The mechanism of damage is thought to involve many diverse pathways, which include oxidative damage, mechanical shearing forces and glutamate excitotoxicity. Although no current treatment exists to reverse the damage caused, these pathways can be targeted by agents that are being trialled for use in post-exposure treatment and prevention, with conflicting results. Other preventative strategies are important in addressing damaging hearing behaviours, including 'safe listening' promotion with advocacy materials for young adults, and promoting safe listening devices.

The prevalence of hearing loss in the UK is approximately 11 million, projected to reach 15.6 million by 2035 (Action on Hearing Loss, 2016). This may be the result of numerous factors, including greater noise exposure at a younger age increasingly contributing to noise-induced hearing loss. The World Health Organization (2016) has estimated that 1.1 billion people in the 12–35-year-old age group are at risk of hearing loss as a result of chronic exposure to recreational loud sound. This article addresses what is becoming an increasingly common issue and explores the mechanisms, treatment, prevention and future management options of noise-induced hearing loss.

Noise-induced hearing loss

Noise-induced hearing loss is the second most common cause of acquired hearing loss after presbyacusis in the UK (Wong et al, 2013). Although it is most common in the older age group (over 65 years of age), there is an increase in the number of children and young adults affected (Shargorodsky et al, 2010). High levels of noise exposure can stem from occupational noise, such as in the construction, mining or aircraft industries. This often consists of 'impulse' sounds, such as gunshots, defined as short duration (<1 s) and high-level sound stimuli. This can be more damaging, with quicker onset hearing loss, than chronic noise-induced hearing loss associated with continuous noise stimuli such as background noise or music (Wong et al, 2013).

More recently, recreational noise exposure has become increasingly significant in noise-induced hearing loss, especially affecting adolescents and young adults (Shargorodsky et al, 2010). It has been suggested that listening to sound at levels exceeding 89 dB for more than 5 hours per week can cause permanent hearing damage over time (Action on Hearing Loss, 2016). Listening behaviours of adolescents and young adults include attendance of music concerts, nightclubs, and the increasing use of personal music players (Jiang et al, 2016; le Clercq et al, 2016), which often exceed this 89 dB threshold, as seen in *Table 1*.

The change in music listening behaviour, with a shift towards a preference for and increased exposure to louder music, may be multifactorial. The influence of gigs and nightclubs (where music is often played at high sound levels) may contribute to a 'loud music culture', and thus establishes a norm for music listening enjoyment. Furthermore, increasing use of personal music players with improving headphone technology allows masking of background noise using high sound levels of music. Concurrently, there is an increase in background sound levels that can further contribute to noise-induced hearing loss, especially in urban environments where major contributors include traffic, construction work and pedestrian activity (McAlexander et al, 2015).

Risk factors for noise-induced hearing loss are thought to include smoking, lack of exercise, poor diet, diabetes, tooth loss and congestive heart disease. Such risks overlap significantly with symptoms of the metabolic syndrome

Table 1. Decibel chart

Decibel level (dB)	Source
0	Quietest sound audible
30	Whisper at 1m
50–65	Normal conversation
75–105	Personal music player
120	Nightclub
110–140	Rock concerts

From Daniel (2007)

