

硒在围产期疾病中的研究进展

冯学然, 黎平*

重庆医科大学附属第一医院麻醉科, 重庆

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摘要

硒作为人体必需的微量元素, 其生理功能主要通过硒蛋白实现, 在维持氧化还原平衡、免疫调节及神经内分泌稳定中发挥关键作用。围产期母体血硒水平呈现进行性下降, 使其营养状态与多种围产期疾病密切相关。本综述系统阐述了硒在妊娠期高血压、妊娠期糖尿病、产后抑郁及早产等围产期疾病中的作用。研究表明, 母体低硒状态是上述疾病的重要风险因素, 其机制与硒蛋白抗氧化能力不足, 导致胎盘氧化应激、胰岛素抵抗及神经炎症加剧有关。结论认为, 维持围产期最佳硒水平对保障母婴健康至关重要。未来研究需致力于确立精准的硒营养窗口、深入探索分子机制并开展分层干预试验, 以期将硒发展为围产期疾病预测与个体化营养干预的新靶点。

关键词

硒, 妊娠期高血压, 妊娠期糖尿病, 产后抑郁

Advances in Selenium Research on Perinatal Disorders

Xueran Feng, Ping Li*

Department of Anesthesiology, The First Affiliated Hospital of Chongqing Medical University, Chongqing

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Abstract

Selenium, an essential trace element, exerts its physiological functions primarily through selenoproteins, playing a critical role in maintaining redox balance, immune regulation, and neuroendocrine stability. Maternal blood selenium levels progressively decline during the perinatal period, linking its nutritional status to various perinatal diseases. This review systematically elaborates on the role of selenium in hypertensive disorders of pregnancy, gestational diabetes mellitus, postpartum

*通讯作者。

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depression, and preterm birth. Studies indicate that maternal selenium deficiency is a significant risk factor for these conditions, primarily mediated by insufficient antioxidant capacity of selenoproteins, leading to exacerbated placental oxidative stress, insulin resistance, and neuroinflammation. In conclusion, maintaining optimal selenium levels is crucial for maternal and infant health. Future research should focus on defining precise selenium nutritional windows, elucidating underlying molecular mechanisms, and conducting stratified intervention trials, aiming to establish selenium as a novel target for predicting and individually managing perinatal diseases.

Keywords

Selenium, Hypertensive Disorders of Pregnancy, Gestational Diabetes Mellitus, Postpartum Depression

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1. 引言

硒(Selenium, Se)是人体必需的微量元素,其生物学功能主要通过硒蛋白介导。足够水平的硒对维持中枢神经系统、生殖系统、内分泌系统及心血管系统等正常生理功能至关重要[1]。研究显示,妊娠及哺乳期间母体硒水平随孕周增加呈进行性下降[2]。深入探索硒的生物学机制,对阐明其与围产期疾病的关系具有重要意义。围产期疾病包括妊娠期高血压疾病(pregnancy-induced hypertensive disorders, PIHD)、妊娠期糖尿病(gestational diabetes mellitus, GDM)、产后抑郁(postpartum depression, PPD)、早产(preterm birth, PTB)及胎儿生长受限(fetal growth restriction, FGR)等,不仅严重影响孕产妇健康,亦对胎儿生长发育构成威胁。因此,揭示其发病机制并制定有效防治策略已成为重要的公共卫生议题。本文围绕硒的生理功能及其在围产期疾病中的作用与临床意义进行综述。

2. 硒的分布及生理功能

硒(Selenium)于1817年发现,直至1957年才被确认为高等动物所必需的微量元素[3]。其在地壳中分布不均,形成了如美国、加拿大等富硒地区,以及中国、巴西部分区域等缺硒地区[4]。这种地理分布差异导致全球人群膳食硒摄入量存在显著波动,范围可从每日7 μg至4990 μg不等[5]。世界卫生组织建议成人每日平均硒摄入量为55 μg,并将可接受摄入上限定为400 μg/日[6]。

硒经胃肠道吸收后转运至肝脏代谢,并用于合成硒蛋白[7]。硒在人体内的功能由25个具有硒代半胱氨酸活性中心的硒蛋白实现,主要包括谷胱甘肽过氧化物酶(glutathione peroxidases, GPx)、硫氧蛋白还原酶(thioredoxin reductases, TrxR)和甲状腺激素脱碘酶(iodothyronine deiodinases, DIOs)等[1]。这些硒蛋白具有抗氧化、抗炎、免疫调节及神经保护等多种生物学功能[8]。妊娠及哺乳期因胎儿发育及母体生理变化,对硒的需求显著增加。妊娠期间,硒通过胎盘主动转运至胎儿,导致母体血清硒水平呈进行性下降,尤以妊娠晚期为著[2];至哺乳期,硒通过乳汁分泌进一步增加其生理需求[8]。

