International Journal of Research in Medical Sciences
Kadavil SV et al. Int J Res Med Sci. 2025 Apr;13(4):1746-1753
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20251012

Review Article

Noise induced hearing loss-a review of literature

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Received: 11 February 2025
Accepted: 12 March 2025

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ABSTRACT

Noise-induced hearing loss (NIHL) is one of the most prevalent occupational and environmental health concerns worldwide. First identified as "boilermaker's disease," NIHL remains the second most common cause of hearing impairment after presbycusis, affecting millions globally. This article explores the pathophysiology of NIHL, including harmful noise levels, cochlear damage mechanisms, oxidative stress, and genetic predispositions. It also reviews current diagnostic approaches, such as pure-tone audiometry, otoacoustic emissions (OAE), and electrophysiological testing, which help in early detection. The prevention of NIHL is discussed in the context of global legislative efforts, hearing conservation programs, and protective devices. Additionally, pharmacological advancements, including antioxidants, anti-inflammatory agents, and emerging gene therapies, are highlighted as potential interventions. With increasing noise pollution from occupational and recreational sources, understanding NIHL and its management is critical for reducing its long-term impact on public health.

Keywords: Noise induced hearing loss, Sensorineural hearing loss, Noise exposure and hearing loss

INTRODUCTION

The first documented mention of NIHL as a medical diagnosis was first named 'boilermaker's disease' due to hearing loss in workers.[1] Recent reports such as the The global burden of disease report estimated that 20.3% of the world population, are affected by some sort of hearing loss, with 62% over the age of 50 years.[2] As NIHL is the second most common cause of hearing loss after presbycusis (age-related hearing loss) , it imposes an enormous burden on individuals and health systems. Studies done at the national institute on deafness and other communication disorders (NIDCD) reveals that about 18% of adults aged 20-69 who have been exposed to prolonged loud noise at work for five or more years' experience speech-frequency hearing loss in both ears. Comparatively, only 5.5% of adults without occupational noise exposure are affected by speech-frequency hearing loss in both ears.[3]

PATHOPHYSIOLOGY

Harmful noise level

Normal human sound discrimination typically begins at 0 dB within the frequency ranges of 20 Hz to 20,000 kHz.[10] Table 1 gives an insight into the damaging effects of sounds around us. Persistent exposure to noise above 70 dB can result in cumulative hearing loss wherein even a short exposure to noise above 120 dB can cause immediate hearing loss.[5] Rampant use of headphones for music has increased the prevalence of risk of NIHL in young people. Imam and Hannan reported that people exposed to sound exceeding 89 dB for more than 5 hours per week can suffer permanent hearing damage over time.[6] The susceptibility to noise induced hearing loss increases in the elderly or people who suffer from sensorineural hearing loss.

Table 1: Damaging effects of sounds around us.

Sound type	Sound level (dB)	Effect on hearing
Breathing	10	No impact on hearing
Normal conversation	60-70	No damaging effect on hearing
City traffic	70-85	Prolonged exposure poses risk
Lawnmower	85-90	Prolonged exposure poses risk
Motorcycle	90-100	prolonged exposure poses higher risk
Chainsaw	100-110	Prolonged damage can cause immediate damage
Rock concert (front row)	110-120	Short exposure can cause immediate effect
Jet engine (near takeoff)	130-140	Short exposure can cause Immediate and permanent hearing loss
Fireworks	140+	Immediate, severe hearing damage with short exposure

This table provides an overview of common environmental noises and their potential impact on hearing based on exposure duration and sound intensity. Prolonged exposure to sounds above 85 dB can lead to permanent hearing loss.[5,7]

Mechanisms of damage in NIHL

Noise trauma can result in two types of injury to the inner ear, depending on the intensity and duration of the exposure: either transient attenuation of hearing acuity which is a temporary threshold shift (TTS), or a permanent threshold shift (PTS). Hearing loss persisting at 14 days after noise exposure, with the upper recovery limit being 30 days,is indicative of PTS.[8] Transient attenuation of hearing with recovery within 24-48 h is called a temporary threshold shift (TTS) and results from more moderate noise damage.

The human cochlea is a spiral-shaped structure divided into three fluid-filled chambers-the scala tympani, the scala vestibuli, and the scala media which makes two and three quarter turns around a central canal called the modiolus.Sound vibrations are transmitted to the duct via the oval window which causes movement of the perilymph and in turn the basilar membrane .Tonotropic organization of the basilar membrane causes lower frequencies to vibrate closer to the apex of the cochlea while higher frequencies produce vibrations closer to the base, near the oval window.

