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Prevention of noise-induced hearing loss in paediatric age groups: CODEPEH recommendations 2025

Prevención del daño auditivo inducido por el ruido en la edad pediátrica: recomendaciones CODEPEH 2025

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CODEPEH Recommendation Documents

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- - CODEPEH (Núñez, F. *et al*.) (2014): "Late Onset and Acquired Hearing Loss in Children: CODEPEH 2014 recommendations". *FIAPAS Magazine No. 151:* Supplement.
- - CODEPEH (Núñez, F. *et al*.) (2015): "Aetiological Diagnosis of Childhood Deafness: CODEPEH 2015 recommendations" *FIAPAS Magazine No. 155:* Supplement.
- - CODEPEH (Núñez, F. *et al*.) (2016): "Diagnosis and Treatment of Otitis Media with Effusion in Children: 2016 CODEPEH recommendations*". FIAPAS Magazine No. 159:* Supplement.
- - CODEPEH (Núñez, F. *et al*.) (2017): "Early Diagnosis and Treatment of Unilateral or Asymmetrical Hearing Loss in Children: 2017 CODEPEH recommendations". *FIAPAS Magazine No. 163:* Supplement.
- - CODEPEH (Núñez, F. *et al*.) (2018): "Update on the Programmes for Early Detection of Childhood Hearing Loss: CODEPEH 2018 recommendations. Level 1 Screening". *FIAPAS Magazine No. 167:* Supplement.
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- - CODEPEH (Núñez, F. *et al*.) (2023): "Postnatal Hearing Loss. Progressive, Late-onset or Acquired Hearing Loss in Children: 2023 CODEPEH recommendations". *FIAPAS Special Supplement No. 181*, 1-20
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Prevention of noise-induced hearing loss in paediatric age groups: CODEPEH recommendations 2025

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SUMMARY

Noise-induced hearing loss is an increasingly concerning public health issue due to its high prevalence and the general lack of awareness regarding prevention. It is particularly troubling the impact on the paediatric population and the risks of developing permanent deafness.

Noise-related damage primarily affects the inner ear, compromising hearing cells, the tectorial membrane, and auditory synapses. Prolonged exposure may lead not only to deafness but also to tinnitus, vertigo, and insomnia, among other consequences.

Understanding the molecular and cellular mechanisms is urgently needed in order to develop effective prevention strategies. Educating children and adolescents about the risks of noise exposure is crucial to fostering safe listening behaviours.

KEY WORDS

Noise-induced hearing loss, hearing cells, childhood hearing loss, preventive measures.

RESUMEN

La pérdida auditiva inducida por el ruido representa un creciente problema de salud pública por su alta prevalencia y la falta de conciencia sobre su prevención. Es especialmente preocupante el impacto en la población pediátrica y los riesgos de presentar una sordera permanente.

El daño por ruido afecta principalmente al oído interno, comprometiendo las células ciliadas, la membrana tectoria y las sinapsis auditivas. Si las exposiciones son prolongadas, además de la sordera, pueden provocar acúfenos, vértigo e insomnio, entre otras consecuencias.

Es urgente conocer los mecanismos moleculares y celulares para desarrollar estrategias preventivas eficaces. Educar sobre los riesgos del ruido, especialmente en niños y adolescentes, es clave para generar conductas de escucha seguras.

PALABRAS CLAVE

Hipoacusia inducida por el ruido, células ciliadas, hipoacusia infantil, medidas preventivas.

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1. INTRODUCTION

Hearing loss due to noise exposure is a public health problem that is now receiving increased attention due to its significant health and social impact, as well as its high prevalence. However, there is little awareness for its prevention at all levels is low (GBD 2019 Hearing Loss Collaborators, 2021).

Research into the adverse effects of prolonged exposure to excessive noise levels has advanced significantly in recent years, along with the development and dissemination of primary prevention strategies (Imam and Hannan, 2017).

Noise exposure is the second most common cause of acquired hearing loss and the first preventable cause. According to the World Health Organisation (WHO), it is estimated that 1 billion children and young people are at risk of developing avoidable and permanent hearing loss, which is largely due to new listening habits and the use of personal devices, electronic games, recreational environments, etc. Hence the higher risk among children and young people. The WHO also stresses that there is an urgent need for a comprehensive understanding of the molecular and cellular mechanisms of this type of hearing loss.

Noise-induced hearing loss (NIHL) affects different levels of the inner ear, from the hair cells and supporting cells and structures that maintain the electrical potential of the cochlea, to the tectorial membrane, which is involved in the mechanical stimulation of the organ of Corti. It has also been found that the synapses of hair cells are vulnerable from the earliest stages of damage from noise exposure, even before hair cell loss is evident. Additionally, spiral ganglion cells, which are responsible for the transmission of auditory signals, may be damaged (Din g*et al*., 2019).

Deafness manifests itself as progressive hearing loss. If the harmful sound levels are transient and moderate, it can be observed that the hearing loss is also temporary; however, if the exposure is prolonged, permanent deafness will be established. This disorder has a great impact on individuals' daily life by associating hearing loss with symptoms such as tinnitus, headaches, vertigo and insomnia, among others.

In order to formulate prevention strategies, it is necessary, in addition to understanding the mechanisms involved, to disseminate information on the risks of such exposure and to promote measures that contribute to the reduction of the effects on hearing and the number of cases of hearing loss.

A key aspect is the education of children, adolescents and young adults about the impact of noise, in order to modify their listening behaviour with personal devices and in noise-polluted recreational contexts.

This CODEPEH Document reviews the molecular and cellular processes of the pathophysiology of noise-induced hearing loss, as well as the presentation and progression of hearing loss and strategies for primary prevention, aimed especially at the younger population and their families, and public administrations and agencies responsible for taking preventive measures.

