

# 语言发育迟缓的危险因素研究现况

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## 摘要

语言和言语是儿童发展或神经发育的两个主要领域, 随着现代青年人育儿精细化, 语言发育迟缓开始逐渐受到家属及医生们的重视, 本文通过PubMed、中国知网两大文献资源库以“语言发育迟缓”为关键词搜索近5年针对儿童语言发育迟缓影响因素共4046篇文献, 综述语言发育迟缓影响因素的目前研究情况。文献检索显示, 目前儿童语言发育迟缓的影响因素主要涉及遗传因素(相关神经综合征、基因缺失/变异)、个人成长环境因素(年龄、性别、围产期因素、眼部/耳部/口腔等器质性疾病等)及家庭环境因素(家族史、母亲教育程度、母亲受孕年龄、出生顺序、家庭语言环境、屏幕暴露时间等)三大方面, 结果表明, 家庭环境因素在儿童语言发育迟缓的影响因素中占据关键地位, 具有进一步开展临床应用与研究的价值。

## 关键词

儿童语言发育迟缓, 个人风险因素, 遗传因素, 环境因素

# Current Research Status of Risk Factors for Language Development Delay

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## Abstract

Language and speech are two main areas of child development or neurodevelopment. With the trend of more precise parenting among modern young people, language development delay has

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gradually attracted the attention of families and doctors. This paper searches PubMed and CNKI using the keyword “language development delay” to find 4046 articles published in the past five years on factors influencing language development delay in children, summarizing the current research status of these factors. Literature retrieval shows that the influencing factors of children’s language development delay mainly involve three aspects: genetic factors (related neurogenic syndromes, gene deletions/variations), individual growth environment factors (age, gender, perinatal factors, organic diseases of the eyes/ears/oral cavity, etc.), and family environment factors (family history, maternal education level, maternal age at conception, birth order, family language environment, screen exposure time, etc.). The results indicate that family environment factors play a key role among the influencing factors of children’s language development delay and have value for further clinical application and research.

## Keywords

Children’s Language Development Delay, Individual Risk Factors, Genetic Factors, Environmental Factors

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## 1. 引言

儿童获得第一语言的速度既取决于他们与生俱来的神经认知能力(可能是由遗传决定的),也取决于他们的环境和以前与人类语言的接触[1]。近年来,社会劣势加剧了早期语言发育迟缓,儿童语言问题的患病率迅速增加,据报道,2至7岁儿童 DLD 的患病率为 2.3%至 19%,大约 9.9%的儿童出现语言发育困难,7.6%没有严重的其他障碍,2.3%与语言相关合并症(例如听力损失)相关[2]-[4]。语言障碍的临床异质性、常见的共病存在以及多年来使用的不一致术语,阻碍了研究和临床实践。语言发育的关键期发生在生命的前六个月儿童早期语言接触显著影响儿童的语言掌握[5],一些 18~24 个月大的婴儿在没有任何重大听力问题、认知发育问题或相关出生史的情况下可能会表现出言语迟缓或词汇量有限[3]。儿童语言发育不仅关乎沟通能力,更是大脑神经可塑性、学习潜能及社会适应力的重要标志。儿童的言语和 DLD 与阅读、写作、注意力和社交困难增加有关,50%以上的发育迟缓持续到青春期,并在以后的生活中面临教育和职业挑战[6],且幼儿严重的言语和语言障碍会对以后的教育成就产生负面影响即使在强化干预后也是如此[7]。

语言发育迟缓(发育性语言障碍 DLD)是指由各种原因引起的儿童口头表达能力或语言理解能力明显落后于同龄儿童的正常发育水平,根据目前研究及文献提出主要分为原发性及继发性两大类,原发性言语和 DLD 的类型包括发育性言语和语言迟缓、表达性语言障碍和接受性语言障碍。继发性言语和 DLD 可归因于其他疾病,例如听力损失、智力障碍、自闭症谱系障碍、身体言语问题或选择性缄默症。同时,非典型语言发展可能是其他身体和发育问题的次要特征,这些问题可能首先表现为语言问题[8]。查阅近 5 年国内外文献总结目前 DLD 危险因素主要包括遗传因素、个人风险因素及家族性、其他环境危险因素等。详见如下(本综述中的潜在风险因素列表并不完整,也可能包括证据不确定的因素)。

## 2. 影响因素分析

语言缺陷有多因素的社会文化和教育因素,但有力证据表明遗传因素也参与其中,具体如下:

## 2.1. 遗传因素

### 2.1.1. 相关综合征

Hao-Fountain 综合征(HAFOUS) [9]、脑肌酸缺乏综合征(CCDS) [10] [11]、SATB2 相关综合征(SAS) [12]、威廉姆斯-博伦综合征(WBS) [13]、2 型神经元蜡样脂褐质沉着症(CLN2 疾病) [14] [15]、GLUT1 缺乏综合征(GLUT1DS) [16]、唐氏综合征(DS) [17]-[20]、儿童言语失用症(CAS) [21]-[23]、孤独症谱系障碍 [24]-[26]、注意力缺陷和多动障碍儿童[27]、脑瘫、结节性硬化症[28]等疾病。

### 2.1.2. 其他相关基因缺失/变异

5p 缺失综合征[29]、6q25 微缺失、8p23.1 复制综合征[30]、15q13.3 微缺失(CHRNA7 和 OTUD7A)、16p11.2 微缺失[31] [32]、近端 16p11.2 重复[33]、22q11.2 缺失综合征(22q11DS) [34]-[36]、SETBP1 单倍不足相关[37]、性染色体三体(SCT) [38]、钴胺素 C (cb1C)缺陷、ABHD14A、ANK3 变异[39]、ANKRD17 杂合子功能丧失变异[40]、ARID4A 的新生移码变异、ATP2C2 基因[41]、BDNF 基因(接触金属可能与 BDNF 基因相互作用, 增加神经发育障碍的风险) [42]、BMP2 基因 1.3~3.7 Mb 微缺失[43]、BCL11A 基因(智力障碍综合征 IDS) [44]、CHL1 基因、CNTNAP2、CONT3 突变[45]、致病性 CDK13 变异[46]、CLCN4 相关[47]、DHX30 变异[48]、DDX47、DDX3X [49] [50]、EMC10 基因[51]、FOXP2、EEF2 相关神经发育障碍[22]、纯合 EMC10 变异[52]、FOXP 基因[53] [54]、IQSEC2、KAT6A 基因(Arboleda-Tham 综合征) [55]、MED13L 相关障碍[56]、MRNIP [57]、NIPBL、PPP2R2C 中的接合位点变异、QRICH1 功能丧失变异(Ververi-Brady 综合征) [58]、NRXN2 [59]、RFX7 基因[60]、ROBO2 [33] RPS6KA3 基因(科芬-洛瑞综合征 Coffin-Lowry)、SOX5 (Lamb-Shaffer 综合征) [61]、SOX30、SATB2 基因[62]、SHANK3 基因相关(Phelan-McDermid 综合征, PMS) [63]、SETD1A [22]、TBR1 基因、WASF1 (黄蜂蛋白家族成员 1) 基因新生突变所致的 NEDAVLS [64]、ZNF292 基因(RCV001260794.4, RCV001292573.11, RCV001879995.6, RCV001261752.3, RCV003353266.2)、ZNF292 [33]等在既往文献中有所提及到的突变均以不同程度语言发育迟缓为首要突出表现。此外, 有研究表明语言功能障碍是部分罕见遗传疾病的显著特征, 尽管这并不一定适用于所有遗传疾病[65]。

## 2.2. 个人风险因素与 DLD 的关系

### 2.2.1. 年龄与性别

儿童早期 DLD 受多种因素影响, 其中年龄和性别是重要的生物学变量。0~3 岁是语言发展的黄金窗口期, 大脑神经可塑性最强。若在此阶段出现明显语言落后(如 24 个月词汇量 < 50 个), 需警惕发育性语言障碍风险。年龄越小, 个体差异越大: 24 月龄时正常儿童语言能力可能相差 6 个月, 但到 36 月龄时差异若持续扩大则更具临床意义。有研究支持该观点, 随着年龄的增长, DLD 儿童 Gesell 5 个能区的发育商(DQ 值)明显下降, 与同龄人的差距加大。

男性也是儿童早期出现语言迟缓的独立风险因素之一, 在临床转诊中男女比例可达 3:1。女性在早期语言发展中普遍表现更优: 平均比男性早 1~2 个月达到里程碑, 尤其在词汇多样性和社交语言使用上, 其原因可能是女性左半球语言区髓鞘化更早, FOXP2 基因表达水平更高或养育者更倾向与女婴进行语言互动, 形成正反馈循环; 男童更多表现为外化行为问题(如多动), 可能掩盖语言需求; 女童则更多使用补偿性社交策略。

### 2.2.2. 围产期因素

围产期因素已被确定为儿童早期语言迟缓的影响因素之一, 如受孕方式[66], 具体介绍以下方面:

1) 胎儿因素: 胎龄、宫内生长发育迟缓(如脐动脉逆流、脐动脉搏动指数、大脑保留(brain sparing)、

脐脑比(the umbilical-cerebral ratio)等)、胼胝体(ACC)孤立异常[67]、孤立性胎儿脑室肥大[68] (孤立性胎儿脑室肥大儿童的神经发育与皮质发育改变有关)等。

2) 孕期因素：围产期接触多不饱和脂肪酸(PUFA)、孕妇摄入碳水化合物的数量和质量[69]、孕期膳食氟摄入水平[70]、孕期情绪(抑郁、焦虑等) [71]-[76]、母亲合并产后抑郁[77] [78]、孕期睡眠时间[79]、酗酒[80]-[82] (产前发育期间酒精暴露会导致大脑功能网络分离和整合的某些方面出现破坏[83]，有报道称产前酒精暴露对左上小脑脚和右上纵束的发展轨迹有显著时间变化，暗示产前酒精暴露儿童白质发育改变[84])、母孕期维生素 B12/叶酸缺乏、妊娠晚期母体血清叶酸浓度、孕晚期合并感染、妊娠期并发症(妊娠期高血压/先兆子痫、妊娠期糖尿病[85] [86]、妊娠期甲状腺功能(TT3 水平与语言发展呈正相关，而 TSH 浓度与语言结构域呈负相[87])、总血清碘(TSI) [88]、产前接触抗癫痫药物[89] [90] (产前接触抗癫痫药物(ASM)可能导致血浆叶酸浓度低，并与神经发育受损有关)、暴露于阿片类药物[91]-[96]、怀孕期间主动和被动吸烟的烟草烟雾暴[97] [98]、产前暴露于室内空气污染(PM) [98]、职业暴露于碳纳米颗粒[99]、HIV 感染[100]-[102] (怀孕期间暴露于 HIV 与早期皮层结构改变有关，研究结果表明 HIV 感染与否所导致的儿童前额叶区皮层厚度发育差异可能是导致语言障碍的途径之一[103])、先天性巨细胞病毒感染[104]-[106]、寨卡病毒感染(CZVI) [107]-[109]、兹卡病毒暴露[110] [111]、孕晚期(33~37 周孕期)感染疟疾[112] (PCR 阳性在孕晚期(33~37 周孕期)与 2 岁以下语言发育受损有关[113])、产前钛暴露[114]、多氟烷基物[22] [115]、有机磷农药/多溴二苯醚接触/拟除虫菊酯农药(PYRs) [116]、双酚 A (BPA)或邻苯二甲酸酯(PAEs) [117]/单乙基邻苯二甲酸酯[MEHP] [118]、产后多环芳烃暴露等。

3) 新生儿期因素：(极)早产[119]-[122]、(极)低出生体重儿[123] [124]、新生儿缺氧缺血性脑病[125]-[127]、(发绀性[128])先天性心脏病[129]、新生儿脑损伤[130]、新生儿低血糖、新生儿高胆红素血症、早产儿视网膜病变(ROP)、母乳喂养[131] (尤其对低出生体重儿，纯母乳喂养与五岁女孩的听力语言发育较高有关[132])、进食和/或吮吸行为[133]、早产儿出血后心室扩张、支气管肺发育不良、生后应用氢化可的松、丘脑和纹状体的不同区域与神经认知功能相关的表观扩散系数(ADC)、镰状细胞病[134]等。

### 2.2.3. 儿童成长环境危险因素

1) 相关器质性病变(唇腭裂[135]-[138]、发音困难疾病、耳聋等先天性听力相关疾病[84] [139] [140]及受眼部白化病(OA)影响[141]或其他视觉障碍[142])：聋哑或听力障碍儿童有言语和语言发育迟缓的风险，听力损失可能延缓这些能力的发展，从而引发潜在的社交和交流挑战[143]-[147]。研究表明人工耳蜗恢复声音，可以促进重度至重度感音神经性听力损失儿童的言语和语言发展。此外患有白化病等视觉障碍也会显著阻碍儿童的语言发展[109] [148]-[150]。从心理角度看，由于视障人士通常无法获得非语言信息，他们无法获得完整的语用知识体系；从心智理论角度看，语用知识缺陷会导致执行功能异常和发展迟缓，从而引发语用障碍[142]。

2) 手势(认知、食指使用)：在自闭症儿童指点手势与表达性语言发展之间的纵向关系中起着关键作用。食指指向和认知是自闭症群体表达语言的直接纵向预测因子。

3) 生物标志物：有报道称语言迟缓相关的生物标志物包括 CRP、IFN- $\gamma$ 、IL-1 $\beta$ 、-2、-4、-6、-10、-12p70、中性粒细胞明胶酶相关脂卡林(NGAL)、粒细胞 - 巨噬细胞集落刺激因子(GM-CSF)和基质金属蛋白酶-9 (MMP-9) [151]及钙、锌、维生素 D [152]、血红蛋白[153]、血清铁蛋白[154]水平，仍需进一步研究确认。

