doi: 10.3969/j.issn.1672-4933.2026.03.011

老年性聋患者心理和认知功能障碍研究进展

The Relationship between Age-Related Hearing Loss, Mental and Cognitive Disorders: A Review and Recent Developments

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【摘要】老年性聋在老年人群中高发且普遍存在。老年性聋患者易出现社交退缩、社会孤立，进而伴随心理障碍与认知功能下降，已成为国内外研究热点。本文系统梳理近年国内外相关研究，总结老年性聋与老年人心理健康及认知功能损害之间的潜在关联，为临床干预与康复提供理论参考。

【关键词】老年性聋；心理障碍；认知功能障碍

【中图分类号】R764.43 【文献标识码】A 【文章编号】1672-4933(2026)03-0272-04

【**Abstract**】The Relationship between Age-Related Hearing Loss, Mental and Cognitive Disorders: A Review and Recent DevelopmentsAge-related hearing loss (ARHL) is highly prevalent in the elderly. Accumulating evidence suggests that older adults with ARHL are prone to social withdrawal and isolation, which further contribute so the development of psychological disorders and cognitive decline. This topic has become a major focus of research worldwide. This article reviews recent domestic and international literature, and systematically summarizes the potential links between ARHL, mental health problems, and cognitive dysfunction in the elderly.

【**Key words**】Age-related hearing loss; Mental disorders; Cognitive disorder

老年性聋又称年龄相关性听力下降(age-related hearing loss, ARHL)，以高频听力损害、言语识别能力下降为主要特征，尤其在噪声环境中言语识别率下降显著[1]。ARHL是老年人常见的慢性感觉功能障碍之一，由听觉系统增龄性退行性改变累积所致。约半数70岁以上人群存在影响日常交流的显著听力损失，并与社会孤立、躯体衰弱、抑郁及痴呆等心理与认知损害密切相关[2,3]。ARHL是痴呆发生的第三大危险因素。终生噪声暴露、遗传易感性及代谢应激被认为是ARHL的主要病因，但具体病理机制尚未完全阐明[3]。

国外一项随机对照研究表明，ARHL可通过减少听觉输入、增加沟通负荷、降低社会参与度，对老年人心理健康产生不利影响，进而诱发抑郁情绪。助听器干预可有效改善听障老年人的抑郁状态，提升生活质量[4]。ARHL导致言语信息感知缺失，进而引发心理与社会适应障碍；助听器配戴及人工耳蜗植入可显著改善老年听障患者生活质量，对预防和缓解心理情绪障碍及认知功能衰退具有重要作用[5]。由此可见，ARHL与老年人心理及认知功能损害关系密切，但其内在关联及具体发病机制仍有待深入研究。本文围绕ARHL与心理障碍、认知功能障碍的相关性及潜在机制展开阐述，旨在为ARHL患者的临床诊疗与康复干预提供理论依据。

1 ARHL与心理障碍的相关性

ARHL患者心理障碍发生率显著升高，听力下降可增加老年人抑郁的发生风险。

抑郁症状与轻度听力损失相关，而与中重度听力损失无明显关联[6]。Lee等在社区老年人群研究中显示，经纯音测听评估的听力阈值水平与抑郁症状相关，而主观听力障碍自评结果则无此关联[7]。一项基于美国国家健康与营养检查调查分析显示，听力障碍与重度抑郁无显著相关，但每周配戴助听器≥5 h者抑郁发生率明显降

基金项目：中国高校产学研创新基金-华通国康医学科研专项“miR-34a/SIRT1/p53信号通路调控老年性聋相关认知功能障碍的机制研究”(2023HT085)

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低[8]。Contrera等将听力测定结果与情绪活力指标关联分析发现，中度以上听力障碍(≥40 dB)者发生情绪障碍的风险较健听者高23%[9,10]。

