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Advances in MicroRNA in Sensorineural Hearing Loss

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Abstract: Hearing loss is one of the global health burdens, and the increased risk of communication barriers, social isolation and depression poses a significant threat to the quality of life of patients. A report by the World Health Organization (WHO) estimated that about 466 million people worldwide are affected by disabling hearing loss, of which 34 million are children [1]. Among them, Sensorineural Hearing Loss (SNHL) is one of the common types of hearing loss with a complex pathogenesis, which is usually caused by damage to cochlear sensory hair cells (HCs). Current treatments for SNHL focus on amplifying sound through hearing aids or electrically stimulating auditory neurons through cochlear implantation (CI) for severe to profound deafness; however, neither approach restores the intrinsic sensory hair cells of the inner ear [2]. In recent years, MicroRNAs (Micro Ribonucleic Acids, miRNAs), as key regulatory molecules of gene expression, are involved in the growth and development of cochlear hair cells, and their role in SNHL has gradually become a hot research topic. In this paper, we systematically review the experimental research progress and clinical application potential of miRNAs in SNHL, including the diagnostic value of miRNAs as biomarkers, and the regulatory mechanism in cochlear cell damage and repair, which is helpful to solve the clinical problems of hearing loss.

Keywords: Sensorineural Hearing Loss; miRNAs; Inner Ear; Biomarkers.

1. Introduction

Sensorineural Hearing Loss (SNHL) is one of the most common types of hearing disorders worldwide, with a complex and diverse etiologic mechanism including genetic factors, noise exposure, age-related degeneration, viral infections (e.g., CMV infection) [3] and cardiovascular diseases [4]. Early diagnosis of SNHL is still challenging, and the lack of specific biomarkers makes it difficult to clarify the etiology and delays intervention in many cases [5]. Recent studies have shown that oxidative stress, inner ear hair cell damage, and altered plasticity of auditory neural pathways are common pathological mechanisms in SNHL [6-8]. Notably, micro Ribonucleic Acids (miRNAs), as a class of small non-coding RNA molecules that regulate gene expression, play a key role in the pathology of sensorineural deafness (SNHL) through post-transcriptional regulatory mechanisms. Studies have shown that miRNAs can be involved in core pathological processes of SNHL, such as hair cell damage and spiral neuron degeneration, by targeting and regulating key molecules such as cochlear potassium channels (e.g., KCNQ4), gap junction proteins (e.g., connexin-43), and neurotrophic factor signaling pathways [6, 9, 10]. Based on their extensive regulatory roles in the auditory system, miRNAs have become a research hotspot for the molecular mechanisms of SNHL [11, 12]. Thus, in-depth analysis of the molecular regulatory mechanisms of miRNAs in the development of SNHL will significantly enhance their application value in clinical diagnosis and treatment. These breakthroughs will not only help to establish an early screening system based on miRNAs, but also promote the molecular typing diagnosis and individualized prognosis of SNHL. In this review, the regulatory mechanisms of miRNAs in SNHL and their molecular networks are systematically reviewed, and based on the existing evidence, the prospects and challenges of their application in clinical translation are objectively discussed.

2. MicroRNA Overview

MicroRNAs (Micro Ribonucleic Acids, miRNAs) are a class of single-stranded, non-coding small molecule RNAs of approximately 21-23 nucleotides in length. miRNAs play a key role in the regulation of gene expression through their complex regulatory mechanisms. In mammals, a single miRNA molecule can complementarily pair with specific binding sites in the 3' untranslated region (3'-UTR) of target genes through its seed sequences to achieve regulation of multiple target mRNAs and control target gene expression through translational repression and mRNA destabilization [13, 14]. It has been shown that the human genome encodes more than 2,500 miRNAs, and these non-coding RNA molecules are involved in regulating the expression activities of about one-third of protein-coding genes [15]. Of particular note, the expression profiles of 102 miRNAs have been detected in inner ear tissues, and these molecules play important regulatory roles both during embryonic development of the auditory organ and postnatal maintenance of its function [16]. Intensive studies in recent years have revealed that miRNAs are involved in the regulation of a variety of important biological processes, including cell differentiation and proliferation, organ development, programmed cell death, and regulation of immune system functions [17-19]. Researchers have shown that disrupting miRNA processing by conditionally knocking down the Dicer gene under the control of the Pax2 promoter can support the role of miRNAs in inner ear morphogenesis and innervation [20]. As the regulatory mechanisms of miRNAs in auditory physiology and pathology have been gradually elucidated, more and more researchers have been devoted to exploring the molecular mechanisms of miRNAs in the development and functional maintenance of the inner ear.

