REVIEW

NOISE POLLUTION AND HEARING LOSS: A SUMMARY OF UNDERLYING MECHANISMS OF DAMAGE AND PREVENTION STRATEGIES OF NOISE-INDUCED HEARING LOSS

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Abstract: *Tinnitus and hearing loss are the most common effects of long-term exposure of the hearing system to a noisy environment. Nowadays, due to continuing exposure to noise, an exquisite number of over 430 million individuals worldwide are experiencing hearing deficits, specifically young people aged between 12 and 35 years who are prone to severe hearing loss [1]. People with hearing loss experience a decrease in quality of life because of the lack of a cure for noise-related hearing loss due to its complicated pathophysiology. This summary focuses on key pathways of hearing loss damage, pharmacological treatments, and current prevention and therapy approaches.*

Keywords: noise pollution, hearing loss, tinnitus, mechanisms of damage, prevention.

DOI https://doi.org/10.56082/annalsarscimed.2023.2.29

INTRODUCTION

Noise now poses significant health risks all over the world. The World Health Organization (WHO) and the United Nations Environment Programme (UNEP) estimate that decades of increasing urbanization and industrialization have amplified occupational noise hazards worldwide. People exposed to continued workplace noises, such as operating a jackhammer, or being around planes taking off, explosive blasts, or gunshots, are at risk of acquiring noise-induced hearing loss (NIHL). In addition, noises caused by human activity such as road and rail traffic, ventilation or cooling systems in buildings

(all known as environmental noises), plus noises from common recreational activities like motorcycling, snowmobiling, and attending loud music venues, all together contribute to one-third of all sources of hearing loss worldwide [1, 2]. Nearly half of people aged 12 to 35 are at increased risk of noise-related hearing loss due to the excessive use of personal listening devices (PLDs) [3].

Chronic noise exposure has prompted special concerns about potential health consequences associated with major health adversities such as anxiety, depression, hypertension, and sleep disorders [4,5]. Permanent and irreversible, NIHL is gradually occurring due to long-lasting exposure to loud sounds. Despite its prevalence, governments, businesses, and civil society must prioritize global hearing loss prevention by establishing guidelines and safe practices for protecting the global population from dangerous noise levels [6].

The goal of this summary is to provide an overview of NIHL, including the underlying processes of harm and preventative treatments.

TERMINOLOGY, EXPOSURE MEASUREMENT AND THRESHOLDS

I. Terminology

When the peripheral auditory organ, the cochlea, is subjected to loud noises for brief or extended periods, the cochlea's hair cells (HCs), which are responsible for hearing, are damaged. As a result, hearing sensitivity may decrease for a short period, a condition known as sensorineural noise-induced hearing loss (NIHL) [7]. NIHL can come on suddenly or gradually and can be short-lived or long-lasting. Because NIHL affects both ears, it is also known as symmetric hearing loss [8].

When noise-induced hearing loss affects only one ear, this is referred to as asymmetrical hearing loss. The current literature on asymmetric NIHL is scarce and some study findings merit to be pointed out in our brief [9, 10]. In a 1983 study, despite the fact that a small sample was tested compared to the total industrial population, 4.7% of 1,461 shingle sawyers had a significant prevalence of asymmetric hearing loss, with 82.6% having the left ear more affected [11, 12]. A truck driver's case series research based on 602 workers' compensation board (WCB) claims determined that noise and air flowing through an open window could cause severe hearing loss in the driver's left ear; however, the sample is quite small compared to the general truck driver population [13]. Biased and incomplete data on noise exposure levels have been reported, leading to inconsistent diagnostic criteria and significant data limitations for asymmetric NIHL [14].

Another auditory disorder, recently described as hidden hearing loss (HHL), is caused by defects in the cochlea. In noisy environments, human subjects with no obvious hearing loss yet experience perceptual difficulties, including hearing acuity and understanding speech. There is an urgent need for rapid progress in understanding the molecular mechanisms of HHL so that a treatment for this disorder will be available shortly since the current treatments principally address deficits in auditory thresholds [15]. Moreover, studies have shown a link between HHL and the development of tinnitus, which can contribute to age-related hearing loss. Therefore, finding treatments for HHL could have a substantial impact on other established hearing disorders that pose a significant risk factor for cognitive decline and dementia [16].

Constant hearing of a sound in the absence of an external source of sound is called tinnitus. The sound is frequently

buzzing, ringing, hissing, or similar to other noises. Tinnitus noises can be heard in one or both ears inside or around the head, or as an outside distinct noise. The noises may be constant or come and go, and they may vary in tone from low humming to high screech [17].

