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噪声性听力损失的干预进展

Progress in the Intervention of Noise-induced Hearing Loss

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【摘要】噪声性听力损失(noise-induced hearing loss, NIHL)是全球职业性和非职业性听力障碍的主要病因之一,约16%的致残性听力损失归因于职业噪声暴露,不仅降低患者生活质量,还与认知功能下降、心理障碍等共病密切相关。随着工业化进程的加快及娱乐噪声暴露的增加,NIHL的患病人数逐年增加。作为一种发病机制复杂但可预防的疾病,有效地干预尤为重要。本文旨在综述NIHL的发病机制及干预研究进展,为该领域的预防及诊治提供参考。

【关键词】噪声性听力损失;干预;发病机制

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【Abstract】Noise-induced hearing loss (NIHL) is one of the leading causes of occupational and non-occupational hearing impairment worldwide. Approximately 16% of disabling hearing loss can be attributed to occupational noise exposure. Not only does NIHL reduce patients' quality of life, but also is closely associated with comorbidities, such as cognitive decline and psychological disorders. With the acceleration of industrialization and increasing exposure to recreational noise, the prevalence of NIHL continues to rise annually. As a disease with complex pathogenesis that is nevertheless preventable, effective interventions are critically important. This study aims to review the pathogenesis of Noise-Induced Hearing Loss (NIHL) and progress in intervention research, providing a reference for early prevention, diagnosis, and treatment in this field.

【Key words】Noise-induced hearing loss; Intervention; Pathogenesis

听力损失已成为全球关注的重大公共卫生问题[1]。噪声性听力损失(noise-induced hearing loss, NIHL)是因长期或急性暴露于高强度噪声环境导致的感音神经性听力损伤,是仅次于老年性聋的第二大常见的获得性听力损失[2]。NIHL通常不可逆转,对患者的生活质量及经济社会产生深远的负面影响。在工业化国家或地区,NIHL造成的经济损失占国内生产总值(gross domestic product,GDP)的0.2%~2.0%[3]。因此,积极采取有效的预防措施降低NIHL的发生率,对保护公众听力健康和减轻社会经济负担具有重要意义。近年来,随着生物技术的快速发展,NIHL的干预研究进入了新阶段。本文就此予以综述,供听力学家、耳科医生和相关研究人员参考,以推动NIHL干预技术的发展。

1 噪声性听力损失的发病机制

1.1 机械损伤

NIHL的机械损伤机制主要基于高强度的噪声对耳部结构产生物理性破坏,当噪声强度超过130 dB时,会引起耳蜗内部结构组织损伤[4]。噪声声波通过听骨链传导至内耳,引发耳蜗内淋巴液剧烈流动,形成涡流,对内耳的听觉感受器,特别是毛细胞造成机械性冲击。导致毛细胞倒伏、脱落、变性乃至坏死,破坏毛细胞与其周围支持细胞间的连接,造成其大量缺失,导致Corti器整体结构坍塌、上皮化[5]。此外,高强度的脉冲噪声还可能引起前庭膜、网状板及基底膜撕裂,造成内淋巴液(富含钾离子)与外淋巴液(富含钠离子)混合,从而破坏耳内离子平衡,进一步影响耳蜗毛细胞功能[6]。由于人类毛细胞几乎不具备再生能力,这种损害通常是不可逆的,可导致永久性听力损失。

1.2 氧化应激

噪声暴露会导致耳蜗内活性氧(reactive oxygen species,ROS)水平显著升高,ROS是氧化应激的主要产物。当身体通过抗氧化防御消灭ROS的能力失衡时,就会发生氧化应激[7],从而引起细胞损伤、功能障碍和细胞死亡[8]。ROS产生在噪声暴露后,持续7~10天,从Corti器官的基底扩展到顶端,增加坏死和细胞凋亡的面积[9]。

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ROS诱导的脂质过氧化（丙二醛和4-羟基壬烯醛）可诱导细胞凋亡和血管活性脂质过氧化，并减少耳蜗血流量[10]。噪声诱导的缺血和随后的再灌注进一步促进了ROS的产生[11]。这种过量的ROS会损害耳蜗内脂质、蛋白质和DNA，引发细胞凋亡或坏死，从而导致听力损失[12]。此外，ROS还可诱导促炎细胞因子，如TNF-α、IL-1β和IL-6的表达，进一步加剧耳蜗的炎症和损伤[13]。