This proposal fits within the actions mandated in our legal framework, which must be developed within the field of prevention and reduction of the appearance of new disabilities or the intensification of pre-existing ones, also being identified as one of the measures*(Conducting studies on the impact of noise on health*) to be developed among the lines of action set out and relating to "Research, Training and Awareness" provided for in the 1st National Plan *for the Healthy Well-being of People with Disabilities 2022-2026* (Ministry of Social Rights and Agenda 2030, 2023). It also has its purpose and aligns with the Convention on the Rights of Persons with Disabilities (CRPD), particularly Articles 25 and 26, highlighting in any case the need for "early identification and intervention, where appropriate, and services aimed at preventing and minimising the onset of new disabilities", both in childhood and adulthood. From a rights, equality and non-discrimination approach, this is the ideal strategic framework and one that brings the precise value to the content addressed in this Paper.

2. PATHOPHYSIOLOGY OF NOISE AND NOISE-INDUCED INJURY

The cochlea is a complex organ located in the inner ear, essential in auditory transduction to convert sound waves into electrical impulses that the brain interprets as sound. It is a spiral duct that wraps around a bone (modiolus), with more than two and a half turns. Its structure includes the otic capsule, with two openings: the round window and oval window. It has a size of 3.1-3.3 cm when unwound. It is divided by the basilar membrane into the scala vestibuli and the scala tympani. The spiral shape allows different frequencies to stimulate specific areas along the cochlea, creating a tonotopic map that is crucial for the discrimination of sound frequencies (Casale *et al.*, 2023; Yan *et al.*, 2024).

The cochlea is fully developed at approximately nine weeks of gestation and is functional from the sixth month onwards. In contrast, the central auditory system has a long postnatal maturation phase.

The cochlea contains two types of fluid: perilymph, present in the scala tympani and scala vestibuli, and endolymph, which fills the scala media. The endolymph is rich in potassium and is essential for auditory function. From a mechanical point of view, the basilar membrane serves to generate and propagate the travelling wave along the cochlea so that, at a characteristic frequency, a corresponding principal site of displacement occurs on this membrane. This is known as cochlear tonotopy, allowing the human ear to perceive a wide range of sounds. High frequencies (around 4 kHz and above) are represented in the base, which is a stiffer area and responds better to these sounds. Low frequencies (below 500 Hz) are located in the apex, which is a more flexible section (Boucher and Avan, 2023; Casale et *al.*, 2023).

The cochlea contains approximately 12,000 outer hair cells, which act by amplifying sound-induced vibrations (Elliott *et al.*, 2012) and enhancing hearing sensitivity and frequency selectivity.

Inner hair cells are the main auditory receptors and their function is enhanced by the optimal transfer of vibrations from the basilar membrane, converting mechanical vibrations into electrical impulses, which are then transmitted to the brain by the cochlear nerve, via the 8th cranial nerve (Casale *et al.*, 2023).

From an acoustic point of view, noise is a sound whose energy is randomly distributed in the frequency spectrum, but in communication it represents an unwanted sound that interferes with the perception of the sound signal. With regard to noise-induced hearing damage, noise is any high intensity sound capable of causing damage, regardless of its spectral acoustic characteristics (harmonic or not).

NIHL is the result of damage to the cochlea, the magnitude of which depends on the characteristics of the exposure: mode, intensity, duration, frequency, and the individual susceptibility of the exposed person (Reynolds and Bielefeld, 2023).

Exposure to high noise intensities can lead to transient or permanent cochlear damage (Kurabi *et al.,* 2017). A temporary change in hearing threshold (TTS, *temporal threshold shift*) is a temporary worsening that recovers to pre-exposure thresholds within a period of time. Noise exposure has been shown to induce structural damage to hair cells, auditory nerve synapses and supporting cells. It has been shown in experimental animals that the synapse between hair cells and the auditory nerve can be damaged, even if the hair cells survive. A permanent change in the hearing threshold (PTS, *permanent threshold shift*) is the irreversible worsening of thresholds due to the destruction of cochlear structures. If the noise exposure leading to the occurrence of a TTS is repetitive, it results in a PTS.

The hair cells are the point of greatest vulnerability and the damage they suffer is due to a metabolic phenomenon. The outer hair cells, responsible for the frequency selectivity of the cochlea, are the most sensitive to damage by biochemical processes that are not yet known in detail. Excessive noise exposure increases the presence of reactive oxygen species

(ROS,) inside the cochlea, which are dangerous free radicals that chemically react with important cellular components such as DNA, proteins, mitochondria and cell membrane lipids. ROS are a by-product of mitochondrial metabolism that is regulated by the presence of antioxidant enzyme systems; their excessive presence alters cellular homeostasis, leading to cellular oxidative stress. Toxic levels of ROS have been found in the cochlea of experimental animals immediately after exposure to noise and up to ten days later. Hair cell damage spreads over time, especially towards the basal aspect of the cochlea (Hu *et al.,* 2002). Excessive ROS production triggers two types of biochemical mechanisms: necrosis and apoptosis, although each process differs in its onset and effects. Necrosis is characterised by passive and accidental cell death with uncontrolled release of inflammatory cell contents (Yakolev and Faden, 2004). Apoptosis results in programmed cell death and the dismantling of ciliated cells without generating inflammation.

In addition to progressive metabolic damage, noise exposure can mechanically damage the cochlea, both at the tissue and organ level. This is known as "acoustic trauma" (Ryan *et al.*, 2016). This phenomenon causes significant damage to the organ of Corti and the basilar membrane, resulting in injury to the reticular lamina and loss of hair cells through mechanically induced apoptosis.