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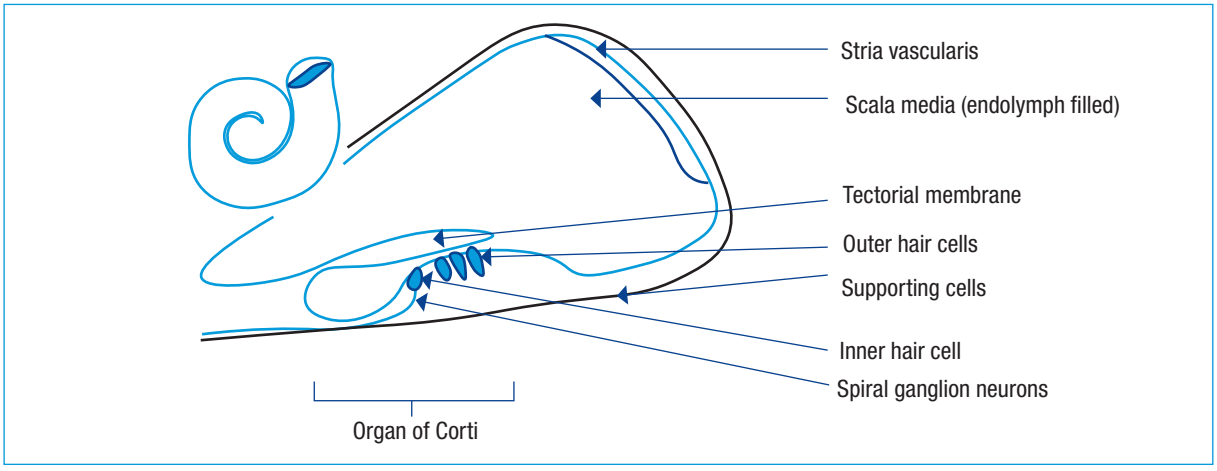

Figure 1. Cross-section through the scala media in the cochlea.

(Daniel, 2007). This refers to the presence (with specific thresholds) of three or more factors, including increased waist circumference, high blood triglyceride levels, hypertension, hypercoagulable states and insulin resistance. It is thought that this can increase the risk of developing type 2 diabetes mellitus 5-fold, and cardiovascular disease (e.g. myocardial infarction, stroke) up to 2-fold during the following 5–10 years (Kaur, 2014). This suggests a potential role of impaired vascular supply to the inner ear and cochlea in the aetiology of noise-induced hearing loss. Thus, a greater prevalence of metabolic syndrome symptoms in the population could be a target for prevention of noise-induced hearing loss. Non-modifiable risk factors of noise-induced hearing loss include age, genetic predisposition and race (Daniel, 2007).

Temporary and permanent threshold shift

Initially, high noise exposure can result in tinnitus and temporary threshold shift (transient sensorineural hearing loss that recovers over approximately 24 hours after removal of the sound stimulus). If this exposure is repeated over a prolonged period of time, with little or no recovery between temporary threshold shift episodes, the hearing impairment can evolve into permanent threshold shift (Wong et al, 2013). This can be assessed via audiological investigations; higher frequencies are most commonly affected, often between the 3–6 kHz range with a characteristic narrow dip around 4 kHz.

During early stages of clinically apparent noise-induced hearing loss, the higher frequency loss results in difficulties with speech discrimination in high levels of background noise, especially higher-pitched voices (women and children) (Mathur, 2016). This damage is greatest with high frequency, high intensity and pure-tone noise stimuli (Wong et al, 2013).

Although there has been controversy about whether loud music exposure does indeed cause a risk of hearing loss, Zhao et al (2010) argue that discrepancy between studies is in part a result of the sensitivity of the audiological measurements used. For example, Meyer-Bisch (1996) proposed that Audioscan is a more sensitive measurement of early cochlear damage than the pure tone audiometry measurements used in older studies. Audioscan involves using frequency sweeps as a variable, relative to constant hearing intensities. This contrasts with pure tone audiometry that uses an intensity sweep with a constant frequency sound stimulus during testing. Hence, a sound stimulus of constant intensity will play, changing in octave increments until response is achieved (the subject presses a button when the sound is heard). Testing is continued with higher intensity sound, testing frequencies with smaller increments, outside of the response range. A detailed audiometric curve can thus show narrower inter-frequency notches than are tested in traditional audiometry, correlating with mild audiological deficits (Meyer-Bisch, 1996). Furthermore, Zhao et al (2010) conclude that sufficient evidence exists to support noise symptoms being closely related to music exposure, based on an analysis of 126 studies of music exposure and hearing loss. Zhao et al (2010) suggest that these noise symptoms (including temporary threshold shift, tinnitus and hyperacusis) can be used as an indicator of a high risk of developing music-induced hearing loss.

