Article's History: Received: 27.10.2025; Revised: 02.02.2026; Accepted: 24.02.2026; Published: 25.03.2026

Bulletin of Medical and Biological Research

Vol. 8, No. 1
2026

ISSN 2706-6282, e-ISSN 2706-6290

Journal homepage: https://bmbr.com.ua/en

UDC 616.28-008.14-053.9

DOI: 10.63341/bmbr/1.2026.17

Neurobiological aspects of hearing deprivation and its impact on quality of life in old age

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Abstract. Age-related changes in the body can cause functional limitations that make it difficult for older people to maintain their lifestyle and fulfil their social and personal roles, affecting their ability to live a full life. This article explored the main mechanisms by which hearing loss affects the quality of life in older people. Scientific publications from 2016-2025 in the MEDLINE/PubMed biomedical research database were reviewed. The analysis showed that age-related hearing loss is primarily caused by neurodegenerative processes. It was found that the degeneration of neurons in the auditory pathway – from the hair cells of the cochlea to the neurons of the neocortex – occurs in a reduction in the cell population, morphological alteration of neurons, and a decrease in the number of synaptic contacts. These changes are accompanied by disturbances in biochemical and electrophysiological homeostasis, dysregulation of intracellular calcium signalling, and decreased levels of key neurotransmitters, including glutamate, glycine, and γ-aminobutyric acid. Further analysis showed that these neurobiological disorders lead to a decrease in impulse activity and a weakening of inhibitory processes, which clinically manifests itself in auditory dysfunction, impaired perception of acoustic signals, and a decrease in the ability to spatially localise sound. The study determined that concomitant microangiopathy is characterised by inhibition of angiogenesis, decreased density of functioning capillaries, thickening of the basement membrane, and endothelial dysfunction, which leads to decreased tissue perfusion. It was found that microcirculatory insufficiency contributes to secondary ischaemic cell damage through the activation of oxidative stress and inflammatory cascades. Generalised age-related degeneration of neurons and microvessels is also evident in the brain structures responsible for cognitive functions. As a result, a recurrent pathophysiological mechanism is formed: auditory deprivation increases cognitive load and accelerates the depletion of neural resources, which in turn exacerbates neurodegenerative processes and structural changes in the brain. Analysis has shown that these processes mutually potentiate each other, causing progressive deterioration of cognitive functions and a decrease in physical and social activity in older people

Keywords: presbycusis; sensorineural hearing loss; dementia; cognitive function; neurodegeneration; angiopathy

INTRODUCTION

Quality of life is one of the key integral indicators of health status. It is a composite multifactorial concept that reflects not only medical aspects, but also functional, psycho-emotional and social components. It is used as one of the leading indicators of the level of social development of countries, which confirms the gradual shift in the global paradigm from a predominantly economic focus and technological progress to a people-oriented priority. Quality of life assessment reflects a person's subjective perception of their psychophysiological state and is based on a system of individual values, life experience, expectations and standards. Given the global ageing of the world's population, research into the factors that affect the quality of life of this age group is of scientific and clinical importance.

The review by Q. Zheng *et al.* [1] emphasised that presbycusis is one of the most common geriatric diseases, second only to cardiovascular disease and degenerative joint disease in terms of frequency. It is reported that approximately two-thirds of people with age-related hearing loss are patients aged 60 and older, with a significant proportion of them having severe or profound hearing loss. The authors analysed in detail the key pathogenetic mechanisms

Suggested Citation:
Bakalets O. Neurobiological aspects of hearing deprivation and its impact on quality of life in old age. Bull Med Biol Res. 2026;8(1):17–27. DOI: 10.63341/bmbr/1.2026.17

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of age-related hearing loss, focusing on mitochondrial dysfunction, energy metabolism dysregulation, histopathological changes in the structures of the inner ear, chronic inflammation and other mechanisms of damage to the cells of the auditory analyser. Considering presbycusis as a common sensory disorder in older adults, the researchers proposed differentiated rehabilitation strategies depending on the leading link in age-related transformation. It is emphasised that the use of an etiopathogenetically sound approach to rehabilitation, including hearing aids, increases its clinical effectiveness and contributes to improving the psychological well-being and functional independence of older people in their daily activities.

At the same time, the literature remains controversial and ambiguous. A.R. Huang *et al.* [2] conducted a secondary analysis of data from a randomised clinical trial to assess the impact of hearing loss correction on eight domains related to quality of life during a three-year follow-up of nearly 1,000 individuals over the age of 70. The study found no statistically significant improvement in physical and mental health indicators as a result of the use of hearing aids and other audiological interventions. The data obtained may indicate that the problem of age-related hearing loss is more complex and systemic in nature and is not limited to a decrease in acoustic perception, and therefore cannot be completely solved solely by increasing sound stimulation of the sensory apparatus.

The study by N. Henderson *et al.* [3] summarised data from 22 studies involving approximately 450 participants. The study determined that the quality of life of adults depends on the functional capabilities of the auditory system. Hearing impairment negatively affects not only its physical component (difficulties with speech recognition, sound localisation, or physical fatigue), but also reduces social (isolation, communication difficulties, reduced confidence and independence) and mental parameters. A cross-sectional study by S. Dedeoglu *et al.* [4] demonstrated that among people aged 18 to 65 with hearing impairment, the prevalence of anxiety disorders is higher, and the severity of psycho-emotional manifestations increases depending on the degree and clinical type of hearing dysfunction. At the same time, it has been established that the subjectively assessed severity of hearing loss has greater prognostic value for the development of psychological distress than objective audiometric indicators. B.J. Lawrence *et al.* [5] also reported a statistically significant correlation between hearing impairment and depression in older adults, with the presence of hearing dysfunction associated with an approximately 1.5-2-fold increase in the likelihood of developing depressive disorders.