1.3 免疫和炎症反应

当个体长时间或高强度暴露于噪声环境，会引起耳蜗毛细胞的机械损伤，进而触发耳蜗内的免疫应答。噪声暴露后，耳蜗内的毛细胞会受损，同时激活内耳的免疫系统。噪声暴露可诱导耳蜗内促炎细胞因子，如肿瘤坏死因子-α（TNF-α）、白细胞介素-6（IL-6）等表达增加，通过激活内耳的免疫细胞，如巨噬细胞和小胶质细胞，引发局部炎症反应[14]。此外，噪声暴露还会导致耳蜗内NLRP3炎症小体激活，进一步促进促炎因子的释放，如IL-1β和IL-18，加剧炎症反应[15]。不仅加剧了毛细胞损伤，还可能通过NF-κB信号通路放大炎症反应[16]，持续的炎症反应会导致不可逆的细胞损伤和听力损失。

1.4 遗传易感性

NIHL是一种复杂的多因素疾病，其发病机制主要涉及环境因素、遗传因素及两者之间的交互作用[17]。在相同噪声暴露环境下，个体的噪声易感性存在显著差异，表明遗传因素可能在NIHL中发挥重要作用[18]。通过同性双胞胎调查发现，噪声敏感性在家庭中具有聚集性，NIHL的遗传易感性约36%[19]。此外，遗传变异导致的个体易感性差异对稳态噪声暴露引起的NIHL贡献率可能超过50%[20]。由于在相同职业噪声环境下收集双胞胎或家庭数据存在较大困难，NIHL的遗传易感性研究主要集中在动物模型。目前，研究发现多种基因与NIHL易感性之间存在关联，如氧化应激相关基因（*CDH23*[21]、*HDAC2*[22]、*CAT*[23]、*GSTP1*[24]、*FAS*[25]）、免疫炎症相关基因（*CARD8*[26]、*STAT3*[27]、*JNK1*[28]）、钾离子通道相关基因（*KCNQ1*、*KCNE1*[29]、*KCNJ10*[30]、*KCNMA1*[31]）、热休克蛋白70基因（*HSPA1A*、*HSPA1L*[32]、*HSPA1B*[33]）等。

2 噪声性听力损失的干预进展

2.1 助听器和人工耳蜗

助听器是补偿听力的辅助设备，其工作原理是基于半导体放大技术，放大外来声源的声强，补偿残余听力，使听障患者感受到原本无法感知的声信号，适用于轻度至中度听力损失患者，不仅可有效改善听力，还能提高其生活质量和社会参与能力[34]。在噪声环境中，通过对是否配戴助听器的患者进行言语识别阈值对比，发现配戴助听器可在一定程度上改善患者噪声环境中的言语识别能力[35]。然而，根据2021年《世界听力报告》显示，全球仅有17%的人从助听器中受益，中国老年人助听器使用率低于10%[36,37]。

对于双耳极重度听力损失患者，若助听器无法满足正常的听觉言语交流需求，人工耳蜗植入（CI）是目前主要和有效的治疗方法[38,39]。这种电子装置可将声音转换为编码形式的电信号，通过植入体内的电极系统兴奋听神经，使双侧极重度听力损失患者恢复感知声音的能力[40]。对重度至极重度感音神经性听力损失患者进行双侧人工耳蜗植入，可显著改善患者的言语感知、言语产生能力提高、阅读成绩和生活质量[41]。Li等[42]对国产CI者进行了10年随访，证明CI后1个月辅助听力阈值显著提高，且10年内保持稳定。

早发现、早诊断、早干预是有效防治听力损失的原则。当发生NIHL时，要及早进行干预。轻度至中度NIHL患者可选择配戴助听器改善生活质量。重度至极重度听力损失患者，尤其是当助听器无法提供足够帮助时，人工耳蜗植入是有效的选择。

2.2 药物疗法

噪声引起的听力损失是导致听觉功能障碍的主要非遗传因素，其发病机制复杂，涉及氧化应激、炎症反应、细胞凋亡等多个病理生理过程。目前，尚无针对NIHL的食品药品监督管理局（food and drug administration，FDA）批准药物，主要通过抗炎、抗氧化、抗凋亡和抗兴奋等途径延缓NIHL恶化。