Physiology of hearing

Important structures in the physiology of hearing include the inner hair cells (*Figure 1*), which are situated in the organ of Corti (sensorineural organ of the cochlea), and comprise actin-containing stereocilia. These move in response to the vibration caused by sound waves and transmit auditory neurotransmitter signals via electrochemical coupling to the spiral ganglion neurons. The greater the intensity of sound, the greater the transmitted vibration of the membranes forming the superior and inferior borders of the scala media (Reissner's and basilar membranes respectively). This results in a greater displacement of the inner hair cells relative to the tectorial membrane, to produce a stronger neuronal response at the spiral ganglion neuron to be transmitted along the cochlear nerve and beyond. Destruction of these cells in noise-induced hearing loss, as well as outer hair cells

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(responsible for amplifying the vibration stimuli), causes sensorineural hearing impairment.

The hair cells in the cochlea do not have the ability to regenerate, unlike those in the vestibular system, or in the cochlea of amphibians, birds, reptiles or fish (Wong et al, 2013). The hair cells in these organisms are found in the lateral line system and inner ears, and are continually regenerated throughout life and in response to damage (e.g. following acoustic trauma) (Monroe et al, 2015). The lateral line system allows detection of movement, water vibration and pressure gradients that are important in orientation, detecting predators and allowing schooling behaviour. This has triggered interest in research into the use of stem cells in regenerating hair cells to replace those damaged, as a potential treatment for noise-induced hearing loss. However, as several factors (to be addressed later) prevent this from currently being a clinically viable treatment, the current management of noise-induced hearing loss places a greater emphasis on prevention rather than treatment, to help face the anticipated rise in incidence.

Mechanisms of damage

There have been several theories proposed in the aetiology of noise-induced hearing loss, which may be clinically significant in future to provide novel targets for the treatment or prevention of hearing loss.

Two types of postsynaptic afferent auditory neurons exist (i.e. spiral ganglionic neurons). The first is a low threshold, high spontaneous activity rate neuron, which is responsible for setting sensitivity levels to auditory stimuli. The other type has a high threshold with low spontaneous activity, so that it is only activated with supra-threshold stimuli. Although both types are affected in temporary threshold shift, there is preferential damage to the high threshold type of spiral ganglion neuron (particularly true in permanent threshold shift at early stages), so that lower threshold stimuli detection is spared for the specific frequencies affected. The higher threshold response range is important for speech recognition, especially in settings of high background noise, which is characteristic of the impairment observed in noise-induced hearing loss (Wong et al, 2013).

The predominant mechanism of damage was, until recently, believed to include mechanical shearing forces resulting in hair cell membrane rupture, with damage to the stereocilia. This is because the amplitude of vibration waves transmitted along the membranous labyrinth is so large that the inner and outer hair cells uncouple from their attachments to the tectorial membrane, with tip link damage. These stereocilia can re-establish their contact points with the tectorial membrane, so that the threshold shift is only temporary. With repeated noise exposure, these forces also cause dendritic swelling in postsynaptic cells (the spiral ganglion neuron), with cellular damage and apoptosis of the stria vascularis and other supporting cells in the organ of Corti, critical for the functioning and survival of hair cells (Wong et al, 2013). This theory co-existed with that of impaired blood flow to the structures of the cochlea resulting in ischaemia, as a result of intense sound stimuli.