3. 硒在妊娠期相关疾病中的作用

3.1. 硒与 PIHD

PIHD 是妊娠期最常见的医学并发症,全球发病率约为10%~15%。PIHD 主要包括妊娠期高血压

(pregnancy-induced hypertension, PIH)和子痫前期(preeclampsia, PE): PIH 指妊娠 20 周后新发高血压(血压 $\geq 140/90$ mmHg), 而 PE 指在高血压基础上伴有明显蛋白尿[9]。研究表明, PIHD 的发生发展与硒状态密切相关。一项病例对照研究显示, 与健康孕妇相比, PE 患者血清硒水平显著降低, 且硒缺乏程度与病情严重性呈正相关[10]。此外, 一项干预性研究将 230 名初产妇随机分为补硒组($60 \mu\text{g/d}$, 以富硒酵母形式)与安慰剂组, 自孕 12 周干预至分娩, 结果显示在低硒人群中补硒可显著降低 PE 发生风险[11]。

PIHD 患者胎盘可出现一系列病理改变, 其核心机制与活性氧(reactive oxygen species, ROS)生成和抗氧化防御系统失衡所致的胎盘氧化应激密切相关, 进而引发促炎细胞因子大量释放[12]。硒作为一种关键的抗氧化微量元素, 在维持机体氧化还原稳态中发挥核心作用, 它通过硒蛋白介导的抗氧化防御系统减轻氧化应激, 从而保护内皮细胞免受 ROS 损伤[10]。研究还表明, 补充硒能够抑制 NF- κ B 信号通路的活化, 下调关键炎症基因的表达, 进而发挥内皮保护作用[11]。

3.2. 硒与 GDM

GDM 是指孕前糖代谢正常或存在潜在糖耐量异常, 在妊娠期首次出现或诊断的糖尿病, 其发病率在我国约为 14.8% [13]。GDM 不仅危害围产期健康, 亦显著增加母亲远期罹患 2 型糖尿病的风险[14]。我们认为, 氧化应激和炎症反应是驱动 GDM 进展的关键病理生理机制[15]。Meta 分析证据显示, GDM 孕妇, 特别是在非欧洲人群以及妊娠中晚期, 其血清硒水平显著低于健康孕妇[13]。为探索其治疗潜力, 一项随机对照试验证实, GDM 孕妇补充硒制剂六周可显著下调淋巴细胞中促炎因子 TNF- α 与 TGF- β 的基因表达[16]。因此, 这些研究为我们提供了新思路: 补硒可能作为 GDM 的辅助治疗手段, 通过缓解氧化应激及炎症反应改善病情。

胰岛组织因超氧化物歧化酶含量在所有组织中最低, 因而对氧化损伤尤为敏感[13]。多数硒蛋白作为抗氧化酶, 通过清除 ROS 在维持胰岛素正常分泌中发挥关键作用。其中, 硒蛋白 P 在胰岛中高表达, 可有效保护 β 细胞免受氧化损伤[17]; 而 GPx1 则通过降解 H_2O_2 减轻其对 β 细胞的毒性作用。GPx1 基因敲除小鼠模型研究显示, 其 β 细胞损伤加剧并伴随胰岛素分泌功能下降[18]。由此可见, 硒及其硒蛋白通过维持胰岛氧化还原平衡, 在保障胰岛素合成与分泌中扮演重要角色, 这为探索硒在 GDM 诊断与防治中的应用提供了理论依据。

3.3. 硒与 PPD

PPD 是一种发生于妊娠期或分娩后 4 周内的抑郁障碍[19], 其全球患病率存在明显地域差异, 高收入国家约为 6.9%~12.9%, 而中低收入国家可超过 20% [20]。伊朗一项随机对照试验表明, 从妊娠早期开始每日补充 $100 \mu\text{g}$ 硒, 可显著提高产后血硒水平, 并有效降低爱丁堡产后抑郁量表(EPDS)评分[21]。PPD 的发病机制涉及多重因素: 患者体内单胺氧化酶活性异常升高, 在催化 5-羟色胺(Serotonin, 5-HT)等单胺类神经递质氧化脱氨过程中产生大量 ROS, 进而通过破坏线粒体功能、诱导神经元凋亡等途径参与 PPD 进程[22]。多数硒蛋白具有抗氧化功能, 以 GPx 为例, 其可催化 H_2O_2 还原为 H_2O , 同时将还原型谷胱甘肽(GSH)氧化为氧化型谷胱甘肽(GSSG), 从而有效清除 ROS、缓解氧化损伤[23]。

神经炎症在 PPD 发病中同样扮演关键角色。分娩过程中的生理应激与组织损伤可引发全身性炎症反应, 促使促炎细胞因子大量释放, 进而诱发抑郁样行为[22]。动物实验表明, 补硒能够显著上调海马区硒蛋白表达, 同时抑制神经炎症相关的氧化应激水平并降低促炎细胞因子表达[24]。由此可见, 硒不仅通过重建氧化还原平衡, 还凭借其抗炎特性在 PPD 防治中展现潜在价值。此外, 研究显示硒还可通过调节肠道菌群组成[25]、促进脑源性神经营养因子(brain-derived neurotrophic factor, BDNF)表达[26]等多重机制参与 PPD 的病理进程, 为其防治提供了新的理论视角。