A. Shukla *et al*. [6] demonstrated a consistent link between hearing loss and increased levels of loneliness and social isolation in adults and older adults. Hearing impairment limits effective interpersonal communication, contributing to reduced social engagement and poorer psychosocial well-being. The authors emphasised the significance of timely diagnosis and rehabilitative audiological interventions as potential strategies for reducing the risk of social isolation and its associated adverse health outcomes. Hearing deprivation in geriatric practice is reliably associated with a decline in cognitive function. M.L. Cantuaria *et al.* [7] found in a cohort study that a decline in auditory perception increases the risk of developing dementia. The use of hearing aids, i.e. effective correction of existing audiological disorders, significantly reduces this risk, i.e. it can prevent or delay the onset and progression of dementia. The report by G. Livingston *et al.* [8] stated that if risk factors are corrected in a timely and effective manner, almost 40% of all cases of this disease can be prevented or delayed. Among the 14 pathological conditions ranked by their significance in the development of cognitive impairment, hearing loss occupies one of the leading places (7%), second only to low education and social isolation and exceeding several metabolic, mental and behavioural factors in terms of contribution. Unlike hyperlipidaemia, which was only included in the list of risk factors in 2024, hearing loss has been considered one of the most significant and potentially modifiable factors in the development of cognitive dysfunction for more than five years.

Recognising the possibility of effective correction of audiological disorders as one of the measures to prevent the development of dementia and improve the quality of life in elderly people encourages active action at all stages of medical care, from primary care physicians to specialised otolaryngological and audiological practices. This, in turn, requires determination of mechanisms of onset and development of audiological disorders. In this regard, the study aimed to identify the key mechanisms of the impact of hearing loss on the quality of life of elderly people based on a synthesis of global experience and analysis of available scientific and information sources.

MATERIALS AND METHODS

To achieve this goal, scientific publications with a high citation index, published in English between 2016 and 2025, were analysed. The search was conducted in September-October 2025 using the MEDLINE/PubMed electronic database of medical and biological publications. To find potentially relevant materials, the following keywords in English were used: “Quality of Life” and “Hearing Loss” (2,481 publications) or “Cognitive Function” and “Hearing Loss” (1,811 publications). Based on the content of their abstracts, 44 studies that met the selected objective were selected for review. Nine articles did not pass the initial screening and were added during the process of explaining the pathogenesis of certain disorders. The study included systematic reviews and meta-analyses; observational epidemiological studies (cross-sectional, prospective/longitudinal, analytical) and experimental studies. The main criteria for inclusion of studies in the systematic review and meta-analysis were established sensorineural hearing loss and cognitive dysfunction of varying severity in older adults, a possible temporal correlation between the two events, and their potential impact on physical, mental, social, and other components of quality of life. The review did not include studies in which: the study population did not include elderly people; the subjects were diagnosed with conductive hearing loss; the relationship between hearing impairment and quality of life indicators was not analysed; the studies were descriptive in nature or were abstracts, comments or publications without full text.

The analysis of scientific sources devoted to the impact of hearing loss on the quality of life of older people was conducted using an integrated analytical approach aimed

at identifying the key mechanisms of the relationship between hearing deprivation and changes in quality-of-life indicators. The review included publications that highlighted the pathogenetic aspects of presbycusis or sensorineural hearing loss and the ways and mechanisms of their impact on the physical, psycho-emotional and social components of quality of life. The selected sources were evaluated according to the criteria of relevance to the research topic, design and methodology, characteristics of the study population, methods used to assess hearing function and quality of life indicators, as well as the analytical significance of the results obtained. The publications were classified and systematised by topic, which made it possible to structure the scientific data according to the main areas of research on the problem. A comparative analysis of the results of various studies made it possible to identify the leading pathophysiological, psychosocial, and functional mechanisms of the impact of hearing loss on the physical, cognitive, emotional, and social components of the quality of life of older people, assess the degree of consistency and inconsistency of scientific conclusions, and identify both the main trends and insufficiently studied aspects of the problem. The results obtained create a conceptual basis for further research and the development of effective strategies for the prevention and rehabilitation of elderly people with hearing loss.

RESULTS AND DISCUSSION

Age-related neurodegenerative processes as a common pathogenetic basis for auditory and cognitive disorders. Impaired sound perception and speech comprehension, which develops with age, is defined as progressive, bilateral, symmetrical age-related sensorineural hearing loss, which mainly manifests itself at high acoustic frequencies [9]. This gradual loss of functionality is the result of genetically determined changes in the neurons of the auditory pathway. The development of presbycusis is associated with the influence of about 300 genes. S. Lee *et al.* [10] identified atonal homolog 1 (Atoh1) as a key transcription factor in the specification, differentiation, and survival of mechanosensory hair cells (both cochlear and vestibular). It coordinates the development, differentiation and functioning of sensory hair cells in the cochlea. The authors showed that Atoh1 plays a central role in the formation of the auditory apparatus during embryogenesis and in maintaining the structural integrity of the sensory epithelium. In addition, the activation of Atoh1 was considered a promising mechanism for the regeneration of hair cells after damage, confirming the importance of Atoh1 as a substantial molecular regulator of pathogenesis and a potential therapeutic target in sensorineural hearing loss.