Oxidative damage

More theories of damage mechanisms emerged, including reactive oxygen and nitrogen species inducing damage, produced by intense metabolic activity (Lim and Melnick, 1971). High mitochondrial activity caused by a high sound stimulus is thought to result in the formation of free radicals within 1–2 hours, which can cause damage to vulnerable cellular DNA. However, there is evidence of a delayed formation of free radicals up to 10 days post-noise exposure, which could be significant in providing a time frame in which to intervene to prevent damage. Several cellular damaging processes may be involved as a result of the formation of reactive oxygen species, including caspase-mediated apoptosis and release of pro-inflammatory cytokines (e.g. interleukin-6, tumour necrosis factor-α). Limiting these processes may be investigated as future therapeutic targets in post-exposure prevention of noise-induced hearing loss. The role of impaired flow to the cochlear structures could also result in production of reactive oxygen species as a result of ischaemia, which would lead to further impaired flow, thus causing a positive feedback loop of free radical formation and ischaemia (Wong et al, 2013). Hence, the metabolic syndrome symptoms and noise exposure may have synergistic roles in ischaemic effects, oxidative damage and noise-induced hearing loss.

Further pathways of damage

Further pathways thought to be involved in noise-induced hearing loss include glutamate excitotoxicity, causing damage to auditory neurons, with evidence of a large release occurring in response to loud noise exposure. An impairment in calcium homeostasis has also been proposed, leading to cell death in the short term via phospholipase A2 activation, with calcineurin-mediated cell damage in the long term.

Hence, since many pathways of damage are implicated, this gives rise to numerous potential targets to allow intervention in the treatment and prevention of noise-induced hearing loss. Furthermore, genetic variation in the pathways between individuals (e.g. function and amount of enzymes implicated in the pathways mentioned) leads to differences in susceptibility to noise-induced hearing loss with equal noise exposure between individuals, which could result in targeted advice and prevention schemes aimed at identified susceptible groups (Le Prell et al, 2007).

Treatment

Owing to the lack of regeneration of the cochlear hair cells, the mainstay of management in noise-induced hearing loss includes the use of hearing aids and potentially cochlear implants. This relies on enhancing remaining cochlear function rather than reversing the damage caused.

Several trials are being undertaken to discover post-exposure treatment options to prevent or treat damage caused in noise-induced hearing loss, revolutionizing future management of noise-induced hearing loss. Potential treatment options include dexamethasone, ebselen (enhances glutathione peroxidase action), and N-acetyl-L-cysteine (repletes glutathione stores) (Lynch and Kil, 2005). Modest benefits have been shown in animal models and at various stages of clinical trials thus far using these agents, although conflicting results between studies show that there is still much research to be done (Oishi and Schacht, 2011). Limitations to efficacy may include the complicated and overlapping pathways implicated in noise-induced hearing loss, so that targeting one pathway may only have a limited effect (Le Prell et al, 2007). Furthermore, the target molecules are not unique to the cochlear tissue, hence significant unwanted side effects may limit use of the treatment and prevent systemic administration. Despite this, emerging treatments include AM-111 (intratympanic injection), which has shown promising phase II trial results, with phase III clinical trials (HEALOS, ASSENT) currently being carried out. Its action is via inhibition of the JNK stress kinase, which is involved in apoptosis and acute inflammation in response to acoustic trauma (Wong et al, 2013).

Neural stem cells also provide a target for future research as a means of replacing damaged cochlear cells (Shi and Edge, 2013). Great advances have already been made, with reports of growth of auditory epithelia with sensory function from murine embryonic cells in vitro. However, barriers such as tumour formation and graft rejection, which are generally common to all stem cell treatments, prevent this from being clinically relevant. Further obstacles include difficulties in assessing extent of damage and delivery (and successful integration) of the generated hair-like cells, posed by the anatomical location of the cochlea (Park, 2015).

Prevention

Because of the lack of current treatment options, the importance of prevention and hearing education has an even greater role. In industries with a high prevalence of occupational noise-induced hearing loss, the employer is required to comply with 'noise at work' regulations. Under the Control of Noise at Work Regulations 2005, legal limits have been set that require action to be taken if daily noise levels exceed 80 dB, including providing noise-limiting equipment and reducing exposure to the noise with adequate rest.

