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· 综 述 ·

母亲产前汞暴露对子代儿童神经发育影响的研究进展

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【摘要】 为了解环境暴露尤其是产前重金属暴露对儿童神经发育的影响, 本研究通过综述近年来国内外关于母亲产前汞暴露及多金属联合暴露与子代儿童神经发育之间的关联, 重点分析汞对子代儿童认知行为和神经发育障碍的影响, 及其可能机制如基因易感性和 DNA 甲基化及氧化损伤, 为儿童神经发育障碍的早期预防提供科学依据。

【关键词】 汞; 神经发育障碍; 研究; 儿童

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Research progress on the effects of maternal prenatal mercury exposure on offspring neurodevelopment

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【Abstract】 To investigate the effects of environmental exposure on neurodevelopment in children, particularly maternal prenatal heavy metal exposure, the review summarizes recent domestic and international research on the associations between prenatal mercury exposure, combined multiple-metal exposure, and offspring neurodevelopment, focuses on analyzing the effects of mercury on offspring cognitive behavior and neurodevelopmental disorders, as well as the underlying mechanisms including genetic susceptibility, DNA methylation and oxidative damage, to provide a scientific basis for the early prevention of childhood neurodevelopmental disorders.

【Keywords】 Mercury; Neurodevelopmental disorders; Research; Child

随着环境重金属污染日益严重, 汞作为常见的环境污染物, 其健康危害受到广泛关注。现有研究报道产前重金属如汞的暴露与子代不良出生结局有明显关联[1], 包括神经管缺陷等神经发育异常[2]。汞的神经毒性可通过胎盘和血脑屏障在胎儿体内蓄积[3], 胎儿神经系统在宫内发育阶段就面临汞的毒性作用, 会产生长远的不良影响。然而, 目前关于产前汞暴露对子代儿童神经发育影响的研究证据尚不充分, 且汞暴露往往以多种金属联合暴露的形式存在, 单一暴露研究可能会低估汞的实际健康风险。基于此, 本文就近年来产前汞暴露及其对子代儿童神经发育影响的研究作综述, 为儿童神经发育障碍的早期预防提供更充实的科学证据。

1 产前汞暴露与子代儿童神经发育评估

1.1 汞的暴露来源及生物标志物 人体汞暴露主要有无机汞(通过牙科的汞合金填充材料)、乙基汞(通过疫苗和医疗制剂)和甲基汞 3 种形式[4]。化妆品中含有多种重金属, 其中具有美白作用的化妆品大多存在汞超标情况, 可通过直接接触皮肤被吸收并在体内蓄积[5]。牙科的汞合金, 低剂量也可引起汞中毒, 孕妇使用汞合金补牙材料也是妊娠期汞暴露的一种来源[4]。妊娠期食用大量的鱼类是孕妇甲基汞暴露的主要途径[6]。此外, 食用汞矿地区大米也是妊娠期甲基汞的暴露来源之一[7-8]。测量汞暴露水平常用的生物标本为孕妇血液[9]及头发[7]、脐带血[10]、胎盘组织[2]等。

1.2 常用的神经发育相关评定量表 包括新生儿神经行为评估(Neonatal Behavioral Neurological Assessments, NBNA)[11]、贝利婴幼儿发展量表(Bayley Scales of Infant Development, BSID)第 II 版(BSID-II)[12]和第 III 版(BSID-III)[13]、韩国版贝利婴幼儿发展量表 II(Korean Adapted Version of Bayley Scales of Infant and Toddler Development II, K-BSID-II)[14]、年龄与阶段问卷第三版(Ages and Stages Questionnaire, Third Edition, ASQ-3)[15]、丹佛发育筛查测试第 II 版

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(Denver Developmental Screening Test Ⅱ, DDST-Ⅱ)[16]、韦氏学前及小学智力量表第三版(Wechsler Preschool and Primary Scale of Intelligence - Third Edition, WPPSI-Ⅲ)[17]、京都心理发展量表(Kyoto Scale of Psychological Development, KSPD)[18]、马拉维发展评估工具(Malawi Developmental Assessment Tool, MDAT)[19]等。