4. 硒与胎儿生长发育

4.1. 硒与 PTB

PTB 指妊娠未满 37 周分娩, 是导致新生儿发病和死亡的主要原因[27]。一项挪威大规模前瞻性队列研究纳入 72,025 名单胎孕妇, 通过孕 22 周时采用的半定量食物频率问卷评估妊娠前半期硒摄入量, 结果显示该阶段较高的膳食硒摄入量与早产风险降低显著相关[28]。早产的发生多由炎症事件触发, 炎症因子上调可促进子宫收缩因子表达与释放, 从而诱导子宫活动[29]。动物实验进一步表明, 硒蛋白在子宫平滑肌收缩调控中具有关键作用, 任一硒蛋白的缺失均可能影响子宫收缩功能[30]。因此, 在炎症水平较高或存在细胞因子失调的妊娠状态下, 维持充足的硒水平可能有助于延长孕周、降低早产发生风险。

4.2. 硒与 FGR

FGR, 也称为宫内生长受限(intrauterine growth restriction, IUGR), 是妊娠期常见的并发症, 影响 10%~15% 的产妇[31]。有证据表明氧化应激和炎症与 FGR 的病理生理学有关, 故一项随机对照试验评估了补硒对有 FGR 风险的孕妇临床体征和代谢状态的影响。根据平均搏动指数(pulsatility index, PI) > 1.45 的异常子宫动脉多普勒波形有 FGR 风险, 结果显示补硒 10 周后, 补硒组 PI < 1.45 的女性比例高于安慰剂组(P = 0.002)。此外, 补硒组血浆总抗氧化能力、GSH 和高敏 C 反应蛋白(high sensitivity C-reactive protein, hs-CRP)水平的变化显著[31]。动物实验进一步发现妊娠期硒缺乏可能通过母体甲状腺激素浓度升高和胎盘甲状腺激素代谢受损而导致 FGR [32]。

4.3. 硒与胎儿神经发育

近期一项波兰研究对 539 对母婴的硒水平与子代神经发育关系进行了追踪分析。该研究量化了妊娠各期、分娩时及脐带血中的硒浓度, 并评估儿童 1 岁和 2 岁时精神运动发育状况。结果显示, 母体妊娠期血浆硒水平呈进行性下降, 且妊娠早期硒水平与 1 岁儿童运动发育评分及 2 岁语言发育评分均呈显著正相关[33]。动物实验进一步支持该结论, 补硒被证明可改善大鼠空间学习记忆能力并保护突触结构可塑性[34]。上述证据共同提示, 生命早期硒营养状态与神经发育存在重要关联, 但其具体机制及临床应用价值仍需通过更深入的流行病学与实验研究加以阐明。

尽管妊娠期硒缺乏与早产及子代神经发育迟缓等不良结局相关, 但过量硒暴露同样可能产生负面影响。一项基于波士顿母婴队列的研究(n = 1550)在产后 72 小时内检测母体红细胞硒水平, 发现其与子代自闭症谱系障碍(autism spectrum disorder, ASD)及注意缺陷多动障碍(attention-deficit/hyperactivity disorder, ADHD)的患病风险均呈正相关[35]。这一结果提示, 产前母体高硒状态可能对胎儿神经发育构成潜在威胁。因此, 维持孕期硒水平的合理范围至关重要, 通过精准调控硒营养状态方可最大化其健康效益并规避潜在风险。

现有证据共同提示, 孕期硒水平与子代神经发育结局之间可能存在一种阈值或倒 U 型的非线性关系。硒水平低于某一阈值时, 随着浓度增加, 神经发育获益; 但当其超过另一阈值后, 进一步增加可能反而带来风险。国内有报道称, 每日硒摄入量超过 850 μg 的人群中发生硒中毒的概率增加。美国医学研究所已将成人硒的可耐受最高摄入量设定为 400 $\mu\text{g}/\text{天}$, 以预防硒中毒的风险[36]。考虑原因可能是当硒水平异常升高时, 硒可能发挥促氧化作用, 直接产生 ROS, 同样引发氧化损伤。

5. 小结与展望

综上所述, 硒作为通过硒蛋白发挥核心生理功能的必需微量元素, 在维持围产期母婴健康中扮演着不可或缺的角色。大量证据表明, 妊娠期母体血硒水平的生理性下降若叠加基础硒营养不足, 会通过削

弱机体的抗氧化与抗炎防御能力, 增加 PIHD、GDM、PPD、PTB 等多种围产期并发症的风险, 并可能对子代的神经发育产生不利影响。其作用机制主要与硒蛋白调控氧化应激、炎症反应及保护内皮与神经细胞等功能密切相关。

展望未来, 本领域的研究突破有赖于构建更精准的硒营养评价体系, 通过大规模前瞻性队列明确围产期各阶段硒需求的“安全窗口”与最佳摄入量。同时, 必须深化对特定硒蛋白分子机制的解析, 并开展基于人群分层的高质量干预研究, 以精准界定补硒的获益人群。此外, 硒的“双重效应”警示我们, 必须要从更全面的视角看待硒, 必须找到最佳硒含量。解决这些关键问题, 将推动硒从一种基础微量元素, 转化为用于围产期疾病风险预警与个体化营养干预的可靠靶标, 最终为提升母婴生命健康提供新支点。

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