A common cause of sensorineural hearing loss around the world caused by aging, known as presbycusis, is characterized by a symmetrical loss of high-frequency hearing that progresses over time in older people. Presbycusis can also be accelerated by repeated short-duration loud-sound stimulation [18].

Although NIHL has been long recognized as an occupational disease, other causes induce hearing loss. Nonoccupational noise, also known as sociocusis, includes sounds from recreation and the outside world (such as from loud music, firearms, power tools, and home appliances) and affects the ear in the same way that occupational noise does [19]. Moreover, hearing loss appears to be aggravated when noise exposure is combined with certain physical or chemical factors such as vibrations, ototoxic drugs, or specific metals [20].

1. Exposure: measurement and thresholds

Sound level intensity is measured in sound pressure levels on a logarithmic decibel (dB) scale from safe to dangerous exposure levels. The decibel graph shows typical ambient noise intensity levels from 0 dB, the quietest audible sound, to 140 dB, which is the pain threshold. Points of reference on the decibel chart are shown in Table 1 [21].

Table 1. Decibel chart and exposure levels

Nr.crt	Decibel level (dB)	Source	Exposure levels
1	0	The softest sound a person can hear with normal hearing	0-40: Soft, safe noise levels
2	10	Normal breathing	
3	20	Leaves rustling, a ticking watch	
4	30	Whisper	
5	40	Refrigerator hum, a quiet office	
6	50	Moderate rainfall	50-60: Moderate
7	60	Normal conversation, dishwasher	
8	70	Vacuum cleaners, city traffic	70-80: Loud
9	80	Police car siren, a noisy restaurant	
10	90	Hairdryers, blenders, power tools,	90-110: Very loud, dangerous over 30 minutes
11	100	Motorcycles, hand dryers	
12	110	Nightclubs, sporting events	
13	120	Thunder, concerts, a jet plane taking off	120-140: Painful, very dangerous noise levels
14	130	Jackhammer, ambulances siren	
15	140	Fireworks, gunshot	

Sound frequency is measured in hertz (Hz), and normal human perception of sound typically starts at 0 dB in the frequency range of 20 Hz to 20,000 kHz [22]. Pure-tone audiometry is the gold standard method of determining the type, degree, and configuration of hearing loss. It is widely available, reliable and relatively easy to test. The assessment evaluates a range of frequencies in a detailed manner and nuances such as mild hearing loss, hidden hearing loss, bilateral and asymmetric hearing loss, and sensorineural hearing loss are identified [23, 24].

Hearing loss can range from mild to severe and profound to total. Moderate to

complete severity hearing loss is greater than 35 decibels in the better hearing ear and is commonly linked with poor functional outcomes in affected individuals [25].

EPIDEMIOLOGY

The second most common cause of hearing loss after presbycusis is NIHL, which has a significant impact on both individuals and the healthcare system all over the world. The NIHL risk is increased in developing countries by greater exposure to occupational and urban noise and it is estimated that approximately 20.3% of the world's population has hearing loss deficits [23]. However, many of the problems go undetected due to limited access to medical care, and testing, and the absence of government policies. In addition, the random use of hearing protection devices prompts a prevalence of NIHL that varies greatly by population and age group [26]. The overall impact of NIHL in developing countries is still to be determined because of insufficient public records and research funding, in addition to a significant lag between the research studies [27]. For example, the outcomes of a 2008 study showed that 76% of traffic police officers in Hyderabad, India, suffered from NIHL as a result of urban noise. More than a decade later, in a 2019-2020 study in Africa, 48% of steel workers in Tanzania were affected by NIHL [28-30].

Developed countries are by no means short of widespread NIHL cases, either. At least 6% of adults in the United States suffer from unilateral or bilateral hearing loss attributable to noise exposure; on a gender basis, males are at higher risk than women because hazardous jobs are mostly performed by men [31]. Eastern and Central Europe have over 34.4 million cases of diagnosed NIHL versus a lesser amount of NIHL cases in Western Europe, as estimated by the 2019 European Union report [32].

Tinnitus and NIHL are more common among military and veteran populations compared to the general population. Nearly every soldier, sailor, aviator, and Marine will be exposed to dangerous noise levels at some point in their career, whether in combat or out of combat [33]. The most common work-related disorders among American veterans are tinnitus and hearing loss, which affect 5.8% and 9.7% of veterans, respectively [34].