All damage is cumulative. According to the principle of equal energy, the total effect of sound is proportional to the total amount of sound energy received by the ear, regardless of the distribution of that energy over time (Berglund *et al*., 1999).

3. RISK FACTORS AND SITUATIONS

Noise exposure and its possible consequences are a problem in paediatric age groups because the auditory system is particularly vulnerable and the effects of noise on hearing, development and learning in these early stages can affect future quality of life.

Given that in many cases exposure to noise often begins in childhood and that its harmful effects are mainly due to repeat exposure to loud noise over long periods of time, greater attention must be paid to its appearance and effect in everyday activities.

There are several categories of noise sources. Ambient noise is noise from the environment generated by human activity (road traffic, aircraft, machinery, industrial areas, among others) or by electrical household appliances and devices. Recreational and leisure noise includes personal listening devices, video games, toys, music at parties, dances and concerts, fireworks displays, sporting events and other hobbies. And occupational noise is noise associated with the performing of an occupational activity (Balk *et al*., 2023).

There are also a number of individual factors that can make someone more susceptible to hearing loss caused by prolonged exposure to loud noise. Among others:

– **Genetic factors**

Susceptibility to NIHL depends not only on the intensity and duration of exposure, but also on genetic factors that may predispose to accelerated and/or more severe hearing damage. Although most genetic studies focus on congenital hearing loss, there is growing evidence that certain genetic polymorphisms increase the risk of noise-induced hearing damage. In these cases, genetic variations occur that make the hair cells or their mechanisms for protecting against oxidative stress less effective. In paediatric age groups, cases with a certain genetic predispositionmay be more vulnerable, even at relatively low noise levels (Mace *et al.,* 1991).

Advances in genetic research have led to the identification of specific genetic markers associated with increased susceptibility to noise-induced hearing damage. Some of the most researched genes and variants are listed in Table 1.

Generally, predisposition to NIHL does not depend on a single gene, but on the combination of

multiple genetic factors together with environmental factors such as noise intensity and duration, as well as the use of hearing protection.

Susceptibility to hearing damage caused by noise is complex and depends on the interaction of different genetic variants. Some people are at greater risk because they accumulate multiple genetic factors that make them more vulnerable to the harmful effects of noise. These alterations influence the normal function of genes that are important for ear development and hair cell protection, increasing the likelihood of hearing loss.

It has also been observed that people with a reduced ability to combat cell damage caused by free radicals are at greater risk of hearing loss due to noise. Several molecular mechanisms such as oxidative stress, inflammation, alterations in ion channels, difficulties in DNA repair or problems in mitochondrial function may act together to increase this damage. Also, genetic factors related to ear structure, cell junctions and communication between auditory cells contribute to susceptibility to NIHL.

- **Epigenetic modifications and noise-induced hearing loss**

In addition to genetic factors, epigenetic modifications, such as DNA methylation and histone modifications, may play a role in an individual's susceptibility to noise-induced hearing damage. These epigenetic changes can influence gene expression and cell function, potentially contributing to the development or progression of NIHL (Śliwinska-Kowalskaand Kotylo, 2007; Tikka *et al.*, 2017).

- **Age and sex**

During infancy, the auditory system is still developing and is therefore more sensitive to loud or prolonged sound stimuli.

Older people may also be more prone to hearing loss due to the sum of presbycusis (age-related hearing impairment) and chronic noise exposure throughout life.

On the other hand, men have a higher risk of NIHL than women, even when age and noise exposure are the same. Several animal studies have provided evidence to support the protective potential of oestrogens in noise-induced cochlear damage. Human studies have concluded that, under comparable conditions of high occupational (industrial) noise exposure, NIHL was significantly more prevalent in males than in females, after adjustment for age, level of noise exposure and other potential behavioural confounding factors. Risk factors for NIHL may differ between men and women (Wang *et al.*, 2021).

– **Pre-existing diseases or health problems**

Conditions such as diabetes, high blood pressure or cardiovascular disorders can compromise the microcirculation in the inner ear. Reduced blood and nutrient supply to the cochlea can exacerbate noise damage. Some infectious diseases, such as chronic otitis, can weaken ear structures and facilitate injury (Balk *et al.*, 2023).

- **Use of Ototoxic medication**

Drugs such as certain antibiotics (aminoglycosides, among others), loop diuretics, platinum-based chemotherapeutic agents and others can be toxic to auditory hair cells. When noise exposure and the use of these drugs are combined, the risk of hearing damage increases markedly (ototoxic synergy) (Núñez-Batalla *et al.,* 2021).

Moreover, joint exposure to noise and certain industrial chemicals (toluene, styrene or organic solvents), used as substances of abuse by adolescents, may enhance hearing damage.

With regard to tobacco, it should be noted that it can influence the blood supply to the cochlea and aggravate noise damage, also in passive smokers, including children and adolescents.

- **Anatomical and physiological factors**

Congenital malformations, whether of the outer, middle or inner ear, may predispose to increased sensitivity to acoustic trauma. On the other hand, ventilation or drainage problems in the middle ear can also create an environment that is more susceptible to hearing damage.

- **Prior exposure to noise**

A history of repeated exposures to high noise levels can cause cumulative subclinical damage, making the ear more vulnerable to future exposures. There is a cumulative effect which, over time, can lead to permanent hearing loss. This is especially important in children admitted to hospital, and above all to neonatal intensive care units (NICUs) and/or paediatric intensive care units (PICUs).

WHO guidelines for hospital environments recommend that noise levels in patient wards should be kept below 35 dB during the day and 30 dB at night. These figures may be difficult to achieve in practice, but they serve as a benchmark for the importance of silence and minimisation of hospital noise.