2　产前汞暴露与子代儿童神经发育的关联

2.1　*产前汞暴露与新生儿神经行为的关联*　新生儿神经行为评估可反映宫内环境暴露对胎儿神经系统的早期影响。上海的一项横断面调查纳入 1 652 对母婴,对出生后第 3 天的新生儿进行神经行为评估,结果显示高水平汞暴露可使 NBNA 的平均得分下降 0.61,小于 5.8 μg/L 的汞暴露和新生儿神经发育的关联无统计学意义[10]。王海燕等[20]在舟山市妇幼保健院选取 418 名新生儿,研究结果也显示脐带血汞水平(≥18 μg/L)是新生儿 NBNA 得分低的风险因素,提示产前较高水平的汞暴露可能对新生儿早期神经行为产生不利影响。

2.2　*产前汞暴露与子代儿童认知行为发育的关联*　Axelrad 等[21]采用贝叶斯分层模型分析母体汞暴露与儿童智商之间的关联,结果显示母亲头发汞含量每增加百万分之一,子女智商降低 0.18 分。Freire 等[22]研究发现,胎盘中汞暴露会增加儿童语言功能较差的概率,并会使言语记忆技能得分较低的概率增加 3.85 倍,此外也对涉及执行和语言功能、定量技能和运动技能的认知子领域产生不良影响。Packull-McCormick 等[9]分别检测妊娠早期、脐带血及同期 3~4 岁儿童的血汞水平,采用 WPPSI-Ⅲ量表评估儿童神经发育状况,发现在母亲产前鱼类摄入量低的男童中,产前低水平汞暴露与操作智商分数降低相关;此外,研究还发现脐带血、儿童血汞浓度与智商呈正相关,这一结果见于女童并可能与产前摄入鱼类的潜在有益作用相关。Llop 等[23]基于西班牙的出生队列研究,对 1 683 名婴幼儿脐带血汞水平和 BSID 评分之间的关联性进行分析,结果显示在 2 倍的总汞暴露水平时并未显示出与智力行为发育迟缓之间的关联;对性别进行分层后,发现汞暴露与女婴的行为发育得分呈负相关。母亲和儿童环境健康队列(mothers and children's environmental health, MOCEH)测量了母体和脐带血汞水平及母体血清中的叶酸水平,通过 BSID-Ⅱ评估婴幼儿的神经发育状况,结果显示,仅在母亲孕期叶酸水平较低且婴幼儿在出生后前 3 年体重快速增长的前提下,产前汞暴露会对婴幼儿神经发育产生显著的不良影响[24]。Rothenberg 等[7]评估中国农村地区由食用大米导致的产前甲基汞暴露与儿童神经发育之间的关联,采集 391 名围产期妇女的头发样本,并采用 BSID-Ⅱ评估儿童神经发育状况,分别于婴幼儿 12 和 36 个月时进行评估,在调整后的模型中,母亲头发总汞暴露水平每增加 1 倍,儿童智力发育指数(mental developmental index, MDI)评分降低 1.3 分;这一不良关联随时间推移逐渐减弱,可能与 36 个月幼儿家庭结构的变化相关。此前,Rothenberg 等[8]另一项研究也观察到孕期食用大米导致的母体甲基汞暴露与子代 12 月龄的认知能力存在不良关联。

尽管多数研究支持产前汞暴露对子代儿童认知行为发育存在不利影响,然而部分研究未观察到其关联。Kobayashi 等[25]基于日本环境与儿童出生队列的研究,均未发现母体及脐带血汞的水平与 6 月龄至 4 岁儿童神经发育迟缓存在关联。一项基于巴西里约热内卢的出生队列研究,对 6 月龄婴儿进行 DDST-Ⅱ测试,也未观察到母体或脐带汞的水平与子代儿童神经发育领域缺陷存在关联[26]。一项纳入了 257 对母婴的前瞻性队列研究得出产前存在汞暴露的新生儿其脑部形态学发生改变,这种关联在纳入长链多不饱和脂肪酸之后有所下降,且该研究未发现脐带血汞的水平与 18 月龄幼儿神经发育评分之间存在关联[27]。一项地中海的队列研究采用 BSID-Ⅲ对 18 月龄幼儿进行测试,发现总汞暴露与幼儿认知和语言结果关联无统计学意义[28]。日本环境与儿童研究出生队列的另一项研究分析脐带血汞的水平(n=3 822)与儿童 2 和 4 岁时的 KSPD 发育评分之间的关联,但未发现有统计学意义[29]。Strain 等[6]对塞舌尔共和国食用较多鱼类的人群进行研究,也未发现产前甲基汞暴露与子代儿童神经系统发育存在不良关联。