此外，约40%的ARHL患者合并耳鸣，其中近20%同时伴随前庭功能及平衡障碍[11]。由于耳鸣、前庭异常与认知衰退[12]及焦虑抑郁障碍[13]风险升高相关，其可能在ARHL相关神经精神损害的发生发展中发挥重要作用。现有神经生物学模型认为，耳鸣与抑郁类似，可能与5-羟色胺耗竭及边缘系统功能异常有关；慢性耳鸣可导致持续应激与生活状态紊乱，进一步增加抑郁风险[14]。综上，ARHL与老年人情绪心理障碍密切相关，通过早期干预改善听力水平，有助于降低心理障碍发生风险。

2 ARHL与认知功能障碍的相关性

ARHL是老年认知功能障碍的高危因素，与痴呆的发生发展密切相关[15]。在痴呆可调控危险因素中，ARHL约占9%，其影响程度高于高血压、肥胖、酗酒、糖尿病及吸烟等。一项为期12年的随访研究显示，听力损失相关痴呆风险为36.4%(研究对象年龄36～90岁)[16]。目前ARHL相关认知损害的神经生物学基础仍有待进一步阐明。

痴呆是超出正常衰老范畴，以认知功能进行性退化为核心的综合征，累及记忆、定向、计算、语言、执行功能及判断能力，常伴随情绪、行为与动机异常，并可早于认知损害出现。其病因多样，以阿尔茨海默病最常见，约占60%～70%。据世界卫生组织统计数据，全球约5500万人罹患痴呆，其中60%以上位于中低收入国家；随着人口老龄化加剧，预计2030年患病人数将增至7800万，2050年达1.39亿[17]。

队列研究表明，ARHL通常较痴呆早5～10年出现，可作为痴呆发生的潜在非侵入性生物标志物与预测因子[18]。ARHL患者听力下降程度与认知障碍的发生风险及严重程度呈正相关，听力损失越重、病程越长、年龄越高，认知损害风险越高、进展越显著，痴呆发生率亦随之升高[19]。因此，早期识别并干预ARHL，对预防老年认知障碍及痴呆具有重要意义。

有研究从认知储备受损机制出发，认为认知皮层可对ARHL患者受损的听觉感知加工进行代偿，而该过程与认知能力下降相关；助听器干预可重塑受损的听觉-认知皮层功能连接[20]。美国一项多中心随机对照研究(aging and cognitive health evaluation in elders，ACHIEVE)结果存在差异，该研究纳入70～84岁未经听力干预、无明显认知障碍的老年人，随机分为听力干预组与健康教育对照组，随访3年发现两组认知变化无显著差异，提示听力干预对认知下降风险较低人群的保护作用可能有限，而对高风险人群更具价值[21]。由于该研究样本量有限，可能存在偏倚，未来需大样本、长期随访的高质量研究进行验证。总体而言，ARHL与老年人认知功能障碍密切相关，其神经机制仍有待深入阐释。

3 ARHL相关心理与认知障碍的可能机制

ARHL是老年人常见的耳科疾病，常伴随听力下降、焦虑抑郁及认知损害，导致社会孤立与沟通障碍，严重影响老年人身心健康与生活质量。作为典型的增龄性疾病，其介导心理与认知损害的具体通路尚未完全明确。随着分子生物学技术发展，ARHL多层面机制逐步被揭示，其与认知损害及老年抑郁风险升高的关联可能涉及以下机制。

3.1 社会孤立与行为适应异常机制

ARHL患者因听力与交流困难易出现社交退缩，进而引发社会孤立、孤独感，继发焦虑、抑郁等情绪障碍。许多老年听障者主动回避复杂社交场景，减少与亲友互动，导致认知刺激不足，最终加速认知功能衰退[22～24]。多项国际观察性研究证实，ARHL与社会功能受损、社会支持减少及情绪相关角色受限显著相关[25,26]。听力障碍可减少老年人户外活动时间，增加休闲活动退出风险[27]，这一现象在社区及养老机构中均存在，重度听力损失老人社交参与度降低的风险升高1.4倍，较少参与机构活动的风险升高1.3倍[28]。美国一项关于与年龄相关的听力损失的报告所指出的，听力障碍对社会功能的影响可能在很大程度上取决于个人环境的需求[29]。