Although the focus in the NICU is on survival and stabilisation, the possibility of hearing damage from excessive noise inherent in routine medical care cannot be overlooked. Continuous exposure to noise exceeding the recommendations (e.g. NICU >45 dB or repeated peaks above 60–70 dB) can damage the developing ear or contribute to hearing impairment in the medium- to long-term (American Academy of Pediatrics, 1997; Sibrecht *et al.*, 2024).

Other deleterious effects of noise in NICU and PICU derive from alterations in neurological and sensory development, as neonates, especially preterm infants, are extremely sensitive to environmental stimuli. Excessive noise can lead to overstimulation, stress, sleep disturbance and long-term negative repercussions (including delays in cognitive and language milestones). Quality sleep and rest are essential for brain growth and maturation. A noisy environment fragments the deep-sleep phases, and hinders newborns' self-regulation and their proper development. In addition, high noise levels have been found to be associated with increased heart rate, blood pressure and alterations in oxygen saturation. In fragile neonates, these variations may complicate recovery and increase clinical instability leading to hearing impairment.

Other non-recreational settings of prolonged noise exposure in paediatric age groups include acoustically poor and particularly noisy school environments or high noise levels from industrial settings, transport or non-recreational activities, which contribute significantly to the impact of noise-induced hearing damage in children. Particular importance should be given to noise levels in nursery schools and educational establishments, where children spend a large part of the day. Although many studies have demonstrated the negative impact of noise exposure on childhood cognitive performance and academic achievement, the actual noise levels in these environments are often overlooked (Klatte *et al*., 2013).

According to the Acoustical Society of America, the speech intelligibility index in many U.S. classrooms is less than 75%.* That is, a normal hearing person, in speech intelligibility tests, would only understand 75% of the words read from a list. Even children with normal hearing are affected by these situations, which are exacerbated for those with learning difficulties or auditory processing problems. These environments, which should be conducive to learning and development, can expose children to excessive levels of noise from a variety of sources, including traffic, construction sites or the activities within the classroom (Bhang *et al.,* 2018).

A survey conducted in the U.S. revealed that a significant number of adolescents are exposed to

loud noise in school environments, 46.5% of them on a regular basis. However, most schools do not provide hearing protection devices in activities and workshops, nor are schoolchildren taught how to protect their hearing (Eichwald and Scinicariello, 2020).

Prolonged exposure to high noise levels in educational settings can also have detrimental effects on attention, memory and academic performance (Shield and Dockrell, 2008; Klatte *et al.,* 2013; Bhang *et al*., 2018).

On the other hand, studies in both the U.S. and Europe have shown socio-demographic differences in relation to noise exposure. Children from vulnerable socio-economic backgrounds are at greater risk of being exposed to high noise levels, which may contribute to the development of NIHL. Factors such as living in noisier neighbourhoods, attending schools and educational institutions without sound insulation, or having limited access to protective interventions, may exacerbate the risk of hearing disability (Bhang *et al.*, 2018; Dreger *et al*., 2019).

In addition to ambient noise, some children may also be exposed to occupational noise due to the work of their family or caregivers at home. Those residing in areas with high levels of residential noise pollution, such as those close to transport hubs or industrial areas, are at an increased risk of developing NIHL (Crandell and Smaldino, 2000; Woolner and Hall, 2010; Hammer *et al*., 2014; Khasawneh *et al.*, 2020).

- **Lifestyle and recreational noise**

While the audiological consequences of noise exposure are commonly associated with the occupational setting, greater attention is now being paid to the impact of loud music. The term “music-induced hearing loss” has been coined to describe this condition due to overexposure to high-intensity sound. Although not many studies are available, existing evidence suggests that loud music can damage hearing. Unlike occupational exposure to noise, exposure to music is intentional among musicians and recreational listeners, increasing the likelihood of hearing damage by actively seeking the highest volume levels and avoiding the use of hearing protection devices.

Recreational noise exposure of children and adolescents comes mainly from personal music players, concerts, discotheques, toys, electronic games and devices, and other everyday or festive noises (fireworks and sporting events and other hobbies) (Balk *et al.,* 2023). These noise sources are a major threat to hearing health.

The use of personal music players and smartphones with headphones has become widespread among children and adolescents, who often use them at high volume and for prolonged periods of time, making them the main source of recreational noise exposure (Twardella *et al.,* 2011; Swierniak *et al.*, 2020).

Attending concerts and discotheques exposes young people to music at volumes that can reach levels above 100 dB. These environments are particularly hazardous due to prolonged exposure (Świerczek *et al.,* 2020; Gáborján *et al*., 2025).

A meta-analysis revealed that the prevalence of hearing loss due to social noise exposure in adolescents and young adults ranged from less than 2% in self-reported studies to between 11.5% and 15.8% in studies using audiometric tests (Costa-Marques *et al.*, 2015).

Another study, involving the Ohrkan cohort over 5 years, reported that the percentage of adolescents exposed to high levels of noise had increased from 32.7% to 63.8%, and that they were exposed to higher levels of noise. Leisure-time exposure was also found to be highest among adolescents aged 17-19 years and decreased with increasing age. Among the main determinants of risk exposure were: nightclub and private party attendance, male gender, higher education levels and smoking (Walser-Reichenbach *et al.,* 2022,

Stadler *et al.,* 2024). A study conducted in Korea found that 17% of adolescents had hearing loss, affecting speech frequencies (11.6%) and high frequencies (10.3%) (Rhee *et al.*, 2019).

Unapproved toys, especially those with built-in loudspeakers, can produce sound levels exceeding 85 dB, the risk threshold for possible hearing damage (Balk *et al.*, 2023).