在抗氧化疗法上面，多项研究报道了针对ROS相关NIHL的抗氧化疗法，该研究涉及多种抗氧化剂，包括N-乙酰半胱氨酸（n-acetylcysteine，NAC）[43,44]、D-蛋氨酸[45]、α-硫辛酸[46]和辅酶Q10[47]等，其在预防和治疗NIHL中显示出良好效果。其中，NAC是动物模型和人体研究中使用最多的抗氧化剂。对暴露于军事噪声5年及以上人员直接给予400 mg NAC治疗，其单耳或双耳的暂时性听力损失风险降低39%[48]。此外，在豚鼠噪声暴露前1小时给予维生素A、C、E和硫酸镁（MgSO4）的ACEMg联合方案，比单独使用维生素A、C、E或MgSO4更能保持听力敏感性，且长期大剂量摄入微量营养素未显示出明显的不良反应，表明联合抗氧化疗法比单药治疗更有效减轻NIHL[49]。

在炎症调控方面，抗炎药物可通过抑制耳内炎症因子表达，减轻内耳炎症反应，从而保护听觉系统。依布硒啉（ebselen）是一种合成的含硒分子，具有抗炎特性，用于治疗各种形式的感音神经性听力损失[50]。在噪声暴露前2天开始，连续4天每日2次口服400 mg依布硒啉，结果

显示噪声暴露后15分钟的4 kHz听觉阈值偏移(temporary threshold shift, TTS)较安慰剂组显著降低68%,且未观察到明显不良反应,证实了其对短暂性噪声性听力损失的有效性和安全性[51]。糖皮质激素是目前研究广泛的抗炎药物。将小鼠暴露于110 dB SPL白噪声下持续2小时,随即腹腔注射地塞米松并连续给药5天。发现与仅接受生理盐水治疗的小鼠相比,地塞米松治疗组小鼠的听觉脑干反应(ABR)在低频声音水平的恢复上更显著[52]。一项临床研究[53]比较了NIHL患者发病3天后,单纯口服泼尼松龙和口服泼尼松龙联合鼓室注射地塞米松对NIHL的防治效果,发现联合治疗组患者的平均听力改善率和言语辨别改善率更高。

神经营养因子(neurotrophic factors)是能够促进神经细胞生长、分化、存活的蛋白质,对内耳的毛细胞和听觉神经元具有保护作用。脑源性神经营养因子(brain-derived neurotrophic factor,BDNF)对听觉神经元具有营养支持作用,小细胞外囊泡作为载体递送BDNF已被证明能减轻噪声引起的内毛细胞带突触和耳蜗神经末梢损失[54],可作为NIHL的治疗策略。此外,BDNF与聚(DL-乳酸-羟基乙酸共聚物)[poly(dl-lactic-co-glycolic acid),PLGA]水凝胶结合,通过单剂量微创中耳注射,在噪声暴露后14小时内给药,可实现NIHL完全修复,显著恢复听力阈值和突触数量[55]。神经营养因子3(neurotrophin-3,NT3)在人工耳蜗支持细胞中的过表达也能诱导部分突触拯救和耳蜗功能恢复。腺相关病毒(adeno-associated virus, AAV)介导的NT3过表达,在噪声暴露前3周给药时,能显著减少突触损伤,尤其在耳蜗某些区域几乎完全恢复突触连接,而暴露后5小时给药效果不显著[56],表明该治疗在暴露前更有效。

由于传统药物递送的局限性,纳米颗粒(nanoparticles,NPs)在耳蜗内药物递送中的潜力受到关注。Zhang等[57]开发了基于超氧化物歧化酶(superoxide dismutase,SOD)和沸石咪唑框架-8(zeolitic imidazolate framework-8,ZIF-8)的纳米复合物(SOD@ZIF-8),该复合物在噪声暴露前1天通过圆窗膜给药,能够显著减轻大鼠的听力损失和毛细胞损伤,表现出预防性保护作用。Zeqi Zhao等[58]研发了ROS响应性纳米粒作为小檗碱(berberine, BBR)的载体磷脂酰胆碱-聚丙硫醚/小檗碱[phosphatidylcholine-poly (propylene sulfide)/berberine, PL-PPS/BBR]用于NIHL的外毛细胞(outer hair cells, OHCs)靶向治疗,发现PL-PPS/BBR在噪声暴露后12小时给药,通过靶向外毛细胞(OHCs)并快速释放(BBR),发挥抗炎和抗氧化作用,显著减少OHCs损伤并改善听力。Xiaogang An等[59]通过圆窗膜给药途径,将LS19肽修饰毛喉素负载纳米粒(LS19 peptide-modified forskolin-loaded nanoparticles, LS19-FSK-NPs)应用于小鼠模型,发现该纳米传递系统可显著降低听力阈值偏移,且在4、8和16 kHz频率下效果显著。然而,现有研究多处于临床前阶段,其有效性和安全性仍需进一步验证。