3. Molecular Mechanisms of miRNA in the SNHL

3.1. Advances in miRNA Research in Inner Ear Development

miRNAs play key roles in mammalian inner ear development and sensory epithelial differentiation. More than one-third of known miRNAs have been identified in the mammalian inner ear, and some of these molecules are highly conserved evolutionarily and closely related to the development and function of mechanosensory cells. For example, specific miRNAs can influence the formation of inner ear structures by regulating the transformation of the primitive ear neuroepithelium to the sensory epithelium [21]. In cochlear hair cells and spiral ganglia, miR-183 family, miR-15a, miR-140 and miR-194 have been demonstrated [22]. Among them, the miR-183 family members (miR183/96/182) are the most important, which play a key role in the development of functional sensory cells, such as inner ear hair cells [23]. miR-183 family is often expressed in hair cells, lateral line neural thalamus, and sensory cells of the eye and nose [23]. Among the members of this family, a single-base mutation in the seed region of miR-96 leads to progressive hearing loss in humans and mice by a mechanism that involves degenerative changes in hair cells and aberrant expression of key cochlear genes (e.g., Slc26a5, Gfi1) [24, 25]. Animal models further revealed that miR-183/96 double knockout (dko) mice not only exhibited peripheral hearing loss, but also reduced nuclear volume in the auditory brainstem, abnormal presynaptic vesicle release, and disturbed distribution of post-synaptic AMPA receptor (GluA1), suggesting that miRNAs affect the function of central auditory pathways by regulating synaptic plasticity and neuronal survival [26]. In addition, upregulation of miR-182 promotes the differentiation of progenitor cells into hair cells, and the function of miR-182 may be related to its potential target Tbx1 [27]. It has been shown that in mice after birth, miRNA is expressed only in hair cells and spiral ganglia, while after maturation, miRNA expression is restricted in hair cells and increased in sites such as the internal sulcus and spiral margin [28]. It has now been found [29] that cochlear hair cells are predominantly enriched for miR-34a family and miR-183 family. This difference in expression also predicts that miRNAs play an important role in inner ear development. Future studies need to deeply analyze the synergistic regulatory mechanism between miRNAs and downstream target genes, explore their regulatory potential in hair cell regeneration, and provide theoretical basis for hearing restoration strategies based on miRNA intervention.

3.2. Progress in Molecular Studies of miRNA-Mediated Regulation of SNHL

Many miRNAs have been shown to be associated with congenital or acquired SNHL [30]. the pathological process of SNHL is highly correlated with cochlear oxidative stress injury. lei Ding et al. found that miR-106a exacerbated glucose oxidase-induced apoptosis and oxidative stress in cochlear limbal cells by targeting and inhibiting connexin-43 (connexin-43) (manifested as elevated MDA and inhibited SOD activity), whereas inhibition of miR-106a activated the Nrf2-Keap1-HO1 pathway and reversed cell damage [10]. In addition, the differential expression of oxidative stress-related miRNAs such as miR-34b/c and miR-449a/b in the cochlear nucleus and inferior colliculus after noise exposure suggests that they may regulate neuroplasticity in the central auditory system through signaling pathways such as MAPK and ErbB [31, 32]. miR-34a and miR-29b were expressed through SIRT1/PGC-1α, SIRT1/p53 and SIRT1/HIF-1α pathways in hypoxia or other oxidative stress-related acquired SNHL [8]. Inhibition of miR-145b ameliorated noise-induced SNHL in mice by upregulating AP2B1 expression [33]. scholarly studies by Wen-Dai Bao et al [34] demonstrated the critical role of miR124/Fpn signaling in iron metabolism and neuronal death after cerebral hemorrhage in an aged mouse model, and that up-regulation of Fpn or inhibition of miR-124 may be a promising treatment for the disease. promising approach. Notably, some differentially expressed miRNAs mostly have important regulatory functions in the nervous system, and their potential target mRNA molecules are mainly enriched in key signaling pathways such as PI3K/Akt and MAPK [35]. From a molecular biological perspective, the PI3K/Akt signaling pathway, as an important self-protection mechanism in the inner ear, has a key role in maintaining the long-term survival of hair cells [36]. Experimental evidence has shown that down-regulation of the expression or functional inhibition of Akt signaling molecules is closely associated with pathological processes such as ototoxic injury, senile deafness, and hair cell apoptosis [37]. miRNA association with oxidative stress and apoptosis provides a target for early intervention in SNHL, but most of the existing studies are based on animal models, and the clinical translation needs to validate the consistency of miRNA expression in humans. In addition, how miRNAs synergize with other markers of oxidative stress (e.g., ROS) still needs to be deeply explored.

