

• 专家笔谈 •

基于认知、神经与遗传的汉语阅读障碍 多维度机制研究

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摘要: 本文回顾了汉语阅读障碍在认知功能、神经生理学及病因方面的最新进展。汉语阅读障碍的核心认知缺陷包括语音意识、语素意识、正字法及快速命名,并在左侧额下回低激活,右侧中央前回/颞中回高激活,提示存在语音处理缺陷及代偿机制。遗传学研究还发现了 *SEMA3F*、*AUTS2*、*EVC* 等新的易感基因,并探讨了环境因素及基因-环境交互作用的重要作用。未来还需要进一步的前瞻性研究阐明汉语阅读障碍普遍和特有的认知、神经生理及病因机制。

关键词: 汉语阅读障碍; 认知功能; 神经生理学; 遗传易感性; 基因-环境交互作用

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Multidimensional mechanisms of Chinese dyslexia from cognitive, neurobiological, and genetic perspectives

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Abstract: This review summarizes recent advances in Chinese developmental dyslexia across cognitive functions, neurophysiological, and etiology. Phonological, morphological awareness, orthographic, and rapid automatized naming constitute core deficits in Chinese dyslexia. Chinese children with dyslexia exhibit reduced activation in the left inferior frontal gyrus and increased activation in the right precentral/middle temporal gyrus, suggesting impaired phonological processing and potential compensatory mechanisms. Genetic research has identified novel susceptibility genes (e.g., *SEMA3F*, *AUTS2*, *EVC*). Furthermore, significant roles of environmental factors and gene-environment interactions were explored. Further prospective studies are warranted to elucidate both the universal and specific cognitive functions, neurophysiological, and etiology underlying Chinese developmental dyslexia.

Keywords: Chinese developmental dyslexia; cognitive functions; neurophysiology; genetic susceptibility; gene-environment interactions

1 研究背景

阅读障碍(developmental dyslexia, DD)是一种常见的神经发育障碍类疾病,是指个体在智力正常、有充足的教育资源、无视听觉损伤的情况下,在阅读、拼写和语言处理方面表现出持续性的困难,其核心是解码困难^[1-2]。中国学龄儿童汉语阅读障碍的患病率约为 3.45%^[3],男生患病率显著高于女生^[2]。阅读障碍儿童通常会面临更多的学业困难,生活质量下降,并更有可能出现如焦虑、抑郁、幸福感降低等心理健康问题^[4],给家庭及社会造成沉重负担。

相较于英语等拼音文字体系,汉语阅读障碍的系统研究起步较晚,但在 21 世纪以来发展迅速,研究成果显著增加,主要集中在阅读障碍的筛查与识别、认知功能、神经生物学、病因学机制以及干预等

方面。鉴于认知功能、神经生物学改变、病因学机制这三个领域对于揭示阅读障碍致病机制,制定有效的早期识别与个性化干预方法至关重要。因此,本文在 PubMed 及中国知网数据库,以 dyslexia/reading problems/阅读障碍等关键词梳理了近 5 年(2021—2025 年)发表的文献,聚焦汉语阅读障碍的认知功能缺陷、神经生物学改变及病因学机制的最新进展,深入理解病因及机制。

2 汉语阅读障碍儿童的认知功能特点

2.1 语言相关认知功能 语音意识是对文字中语音成分辨认、分析和操作的能力^[1]。语音意识缺陷是汉语阅读障碍的核心缺陷^[1-2]。阅读障碍儿童在音位删除,声母、韵母及声调识别等语音意识任务中表现均差于正常儿童^[5-9]。此外,其韵律敏感性较

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差,在声调和语调意识方面存在缺陷,影响字形-语音对应关系的建立^[10]。语素意识是个体对最小意义单元的感知和操作能力,也是汉语阅读障碍的核心缺陷^[9]。研究表明,阅读障碍儿童在语素产生与辨别任务中的表现较差^[9,11],事件相关电位研究也进一步提示其语素加工存在缺陷^[12]。正字法意识是个体对文字构件组合规则的理解能力。阅读障碍儿童在正反字/真假字辨别等正字法意识测试上的表现比正常儿童更差^[6-8,11]。但黄爽等^[9]发现 3 年级阅读障碍儿童与对照儿童在真假字测试上无显著差异,可能与样本量、筛查标准或年龄有关。此外,形声字的部首传递亚词汇的正字法、语义和语音信息^[13],阅读障碍儿童在亚词汇层面的正字法、语音和语义意识也存在缺陷,在文字识别过程中起重要作用^[14]。快速命名即命名熟悉对象的能力,最初认为其反映语音提取能力,现认为其独立于语音意识与阅读障碍相关^[1],是阅读障碍的核心缺陷^[5]。阅读障碍儿童在字母、数字、颜色、物体等快速命名任务中表现比对照组更差^[5-9,11]。此外,阅读障碍儿童在小学各年级均存在句法意识与词汇知识缺陷^[15],Wang 等^[16]指出汉字感知能力也是独立的认知缺陷维度,反映对视觉特征的区分效率,如形近字识别。