K.L. Elliott *et al.* [11] demonstrated that the formation of sensory and neuronal components of the auditory system depends not only on Atoh1, but also on the structural and functional state of other transcription factors: Neurogenin 1 (Neurog1) and Neuronal Differentiation 1 (Neurod1), and their direct or indirect interdependent influence. According to them, Neurog1 is a key regulator of neurogenesis and differentiation of spiral ganglion neurons, while Neurod1 ensures their further specialisation, survival and formation of neural connections in the auditory nuclei. Atoh1, whose expression increases from the cochlear base to the apex, determines the development and differentiation of sensory hair cells in the cochlea, ensuring the functional integrity of the peripheral auditory analyser. Accordingly, dysregulation of these genes leads to impaired neurosensory integration, forming the molecular basis for the development of neurosensory hearing loss.

K.L. Elliott *et al.* [12] demonstrated age-related cochlear degeneration by comparing different profiles of sensory deficits in elderly people and in experiments. The researchers compared age-related changes in a 70-year-old person with similar changes in an old 2-year-old mouse. Progressive age-related hearing loss, mainly at frequencies above 4 kHz, was established using tonal audiometry and confirmed histologically by delayed and progressive loss of inner (IHCs) and outer hair cells (OHCs). Based on changes in the cellular expression of related basic helix-loop-helix (bHLH) genes: Atoh1, Neurog1, the authors found that for outer hair cells, the vector of degeneration is directed from the base to the apex, while for inner hair cells, the morphological and functional changes are characterised by an apical-basal direction, leading to age-related auditory sensory deprivation at high frequencies. However, this is not a simple linear mechanism. The process of age-related degeneration begins with the outer hair cells and subsequently spreads to the inner hair cells. Therefore, the rate of cell loss is uneven: at the age of 75, the number of inner hair cells at the base of the cochlea decreases by about half, while in the same area, the number of outer hair cells decreases by 40%. In addition, degenerative processes spread to the central parts of the auditory analyser. With age, not only does the number of cells in the inner ear, cochlear nucleus, superior olivary cells, dorsal cochlear nucleus, medial nucleus of the trapezoid body, and other neurons of the auditory pathway decrease, but the number of synaptic terminals also decreases, along with other morphological changes. The study emphasised the role of glutamate excitotoxicity, mitochondrial dysfunction, and oxidative stress in the development of cochlear synaptopathy and neuronal degeneration. In other words, the interaction of sensory and neural biochemical disorders determines the progressive nature of presbycusis and the heterogeneity of its pathophysiology and clinical manifestations.

J.B. Dewey *et al.* [13] demonstrated the decisive role of the active mechanical response of outer hair cells in the mechanisms of cochlear amplification, which ensures high sensitivity and accuracy of auditory perception. The central molecular component of this process is the motor protein prestin, which is specifically expressed on the membranes of outer hair cells and provides rapid electromechanical conversion in response to changes in membrane potential. The cells rapidly change their length and stiffness, converting electrical energy into mechanical energy, i.e., acting as a physiological electromechanical transducer. The authors showed that prestin activity causes amplification of the movements of the organ of Corti at each sound wave cycle, especially in the high-frequency range. Accordingly, prestin dysfunction leads to a decrease in the efficiency of mechanical amplification of the sound signal and impaired frequency selectivity of hearing. J. Zheng *et al.* [14] explained the electromechanical activity of prestin by the fact that it is accompanied by a specific “gating current”, which reflects voltage-dependent conformational

changes in the molecule and its charge movements in the membrane. The detection of gating current confirmed the molecular mechanism of converting electrical signals into mechanical contractions of outer hair cells. Thus, prestin dysfunction is considered by scientists to be one of the key molecular links in the pathogenesis of age-related and neurosensory hearing loss.

R.H. Asli *et al.* [15] confirmed in a case-control study the role of prestin as a molecular link in the disruption of cellular and biochemical processes underlying the pathogenesis of sensorineural hearing loss. Studying the relationship between serum prestin concentration in individuals younger and older than 50 years with varying degrees of sensorineural hearing loss, it was determined that in both groups, serum prestin levels were significantly higher than in the control group, which may indicate a biochemical basis for functional insufficiency of outer hair cells. In addition, as prestin concentration increases, so does the severity of audiological disorders (odds ratio was 1.009 with a 95% confidence interval of 1.005-1.013). The authors emphasised that changes in the expression and functional activity of prestin, as a key motor protein, affect cell electromobility and calcium-dependent mechanisms of cochlear amplification. Therefore, the study proposed considering prestin levels as a quantitative biomarker of the severity of hearing disorders.

Y.J. Hu *et al.* [16] systematised role of Ca^{2+} signalling in the molecular and cellular mechanisms of sensorineural hearing loss. The study demonstrated that disturbances in intracellular Ca^{2+} regulation, dysfunction of voltage-gated calcium channels, pumps, and Na^{+}/Ca^{2+} exchangers lead to changes in synaptic transmission, neurotransmitter balance, and energy metabolism in hair cells. Outer hair cells are connected to efferent neurons and amplify incoming sound signals, while inner hair cells, innervated by afferent nerve fibres, form synapses with 90-95% of the auditory nerve fibres and transmit virtually all acoustic information to the central nervous system. Given this, changes in Ca^{2+}-dependent intracellular processes lead to impaired cochlear amplification and neural transmission of acoustic signals.