The cochlea plays a key role in auditory transduction by maintaining distinct ionic compositions in its fluid-filled chambers: the scala vestibuli and scala tympani contain sodium-rich perilymph, while the scala media contains potassium-rich endolymph, separated by Reissner's and the basilar membranes. Hair cells in the cochlea, particularly inner and outer hair cells, are essential for hearing. Sound vibrations cause stereocilia on hair cells to move, opening ion channels and allowing potassium and calcium to flow in, depolarizing the cells. This leads to the outer hair cells amplifying sound sensitivity, while inner hair cells release glutamate to activate spiral ganglion neurons, transmitting the auditory signal to the brain. This process enables frequency discrimination and sound detection.

Exposure of the hair cells to loud noise can result in mechanical damage of cochlear structures, reduction in blood flow, sterile inflammation, and oxidative stress and excitotoxicity due to overstimulation of hair cells and nerves. Apoptosis is the major contributor to the loss of hair cells causing permanent hearing loss. The nature of tonotopic organization means that noise at specific frequencies can cause discrete areas of hair cell damage which manifest as frequency-specific hearing deficits.[9]

Understanding the pathophysiology and molecular mechanisms give an insight to the management of noise induce hearing loss which can be witnessed lower down in the pharmacological advances.

Mechanical damage

Hair cells are subject to mechanical shearing forces due to their position between the basilar and tectorial membranes, leading to gradual damage which explains age-related hearing loss. Intense or sustained noise exposure accelerates this damage by causing stereocilia breakage, tip link destruction, and premature hair cell death. PCDH15 and CDH23 proteins have been found to have reparative effect on tip links. Noise exposure also affects supporting cells, weakening their rigidity, which further disrupts hair cell function and contributes to NIHL.

Noise exposure leads to a spike in intracellular calcium, which can trigger cell death pathways. Calcineurin, a calcium-dependent protein, plays a role in this process by activating pro-apoptotic proteins, but inhibitors like FK506 can reduce hair cell death. Voltage-gated calcium channels (VGCCs), which regulate calcium influx, are involved in noise-induced calcium overload. Some studies suggest that T-type calcium channel blockers (e.g., ethosuximide) may protect against hearing damage, while others argue that L-type blockers are more effective in reducing NIHL.[10]

Oxidative stress

Mitochondria play a critical role in apoptosis and oxidative stress, especially in response to noise-induced trauma. Noise exposure triggers the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS), which

activate pro-apoptotic pathways and can lead to hair cell death. Free radicals, including superoxide and peroxynitrite, continue to damage cochlear structures for days post-exposure. Antioxidant enzymes like superoxide dismutase (Sod1) protect against ROS, and its deficiency increases susceptibility to NIHL. Noise also disrupts blood flow in the cochlea, especially affecting the stria vascularis, which regulates the endocochlear potential. Imbalances in ions, particularly potassium and sodium, can lead to cellular edema and further damage. Magnesium deficiency exacerbates cochlear trauma by increasing calcium influx and blood viscosity, worsening oxidative stress and vasoconstriction in response to acoustic trauma.[10]

Inflammation

Increased levels of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukins, and chemokines like CCL2 and recruitment of inflammatory cells, particularly macrophages, to the cochlea, contribute to the inflammatory response and potential cochlear damage.

Recent studies, such as Housley et al suggest that low-level TTS is mediated by ion channels, specifically the P2RX2 receptor, activated by ATP and more extensive TTS (up to 50 dB), involves additional mechanisms like Morphological changes, such as uncoupling of outer hair cells (OHCs) from the tectorial membrane and excitotoxic swelling of inner hair cell (IHC) afferent endings, have been linked to higher TTS levels.[11]

Metabolic overstimulation also contributes to TTS. Cheng et al demonstrated that antioxidants, like D-methionine, protect against TTS by reducing ROS. Decreased activity of ion transporters in the cochlear lateral wall further suggests that reduced endocochlear potential may also play a role in mediating TTS.[12]

Genetic

Although in its early stages, work on NIHL susceptible genes show promising results in the prevention or treatment of NIHL. Individuals with these genetic predispositions may be identified and the risk can be significantly reduced by keeping them away from intense noise exposure.few groups of NIHL susceptible genes have been listed.[13]

Antioxidant genes: These genes, including APEX1, ATP2B2, and CAT, are involved in protecting cells from oxidative damage, which can contribute to hearing loss. Their loci include variants like rs1130409 and rs1719571.