MECHANISMS OF DAMAGE

1. Environmental factors

The development of hearing dysfunction following exposure to a loud sound is complex and still under investigation. However, the most important pathological changes in NIHL are thought to result from the range of biological damage to the cochlea caused by noise exposure. The hair cells that lie within the cochlea are highly differentiated cells that cannot regenerate. Once they have become permanently damaged, they no longer perceive the vibration, amplifying the sound and translating it into a signal that the brain interprets [35]. Threshold shift is considered when the amount of signal required to detect or generate a response increases after noise exposure. Transient threshold shift (TTS) refers to reversible HC damage that can be reversed over minutes, hours, or even days. When TTS is not recovered, permanent hearing loss occurs which is known as permanent threshold shift (PTS). Both TTS and PTS are considered to have different causes and underlying mechanisms attributed to mechanical disruption of HCs and metabolic damage of the cochlea, depending on the sound level and duration of the exposure. Therefore, these two phenomena,

permanent and temporary threshold shifts are still not well understood [36,37].

2. Genetic factors

Recent studies have shown that interactions between genetic and environmental factors also contribute to NIHL. Genes associated with susceptibility to NIHL have been extensively studied to provide insights for further research on NIHL prevention and individualized treatment [38].

Oxidative stress plays an important role in metabolic damage in the cochlea and contributes to NIHL. Mutations in genes associated with oxidative stress inevitably impair hearing function by unbalancing the oxidative and antioxidant systems of the cochlea, ultimately leading to hearing loss [39]. In the cochlea, the enzyme glutathione-S-transferase (GST) catalyzes the binding of compounds to reduced glutathione, an important hairy cells' antioxidant. To ascertain the relationship between polymorphisms and susceptibility to NIHL, Shen et al. (2012) examined the GST gene polymorphisms in a sample of 444 NIHL workers and 445 workers with normal hearing. The findings demonstrated a higher risk of NIHL in those with a GST null genotype, compared with the wild-type genotype [40].

Numerous studies have indicated that stressful situations such as extreme noise, ototoxic medications, and high temperatures, govern the extensive expression of a protein family named heat-shock proteins (HSPs); in addition, the HSP70-1, HSP70-2, and HSP70-hom genes have an essential role in HSPs synthesis [41,42]. In animal studies, when the HSP70 proteins were first expressed by exposure to moderate sound levels, the proteins in the control group were much higher than those in the experimental group, indicating that HSP70 gene expression plays a protective role against NIHL [43].

In 2009, a comprehensive investigation of single nucleotide polymorphisms in the protocadherin 15 (PCDH15) and myosin 14 (MYH14) genes was conducted in two distinct populations (Swedish and Polish). PCDH15 molecules establish the angle linkages between sensory hair cells in the cochlea that are required for mechanoelectrical transmission. The MYH14 gene encodes one of the myosin superfamily proteins, which are actin-dependent motor proteins that regulate cochlear hair cell movement and polarity. In humans, mutations in the PCDH15 gene are the origin of both syndromic and non-syndromic hearing loss, whereas mutations in MYH14 cause autosomal dominant hearing loss [44-46].

THERAPIES FOR NIHL

1. Prevention measures

Because NIHL is an irreversible disease with no viable treatment at the moment, prevention is the best strategy for preventing hearing loss. Promoting hearing conservation educational programs in schools, funding a safe and healthy employment environment, endorsing legislation implementation, and encouraging the use of personal hearing protection devices (HPDs) are all NIHL preventive approaches [47].

Occupational noise exposure poses the greatest risk of NIHL, accounting for 16% of adult cases of hearing loss. According to previous studies, people who are most at risk from acquiring NIHL are those working in the construction, manufacturing, mining, agriculture, utilities, and transportation sectors, as well as armed forces personnel and musicians [48]. Hearing loss is more common in men than in women, owing to the higher percentage of men employed in these occupational groups and their lifetime work history. Furthermore, the age groups 30-44 and 45-59 years are at increased risk when exposed to work-related noise, thus matching the period of peak labor force involvement as

research data suggests [49]. Equipment upgrades and physical barriers surrounding loud sound sources are examples of workplace interventions. Exposure to continuous noise is measured using a weighted decibel scale (dBA), which takes into account the sensitivity of the human ear to different frequencies of sound. Although highly recommended by the US Environmental Protection Agency (EPA), the exposure limits to 80 dBA and a maximum of 40 hours a week are not standards or regulations, but they do give state and local governments the basic information for setting their criteria [50].

The National Institute for Occupational Safety and Health (NIOSH) has legally enforced recommended exposure limits (RELs) starting at an average of 85 dBA over 8-hour time period in many industries, as seen in Table 2 [51].

Table 2. Recommended exposure limits (REL) over time

Nr.crt	Time	Exposure Level per NIOSH (RELs)
1	8 hours	85 dBA
2	4 hours	88 dBA
3	2 hours	91 dBA
4	1 hour	94 dBA
5	30 minutes	97 dBA
6	15 minutes	100 dBA

Although current hearing conservation programs (HCPs) have proved beneficial in increasing knowledge about the hazards of noise exposure and improving the usage of HPDs, NIHL is still high, reaching about 18% of overexposed workers. The 2017 Cochrane review concluded that better implementation of legislation linked to preventive programs is required to prevent NIHL [52]. For example, in the mining industry, revised regulations have resulted in a 4.5 dBA decrease in noise levels, along with lower trends in noise exposure amounts per year [53].