D.A. Godfrey *et al.* [17] emphasised that ageing causes a significant restructuring of the neurochemical profile not only in the peripheral cells of the auditory analyser. Structural changes in the neurons of the central auditory system are also accompanied by disturbances in intracellular biochemical and electrophysiological processes. Disturbances in glutamatergic neurotransmission manifest themselves in changes in glutamate concentration and metabolism, leading to a decrease in the efficiency of excitatory transmission and the potential development of excitotoxic processes. At the same time, there is a dysregulation of inhibitory mediator systems, GABAergic and glycinergic, which causes an imbalance between excitatory and inhibitory influences and a degradation in the accuracy of neural encoding of acoustic information. Along with this, age-related modification of cholinergic neuromodulation, associated with changes in acetylcholine metabolism, limits the adaptive capabilities of auditory neural networks, reduces the level of synaptic plasticity and the efficiency of central processing of sound signals. In addition, the restructuring of the amino acid profile (aspartate, taurine, serine and other biologically active compounds) reflects systemic dysregulation of neurochemical homeostasis, which forms the biochemical basis for progressive dysfunction of the auditory pathways and the pathogenesis of age-related hearing loss.

A study by J.K. Mittelstadt *et al.* [18] also found that age-related audiological changes in ageing are the result of auditory cortex dysfunction. Therefore, the disruption of spectral and temporal processing of acoustic signals is caused not only by the weakening of inhibitory neural mechanisms, but also by a profound reorganisation of cortical neural networks. Magnetic resonance spectroscopy determined that the reduction of inhibitory neurotransmission in the auditory cortex is reliably associated with impaired speech perception in conditions of acoustic noise, reflecting age-related dysregulation of the balance between excitatory and inhibitory processes in cortical neural networks. Changes in neurotransmitter activity and receptor expression (modification of acetylcholine receptor subunit expression, decreased glutamate decarboxylase activity, decreased serotonin, increased acetyl transferase enzyme, impaired norepinephrine and dopamine activity, etc.) have been detected in auditory cortex neurons.), which, in combination with the restructuring of cortical neural networks, leads to a deterioration in the spectral and temporal processing of sound signals. Together, these processes form the neural basis for age-related decline in auditory discrimination and speech perception.

Systematic analysis of the experimental and clinical data presented reconstructed a generalised pathogenetic cascade of intracellular disorders (Table 1) underlying progressive neurosensory degeneration. Conceptually, it can be represented as a sequence of interrelated processes: genetic dysregulation → disruption of ion homeostasis and ion-dependent signalling → neurotransmitter imbalance → metabolic and mitochondrial dysfunction → energy deficiency, oxidative stress and impaired cellular signalling → cochlear synaptopathy → apoptosis of sensory and neuronal cells. The combination of intracellular metabolic, energy and regulatory changes is activated in stages at different levels of the auditory analyser, leading to the formation of a multicomponent, mutually potentiated pathological cascade.

Table 1. Key aspects of the pathophysiology of neurodegeneration in presbycusis

Level of damage	Key mechanisms	Pathophysiological effects	Functional audiological consequences for hearing
Genetic	Dysregulation of genes involved in auditory system development; deficiency of transcription factors.	Changes in the expression of Atoh1, Neurog1, Neurod1, deficiency of transcription factors and disruption of their regulation.	Impaired differentiation and specification of hair cells (HC), reduced neurogenesis, regeneration and plasticity of sensory and neuronal cells, defects in the formation of neural connections in the auditory nuclei.

Table 1. Continued

Level of damage	Key mechanisms	Pathophysiological effects	Functional audiological consequences for hearing
Intracellular	Dysregulation of Ca^{2+} signalling.	Disruption of Ca^{2+} -dependent regulation, cellular homeostasis, synaptic transmission, etc.	Decreased speed and accuracy of perception, transmission and analysis of acoustic information.
	Prestine dysfunction.	Electromechanical transduction disorders; gating current; decreased electromotility of OHCs.	Loss of cochlear amplification, decreased frequency selectivity.
	Imbalance of mediators: glutamate, gamma-aminobutyric acid, glycine, etc.	Excitotoxicity, disruption of excitation-inhibition processes.	Development of cochlear synaptopathy; neurodegeneration.
	Mitochondrial dysfunction.	Decreased production of adenosine triphosphate, accumulation of active forms of oxygen.	Energy deficiency of the brainstem, auditory pathway neurons, and auditory cortex.
	Oxidative stress.	Excess free radicals damage membranes, proteins, and DNA.	Apoptosis of hair cells and neurons.
Cochlear (sensory)	Reduction in number, structural and functional degeneration of OHCs.	Decreased cochlear amplification.	Decreased cochlear amplification and frequency selectivity.
	Decrease in number, structural and functional degeneration of IHCs.	Disruption of synaptic transmission to neurons in the spiral ganglion.	Decreased speed and accuracy of acoustic information transmission.
Retrocochlear (auditory pathway)	Decrease in the number, structural and functional degeneration of neurons in the auditory pathway, synaptic terminals, etc.	Decreased neural transmission efficiency, impaired synaptic integration and neuroplasticity.	Impaired neurosensory integration.
Central	Structural and functional degeneration of neurons in the auditory cortex.	Changes in neurotransmitter activity and receptor expression, impaired synaptic integration and neuroplasticity.	Changes in neuroplasticity, increased spontaneous neural activity, decreased inhibitory neural activity, imbalance between excitation and inhibition processes, decreased synaptic plasticity, etc.

Source: compiled by the author

Neurodegenerative processes are not limited to the structures of the auditory analyser. The nonspecific nature of age-related cellular and molecular changes gives reason to interpret them as a manifestation of pan-neuronal involution. A study by Z. Jafari *et al.* [19] highlighted the pathophysiological mechanisms linking presbycusis with cognitive decline based on magnetic resonance imaging and cellular studies. Based on the integration of neuroimaging and cellular neurobiology data, the authors demonstrated that auditory deprivation is associated with neuroanatomical and functional changes in the central nervous system, in particular, remodelling of the auditory cortex with impaired integration of sensory and cognitive networks, as well as a reduction in grey and white matter volume. The pooled data indicated that age-related hearing loss may be an independent and potentially modifiable risk factor for cognitive decline and dementia, justifying the need for early detection of hearing impairment and the implementation of multidisciplinary preventive and therapeutic strategies.