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Hearing education programmes

Owing to the increasing relevance of recreational noise exposure in contributing to noise-induced hearing loss, the effectiveness of achieving behavioural change via hearing education programmes targeting school-aged children has been explored, although limited research currently exists. Keppler et al (2015) looked at the effect of behavioural change in young adults 6 months after a hearing conservation programme, with knowledge of their hearing status. There was a modest increase (12%) in the self-reported use of hearing protector devices, although the lack of a control group makes the results difficult to interpret. This could be an area for future research, encouraging the establishment of national hearing conservation programmes in secondary schools.

Several difficulties may still persist despite the use of educational schemes. For example, it may be difficult to sustain safe listening practices, unlike in occupational noise-induced hearing loss where organizations (i.e. employers) are accountable for following regulations. Furthermore, compliance with hearing protection devices (e.g. earmuffs or earplugs) may be poor in young adults as a result of discomfort, peer pressure, cost and difficulty in hearing speech. Manufacturers may be able to address these concerns by designing more comfortable and 'stylish' protection devices (Keppler et al, 2015).

Safe listening initiatives

Action on Hearing Loss have encouraged 'safe listening' by setting out regulations (in association with the European Union) for MP3 player manufacturers. These include providing guidelines to inform users about safe listening on the packaging and in custom settings of the product (Action on Hearing Loss, 2016). Furthermore, the European Commission in 2009 set a standard of 85 dB as the output level in new audio devices (World Health Organization, 2015a). However, it is worth noting that this level is higher than the 80 dB level used as a threshold for intervention in occupational noise exposure.

Furthermore, the World Health Organization launched a 'Make Listening Safe' initiative in March 2015, which includes providing advocacy materials for young people, promoting safe listening devices, developing software for safer listening and advancing research in this field (World Health Organization, 2015b). The overall aim is to limit the potential harm that young adults subject themselves to by targeting and regulating manufacturing of personal listening devices and headphones, setting maximum

KEY POINTS

- There is a rise in the incidence of hearing loss, with a higher proportion of young adults and children being affected by noise-induced hearing loss than previously.
- There are new listening behaviours in young population groups, including attending music concerts and night clubs, and using personal music players with headphones.
- Several pathways are thought to contribute to the pathology of noise-induced hearing loss, including mechanical shearing forces and oxidative damage from reactive oxygen species.
- No effective treatment currently exists to reverse damage caused, but post-exposure treatment and prevention is being trialled for several agents, including AM-111 and neural stem cells.
- Prevention is important because of the lack of treatment, and initiatives by the World Health Organization and Action on Hearing Loss are targeting this with 'safe listening' promotion.

volume levels in nightclubs and concerts with the provision of hearing protection devices, and promoting 'rest areas' with music at a lower volume.

Smartphone applications (apps) can be used to measure sound levels, providing a quick and convenient means of identifying unsafe sound levels to determine when noise-limiting equipment would be warranted. Although testing has shown the best performing apps to reach an accuracy within a 2 dB range of true noise levels (SLA-lite, SoundMeter), performance limitations include phone age, user calibration of the app, and varying phone models, with Android systems generally producing poorer results. Thus, the risk of incorrectly identifying a harmful noise level as safe may result in the widespread use of these apps causing more harm than providing benefit (Murphy and King, 2016).

Conclusions

With the increasing use of personal music players, along with compounding high noise exposure in nightclubs and pubs, new population groups are being affected by noise-induced hearing loss. Treatment to reverse cochlear damage is limited, and although ongoing research into post-exposure treatments is proving promising, the most effective management strategy is likely to focus on prevention of loud noise exposure. Accordingly, responsibility for tackling this increasingly prevalent issue is widespread, including that of the individual, pub and nightclub managers, manufacturers (headphones, personal listening devices) and the government (education programmes, legislation for maximum volume settings). Facing a potential future of a young population afflicted by hearing loss and its socioeconomic consequences, awareness of noise-induced hearing loss should be a pressing concern. **BJHM**

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