有研究提出整合行为与神经层面的理论模型，用以解释ARHL与认知及情绪障碍的关联[30]。一方面，社交退缩导致社会孤立、孤独感与抑郁；另一方面，ARHL使中枢听觉通路激活减少，引发认知控制网络代偿性激活增强、听觉-边缘系统功能连接异常及额叶区域失神经性萎缩。上述病理改变通过降低认知储备、加重执行功能损害、破坏情绪调节通路，共同促进认知衰退与抑郁发生。因此，社会孤立与行为适应异常是ARHL诱发心理及认知障碍的重要机制。

3.2 脑萎缩、结构重塑与整合功能下降

ARHL可导致脑结构改变与脑萎缩，进而影响大脑高级整合功能。一项平均随访6年的研究发现，ARHL患者右侧颞上回、颞中回、颞下回(参与言语处理、语义记忆及感觉整合的关键脑区)及全脑萎缩速率显著加快，与认知及精神心理障碍密切相关[31]。ARHL与右侧海马及左侧内嗅皮层灰质体积下降相关，而这些脑区正是阿尔茨海默病早期受累部位[32]，提示ARHL可能通过影响颞叶

结构，对邻近认知相关脑区产生级联效应。ARHL导致耳蜗声音编码能力下降[33]，听觉输入减少与耳蜗损伤可引发皮层形态与功能重塑。动物实验证实，耳蜗损伤可引起中枢神经元结构改变，听觉信号输入不足及毛细胞退行性病变可导致听觉与认知整合相关脑区萎缩[34]。流行病学研究提示，早期阿尔茨海默病相关脑结构改变，尤其是颞叶区域损伤，可能在ARHL与痴呆及精神障碍之间起部分介导作用[35]。

3.3 氧化应激及相关信号通路机制

ARHL以耳蜗毛细胞、螺旋神经节神经元及血管纹细胞不可逆的损伤为病理特征，显著影响65岁以上老年人生活质量，并增加抑郁、认知障碍及痴呆风险[36]。因此，阐明ARHL分子机制、寻找治疗靶点具有重要临床意义。

ARHL与认知障碍均为衰老相关神经退行性改变，活性氧（reactive oxygen species，ROS）增多及氧化应激增强是其共同核心机制。ROS清除能力下降导致氧化还原失衡，引发线粒体功能障碍与大分子损伤，进而促进细胞凋亡与组织退行性变[37]。内耳同样存在增龄性氧化应激失衡，被认为是毛细胞损伤、听觉系统衰老、脑白质变性及后续认知与情绪障碍的重要原因[38]。

多项分子研究揭示了关键调控靶点，分别为：① lncRNA Gm44593可通过靶向miR-29b减轻氧化应激，在内耳毛细胞损伤及ARHL进程中发挥重要作用[39]；② m^6A甲基转移酶METTL3可通过调控SIRT1的m^6A修饰水平，减少氧化应激诱导的细胞凋亡，在ARHL中具有保护效应[40]；③ Bcl2缺失可影响听觉皮层脂质代谢，导致突触功能异常与神经变性，造成内耳神经传导异常与听力下降，继而引发情绪及认知改变，提示Bcl2可能成为ARHL潜在干预靶点[41]。上述研究为ARHL的分子机制与靶向治疗提供了新思路。

4 结论与展望

ARHL与老年人焦虑、抑郁等心理障碍及认知损害、痴呆发生显著相关，听力损失是老年认知障碍与痴呆的重要危险因素。在人口老龄化持续加剧的背景下，加强ARHL早期筛查与及时干预具有重要公共卫生意义。进一步探索ARHL与心理、认知障碍之间的因果关系及分子机制，对构建精准诊断、预防与治疗策略至关重要。

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收稿日期 2024-12-11
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