Thus, the combination of generalised age-related neurodegeneration of the auditory analyser, which causes the development of presbycusis, with progressive neurodegenerative changes in the parts of the brain responsible for cognitive functions, forms a clinically significant sensory-cognitive deficit. This recurrent pathophysiological mechanism, which consists of auditory perception and deprivation, decreased cognitive performance, limited functional autonomy of patients and other related processes, collectively manifests clinically as a degradation of integral indicators of quality of life in the ageing process. Thus, the decline in auditory function in older adults not only impairs their social integration but also significantly limits their ability to perform daily tasks independently, which further exacerbates their psycho-emotional stress and leads to an increased risk of developing depressive disorders.

Age-related changes in microcirculation as a systemic pathogenetic factor in neuronal and tissue involution. Age-related metabolic transformation extends beyond neurons. X. Lu *et al.* [20] called ageing a systemic multi-organ process based on changes in cellular metabolism, mitochondrial dysfunction, inflammation, oxidative stress, and other pathological mechanisms triggered by energy homeostasis disruption. This work systematised current ideas about the role of exergines as key molecular mediators of a whole range of anti-ageing effects in gerontological practice. It has been proven that physical exercise induces the secretion of biologically active molecules such as brain-derived neurotrophic factor, Irisin, Fibroblast growth factor 21, Phospholipase D1 and a number of others, which trigger molecular cascades aimed at increasing cell resistance to oxidative stress and slowing down the processes of biological ageing. A leading role in this is played by the signalling systems of adenosine monophosphate-activated protein kinase, calcium/calmodulin-dependent protein kinases (CaMKs) and sirtuins, which ensure the induction of mitochondrial biogenesis, optimise energy metabolism, activate autophagic mechanisms of cell repair, and suppress NF-κB-mediated inflammation, while improving endothelial function and microcirculation. Therefore, optimisation of microcirculatory blood flow, including through physical activity, ensures effective delivery of oxygen and metabolic substrates to neuronal and sensory cells, which contributes to the stabilisation of

mitochondrial function, maintenance of ionic and redox homeostasis, regulation of neurotransmitter activity and activation of cellular adaptation mechanisms, resulting in the normalisation of intracellular metabolic processes and increased functional resistance of neural structures.

B.S. Tsai Do *et al.* [21] also showed that the development of bilateral presbycusis is caused by degenerative changes in the structures of the inner ear and auditory nerve. However, among the main pathogenetic involutional mechanisms, in addition to damage to the hair cells of the cochlea and impaired function of the spiral ganglion, they emphasised age-related changes in the vascular strip, which normally produces endolymph with a characteristic high concentration of acetylcholine and potassium ions and a low sodium content. The vascular strip of the inner ear is the only epithelial tissue in the body that contains blood vessels. Therefore, impaired blood supply can also be considered a factor which, in combination with neurodegeneration, indirectly leads to a decrease in the efficiency of transduction and neural transmission of sound signals. The authors emphasised the multifactorial nature of age-related hearing loss and noted that its rate of progression is influenced by genetic factors, cumulative noise exposure, systemic metabolic disorders and concomitant diseases.

Neurons are characterised by high energy requirements and limited internal energy reserves, so their functioning is critically dependent on a continuous and efficient blood supply [22]. Therefore, it would be logical to assume that impaired tissue perfusion may be a common aetiological factor or catalyst for the progression of sensory and cognitive impairments in older adults. In support of this, Y. Uchida *et al.* [23] presented vascular brain damage, including stroke and microvascular infarction, as one of the pathophysiological mechanisms linking sensory and cognitive decline. In particular, a statistically significant association between moderate and severe hearing loss and stroke was found in elderly participants in a population study. The authors emphasised the importance of microcirculatory disorders (atherosclerotic, metabolic or other) in the development of auditory analyser dysfunction and cognitive decline, reflecting the commonality of vascular-metabolic mechanisms in their pathogenesis.

K. Połtyn-Zaradna *et al*. [24] consider age-related hearing loss to be a potentially modifiable risk factor for dementia, pathogenetically linked to vascular insufficiency and haemodynamic disorders. The study showed that sensorineural hearing loss is associated with an increased prevalence of vascular and metabolic pathology, in particular arterial hypertension, ischaemic heart disease, angina pectoris, myocardial infarction, and metabolic syndrome, confirming its role in the formation of vascular-mediated mechanisms of cognitive decline. The ageing process is accompanied by the risk of not only sensory and cognitive changes. Older people are prone to multimorbidity, when metabolic disorders intensify, and cardiovascular and degenerative geriatric diseases appear [25]. The risk of developing age-related audiological changes in individuals with a cardiometabolic profile was explained by D.W. Maidment *et al*. [26] as being due to common microvascular disorders characteristic of these diseases. Microangiopathy is manifested by inhibition of angiogenesis, a decrease in the number of functioning capillaries, thickening of their basement membrane, endothelial dysfunction, and other structural changes that limit the perfusion of the metabolically active cochlea.