Potassium ion cycling related genes: These genes (e.g., KCNQ1, KCNQ4) are involved in maintaining ion balance in the cochlea. Variants such as rs800336 and rs34287852 are linked to susceptibility to NIHL.

Cilia structure related genes: Genes like CDH23 and PCDH15 are related to the structure and function of cilia in the inner ear, with gene variants like rs1227049 influencing hearing loss risk.

Heat shock protein genes: These genes (e.g., HSPA1A, HSPA1L) protect cells from stress, and variants such as rs1043618 are linked to NIHL susceptibility.

DNA damage repair genes: Genes like DNMT1 and OGG1 repair damaged DNA, and variants such as rs2228611 affect the repair processes, contributing to NIHL risk.

Apoptosis-related genes: Genes like CASP3 and ERK2 play roles in cell death pathways, with variants influencing the impact of noise exposure on hearing.

Other NIHL susceptible genes: This category includes various other genes, such as AUTS2 and CARD8, with distinct genetic loci influencing susceptibility to hearing loss.

AUDIOLOGICAL INVESTIGATIONS

Pure tone audiometry

Individuals with early or moderately advanced NIHL often exhibit the characteristic ‘boilermakers’ notch around 4 kHz.

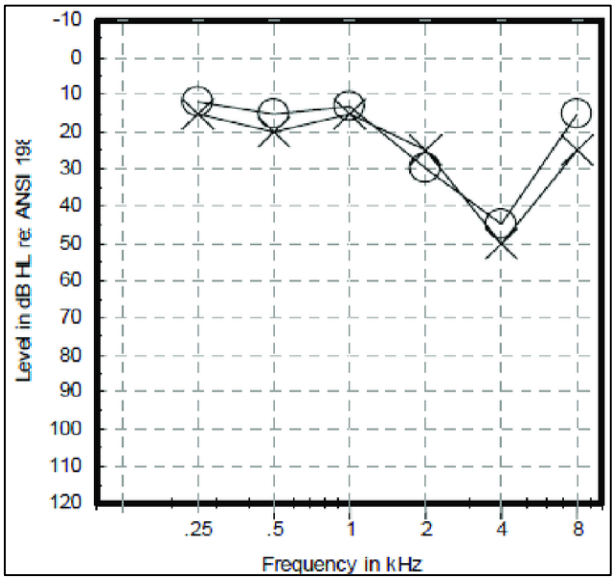

Figure 1: Audiogram in NIHL with notch 4 kHz.[14]

The outer ear canal has a resonance frequency of around 3 kHz to 4 kHz which means that sounds within this frequency range are amplified more effectively by the ear’s anatomy, making the ear more sensitive to sounds at these frequencies. But chronic noise exposure can affect adjacent frequencies and even show notches from low frequencies, like 0.5 kHz to 8 kHz.[15]

NIHL typically reaches an average of no more than 75 dB in the high frequencies and no more than 40 dB in the lower frequencies. However, studies have reported severe to profound SNHL in noise-exposed individuals, with prevalence rates ranging from 1% to 15%, much higher than the general population's prevalence of 0.5% in the United States and 0.7% in the United Kingdom.[16] Differences in the severity may arise from genetic factors or variations in the intensity, type, and duration of noise exposure.

Hearing Thresholds at extended high frequencies, commonly identified as the most susceptible to noise-induced changes across the ten studies, were 12, 14, and 16 kHz.[17]

Another point to be considered in audiometric findings is asymmetrical hearing loss. Hearing Threshold levels of 15 dB or more, between the two ears in normal population is 1% but a comparison of various studies show that the incidence of asymmetrical loss in NIHL was up to 36%.[15] External factors, such as varying levels of ambient noise exposure, internal or anatomical factors like the head shadow effect, individual handedness have been postulated to cause this asymmetry.

Speech recognition

Pure tone audiograms have been relied upon to analyze NIHL, which has miscalculated the prevalence of the condition. Damage to the connections between inner hair cells and low-spontaneous-rate auditory nerve fibers, essential for temporal processing has been studied to cause reduction in speech recognition abilities in quiet and noisy environments even when the pure tone audiograms are normal.[18]

Hence, to increase accuracy of assessment of NIHL, speech recognition tests in quiet and noisy background would be warranted along with pure tone audiometry.[15]

Otoacoustic emission

OAEs is an easily available, objective option for diagnosis of NIHL with high sensitivity. A study done on a large sample size showed that the emissions range narrowed, and amplitude decreased as the severity of NIHL increased. OAEs had more sensitivity to acoustic trauma than behavioral audiometry with a sensitivity up to 95%) and specificity up to 87%.[19]

The 82% sensitivity and 92.5% specificity is shown by distortion product OAEs at 2,3 and 4 kHz. Research indicates that OAEs may even detect noise-induced cochlear damage than audiometry.[20,21] However, audiometry is essential if the hearing is already severely impaired or OAEs are absent, there may be little or no remaining capacity to assess changes effectively using OAEs.