Exposure to recreational noise has been shown to reduce the amplitude of otoacoustic emissions, indicating possible damage to the inner ear (Twardella *et al.,* 2011; Gáborján *et al*., 2025).

In addition to the direct effects on hearing, exposure to recreational noise has other consequences that impact general well-being. Loud noises can provoke physiological responses to stress, including increased heart rate and cortisol levels. Chronic exposure can lead to anxiety and other emotional disorders (Świe *rczek et al.;* 2020; Balk *et al*., 2023). It can also disrupt sleep patterns, of particular concern for children and adolescents who need sufficient sleep for growth and development (Rhee *et al*., 2019; Balk *et al*., 2023).

Background noise can interfere with cognitive functions such as concentration and memory, which can negatively affect academic performance and social interactions (Balk *et al*., 2023).

Many children and adolescents are unaware of the risks associated with exposure to recreational noise. This lack of awareness can lead to unsafe listening habits (Antonini-Santana *et al*., 2016; Balk *et al*., 2023). The prevalence of recreational noise hearing loss among U.S. adolescents ranges from 12.8 % to 17.5 %, indicating that between one in six and one in eight students has a hearing loss (Eichwald and Scinicariello, 2020).

A study of 10,460 subjects in Canada found that amplified music in homes and vehicles was the first and third most frequent source of recreational noise (DIY tools being the second), especially among young adults aged 20-40 years. The AYE*(acceptable yearly exposure)* measurement is the annual dose accumulated during an eight-hour working day over 220 working days per year. For noise, the maximum AYE considered safe is defined by the 85 dBA limit. If the annual noise exposure dose is exceeded, hearing impairment manifests itself between the ages of 50 and 79 years. However, this study showed that AYE is not associated with hearing impairment in this younger age range, but manifests itself later in life (Feder *et al.,* 2023).

4. CLINICAL PRESENTATIONS

The characteristic pattern of NIHL has been known since the development of tone audiometry because, due to the non-linear response of the cochlea, damage from exposure to broadband noise, whether stationary or intermittent, is most evident at frequencies above the exposure frequency (Cody and Johnstone, 1981).

For typical broadband-noise exposures, the first audiometric sign is a symmetrical scotoma at 4 kHz, since broadband noise preferentially affects around 4 kHz owing to the resonance of the outer ear and the mechanical properties of the middle ear. This scotoma is a very useful clinical sign to identify NIHL. If noise exposure persists, the scotoma expands to affect neighbouring frequencies. Over time, as NIHL combines with presbycusis, the scotoma becomes more evident in the lower frequencies and the audiometric configuration takes on a ”bucket-shaped” appearance.

Noise exposure does not usually cause hearing loss with thresholds greater than 75 dB at the first audiometric scotoma or 40 dB at low frequencies (Mi*rza et al.*, 2018). The degree of noise-induced hearing loss usually increases rapidly in the first 10–15 years of exposure and then slows down over time, while the degree of hearing impairment associated with age typically accelerates as time passes. Current evidence suggests that early noise exposure increases the risk of presbycusis (Basner *et al.*, 2014).

Until recently it was thought that if the audiometric threshold was not altered, noise-induced auditory system damage could be ruled out (Themann and Masterson, 2019). However, studies in experimental animals have shown that in both TTS and PTS, the primary lesion occurs at the level of the synapse between inner hair cells and spiral ganglion neurons (known as "cochlear synaptopathy"), which is not reflected in tonal audiometry (Liberman and Kujawa, 2017). This cochlear synaptopathy may explain why some people report poor intelligibility in noisy environments despite having a normal audiometric threshold (Kobel *et al*., 2017), known as "hidden hearing loss".

It has been shown that noise can damage synapses and neurons directly, even when hair cells retain their function (Kujawa and Liberman, 2015). Noise-induced damage to synapses and afferent terminals is rapid and permanent, while damage to neurons is comparatively slower and may be "primary" (without hair cell injury) or "secondary" to hair cell loss. The spiral ganglion neurons most sensitive to cochlear synaptopathy are neurons with high thresholds and low or intermediate spontaneous discharges. These neurons are not required for the detection of auditory signals in quiet environments, so tonal audiometry is not affected if they are damaged, but they are required for more complex auditory functions, such as decoding signals in noisy environments, as neurons with high spontaneous discharges are saturated.

The diagnosis of a hidden hearing loss in humans is very complex because studies on cochlear synaptopathy have been carried out in animals. In humans, it would be necessary to analyse the tissues *post mortem* to identify the state of the synapses. Therefore, longitudinal studies are needed to determine what level of noise can cause a hidden hearing loss and to identify the most appropriate diagnostic tests.

Noise exposure also produces more subtle damage that manifests itself in more complex auditory situations (Themann and Masterson, 2019). On the one hand, there is a worsening of frequency resolution, which hinders the ability to distinguish one sound from another and manifests itself as difficulty in sound localisation and poorer intelligibility in noisy environments. On the other hand, there is a worsening of auditory temporal resolution, which reduces the ability to differentiate auditory stimuli that are presented in rapid succession. There is also a reduction in spatial resolution, making it difficult to localise sound even in quiet environments.

Recreational exposure to music should be considered a prominent cause of sensorineural hearing loss. Although it does not always result in acute cochlear injury, it contributes to the establishment of a loss that accumulates over a lifetime and is associated with age-related hearing loss (Reynolds and Bielefeld, 2023).