2.3　*产前汞暴露与儿童孤独症谱系障碍、注意缺陷多动障碍之间的关联*　MOCEH 队列纵向测量母亲及子代儿童多个时点的汞暴露,通过社会反应量表(Social Responsiveness Scale, SRS)评估孤独症行为,结果发现孕晚期和脐带血汞水平与 SRS T 评分呈正相关,表明孕晚期和儿童早期血汞水平与 5 岁儿童更多的孤独症行为相关[30]。一项在马萨诸塞州开展的出生队列研究发现产前低水平汞暴露与 8 岁儿童的注意力不集中、冲动/多动的高风险相关联[31]。而一项大型的挪威母婴出生队列研究发现母亲产前接触汞合金填充物与子代 3 和 5 岁儿童注意缺陷多动障碍(attention deficit hyperactivity disorder, ADHD)之间相关性无统计学意义[32]。基于挪威的母亲、父亲和儿童队列研究[包括 705 例 ADHD 患者、397 例孤独症谱系障碍(autism spectrum disorders, ASD)患者和 1 034 例对照组儿童],报道了孕期汞暴露的增加与子代患 ASD 和 ADHD 的风险降低相关[33]。

2.4　*汞与其他金属联合暴露对子代儿童神经发育的影响*　Farías 等[34]纳入 253 对母婴,通过测量孕妇的血汞、铅和锰水平,在婴儿第 1,3,6 和 12 个月时使用

BSID-III 评估其神经发育情况，结果显示，母亲血铅水平每升高 1 μg/dL，子代儿童语言发展系数下降 1.5 分，这一关联强度在汞>1.9 μg/L 的儿童中更为显著；当纳入汞、铅和锰的交互项时，子代儿童认知和运动发育与母亲血铅水平呈负相关，提示汞的联合暴露会增强铅的神经毒性。Shah - Kulkarni 等[35]基于 MOCEH 队列纳入了 523 对母婴，使用 K-BSID-II 评估神经发育水平，结果显示，在汞共同暴露的条件下，妊娠晚期铅暴露对 6 月龄婴儿的 MDI 得分存在显著的负向影响，此关联在贝叶斯核机器回归和广义加性模型均得到验证；分层分析显示，妊娠晚期血汞浓度超过 P_{50}，则会加剧铅暴露对 6 月龄婴儿 MDI 和精神运动发育指数的不良影响，进一步证实汞在金属联合暴露中的协同毒性作用。Nyanza 等[36]在坦桑尼亚西北部手工和小型金矿开采区开展一项纵向随访研究，310 名研究对象为 2015 年在盖塔地区加入坦桑尼亚矿业与健康队列的母亲所生的 3~4 岁儿童，研究采用 MDAT 进行评估，结果显示，与单一暴露相比，汞与铅、镉和砷的联合暴露导致儿童粗大运动能力下降 17.78%、语言能力下降 55.36%、总体发育里程碑（综合评估大运动、精细运动、语言能力、社交能力的发育情况）达标率下降 13.36%。铅和汞的独立神经毒性效应在镉和砷的存在下被进一步放大，提示在多金属共暴露的实际环境中，单一元素的健康风险评估可能低估其真实危害。Nyanza 等[37]另一项研究也证实产前较高浓度的汞暴露可使婴儿 6 和 12 月龄的神经发育障碍风险增高，且高浓度汞与铅或汞与砷的联合暴露会对子代儿童神经发育产生更强的负面影响。

综上所述，尽管有部分研究未发现母亲产前汞暴露与子代儿童神经发育之间的关联，但多数研究提示产前汞暴露对子代儿童神经发育存在不利影响，这种关联可能受到性别、营养因素及联合暴露重金属的修饰或交互作用影响。