Vascular alteration leads to chronic tissue hypoperfusion, impaired transport of oxygen and metabolites, local hypoxia, and activation of inflammatory-oxidative cascades, which together contribute to the progression of organ dysfunction and neurodegenerative changes [27]. Y. Li *et al.* [28] described in detail the molecular mechanisms of metabolic vascular damage, which cover both the macro- and microcirculatory beds. They described their key pathogenetic links: endothelial dysfunction, chronic low-grade inflammation, oxidative stress, activation of the polyol and hexosamine pathways, as well as dysregulation of angiogenesis and vascular wall remodelling, which together lead to chronic hypoxia, metabolic failure and progressive organ dysfunction. The authors emphasised that diabetic microangiopathy is a universal mechanism of damage to various organs and systems and a substantial therapeutic target for strategies aimed at restoring vascular function and preventing neurodegenerative and sensory disorders.

Hemodynamic disturbances occur not only in peripheral areas. A study by O.Y. Bang *et al.* [29] showed that cerebral microangiopathy and macroangiopathy have common risk factors and biomarkers, indicating the unity of the pathogenetic mechanisms of vascular damage to the brain. The authors found that arterial hypertension, diabetes mellitus, dyslipidaemia, and inflammation are associated with both microvascular damage and large artery pathology, and that the key biological markers are indicators of endothelial dysfunction, oxidative stress, and systemic inflammation. The study determined that micro- and macrovascular disorders form a continuum of vascular pathology, which determines the progression of structural and functional changes in brain tissue, including ischaemic damage, neuronal dysfunction, and cognitive decline. The results confirm the concept of the systemic nature of vascular mechanisms of neurodegeneration and substantiate the role of microcirculatory insufficiency as a key link in the pathogenesis of age-related sensory and cognitive disorders.

Impaired blood flow is accompanied by activation of the coagulation cascade and the development of hypercoagulable syndrome, which further exacerbates microcirculatory disorders and tissue hypoxia. Increased blood clotting in the veins is clinically manifested mainly by pulmonary embolism, deep vein thrombosis, usually in the legs, and arterial thrombosis, which leads to ischaemia and tissue necrosis. Since the hair cells of the inner ear and the neurons of the auditory pathway and cortex are very sensitive to ischaemia, macro- and/or microthrombosis, for example, in the terminal capillary bed, which originates from the labyrinthine artery and provides vascularisation of the inner ear, leads to cochlear sensorineural hearing loss, and in the basin of the middle cerebral or anterior temporal arteries, i.e., the vessels that supply the upper auditory pathways, it can lead to central perceptual hearing loss [30].

Morphological changes in the capillary wall and hypercoagulation lead to hypoxic changes in cells, disrupting the transport of glucose, proteins, ions, and other metabolites necessary for electrophysiological processes. This pathophysiological cascade can cause direct damage to

metabolically active cells in the inner ear. Even temporary hypoxia has a stressful effect on cochlear cells. Changes in molecular transport are exacerbated by the accumulation of products of concomitant inflammation and oxidative stress, causing additional damage to cochlear cells [31]. C.Y. Förster *et al.* [31] showed that age-related hearing loss and Alzheimer's disease have a common vascular pathogenetic basis caused by dysfunction of the cerebrovascular system and microcirculation disorders. The authors substantiate the role of endothelial dysfunction, oxidative stress, and neuroinflammation as key mechanisms contributing to the simultaneous degeneration of the auditory analyser and cognitive structures of the brain. C.Y. Förster *et al.* noted that in older adults, cardiovascular disease, diabetes mellitus and other metabolic disorders, and hearing loss, among other things, are modified risk factors for cognitive disorders, with possibly identical mechanisms of development. During the search for common pathogenetic mechanisms in patients with Alzheimer's disease and presbycusis, MRI data revealed cortical microhaemorrhages, signs of cerebral hypoperfusion, and impaired blood-brain barrier permeability. Given the functional and structural similarity of the blood-brain and blood-labyrinth barriers, the study suggested that their damage is caused by the accumulation of β-amyloid or other neurotoxic metabolites, in particular active forms of oxygen, which contribute to the progression of neurosensory and cognitive disorders.

Potential dysmetabolic molecular mechanisms of hearing impairment associated with decreased microcirculation and neurodegeneration were demonstrated by R. Mittal *et al.* [32] using the example of hearing loss in the context of diabetes mellitus. The pathogenesis of changes caused by hyperglycaemia includes processes that are typical of atherosclerotic, vascular and metabolic microangiopathies, neuropathies and oxidative stress. These pathological reactions can damage sensory structures in the cochlea, including the vascular strip, spiral ganglion neurons, and hair cells, and may be the result of cochlear synaptopathy, microangiopathy, neuropathy, oxidative stress, mitochondrial abnormalities, and apoptosis-mediated cell death.

The multi-organ nature of this condition is accompanied by a decline in the function of the affected organs and, consequently, the entire body. H. Zhang *et al.* [33] conducted a cohort study in Dongfeng-Tongji, China, observing 18,625 individuals over a period of 5.5 years. They found that the adjusted mortality risk ratio, including cardiovascular problems, increased with the progression of hearing deprivation. In addition, the risk increased even more when combined with above-average hearing loss, noise exposure, diabetes or hypertension.

Thus, micro- and macroangiopathies in older individuals may, on the one hand, be the result of physiological changes in the ageing body, i.e., one of the causes of presbycusis, and, on the other hand, they may be the result of concomitant age-related metabolic and cardiovascular diseases that contribute to sensory deprivation. The question of which of these is primary and which is secondary is debatable. However, it is an indisputable fact that the combination of these processes has a mutually aggravating effect, reducing quality of life and increasing the risk of death. In addition, these changes significantly complicate clinical management and require a comprehensive approach to treatment.