4) 睡眠时间[155]：与短而可变的夜间睡眠轨迹相比，长而稳定的夜间睡眠轨迹与认知和语言得分较高相关，而中度/长时间持续的夜间睡眠轨迹则与较高的语言和综合得分相关。具有较长且稳定总睡眠轨迹的儿童，其认知和表达语言和言语及复合得分高于总睡眠轨迹短的儿童。白天睡眠时间长且可变的儿

童在认知和精细运动和语言和综合得分低于短且稳定的白天睡眠轨迹的儿童。

5) 鼓室造口管[156]: 鼓室造口管可以预防中耳炎的后遗症, 这些后遗症对儿童的长期听力和语言发展产生不利影响。这些负面结果加剧了已经被诊断为发育障碍儿童所面临的困难。

6) 铅[157]、汞、锰暴露[152]、拟除虫菊酯杀虫剂(PYR)等化学物质暴露: 婴儿期儿童暴露于拟除虫菊酯农药(PYRs)同样可能影响其4岁时的神经发育结果, 2岁可能是暴露于PYRs的敏感窗口, 此时PYRs的暴露可能对运动和语言发展产生负面影响[158]。

7) 首句习得时间: 首句习得时间(First Word Combinations, FWC)通常指儿童首次将两个及以上词汇组合成有意义的短语(如“妈妈抱”), 是评估儿童早期语言发育的重要里程碑, 其延迟可作为DLD的敏感指标。多数儿童在18~24月龄出现首句组合(平均20月龄), 若 $\geq 30$ 月龄仍未出现, 则被定义为显著延迟在控制儿童年龄时, 24个月后说出第一句的儿童患DLD的几率高出6倍。

还有部分尚未验证的因素如头部外伤[159]右撇子、学龄前儿童以高脂肪、高盐和高糖为特征的食物等。

## 2.3. 家族性及家庭环境危险因素与DLD的关系

### 2.3.1. DLD 家族史

语言问题的家族史经常被记录为DLD的主要非环境风险因素, 其所致DLD儿童较无家族史儿童高出两倍以上, 据统计, 当家族有语言习得困难史时, DLD的发病率为32%, 即有家族史儿童表现出较差的表达词汇量和较高的语言迟缓患病率[160], 该指标可作为预测因子进一步预测发病概率, 其影响机制涉及遗传(见上文)、表观遗传及家族语言环境的交互作用。因此对于对有一级亲属语言迟缓史的儿童, 建议在18~24月龄启动标准化筛查(如MacArthur-Bates CDI)。合并家族史的晚期说话者应优先转诊遗传检测(如WES检测FOXP2变异)。

### 2.3.2. 母亲教育程度

母亲教育程度(Maternal Education Level)是影响儿童早期语言发育的核心环境因素之一, 较低的母亲教育程度与DLD和病理性语言发展有关[161], 其作用机制涉及语言输入质量、家庭认知刺激及社会资源获取等多重路径。高中毕业(12年教育)是显著分界点: 低于此阈值的母亲, 子女语言迟缓风险骤增2.3倍。对于此种影响作者猜测可能与高教育母亲较多使用多样性词汇、开放性提问及叙事性语言等有关。同时有研究表明只有当母亲也具备较强的发展知识时, 母亲的自我效能感感知(自我效能指数-幼儿量表-“教学”和“游戏”子量表)才与儿童语言分数呈正相关[162]。

### 2.3.3. 母亲受孕年龄

母亲受孕年龄(Maternal Age at Conception)作为重要的生物学与社会环境交互因素, 对儿童早期语言发育的影响呈现U型曲线, 其作用机制及生理、心理及社会多维度路径。年龄较小及年龄较大母亲都被确定为语言障碍的风险。年龄较大的孕母其卵细胞老化, 线粒体DNA突变累计可能影响胎儿神经发育, 且高龄产妇的妊娠期糖尿病及高血压发生率较高, 可导致胎儿脑氧供应减少从而导致脑损伤。年龄较小的孕母(低龄妊娠 $< 20$ 岁)情绪调节与育儿计划能力较弱, 语言输入质量显著降低, 且低龄母亲更易陷入贫困, 研究表明 $\leq 18$ 岁母亲子代36月龄语言筛查未通过率是25岁组的2.1倍。