Synaptopathy has found to reduce responses of suprathreshold wave 1 ABR after noise exposure in animals with normal auditory thresholds, at the frequencies tonotopically related to the synaptic loss.[22,23] Similarly acoustic reflexes threshold shifts may also indicate Synaptopathy.[24] Hence, more work needs to be done on assessing the role of these objective electrophysiological tests in NIHL.

PREVENTION OF NOISE INDUCED HEARING LOSS

In today's era of industrial revolutions and air traffic, preventive measures to control noise trauma is imperative. Legislations for regulation of noise exposure and hearing protection devices are the key to prevention. There are variations between legislations and regulations of noise control across the world, while some countries have hardly any legislation. In most northern and south American countries, the permissible exposure limit (PEL) is established at 85 dB(A) for an 8-hour work shift.[25]

A prospective study done in school children indicates that hearing conservation programs for elementary school children created a positive impact in long term noise reduction and use of hearing protection.[26] There is low-quality evidence suggesting that stricter legislation may help reduce workplace noise levels.[27]

Hearing conservation programs use an exchange rate to determine how changes in noise levels affect permissible exposure times, typically reducing allowable exposure by half for every 3 to 5 dB increase in intensity.

While most regulations restrict impulse noise at 140 dB, there is no universal agreement on the exchange rate, though research suggests a 5 dB rate may better estimate the risk of NIHL than 3 dB.[28]

The efficacy of hearing protection devices relies on proper training and correct usage. The use of Earmuffs, earplugs are limited by discomfort or inconvenience.[29] This inconvenience can be addressed by customization, fit testing or even newer advances like 3D-printed earmuffs with lightweight materials, acrylonitrile butadiene styrene/clay nanocomposites.[30,31]

Improved implementation, enforcement of HLPPs, and robust evaluations of technical interventions and their long-term effects are necessary.[27]

SURGICAL TREATMENT

Cochlear implantation

Cochlear Implantation through electrical stimulation and or acoustic stimulation may be considered in cases of severe synaptopathy with severe hearing loss and severely impaired speech recognition.[32]

Pharmacological management of NIHL

Various anti-apoptotic and anti-inflammatory agents, including glucocorticoids, have been suggested as potential therapeutic targets for preventing NIHL. Among these, several natural substances have been explored for their effectiveness in mitigating HC degeneration.[33]

Antioxidants

D-methionine (D-Met): Between 2021 and 2023, Campbell et al studied that optimal preloading of D-Met for impulse and steady state acoustic trauma, although with appropriate windows at different start points reduced ABR threshold shifts.[34]

N-acetyl-L-cysteine (NAC)-combined antioxidant treatments: Campbell et al demonstrated that NAC/HPN-07 effectively reduced auditory thresholds across frequencies for up to 21 days post-noise exposure, highlighting its otoprotective potential.[35]

Dose-dependent effects: Choi et al and Ada et al reported that NAC, alone or combined with other agents, reduced cochlear damage, outer hair cell loss, and threshold shifts, emphasizing its protective role against acoustic trauma.[36,37]

Berberine: Zhao et al berberine, delivered via PL-PPS/BBR nanoparticles, resveratrol, 1-(5-Hydroxypyrimidin-2-yl) pyrrolidine-2,5-dione (HK-2), rosmarinic acid (RA), statins, SOD ZIF-8, Ginkgo Biloba, coenzyme Q10, Idebenone are being researched for their roles in NIHL treatment.[15]

ANTI-INFLAMMATORY DRUGS (GLUCOCORTICOSTEROIDS AND NON-CORTICOSTEROIDS)

Glucocorticoids, especially intratympanic dexamethasone, has been showing the most promising effects on NIHL when given before or after noise exposure in animals. However, it cannot be a longstanding solution for chronic NIHL due to occupation.[15,38,39]