To effectively prevent NIHL, personal HPDs must fit well and consistently be on during hazardous noise exposure [54]. Several varieties of HPDs based on noise reduction rating (NRR) and manufacturer are as follows:

1. Reusable, premolded plugs.
2. Earmuffs and Electronic/Noise Cancellation (Active) Earmuffs: over-the-ear HPDs that form an airtight seal around the ear.
3. Attenuation by Selection Earplugs: These are designed to be worn in the presence of high-frequency noises such as gunfire or explosion blasts.

2. Pharmacological prevention

While NIHL can't be cured, preliminary evidence from animal models supports the use of several classes of pharmacological agents to prevent or improve the effects of hearing loss caused by acoustic trauma. Nonsteroidal anti-inflammatory drugs (NSAIDs), antioxidants, minerals and calcium antagonists, vitamins, and hemodilution agents can all play a significant role in avoiding or ameliorating hearing loss [55].

When administered three days post-exposure, NIHL was also reduced to some degree when treatments were combined with reactive oxygen species (ROS) [56]. The thiol N-acetylcysteine (NAC), which provides cysteine for the synthesis of intracellular GSH, has likely undergone the most extensive testing in both human trials and animal models and showed reduced noise trauma when given before and after noise exposure and given under a variety of conditions and dosages [57].

DEVELOPING INTERVENTIONS TO PREVENT OR ATTENUATE NIHL

Emerging therapeutics involving human clinical studies have indicated potential for employing pharmacological medicines to prevent NIHL throughout the last two decades. According to the US ClinicalTrials.gov registry system, there were 12 clinical trials completed in January 2023 and only five reported their results on the website or in publications [58]. A couple of recent studies' findings are worth considering in more detail. A significant reduction in TTS at 4 kHz for 83 adults with normal hearing when 400 mg of ebselen was administered 15 minutes post-sound exposure for 4 days was reported in a 2017 Kil et al. study [59]. Researchers at Chang Gung Memorial Hospital, China, conducted for 2 months a study of orally administering 40 mg zinc daily to twenty adults diagnosed with NIHL-related tinnitus. The findings of this study revealed that zinc only improved the tinnitus handicap inventory (THI) score from baseline but not the hearing threshold [60].

Outside of the US registry system, clinical trials conducted all over the world have published additional research results. In 2004, studies from Italy and Israel focused on the effects of micronutrients with antioxidant or anti-inflammatory capacity, such as vitamin B12 and magnesium (Mg), respectively. Both studies found a significant reduction in TTS magnitude for treated groups vs. placebo and no-intake groups [61, 62]. A study conducted in 2010 in Belgium examined the impact of hyperbaric oxygen therapy (HBOT) and regular medical therapy (consisting of corticosteroids and piracetam treatments) administered to Belgian soldiers exposed to acute acoustic trauma (AAT). The study findings concluded that both regimens of HBOT plus medical therapy were superior to medical therapy alone and the HBOT treatment delivered shortly after AAT is critical for beneficial results [63].

CONCLUSIONS

Although to a great extent preventable, NIHL is still a common and permanent form of sensorineural hearing loss and more widespread than has already been recognized. Individuals affected by noise-induced trauma have a decreased quality of life, thus generating a considerable financial burden for society at large. Since there is no current cure, there is increased awareness of the need for NIHL prophylaxis. Primary prevention of NIHL includes avoiding extended exposure to harmful noise levels and wearing personal hearing protection when needed, combined with regulations, legislation and education in schools. While pharmacological agents' studies on both animal models and human clinical trials have shown promising results, the effectiveness and long-term effects of existing pharmacotherapeutic alternatives are yet to be determined. Future research is required to address a better understanding of the complex mechanisms of NIHL that may allow additional exploration into innovative prevention techniques and treatments.

Author Contributions:

O.C.T. conceived the original draft preparation. O.C.T. and C.A.S. were responsible for the conception and design of the review. O.C.T., I.A.V., and C.A.S. were responsible for the data acquisition. O.C.T. and I.A.V. were responsible for the collection and assembly of the articles/published data, and their inclusion and interpretation in this review. O.C.T. and C.A.S. contributed equally to the present work. All authors contributed to the critical revision of the manuscript for valuable intellectual content. All authors have read and agreed with the final version of the manuscript.

Compliance with Ethics Requirements:

The authors declare no conflict of interest regarding this article

Acknowledgements: *None*

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