### 2.3.4. 出生顺序

出生顺序(Birth Order)对儿童早期语言发育的影响呈现出复杂的动态模式, 涉及语言输入量、互动机会及家庭资源配置等机制。在家庭等级方面, 相较于第一个孩子, 他/她的弟弟妹妹患有DLD的概率稍有增加, 其原因大致可能为首胎优势, 即第一个孩子出生将独享期的语言输入量相比二胎、三胎多32%, 且多胎家庭的父母对单个儿童的语言响应时间减少25%。但也有相反意见。

### 2.3.5. 家庭(语言)环境

父母关系、母亲亲密伴侣(即家庭暴力[6] [163] [164])、单亲家庭、双语家庭、移民母亲、少数族裔[165]、亲子沟通少[166]、社会家庭环境[167]-[172] (地区层面的社会经济贫困程度越高, DLD 的比例越低)、住宅绿地与交通暴露相关及有公共和私人保险均是目前文献所提及的语言障碍的影响因素, 但对于其与语言发展之间的联系目前尚无具体解释方法, 不过有研究提出患有(怀疑) DLD 的幼儿在家中发声比患有 TD 的儿童少。他们听到的成人话也更少, 谈话转折也更少。患有 DLD 语言成果的儿童在有限程度上与家庭语言环境相关[173], 但具体如何产生影响及相关机制尚不明确。

### 2.3.6. 屏幕暴露时间(电子产品、电视)、儿童游戏活动类型和内容

儿童屏幕暴露内容与和照顾者互动共观的内容是早期儿童发展的重要决定因素, 鉴于数字设备的普及和多样化的数字内容, 制定能够最大限度地减少对儿童发展负面影响的屏幕使用习惯, 对于促进儿童健康屏幕使用至关重要[174]。屏幕时间与交流方式(口头/非语言)之间没有显著关联, 但是, 屏幕时间增加的儿童中有明显数量的儿童具有口头以外的其他主要交流方式(指点/手势) [175]。过多的屏幕时间(使用智能手机、电视、电脑和/或视频游戏)与言语和语言发育迟缓有关[176] [177], 在发育、行为或情感问题的一项研究中, 存在屏幕观看时间(SVT)的学龄前儿童超过一半(51.1%)的儿童具有语言发育迟缓的临床特征, 且 SVT 较高的儿童注意力较差, 攻击性更强, 行为问题增加。研究发现[178] [179], 每天一小时或以上的屏幕使用时间与较差的语言发展分数及语言理解困难和表达性语言技能困难的概率增加。平均每天屏幕时间为 6~8 小时的儿童更有可能有言语发育迟缓病史并被诊断出患有口语障碍。也有其他相关文献支持儿童屏幕暴露时间与儿童口语表达呈负相关且显著相关[180]。屏幕的一些负面影响可能取决于曝光时间, 但更重要的是取决于孩子的特征、观看内容的类型以及观看内容的背景, 大量使用的屏幕说服设计和深色图案会激发更频繁的使用, 吸引孩子和父母的注意力, 从而严重干扰亲子关系。有幼儿的家长使用视频聊天的方式与其他屏幕媒体不同, 这与专家建议(例如美国儿科学会传播与媒体委员会)一致, 这些建议认为视频聊天与其他屏幕媒体不同, 18 个月以下儿童是可以接受的。视频聊天在屏幕媒体中独树一帜, 因为它允许偶然的(时间敏感性和内容敏感性)社交互动[181]。移动设备已成为日常生活中不可或缺的一部分, 甚至在年幼儿童生活中也无处不在, 减少屏幕时间可以显著改善言语发育迟缓的儿童[182]。

除此之外, 仍有部分文章提出其他相关因素, 需进一步明确有无相关性。

## 3. 小结

本文综述儿童语言发育迟缓因素如上, 对于没有达到预期的言语和语言里程碑的儿童, 全面的发育评估是必不可少的, 早期转诊进行评估可以减轻长期沟通障碍的发展以及对社会和学业发展的不利影响。所有有言语和语言迟缓问题的儿童都应转诊到言语语言病理学和听力学进行诊断和治疗。目前研究中通过比较临床组来更好地记录与 DLD 共发的重要性, 与此同时未来的研究方向应适当包括风险因素和保护因素, 如语言缺陷水平等指标, 来验证目前存在的研究结果。同时也呼吁广大研究者进行更多研究, 未来的研究应该探索如何更好地使治疗与儿童的偏好和发展需求保持一致, 多倾听孩子们的声音, 提高普通教育教师和公众对儿童语言发展和障碍的认识和教育。

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