Hearing deprivation as a determinant of reduced physical and social activity. A systematic review by P. Martinez-Amezcua *et al.* [34] analysed the relationship between hearing impairment and physical activity levels, measured using objective methods, in an elderly population. The authors included five studies based on data from the National Health and Nutrition Examination Survey and used accelerometry to quantify physical activity. The pooled results show that hearing impairment, especially moderate to severe, is associated with reduced physical activity and increased sedentary behaviour. D.S. Chen *et al.* [35] indicated that the presence of hearing dysfunction is associated with a deterioration in mobility, strength, and functional independence, which increases the risk of disability in women by 30%. Therefore, hearing deprivation is an independent predictor of functional decline, indicating its systemic impact on the processes of age-related decline in physical reserve. According to B.S.Y. Yeo *et al.* [36], the presence of presbycusis is significantly associated with an increased risk of falls in older adults. The identified relationship is due to a complex of pathogenetic mechanisms, in particular, impaired sensory integration between the auditory, vestibular and proprioceptive systems, decreased postural stability and compensatory redistribution of cognitive resources for processing acoustic information. Concomitant neurodegenerative and vascular processes contribute to functional vulnerability and impaired motor control. Fear of falling and feelings of helplessness force people to lead a more sedentary lifestyle [21].

The recommendations published by the World Health Organisation [37] stated that to achieve optimal health benefits, older adults need moderate-intensity aerobic physical activity for 150-300 minutes per week or high-intensity activity for at least 75-150 minutes. However, according to a longitudinal study of ageing conducted by M.V. Goodwin *et al.* [38], British people over the age of 50 with hearing loss move significantly less, and the decline in this activity occurs faster in them compared to those who did not report hearing loss. Decreased physical activity may also be a consequence of age-related reduction in skeletal muscle mass and strength. In addition, alteration of their capillary network contributes to reduced blood flow and oxygen delivery to contracting muscle fibres. C.M. Hearon & F.A. Dinenno [39] described an age-related impairment of "functional sympatholysis", the ability of muscles to contract despite sympathetic vasoconstriction, leading to reduced blood flow and oxygen delivery during physical exertion. This process is critically relevant for the proper regulation of tissue blood flow distribution and oxygen transport. A decrease in physical activity during ageing is accompanied by a reduction in the oxygen demand for muscle contraction due to a slowdown in oxidative metabolism, which in turn leads to a weakening of the haemodynamic response and a decrease in the stimulation of muscle blood flow.

The progressive decline in cognitive and physical functions contributes to the deterioration of mental health and increases the risk of developing depressive disorders and psychological distress. J.S. Golub *et al.* [40] found that more than a third of the 5,328 participants in a cross-sectional study had clinically significant depressive symptoms.

According to J.A. Holman *et al.* [41], hearing loss causes increased fatigue, which in turn affects the level of physical activity and subjective well-being of people with hearing impairment. The study established that hearing dysfunction is indirectly associated with decreased activity and impaired quality of life due to increased cognitive load and energy expenditure on auditory perception. Fatigue associated with auditory load is a relevant mediator between hearing impairment and psychophysical state.

Emotional well-being is a fundamental component of human life and a relevant determinant of mental and physical health. A positive emotional state, formed through feelings of confidence, involvement, satisfaction and the achievement of personally meaningful goals, contributes to maintaining psychophysiological balance. Harmonious interpersonal interaction and positive emotions are central in shaping social activity and the adaptive potential of the individual [42]. With age, the cumulative effect of exogenous and endogenous factors on auditory function is primarily manifested by an increase in hearing thresholds in the high-frequency range of the sensorineural type, which progresses at an individually variable rate, involving the frequencies of the main speech spectrum and the low-frequency range. As the degree of hearing loss increases, speech intelligibility deteriorates, which significantly complicates communication, especially in conditions of background noise. According to B.H.B. Timmer *et al.* [42], in such conditions, people with hearing impairments more often experience feelings of insecurity, discomfort and frustration, which leads to avoidance of socially complex situations, in particular interaction in large groups or noisy environments. The fear of social stigmatisation limits involvement in interpersonal interaction, reduces motivation to communicate and communicative activity, which negatively affects the social well-being of people with audiological impairments.

Studying the impact of hearing loss on communication, A. Shukla *et al.* [6] described an increased risk of loneliness and social isolation. Social isolation is described as a decrease in the number of social contacts and the frequency of interaction between them, i.e. it is a kind of measure of a person's social network. Loneliness, on the other hand, is a subjective emotional response to a perceived mismatch between the actual and desired levels of social connections. P.L. Ramage-Morin [43] found that hearing deprivation in people aged 45 and older is significantly associated with increased levels of social isolation. The study found that people with hearing impairments are more likely to report limited social contacts, reduced involvement in interpersonal interactions, and emotional alienation compared to people without hearing problems. According to A. Chern & J.S. Golub [44], hearing loss causes feelings of emotional distance and complicates communication in a social environment, forming persistent communication barriers. To adequately perceive speech, people with hearing impairments are forced to engage additional cognitive resources, which is accompanied by increased cognitive load and the development of hearing-induced fatigue. Prolonged overload of cognitive mechanisms contributes to the progressive depletion of cognitive reserves, which can accelerate the formation of cognitive deficits and psychosocial disorders.