在传统医学领域,郭集军等[60]将活血化瘀中药配合针灸治疗NIHL患者,有一定疗效,且在6 k及8 kHz较常规营养疗法更佳。陈志刚等[61]使用缪刺法联合高压氧治疗,有效提高了特勤官兵职业噪声性聋的疗效。因此,可中西医协同作用干预NIHL的进展。

2.3 基因疗法

基因疗法是治疗听力损失的新兴方法,通过直接修改或替换病变基因,恢复或改善细胞的正常功能。在听力损失治疗中,基因治疗的目标是将正常的基因传递至内耳毛细胞或相关细胞中,以恢复或改善听力。NIHL的易感性与多种基因的单核苷酸多态性(single nucleotide polymorphism,SNP)密切相关,涉及的基因包括*GAPDH*、*SOD2*、*SOD1*、*CAT*、*CASP3*和*IL6*等[62]。Mukherjee等[63]使用AAV2(quad Y-F)-BDNF局部磁性靶向新方法,成功逆转了NIHL后ABR I波振幅和耳蜗突触的降低。He等[64]确定了FK506衰减NIHL的下游机制,发现FK506治疗不仅抑制钙调磷酸酶活性,减轻了中度噪声诱导的外毛细胞流失和听力损失,还抑制了ROS并激活自噬。通过AAV介导的神经营养因子-3(neurotrophin-3,NT-3)过表达,可有效减少噪声对螺旋神经节神经元与内毛细胞突触的损伤,显著减轻NIHL后果[65]。将腺病毒血清型1介导(adeno-associated virus serotype 1, AAV1)的VEGFA165病毒载体输送到缺失周细胞或暴露于噪声的动物体内,可防止和再生缺失的周细胞,改善血液供应,减轻听力损失[66]。此外,Rodrigues等[67]通过基因沉默技术阻断了肿瘤坏死因子α的表达,成功预防了噪声引起的听力损失,为基因疗法在NIHL防治中的应用提供了新思路。Fu等[68]使用CRISPR/Cas9技术在CBA/CaJ背景下建立了Myh14敲除小鼠(Myh14-/-小鼠),发现Myh14-/-小鼠更易受高强度噪声影响,表明Myh14可能在CBA/CaJ小鼠声学过度刺激后的耳蜗保护中发挥有益作用。目前,NIHL的基因治疗尚未进入临床试验,只在动物模型上取得了研究进展,针对NIHL的基因治疗仍需克服其科学和伦理挑战,进一步验证其长期安全性。

2.4 干细胞疗法

哺乳动物内耳毛细胞无法自然再生,传统治疗手段(如助听器或人工耳蜗)存在局限性,干细胞具有自我更新和分化成多种细胞类型的能力[69],能够分化成各种类型,包括内耳毛细胞和神经细胞,为修复或替换受损细胞

提供了潜在可能。目前，常用于治疗的干细胞有成体干细胞（adult stem cells，ASCs）、胚胎干细胞（embryonic stem cells，ESCs）和诱导型多能干细胞（induced pluripotent stem cells，IPSCs）[70]。Chen等[71]使用CRISPR/Cas9技术对听力损失患者iPSC中线粒体DNA中的基因进行修正，修正后的iPSC可分化为耳上皮祖细胞和HC样细胞，表现出正常的电生理特性。Kim等[72]将人胚胎干细胞衍生的间充质干细胞（ES-MSCs）通过成年大鼠的尾静脉系统给药，发现ES-MSC+噪声大鼠在4、8和16 kHz时表现出比噪声大鼠更低的ABR阈值，全身注射ES-MSCs不仅可保持听力水平，还能减弱NIHL大鼠的部分器官衰竭和细胞凋亡。Fan等[73]将嗅觉上皮神经干细胞（oe-NSCs）植入噪声暴露7天的大鼠体内，发现其不仅能够存活，而且在螺旋神经节神经元周围迁移，噪声暴露引起的听力损失得到恢复。干细胞治疗NIHL有一定潜能，但目前研究仍处于早期阶段，面临技术和伦理挑战，需更多的研究验证其作为常规治疗手段的可行性，治疗的长期效果和潜在副作用也需进一步研究和监测。