At present, the characteristic effects on the auditory system of recreational exposure to loud music, compared to other noise sources, are not known exactly. However, there is evidence to suggest that music and noise impact the auditory system in different ways. Much research has been carried out to determine whether exposure to loud music produces different TTS from other sources of noise. Lindgren and Axelsson (Lindgren and Axelsson, 1983) conducted an experiment with volunteers who were exposed to both noise and music at the same intensity (106 dBA for 10 minutes) in order to measure audiometric thresholds at 1000 Hz two minutes after exposure and thus determine the degree of TTS. Music-induced TTS were found to be less severe than those caused by noise. These findings were later replicated by Strasser (Strasser *et al.*, 2003) and, indeed, other research suggests that there are emotional and psychological implications that may influence the development of acquired hearing loss (Hörmann *et al.., 1970). It is not clear whether such a disparity in TTS caused by music or noise is due to the acoustic properties of the two sources or whether it is due to other non-acoustic factors that may contribute to the degree of TTS and the time to recovery (Reynolds and Bielefeld, 2023).*

Noise exposure can also cause tinnitus, which is the perception of a sound in the absence of a simultaneous

acoustic or electrical signal. The prevalence of tinnitus in noise-exposed adults varies between 5% and 30% according to different studies (Shargorodsky et al., 2010; Bhatt et al., 2016).

Hyperacusis is another symptom induced by noise exposure. According to Duarte's study (Duarte et al., 2015), conducted on 364 workers exposed to occupational noise (mainly metalworkers), more than 50 % had hyperacusis. Most studies on hyperacusis have been conducted with professional musicians and, according to a recent review, between 2% and 45% of musicians reported it, with the risk being higher in pop/rock musicians (Di Stadio et al.) 2018).

Noise-induced hyperacusis can also affect children with tinnitus, Williams syndrome, epilepsy, sensorineural and conductive hearing loss, migraine, head injury, cerebral palsy, Down syndrome, prematurity, hydrocephalus, Klinefelter syndrome, Leigh syndrome, otitis media, hypercalcaemia, dyspraxia, microcephaly and microdeletions.

The vestibular organs can also be affected by noise exposure. High noise levels have been shown to induce damage to the hair cell stereocilia of the utricular and saccular maculae of hair cells and the ampullary crests of the semicircular canals. In Ylikoski's study (Ylikoski *et al*., 1988), greater instability was detected in patients with noise-induced hearing loss without associated vestibular symptoms compared to subjects without hearing loss.

5. PREVENTIVE AND PROTECTIVE MEASURES

Prevention of NIHL in childhood and adolescence requires a multifaceted approach that includes training, regulation and acoustic protection tailored to children's characteristics and environments (Suł kowski, 2009; Harrison, 2012; Taljaard *et al.,* 2013; Śliwinska-Kowalskaand Zaborowski, 2017; Brennan-Jones *et al*., 2019; Balk *et al*., 2023).

The most important preventive measures are listed below:

- **Awareness raising and training**

  It is crucial to raise awareness about the risks of noise exposure. Education programmes should emphasise the importance of practising safe listening (Antonini-Santana *et al*., 2016; Gáborján *et al*., 2025). Training should be provided to families, teachers and caregivers explaining the importance of protecting children's hearing, especially in school and recreational settings, as well as the proper use of games, toys and electronic devices. The prevention and care of hearing health should also be included in the educational curriculum, and workshops or lectures should be organised.

  On the other hand, it is necessary to carry out regular awareness-raising and information campaigns through mass media and social networks, pointing out the need to protect hearing, highlighting the short and long-term consequences of hearing loss.

- **Noise control in the environment**

  Complying with regulations on noise levels in products, buildings, educational, recreational and sporting spaces and activities reduces the risk of hearing loss. This regulation should be based on scientific evidence and international standards (Balk *et al*., 2023; Gáborján *et al.,* 2025) that include volume control and sound intensity limitation in toys, games and electronic devices, as well as those related to the design and acoustic conditioning of spaces and built environments.

  Music and video-game players must have safe volume modes and/or applications that lock the volume at 60% of maximum capacity.

  It is necessary to check labels or warnings on toys that generate loud sounds and to educate on the safe handling of toys and sound devices, avoiding their continuous use and keeping them more than 25 cm from the face and ears.

Acoustic design and conditioning are favoured by the use of materials that reduce reverberation and ambient noise (curtains, sound-absorbing panels, carpets…).

In classrooms and educational spaces, the level of noise caused by dragging furniture, the use of school supplies, metal surfaces and equipment without proper maintenance should be controlled, and microphone systems and other organisational measures may be used to allow adequate communication between those present (FIAPAS, 2022a; FIAPAS, 2022b).

In healthcare environments, it is particularly important to encourage the development and use of quieter technologies and equipment, as well as more efficient protective devices. It is also necessary to check and maintain equipment to prevent loud or continuous noise and to check the calibration of medical alarms or other sound signals.

- **Otoprotection**

The use of hearing protection devices, such as earplugs, should be encouraged in noisy environments to reduce the risk of hearing loss (Portnuff, 2016; Balk *et al.,* 2023). It is important to ensure that these protectors are correctly fitted and age-appropriate, and to monitor their continued use while in a noisy environment. If hearing protection equipment is not available, rotate or alternate activities in less noisy areas and plan breaks of at least 10-15 minutes every hour if exposure to noise is prolonged.

Promoting safe listening habits, such as keeping the volume at reasonable levels and taking regular breaks to avoid loud noises, can help mitigate the risks associated with recreational noise exposure (Antonini-Santana *et al*., 2016; Portnuff, 2016). In relation to personal electronic listening devices, the '60/60' rule is recommended (do not exceed 60 % of the maximum volume and limit continuous use to 60 minutes), with circum-aural rather than in-ear headphones being preferable.