This creates a pathological circle: difficulties in processing auditory perception cause excessive cognitive load, which, in turn, exacerbates the development of neurodegenerative processes (i.e., exacerbates existing impaired auditory neurosensory function) and causes structural changes in the brain, further impairing cognitive processes. In addition, hypothetically, such excessive load can lead to cognitive decline due to the fact that in such conditions there is a redistribution of cognitive resources towards the processing of auditory information, "robbing" other mental processes, such as working memory [23]. The forced need to perceive and analyse overly quiet sound signals increases the load not only on cognitive resources. The attention resources involved in controlling posture and balance are also overloaded. B.S. Tsai Do *et al.* [21] presented the results of a cross-sectional study that showed a positive correlation between an increased probability of social isolation and the degree of hearing loss in elderly Americans. Moreover, the desire for solitude does not depend on whether such individuals receive correction for audiological disorders or not. Under such conditions, the risk of cognitive decline increases: brain stimulation is limited, and the mechanisms involved in overcoming not only mental but also physical difficulties are reduced. In other words, communication disorders associated with presbycusis have a direct impact not only on social activity but also on the overall quality of life, including physical health. In other words, communication problems associated with presbycusis have a direct impact not only on social activity but also on overall quality of life, including physical activity.

Hearing loss is just one of the symptoms of age-related deprivation, which has several common pathogenetic links with cardiovascular and metabolic diseases. Improving hearing function contributes to increased social activity, optimisation of cognitive resources and a reduction in hearing-related fatigue, which has a positive overall effect on the quality of life of older people. Timely audiological rehabilitation indirectly improves physical functioning, psycho-emotional state, and vascular-metabolic indicators. Thus, correction of hearing dysfunction is a substantial component of strategies for maintaining functional independence and preserving a high quality of life in the ageing process.

CONCLUSIONS

An analysis of scientific data has revealed that age-related hearing loss is becoming a global medical and social issue. The analysis showed that disabling hearing loss is one of the key problems in geriatrics, requiring improved approaches to early diagnosis and comprehensive correction. The study proved that the leading link in the pathogenesis of presbycusis is degenerative changes in the neurons of the auditory pathways from peripheral receptor structures to cortical centres, the development of which is exacerbated by age-related microvascular disorders, hypoperfusion and hypoxia of the cells of the auditory and vestibular apparatus. Genetically determined alteration is a consequence of changes in the expression of Atoh1, Neurog1, Neurod1, deficiency of transcription factors and their regulatory disorders, which lead to disruption of intracellular biochemical and electrophysiological processes, mitochondrial dysfunction, excessive oxidative activity, etc. The study

established that similar pathophysiological mechanisms underlie age-related cognitive disorders, which often develop in the context of hearing loss or in parallel with it, demonstrating a mutually reinforcing negative effect. Study determined that auditory deprivation causes excessive cognitive load, leading to depletion of cognitive reserve, decreased physical activity, and limited social engagement. The analysis showed that the multi-organ nature of age-related changes determines the need for a multidisciplinary approach to the prevention and treatment of hearing disorders and associated diseases to preserve the functional independence and quality of life of older people. Prospects for further research lie in an in-depth study of the causal relationships between hearing dysfunction and cognitive decline, as well as in the development of integrated models for the early diagnosis and rehabilitation of age-related sensory and cognitive disorders.

ACKNOWLEDGEMENTS
None.

FUNDING
None.

CONFLICT OF INTEREST
The author declares no conflict of interest.

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Нейробіологічні аспекти слухової депривації та її вплив на якість життя у похилому віці

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Анотація. Вікові зміни в організмі можуть призводити до функціональних обмежень, які ускладнюють старшим людям підтримку способу життя та виконання соціальних та особистісних ролей, що впливає на здатність жити повноцінно. Стаття присвячена з'ясуванню основних механізмів впливу зниження слуху на якість життя у осіб похилого віку. Було опрацьовано наукові публікації за 2016-2025 роки у електронній базі даних біомедичних досліджень MEDLINE/PubMed. Аналіз показав, що вікова втрата слуху передусім зумовлена нейродегенеративними процесами. Було з'ясовано, що дегенерація нейронів слухового шляху – від волоскових клітин кохлеї до нейронів неокортексу – проявляється редукцією клітинної популяції, морфологічною альтерацією нейронів і зменшенням кількості синаптичних контактів. Ці зміни супроводжуються порушенням біохімічного та електрофізіологічного гомеостазу, дисрегуляцією внутрішньоклітинної кальцієвої сигналізації та зниженням рівнів ключових нейромедіаторів, зокрема глутамату, гліцину та γ-аміномасляної кислоти. Подальший аналіз засвідчив, що зазначені нейробіологічні порушення призводять до зниження імпульсної активності та ослаблення гальмівних процесів, що клінічно проявляється слуховою дисфункцією, погіршенням сприйняття акустичних сигналів і зниженням здатності до просторової локалізації звуку. Було встановлено, що супутня мікроангіопатія характеризується пригніченням ангіогенезу, зменшенням щільності функціонуючих капілярів, потовщенням базальної мембрани та ендотеліальною дисфункцією, що зумовлює зниження тканинної перфузії. Було з'ясовано, що мікроциркуляторна недостатність сприяє вторинному ішемічному ушкодженню клітин через активацію оксидативного стресу та запальних каскадів. Генералізована вікова дегенерація нейронів і мікросудинного русла виявляється також у структурах мозку, відповідальних за когнітивні функції. У результаті формується рекурентний патофізіологічний механізм: слухова депривація підвищує когнітивне навантаження та прискорює виснаження нейронних ресурсів, що, своєю чергою, посилює нейродегенеративні процеси й структурні зміни мозку. Аналіз показав, що ці процеси взаємно потенціюють одне одного, спричиняючи прогресивне погіршення когнітивних функцій і зниження фізичної та соціальної активності у осіб похилого віку

Ключові слова: пресбіакузис; сенсоневральна приглухуватість; деменція; когнітивна функція; нейродегенерація; ангіопатія