2.5 生活方式干预

吸烟、饮酒、体育锻炼、饮食均是NIHL的影响因素[74~76]。饮食因素与NIHL密切相关，摄入高烟酸和视黄醇及低碳水化合物可降低NIHL的风险[77]，维生素B12、叶酸和NAC对职业性NIHL有保护作用[78]。此外，NIHL患者的BMI、三酰甘油、空腹血糖、血压往往较高[79]，这些异常指标可能与不健康的饮食习惯密切相关。因此，NIHL患者应控制饮食，避免高热量、高脂肪、高糖食物的过量摄取。尼古丁和酒精会加重噪声对耳蜗血管的收缩效应，促进自由基积累，从而加剧听力损伤。一项纳入27项研究（共30，465名受试者）的荟萃分析显示，当前吸烟者合并比值比（odds ratio，OR）高于既往吸烟者，且每日吸烟量和NIHL之间存在剂量反应关系[80]。规律运动可减少氧化应激[81]，保护耳蜗细胞免受损伤。较高的肌肉和体能与较低的听力损失发生率相关[82]。因此，长期暴露于噪声的工作人员在生活中应加强体育锻炼，减少摄入高热量食物，同时补充微量营养素，戒烟限酒，规律饮食。

2.6 噪声防护

在全球范围内，约16%的听力损失可归因于职业噪声暴露[83]。NIHL是噪声长期暴露导致，因此，做好噪声防护至关重要。个人听力保护装置（hearing protection devices，HPDs）的正确使用已被证明是减少噪声暴露和听力损失的有效手段。佩戴耳部保护装置（ear protection devices，EPDs）的牙医在高频噪声暴露后的即时阈值偏移（temporary threshold shift，TTS）显著低于未佩戴EPD的对照组，表明EPD能够有效降低即时TTS，减少听阈变化[84]。Shu等[85]评估了HPD（预成型耳塞和泡沫耳塞）对NIHL的作用，发现在嘈杂环境中错误佩戴HPD的人仍有患NIHL的风险，强调正确佩戴动作和持续佩戴HPD的重要作用，尤其是泡沫耳塞的滚动和拉动动作。一项对15项研究（包括79986名参与者）的Cochrane评价得出结论[73]，更严格的立法能够降低工作场所的噪声水平，更好地使用HPD作为听力损失预防计划的一部分，可显著降低听力损失的风险。此外，加强环境噪声污染治理也至关重要。目前，我国出台了《中华人民共和国噪声污染防治法》[86]，从立法层面规范工业噪声排放、落实企业防治责任、推广低噪声工艺并强化监管。《工业企业噪声控制设计规范》（GB/T 50087-2013）[87]明确了工业企业噪声控制设计要求与工作场所噪声限值，确立了声源优先控制、辅以隔声消声等工程防控措施。因此，为有效预防NIHL，一方面需重视并正确佩戴各类听力保护装置，另一方面要严格落实相关法律法规与技术规范，加强立法监管，完善听力损失预防计划，切实保障劳动者听力健康。

3 展望

NIHL作为一种常见的环境性和职业性疾病，其发病机制复杂，涉及多种生物学途径。目前，针对NIHL尚无有效的治疗方法，主要依赖于噪声控制和听力保护设备预防，其诊断主要依赖于听力测试，无法在早期发现潜在的损伤，未来需要开发更敏感的生物标志物，以便在噪声暴露后尽早进行干预。此外，加强公众对噪声危害的认识，提高噪声环境下的自我保护意识，也是预防NIHL的重要环节。未来应加强多学科合作，整合医学、生物学、工程学等领域的研究成果，共同推动NIHL的研究和干预治疗进展。

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