tems, including the structural similarity of their receptors, the anatomical proximity of the sound energy transmission system to the vestibular system, and their common blood supply. (Via anterior inferior cerebellar artery) (19, 21, 60).

Cochlear pathologies or hearing loss mechanisms due to noise exposure can be classified into mechanical damage and metabolic damage (61). Mechanical damage occurs at high noise levels and can damage cochlear structures (e.g. damage to stereocilia tips, cochlear vessels, tip links, and supporting cells) by transmitting excessive vibration to these structures (62). Due to the proximity and anatomical connections between the auditory and vestibular organs, the mechanical vibrations generated by noise can therefore be transmitted to the vestibular organs and damage their structures. Animal experiments have reported the loss of sensory stereocilia in the otolith organs and SCCs (24), and the degeneration of epithelial cells and the separation of their layers in the saccular maculae (22) due to mechanical damage at high noise levels (116 and 120 dB sound pressure level). On the other hand, an abnormal VEMP response and its association with histological damage in the saccular organ has also been reported after exposure to 115 dB (63). Therefore, the damage to the end organs of the vestibular system due to mechanical damage from loud noise reported in the studies included in this review (20, 54-57) can be attributed to the proximity and structural and functional similarities of the two organs of auditory and vestibular function.

On the other hand, metabolic changes are the main mechanism of NIHL because of chronic noise exposure. Therefore, metabolic changes including reactive oxygen species (ROS), ischemia, free radicals, reactive nitrogen species (RNS), Lipid peroxidation induced by ROS, and metabolic overloads in the organ of Corti are the main mechanism of cochlear damage due to noise exposure (or continuous noise) (61, 64-66). Chronic exposure to noise leads to the generation of free radicals, such as ROS and RNS, from the mitochondria to the cytoplasm of the hair cells and the production of pre-apoptotic factors and eventual apoptosis of hair cells (62, 65, 66). The free radicals generated can persist for days after exposure has ended, leading to progressive damage to the cochlea (66). Due to the same blood supply pathway of the auditory and vestibular systems, it is therefore to be expected that metabolic changes could induce similar damage to the vestibular sensory receptors (67). In addition, the ototoxicity and vestibulotoxicity effects of drugs such as aminoglycoside (68), and carboplatin (69) have recently been demonstrated. The mechanism of ototoxicity of these drugs has been explained by their entry into the cochlea and hair cells through the bloodstream and cell damage and apoptosis due to ROS formation (70). Due to the longer clearance times of the ototoxic drugs from the inner-ear fluids than from the blood, they can remain in the cochlea for several months (with a half-life of 5 to 6 months) after stopping treatments (71, 72). Due to the anatomical proximity and the structural and functional similarity of the two vestibular and auditory organs, the factors that damage the auditory organs can also damage the vestibular organs (19, 21-24). Therefore, the dysfunction of the vestibular system reported in the studies included in this review could be attributed to the mechanism of metabolic damage due to noise exposure (or continuous exposure to noise) (40, 42-47, 49-51).

On the other hand, the absence of symptoms (vertigo or dizziness) in individuals with vestibular dysfunction is likely due to the compensatory mechanism. Thus, the cerebellum seems to play an important role in initiating this mechanism. Although vestibular compensation plays an important role in maintaining balance, it should not be mistaken with full recovery (67). This is because peripheral vestibular abnormalities can result in decreased performance, particularly in unusual situations such as reduced visual input, use of rapid head movements, and the need for spatial navigation skills (34-37).

The inner ear consists of two portions separated by a thin membrane called the membrana limitans including the pars superior (utricle and SCCs) and the pars inferior (saccule and cochlea) (73). The membrane limitans as a partial barrier protecting the utricle and SCCs from acoustic

trauma. In other words, the susceptibility of the saccule to noise-induced damage is due to the anatomical proximity of the saccule to the stapes footplate (sound energy transmission system) (22, 24, 74). Moreover, the pars inferior is more susceptible to damage than the pars superior due to high-intensity noise exposure (63). Therefore, due to differences in the susceptibility of the vestibular portions, one part can precede other parts and this can result in discrepancies between evaluation findings of different portions of the vestibular system.

Limitation

The main limitation of this review is the heterogeneous data that precludes meta-analysis. Heterogeneity includes different examination methods, different diagnostic criteria, variability in industry/workplace population, different age groups and variability in sample size.

Conclusion

Almost all studies in this overview have shown a significant association between NIHL and VD among the workplace population exposed to occupational noise. Because of the crucial role of the function of the vestibular system in maintaining balance and gaze stabilization, hearing protection legislation should therefore be extended to cover the vestibular system of individuals who are at risk of damaging it. In view of the importance of medical competence and fitness for work, especially in professions with high demands on balance and gaze stabilization, it is recommended to supplement the obligatory medical examinations with an assessment of the balance function. However, conclusions from the present study needs to be treated with caution. Further research is needed to investigate the association between the occurrence of VD due to occupational noise exposure or concomitantly with NIHL.

Journalism Ethics considerations

Ethical issues (Including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc.) have been completely observed by the authors.

Acknowledgements

There was no financial source of this study. This systematic review was part of Ph.D. thesis (with Ethics Code No: IR.MODARES.REC.1399.037) of Tarbiat Modares University, Tehran, Iran.

Conflicts of interest

The authors declare that there are no conflicts of interest.

References

1. Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, Stansfeld S (2014). Auditory and non-auditory effects of noise on health. *Lancet,* 383:1325-1332. doi: 10.1016/S0140-6736(13)61613-X.
2. Nelson DI, Nelson RY, Concha-Barrientos M, Fingerhut M (2005). The global burden of occupational noise-induced hearing loss. *Am J Ind Med,* 48:446-458. doi: 10.1002/ajim.20223.
3. World Health Organization (2018) Addressing the rising prevalence of hearing loss. World Health Organization. Geneva. Available at https://apps.who.int/iris/handle/10665/260336.
4. World Health Organization (WHO) (2017). *Global costs of unaddressed hearing loss and cost-effectiveness of interventions. World Health Organization. Geneva.* Available at https://apps.who.int/iris/handle/10665/254659. ed. World Health Organization.
5. May JJ (2000). Occupational hearing loss. *Am J Ind Med,* 37:112-120. doi: 10.1002/(SICI)1097-0274(200001)37:1<112::AID-AJIM9>3.0.CO;2-%23.
6. Mirza R, Kirchner DB, Dobie RA, et al (2018). Occupational noise-induced hearing loss. *J*