4. miRNAs as Disease Markers in SNHL

SNHL, as a common sensory disorder, most researchers began to work on finding reliable biomarkers to assist in the early diagnosis and prognosis assessment of SNHL. Currently, some inflammation-related factors, inner ear-specific proteins, and oxidative stress markers are associated with SNHL. For example, pro-inflammatory factors such as IL-6 and TNF-α [38, 39], inner ear-specific prestin proteins [40], reactive oxygen species (ROS), and antioxidant enzymes (e.g., SOD, GSH-Px) [41, 42] have been shown to have varying levels of expression during the pathogenesis of SNHL [43]. Notably, the differential expression of miRNAs in the inner ear and the cross-regulation of downstream target genes allow them to be used as novel molecular markers of sensorineural deafness (SNHL), showing significant potential in its early diagnosis and mechanism resolution. miRNA peripheral expression profiles correlate significantly with pathologic changes in the inner ear. Several studies have revealed the aberrant expression patterns of specific miRNAs in SNHL and their clinical value: in oxidative stress-related mechanisms, miR-34a and miR-29b are involved in inner ear hair cell damage by regulating the ROS (reactive oxygen species) pathway, and their expression levels correlate significantly with the progression of SNHL disease [8]. Exosome-derived miRNAs (e.g., PC-5p-38556_39, PC-5p-29163_54, and miR-93-3p) have been shown to serve as potential diagnostic markers of sudden SNHL by carrying signals from the cochlear microenvironment [44]. The regulatory network of miRNAs is characterized by multi-targets, for example, miR-145b has

been shown to be a key regulator of noisy SNHL by affecting auditory synaptic transmission through targeting the AP2B1 gene [33]. In terms of clinical translational research, Sun Mok Ha's [45] team found that serum miR-183, miR-210 and other molecules can dynamically reflect the degree of inner ear hair cell damage, and their expression profile characteristics provide a new idea for early noninvasive diagnosis of SNHL. Meanwhile, Desmond A. Nunez and other scholars showed that the differential expression of eight miRNAs, such as hsa-miR-590-5p and hsa-miR-186-5p, in the sera of SSNHL patients had significant specificity through cohort analysis [35]. miRNA markers research not only provides a new perspective for molecular typing of SNHL, but also its high sensitivity, accessibility also lays a theoretical foundation for the development of minimally invasive diagnostic tools.

5. Therapeutic Promise of miRNAs in SNHL

Currently, many miRNA-specific modulations provide new ideas for SNHL treatment. For example, whereas miR-183/96 cluster deletion resulted in an enlarged presynaptic vesicle release pool and abnormal postsynaptic receptors, suggesting that it plays a key role in correct synaptic transmission and development of the auditory hindbrain [24]. In addition, the identification of differentially expressed miRNAs such as miR-200 family and miR-34b/c in the HEI-OC1 cell differentiation model provides new targets for in vitro screening of otoprotective drugs [32]. Dysregulation of exosomal miRNAs (e.g., miR-19a, miR-19b) in head and neck cancers suggests that similar mechanisms may be applicable to SNHL [46]. miR-34a/miR-29b modulation of oxidative stress pathways [8] may be a therapeutic direction for SNHL. Interaction of long chain non-coding RNAs (e.g., SNHG17) with miRNAs predicts survival in lymphoma patients, and similar approaches may be applicable to SNHL [47]. Although animal experiments have shown the effectiveness of miRNA interventions, cochlear targeting and safety of their delivery systems (e.g., liposomes, exosomes) remain bottlenecks. In addition, miRNAs as non-invasive biomarkers require standardized detection methods (e.g., PCR, fluorescence sensing platforms) [48, 49], and highly specific delivery strategies need to be developed.

6. Concluding Remarks and Future Outlook

As key molecules regulating the pathological process of SNHL, miRNAs are of great value in the fields of diagnostic marker screening and targeted therapy. Currently, it is necessary to integrate multi-omics technology to promote its clinical application: at the level of mechanism research, single-cell sequencing combined with spatial transcriptome analysis can be used to elucidate the dynamic regulation of miRNAs in different cell subpopulations of the cochlea; in the development of therapeutic strategies, the construction of a new type of nano delivery platform to achieve precise targeting of miRNAs is expected to break through the bottleneck of the intervention of drug-induced or noise-induced hearing damage; optimization of diagnostic technology. As for the optimization of diagnostic technology, the current research may require the construction of a large-scale miRNA expression profile database and the establishment of a highly sensitive diagnostic system through the joint detection of serum miRNAs and traditional biomarkers (e.g. prestin protein). Therefore, miRNA research is expected to move from mechanism exploration to individualized medical treatment, opening up a brand new path for precise typing, early intervention and gene editing treatment of SNHL.

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