2.2 非语言相关认知功能 阅读障碍儿童在非语言层面如视觉、听觉、工作记忆也存在缺陷。近年研究发现阅读障碍儿童存在视觉感知能力受损,在纹理辨别任务中的阈值明显更高,且这一阈值与阅读流畅性显著负相关,提示其缺乏将视知觉与阅读流畅性联系起来的能力^[17]。另外,阅读障碍儿童在视觉选择性注意及时空视觉注意调节上存在缺陷,表现为注意选择和抑制的功能失衡^[18],这种缺陷还会持续至成年期,表现为整体加工效率下降^[19]。视觉注意广度(visual attention span, VAS)缺陷也与阅读障碍有关,阅读障碍儿童在 visual-1-back 任务中表现更差^[8],但两项 Meta 分析的结论不一:一项发现 VAS 缺陷与汉语阅读障碍有关,但汉语中效应量较小^[20],而另一项发现 VAS 缺陷与表意文字阅读障碍无关^[21]。Cheng 等^[5]则发现仅语言 VAS 缺陷与阅读障碍相关。工作记忆是指短时加工和储存信息的能力。阅读障碍儿童存在工作记忆缺陷^[11],并在语言工作记忆和视觉空间工作记忆上的表现更差^[22]。其中,中央执行系统的研究结论不一致:祝可等^[23]发现阅读障碍儿童在刷新、抑制、转换 3 个维度上均存在缺陷,抑制是核心缺陷,另一研究仅发现在刷新维度存在缺陷^[24]。

3 汉语阅读障碍儿童的神经生理学

3.1 汉语阅读障碍儿童的脑结构异常 汉语阅读

障碍儿童在右颞上回、左内侧额回及左尾状体的白质完整性降低^[25],左侧颞顶皮层、左侧距状皮层、左侧额中回的灰质体积减少^[26],右侧颞上回的灰质体积增加^[26],同时钩状束连接中断^[8]以及额叶-丘脑白质纤维束更强的右侧偏侧化^[27]。

3.2 汉语阅读障碍儿童的脑功能异常 两项 Meta 分析显示阅读障碍儿童在左侧额下回(岛盖部、背侧部)、左侧额中回、左侧缘上回、左侧颞下回、前扣带回、中扣带回、辅助运动区表现出低激活;在右侧中央前回、中央后回、右侧颞中回和左侧直回表现出高激活^[26, 28-29]。在语音任务中表现出左侧额下回岛盖部低激活^[29];在视觉任务中表现出大细胞-背侧通路的左侧中颞区视觉运动区低激活^[30],右侧舌回/枕下回^[29]、视觉皮层^[17]高激活,提示其需要更多神经资源代偿视觉处理效率不足^[17]。

阅读障碍儿童还存在小脑-大脑皮层连接异常,如小脑蚓部 IX 区与右侧额上回/左侧额中回之间的静息态功能连接增强,并与语音意识和快速命名能力负相关,可能是代偿机制^[6]。对照组视觉皮层与左额中回的静息态功能连接强度与视觉任务和阅读流畅性相关,但在阅读障碍组缺失,提示其无法利用此通路链接视觉感知与阅读^[17]。皮层下区域的左侧苍白球与额下区域连接降低与语音加工有关,而与语义加工无关^[31]。Granger 因果分析发现阅读障碍儿童在左中央前回到右楔前叶/左前扣带回/旁扣带回,左距状裂到右角回,以及左额中回到左距状裂之间功能连接减弱,在右楔叶到左额下回三角部的功能连接增强^[32]。此外,语素意识缺陷与腹侧通路(右侧下额枕束)和视觉空间通路(右侧丘脑-枕叶束)轴突变性有关^[33],在书写过程还表现出感觉运动网络与视觉网络之间的网络连接减弱,默认模式网络与腹侧注意网络之间的网络连接减弱^[34]。

4 汉语阅读障碍的病因学研究进展

4.1 遗传因素 阅读障碍有显著的遗传性,家系研究表明其遗传度可达 70%^[35]。既往研究已经确定了多个阅读障碍易感基因,如 *DYX1-9* 基因座、*KIAA0319*、*DCDC2*、*FOXP2* 基因等^[2],候选基因研究也揭示了 *KIAA0319*、*DOCK4* 等基因与汉语阅读障碍的关联^[36]。近几年开展了全基因组关联研究(genome-wide association study, GWAS)及候选基因验证研究,发现了汉语阅读障碍新的易感基因和单核苷酸多态性位点(single nucleotide polymorphism, SNP)。

2022 年发表的目前最大的涵盖中国和欧洲人群的 GWAS,确定了 42 个阅读障碍相关 SNPs,其中新发现了 27 个 SNPs;如调控 *SEMA3F* 基因的

rs2624839、调控 *AUTS2* 的 rs3735260、调控 *POU6F2* 的 rs62453457 位点等^[37]。此研究还独立验证了 75 个已报道 SNPs 中的 19 个位点在新样本中仍与阅读障碍显著相关^[37]。2023 年针对中国人群的 GWAS 也报道了一个调控 *EVC* 基因的 rs6446395 位点与汉语阅读障碍维度—阅读流畅性相关^[38]。该数据集还验证了位于 *KIAA0319* 基因的 rs2760157 和 rs16889506 位点与阅读障碍的快速命名功能相关^[38]。