In terms of pharmacological otoprotection, several classes of compounds show promise, including antioxidants, vasodilators and glucocorticoids (Tieu and Campbell, 2012). OTO-104 is asustained-releaseformulation of dexamethasone, which demonstrated significant protection against NIHL in guinea pigs when administered intra-tympanically before or after acoustic trauma (Tieu and Campbell, 2012; Harrop-Jones *et al.,* 2015).

Other potential otoprotectants include D-methionine, N-acetylcysteine and ebselen (Tieu and Campbell, 2012). Research has also explored otoprotection strategies specifically for hearing loss caused by blast noise, which may require different interventions due to its unique injury patterns (Bielefeld *et al*., 2019). Although no otoprotective agent has yet received FDA approval, ongoing preclinical and clinical studies are rapidly expanding, with several compounds shown to be effective for both NIHL and drug-induced hearing loss (Tieu y Campbell, 2012; Harrop-Jones, 2015; Bielefeld *et al*., 2019; Le Prell, 2019; Le Prell *et al.,* 2020; Núñez-Batalla *et al*., 2021; Le Prell, 2022).

- **Audiological detection and monitoring**

It is important to have regular hearing tests during childhood and adolescence, even if the result of neonatal screening has been normal, as damage induced by noise exposure appears early and progressively. Detection of this damage makes early treatment possible.

Similarly to adults, the hearing of children and young people who are frequently exposed to high noise levels in their environment should be monitored. Attention should also be paid to complaints of ringing in the ears (tinnitus) or a sensation of blocked ears following the use of headphones or attending a noisy event. And consult a specialist if difficulties in hearing and understanding are suspected, or the child or adolescent him/herself makes this apparent, turns up the volume of

devices and television and/or asks for frequent repetition of what is said or asked, especially in the classroom or at home.

Close monitoring of children with added risk factors is necessary. Non-modifiable risk factors for NIHL include age, male sex, ethnicity and genetics (Daniel, 2007).

As the understanding of the genetic determinants of NIHL advances, personalised approaches to prevention and treatment may emerge in the future. The identification of these polymorphisms and mutations would allow interventions to be personalised to an individual's genetic profile, improving efficacy and outcomes. From this genetic perspective, strategies can be devised to identify individuals at higher risk of NIHL, facilitating early intervention, personalised treatment approaches and better protection of susceptible individuals (e.g. genetic screening in highly exposed populations) (Bovo *et al.,* 2007; Daniel, 2007; Śliwinska-Kowalska and Pawelczyk, 2013; Öztanand Issever, 2021; Samara *et al*., 2024).

- **Public health policies and campaigns**

In global data provided by the World Health Organisation, which has been warning about the issue for more than twenty years, the situation of young people is concerning as more than half of this population between the ages of 12 and 35 in developed countries are at risk of hearing loss due to noise and acoustic pollution in recreational and leisure contexts (WHO, 2022).

Public health policies should therefore focus on knowledge about hearing loss, noise-induced damage and the impact of noise pollution, as well as on the most effective preventive measures in each case.

These public health policies need to converge with educational, social and environmental policies in a framework of inter-administrative cooperation in which both educational and training programmes for professionals, as well as campaigns and visibility in the media to encourage prevention and the promotion of healthy habits from an early age, in all types of environments (educational, recreational and leisure, etc.), can be effective .

In this context, it would also be desirable to integrate these public policies into a shared agenda aimed at raising awareness, regulating noise levels, updating the regulatory framework on noise prevention and control in all types of products, services and environments, promoting acoustically healthy environments in school and recreational activities, and carrying out regular assessments of hearing status throughout childhood.

6. CODEPEH RECOMMENDATIONS 2025

Exposure to noise, including recreational noise, poses a risk to the hearing of children and adolescents. Noise-induced damage is cumulative and irreversible.

CODEPEH therefore makes the following recommendations:

- Prevention of noise-induced hearing loss in the paediatric population requires a multidisciplinary approach, targeting families, health professionals, educators and authorities, providing a variety of strategies to ensure the integrity of hearing health.
- Key measures for the prevention of noise damage are: awareness and education, limiting noise exposure, use of hearing protection, responsible use of personal music players.
- The competent public administrations and bodies must regulate and adopt primary prevention measures, especially aimed at the younger population and their families.
- Public health strategies need to be planned and implemented to specifically address this challenge of our time and the close monitoring of children and adolescents with added risk factors.
- Regular hearing tests are necessary for the early detection of noise-induced hearing loss.