3 汞暴露对子代儿童神经发育影响的可能机制

汞通过胎盘屏障进入胎儿体内，可直接作用于发育中的神经系统，也可通过损害胎盘功能间接影响胎儿神经发育[3]。目前关于神经毒性的分子机制研究主要聚焦于以下两个方面。

3.1 基因易感性和 DNA 甲基化 近年来国内外研究发现，基因的易感性及 DNA 甲基化可能是产前汞暴露对胎儿神经系统发育损伤的基础[38]。有研究发现，不同个体对汞毒性的敏感性可能与基因有关，汞对胎儿的神经发育毒性是基因与汞交互作用的结果[23]。我国台湾地区北部一项前瞻性队列研究发现，产前汞暴露对携带载脂蛋白 E 等位基因 ε4 的 2 岁儿童认知、社交和发育商数均存在不良影响[39]，提示基因和环境的交互作用会增加神经系统发育损伤的风险。Maccani 等[40]研究显示，胎盘中 *EMID2* 基因的低甲基化可能是连接宫内汞暴露与新生儿神经行为发育损伤的一种新机制。塞舌尔儿童发育研究营养队列 2 研究发现，产前甲基汞暴露与 12 个 CG 二核苷酸的胞嘧啶位点（CpG）中 1 个 *GRIN2B CpG* 和 2 个 *NR3C1 CpG* 的 DNA 甲基化存在关联，DNA 甲基化改变会导致基因表达下调[38]。

3.2 氧化损伤 动物研究发现，甲基汞诱导的神经毒性主要通过氧化应激诱导活性氧过度生成，进而引发脂质过氧化、DNA 损伤，最终导致神经元功能障碍或死亡[41]。王欣梅等[42]通过动物实验发现，甲基汞暴露会对大鼠脑组织的超氧化物歧化酶、谷胱甘肽过氧化物酶和一氧化氮合酶活性产生影响，从而对脑组织发挥氧化损伤作用。

4 拮抗汞毒性的相关研究

动物研究发现“硒+汞”组幼鼠脑的汞水平和硫代巴比妥酸反应物质的脂质氧化水平均降低，提示硒预处理有助于降低哺乳大鼠组织中汞的含量，可防止大脑因汞暴露所致的脂质氧化损伤[43]。人群研究发现，较高硒水平可降低汞对新生儿的头围和出生体重的不利影响[44]，硒作为一种抗氧化特性的微量元素可拮抗汞所致的氧化损伤，并且该作用可能受到谷胱甘肽相关基因遗传多态性的影响[45]。

Choi 等[46]也发现在调整 N-3 多不饱和脂肪酸（polyunsaturated fatty acids，PUFAs）后，产前甲基汞暴露与学龄期儿童的神经行为损伤之间的关联性增强，可能是 N-3 PUFAs 能够促进神经发育或改变胎盘的炎性环境，从而调节产前甲基汞暴露对儿童神经发育的损伤。MOCEH 队列研究显示，产前汞暴露对婴儿神经发育的不良影响仅出现在产前母亲叶酸水平较低的儿童中，提示叶酸对汞毒性起到保护作用[24]。中国上海一项研究显示，产前低水平汞暴露对新生儿神经行为发育的负面影响仅在二十二碳六烯酸（docosahexaenoic acid，DHA）水平<45.54 μg/mL 的儿童中显现[47]，提示产前 DHA 水平可能对汞暴露起到保护作用。

5 小结

综上所述，现有证据支持产前汞暴露、多金属联合暴露可能对子代儿童神经发育产生不利影响。但由于研究设计、暴露评估方法、结局测量工具、混杂因素控制及人群特征等方面的差异，研究结论尚存在不一致性。未来研究应重点关注以下方面：（1）采用统一规范的暴露评估和结局测量方法，明确产前汞暴露的关键阈值及其与子代儿童神经发育的剂量-反应关系；（2）开展大规模前瞻性队列研究，充分考虑多金属联合暴露、性别差异及营养因素修饰作用；（3）深入探

索汞对神经发育损伤的生物学机制，特别是表观遗传调控、胎盘介导作用及基因-环境交互作用；(4)加强汞毒性拮抗策略的人群研究，评估硒、叶酸、多不饱和脂肪酸等营养素的保护效应及其适用条件，为儿童神经发育障碍的围生期预防提供更充分的科学依据。

利益冲突声明　所有作者声明无利益冲突。

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