近年来还有多篇采用候选基因策略的位点验证研究。赵华等^[39]在汉族儿童中验证了位于 *KIAA0319* 基因的 rs6935076、rs3756821、rs3212236 位点与阅读障碍相关。此外,有学者还揭示了其他神经发育障碍类疾病相关基因与阅读障碍的关联:位于 *SLC6A3* 基因上的 rs2975226 位点^[40]、*BDNF* 基因上的 rs6265 位点^[41]及 *GRIN2B* 基因上的 rs1805502 位点^[42]与阅读障碍相关。

4.2 基因-环境交互作用 阅读障碍的病因涉及复杂的生物学和环境因素相互作用。除遗传因素外,环境因素在汉语阅读障碍的发生发展中扮演着重要角色。多项研究强调了基因-环境交互作用在汉语阅读障碍发病机制中的关键意义,例如家庭环境及其与基因的交互作用已被证实共同影响阅读障碍的发生^[43]。近期研究多探索环境污染物暴露及基因-环境交互作用与阅读障碍的关系。

金属暴露与阅读障碍的关联研究结论存在异质性,但较多研究指出,汞、铅等高浓度暴露与阅读障碍患病风险增加相关^[41, 44],而锌、钴可能具有保护作用^[44]。此外,锰与阅读障碍的患病风险无关^[40-41, 44],但与阅读能力有关^[40]。尽管存在异质性,但多项研究指出金属暴露与基因的交互作用与阅读障碍有关。Zhu 等^[40-41]揭示了 *SLC6A3* 基因与锰的交互作用以及 *BDNF* 基因与铜的交互作用与阅读障碍的关联;携带 *SLC6A3* 基因 rs27072 位点 CT/TT 基因型与高浓度锰暴露、携带 *BDNF* 基因 rs6265 位点 TT 基因型与高浓度铜暴露均会显著增加阅读障碍的患病风险。Liu 等^[42]则发现锌与阅读障碍的关联受 *GRIN2B* 基因的调节。

对有机污染物与阅读障碍的关联也进行了广泛探索。有机磷杀虫剂毒死蜱代谢物三氯吡啶酚与阅读障碍患病风险增加有关(最高暴露组 vs. 最低暴露组 $OR=1.68$),可能通过脂质过氧化过程与阅读障碍有关^[45],但未发现拟除虫菊酯、新烟碱类杀虫剂与阅读障碍的关联^[45]。环氧乙烷、邻苯二甲酸酯替代物、苯类的代谢物均与阅读障碍患病风险正相关,而甲苯、乙苯、二甲苯的代谢物与阅读障碍患病风险无关^[46-48]。Xiang 等^[49]发现双酚 A 暴露与阅

读障碍患病风险相关(最高暴露组 vs. 最低暴露组 $OR=1.23$)。该研究基于既往 GWAS 构建了多基因风险评分(polygenic risk score, PRS),发现与低 PRS 和低双酚 A 暴露的儿童相比,高 PRS 和高双酚 A 暴露儿童的阅读障碍风险更高,尽管该研究未发现 PRS 与双酚 A 暴露存在交互作用。此外, Yang 等^[50]通过生物信息学分析,探索性地揭示了多种环境污染物(金属、持久性有机污染物、多环芳烃、农药等)与阅读障碍遗传易感性的关联,提示这些污染物可能通过干扰阅读障碍相关基因的表达或功能参与发病过程。

尽管在筛查标准和语言文字体系上存在差异,但阅读障碍的认知功能、神经生理学基础及遗传因素显示出跨文化的相似性和独特性。单一认知缺陷理论不足以解释阅读障碍,阅读障碍儿童通常表现出包括语音意识、语素意识、正字法意识、快速命名等在内的多种核心缺陷,同时可能伴有视知觉缺陷和工作记忆问题。针对汉语阅读障碍的神经生理学研究虽然有限,但已发现左侧额下回的低激活以及在右侧中央前回/颞中回的高激活,提示存在典型的语音处理缺陷和特异性代偿机制。汉语阅读障碍遗传学研究也取得了重要进展。大规模的 GWAS 揭示了新的易感基因(如 *SEMA3F*、*AUTS2* 等),并验证了 *DCDC2*、*KIAA0319* 等经典易感基因。同时,多项研究证实了环境因素及基因-环境交互作用的关键角色。

虽然汉语阅读障碍的进展迅速,但目前对于感知觉处理缺陷、环境因素与汉语阅读障碍的关联仍存在异质性,神经生理学研究相对较少,并且有很大一部分遗传度尚未得到解释。未来仍需结合前瞻性设计方法在大样本中进行验证,深入理解汉语阅读障碍独特和普遍的认知功能、神经生理学特征、病因机制等。

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