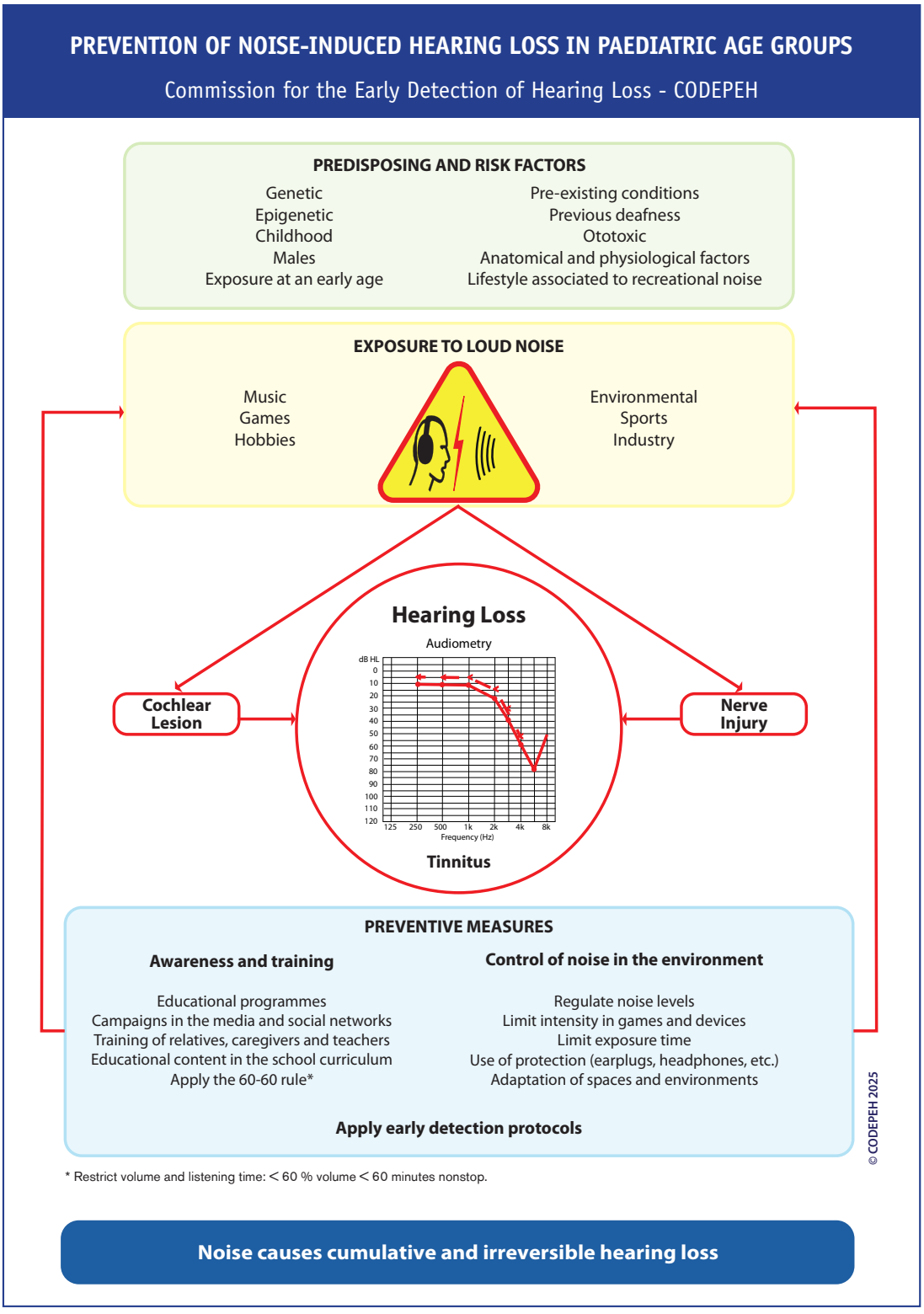
7. TABLE
Genes and noise-induced hearing loss (NIHL)

CATEGORY	MAIN GENES	FUNCTION / IMPORTANCE	RELATIONSHIP WITH NIHL
Oxidative stress response genes	**SOD (Superoxide dismutase) CAT (Catalase) GST (Glutathione S-transferase) GPX (Glutathione peroxidase) PON (Paraoxonase) NFE2L2 */HSP70 ****	They metabolise and detoxify ROS generated by noise. * NFE2L2 regulates the expression of other antioxidant genes. **HSP70 promotes the proper folding of antioxidant proteins.	These enzymes help neutralise free radicals. Decreased antioxidant activity increases the vulnerability of hair cells.
Ion channel genes and cochlear proteins	**KCNQ4/KCNMA1 GJB2 (connexin 26)**	They regulate cochlear electrical potentials (potassium channels, etc.).	Mutations enhance noise-induced damage due to ion imbalances.
Structural genes of the cochlea	**TECTA/MYO7A OTOF/CDH23 (cadherin 23)**	They encode proteins essential for the tectorial membrane, hair cell motor function and synaptic vesicle release.	Any structural defect would predispose to increased sensitivity to damage from high sound levels.
Genes related to inflammation	**TNF/IL-6 STAT3 JNK /TAB2**	They are involved in the inflammatory response and cytokine regulation within the inner ear.	Hyperinflammation promotes degeneration of auditory cells and stereocilia.
Mitochondrial genes	**UCP/MT-CO2**	They regulate mitochondrial energy metabolism and the production of reactive oxygen species (ROS).	Increase susceptibility to NIHL by enhancing oxidative damage to the cochlea.
DNA repair genes	**hOGG1/APEX1 XRCC1**	They repair DNA damage, counteracting mutations and breaks caused by oxidative stress.	A deficient DNA repair system increases vulnerability to noise.
Tight junction genes	**MARVELD2 ILDR1/*CLDN14***	Its alteration causes disruption of the endocochlear microenvironment and the reticular barrier of the cochlea.	If the reticular barrier is disrupted, noise can further aggravate cell damage and ionic homeostasis.
TBC1D24 variant	**TBC1D24**	Involved in synaptic vesicle transport and neuronal processes.	Excessive environmental noise worsens the phenotype in carriers of this variant.
Wnt signalling pathway	**Includes 19 Wnt genes, grouped into 12 families**	It is involved in the development and function of the inner ear.	Variations in this pathway increase susceptibility to NIHL.
WHRN	**WHRN rs12339210**	whirlin isoforms contribute to stereocilia length and stability.	The normality of the stereocilia is essential for auditory function.
Genes with a protective effect	**NOX3/IL-6 CARD8/CASP3 FAS**	They attenuate the inflammatory or apoptotic response. NOX3 balances the generation of reactive oxygen species (ROS) in the cochlea.	They decrease NIHL, either by limiting oxidative stress, inflammation or cell death.

SOURCE: (Balestrini *et al.,* 2015; Jiang *et al.,* 2021; Li *et al.,* 2024).

8. FIGURE

Prevention of noise-induced hearing loss in paediatric age groups



SOURCE: own work, CODEPEH 2025.

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