In a study, the efficacy of combining systemic prednisolone (PD) at a dosage of 60 mg/day for 10 days with intratympanic steroid injections (ITSI) in patients who experienced NIHL due to gunshot noise exposure showed good improvement.[40]

Choi et al in military personnel, high-dose systemic PD (60 mg/day for 10 days followed by tapering) promoted optimal hearing recovery after acute acoustic trauma caused by a blast.[41]

Studies show that combining DEX with vehicles like poloxamer hydrogel and air-core microbubbles improves drug delivery and hearing recovery in animal models.[42] Hyaluronic acid (HA) enhanced the effects of DEX, while methoxy block copolymers offered longer drug retention.[43] Additionally, sodium caprate significantly increased cochlear drug concentration and accelerated recovery after acoustic trauma.

In a recent study, hyperBaric oxygen combined with retroaural injection of 20 mg methylprednisolone sodium succinate was proven to have good clinical outcome in treatment of NIHL.[44]

Methylprednisolone sodium succinate helps block viral proliferation and ensures oxygen and blood supply to the cochlea, promoting hearing recovery. HBO therapy improves microcirculation, reduces ear edema, and enhances tissue metabolism. Retroaural drug administration targets the inner ear, ensuring optimal drug concentration while minimizing side effects. Together, these treatments support hearing restoration and inner ear health.

RECENT ADVANCES IN DRUGS

The phase 2 trial of Ebselen, a GPx1 mimic designed to reduce oxidative stress, showed promising results for protecting against NIHL.[45]

An artificial analog of the antioxidant coenzyme Q10, Qter aided in restoring the morphology of spiral ganglion cell dendrites and cortical neurons by scavenging free radicals and regenerating antioxidants like reduced glutathione (GSH).[46]

A peptide derived from human telomerase called GV1001 attenuates sensory hair cell death targeting the noise-induced excessive ROS/RNS accumulation, as suggested by the byproducts of lipid peroxidation (4-HNE) and protein nitration (3-NT).[47]

MEDICINE TARGETING INFLAMMATION

Avenanthramide-C (AVN-C), a natural flavonoid derived from oats, exhibits anti-inflammatory and antioxidant properties in vitro. Its high-water solubility and ability to cross the blood-labyrinth barrier suggest it has significant pharmaceutical potential for treating NIHL.[47]

MEDICATIONS TARGETING HEARING PROTECTIVE GENES

Isl1, a gene expressed during otocyst development but not in the postnatal cochlea, has been shown to reduce noise-induced hair cell loss when overexpressed in postnatal hair cells. Studies suggest that enhancing acoustic progenitor developmental genes in the adult cochlea could strengthen hair cell defences against noise stress.

B vNanomedicine is being explored for treating NIHL. Nanosystems, such as polyethylene glycol-coated poly lactic acid (PLA) nanoparticles and zeolitic imidazolate nanoparticles, have been developed to deliver steroid

drugs to the inner ear. These nanoparticles demonstrate significant protective effects and improved stability and biocompatibility compared to free steroid drugs. However, further research is needed to confirm the efficacy and safety of these nanoparticle-based treatments.[47]

NIHL AND AGE

Exposure to different types of noise from early childhood might have cumulative effects on hearing impairment in adulthood. Evidence is increasing that early social and biological factors might affect hearing in middle age (eg, a study of patients assessed at age 45 years).[28] Prevalence of hearing loss is highly related to age.[29] Data suggest that pathological but sublethal changes from early noise exposure substantially increase risk of inner ear ageing and related hearing loss.[30,31] In addition to noise, factors such as alcohol and tobacco use and hyperglycaemia are associated with age-related hearing loss.

CONCLUSION

NIHL is a concerning public health concern, in the modern ears especially due to its unavoidable link to various occupational and environmental settings. The pathophysiology of NIHL reveals a complicated network of mechanical damage, oxidative stress, inflammation, and genetic factors that contribute to cochlear damage and permanent hearing loss. Audiological investigations, including pure tone audiometry and otoacoustic emissions, provide critical diagnostic tools to detect early signs of NIHL. Preventive measures like hearing conservation programs, noise regulations, and the use of hearing protection devices, have promising effects in this regard. Pharmacological treatments, including antioxidants, anti-inflammatory agents, and emerging therapies like cochlear implantation are being explored. There is ongoing research into the genetics of NIHL, along with advances in drug delivery systems like nanoparticles.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Kadavil SV, Nasif M, Sirajudeen J. Noise induced hearing loss-a review of literature. Int J Res Med Sci 2025;13:1746-53.