

• 综述 •

孕期碘营养与妊娠期代谢性疾病关系的研究进展

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摘要: 碘是人体甲状腺激素合成和生长发育所必需的微量营养素。研究发现, 碘可在甲状腺外发挥生物学功能, 通过调节氧化应激和肠道菌群丰度影响机体代谢稳态, 孕期碘营养失衡与妊娠期代谢性疾病的发生发展密切相关。本文收集 1980—2025 年关于孕期碘营养与妊娠期代谢性疾病关系的相关文献, 对孕期碘营养与妊娠糖尿病、妊娠高血压及子痫前期、妊娠期超重或肥胖、妊娠期血脂异常等妊娠期代谢性疾病的关系及其潜在生物学机制进行综述, 为制定妊娠期代谢性疾病的防控策略提供依据。

关键词: 碘; 妊娠期; 代谢性疾病

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The relationship between iodine nutrition during pregnancy and gestational metabolic diseases: a review

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Abstract: Iodine is an essential micronutrient for the synthesis of thyroid hormones and for growth and development in the human body. Recent studies have revealed that iodine can exert biological functions beyond the thyroid. By modulating oxidative stress and gut microbiota abundance, it influences systemic metabolic homeostasis. Imbalance in iodine nutrition during pregnancy is closely associated with the onset and progression of gestational metabolic diseases. Based on studies pertaining to the relationship between iodine nutrition during pregnancy and gestational metabolic diseases from 1980 to 2025, this review summarized the relationship of iodine nutrition during pregnancy with gestational metabolic diseases, including gestational diabetes mellitus, gestational hypertension and preeclampsia, gestational overweight or obesity, and gestational dyslipidemia, and described the underlying biological mechanisms, so as to provide the evidence for formulating prevention and control strategies for gestational metabolic diseases.

Keywords: iodine; pregnancy; metabolic diseases

妊娠期代谢性疾病是一组与妊娠期代谢紧密相关的疾病, 包括妊娠糖尿病、妊娠高血压及子痫前期、妊娠期超重或肥胖、妊娠期血脂异常和妊娠期代谢综合征。碘是人体甲状腺激素合成和生长发育

所必需的微量营养素, 可在甲状腺外发挥生物学功能, 参与机体代谢稳态调控^[1]。妊娠期女性对碘的需求量明显增加, 同时伴随一系列特有的代谢及生理变化, 这些因素的共同作用可能增加妊娠期代谢性疾病的发生风险^[2-3]。本文通过检索中国知网、PubMed 数据库, 系统综述 1980—2025 年关于孕期碘营养与妊娠期代谢性疾病关系的相关研究, 探讨孕期碘营养与妊娠期代谢性疾病的关系, 以及潜在的生物学机制, 为制定妊娠期代谢性疾病的防控策略提供依据。

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1 孕期碘营养与妊娠期代谢性疾病关系的流行病学研究

1.1 孕期碘营养对妊娠糖尿病的影响

妊娠糖尿病 (gestational diabetes mellitus, GDM) 是在妊娠期首次发生, 因糖耐量异常而导致不同程度的血糖升高。16% 的 GDM 患者会在产后一年进展为代谢综合征, 且再次妊娠时 GDM 复发率达 33%~69%^[4]。孕期碘缺乏 (尿碘浓度 $<150\ \mu\text{g/L}$)、碘超足量 (尿碘浓度为 $150\sim<500\ \mu\text{g/L}$) 或碘过量 (尿碘浓度 $\geq 500\ \mu\text{g/L}$) 均可能增加 GDM 发生风险。一项中国的前瞻性队列研究结果显示, 孕早期轻度碘缺乏是 GDM 的独立危险因素 ($OR=1.669$, 95%CI: 1.114~2.501)^[5]。通过动态监测尿碘浓度, 可进一步发现 GDM 确诊前持续处于低尿碘水平的孕妇 GDM 发生风险显著增加^[6]。此外, 一项巴西的队列研究采用 3 次随机尿样的尿碘中位数评估孕早期碘营养, 发现与碘充足组相比, 碘超足量或碘过量组孕妇 GDM 发生风险更高^[7]。但尚未发现孕中期尿碘浓度与 GDM 的关联^[8], 提示应进一步关注孕期碘营养与 GDM 关联是否存在暴露窗口期。

1.2 孕期碘营养对妊娠高血压及子痫前期的影响

妊娠高血压是妊娠 20 周后新发的高血压。孕期碘营养失衡可导致亚临床甲状腺功能减退, 增加妊娠高血压发生风险^[9]。SILVA 等^[7]研究结果显示孕早期碘超足量或碘过量是妊娠高血压的独立危险因素; 另有研究结果显示孕晚期碘缺乏与妊娠高血压有关^[10]。子痫前期是在妊娠高血压的基础上同时合并蛋白尿或多器官功能损伤。孕期碘缺乏可能增加子痫前期的发生风险。BUSINGE 等^[11]研究结果显示, 子痫前期患者的尿碘浓度显著低于血压正常的对照组, 且尿碘浓度随子痫前期严重程度的增加呈下降趋势^[12]。一项纳入 49 187 名孕妇的队列研究结果显示, 低碘摄入量与较高的子痫前期发生风险有关^[13]。

1.3 孕期碘营养对妊娠期超重或肥胖的影响

妊娠期超重或肥胖指妊娠期女性体内脂肪组织过度蓄积的状态, 包括妊娠前超重或肥胖 (孕前体质指数 $\geq 24\ \text{kg/m}^2$), 以及孕期过度增重。尿碘浓度在超重或肥胖孕妇和正常体重孕妇间的差异无统计学意义^[7-8]。然而, 多项研究表明孕期碘营养与孕期增重呈负相关^[14-16]。NEVEN 等^[14]的研究结果表明孕期增重较多与胎盘碘储存量较低相关; LIU 等^[15]通过队列研究发现孕期血清碘浓度与孕期增重呈负相关, 其中孕中期血清碘浓度对孕期增重影响最

大^[16]。孕期碘营养可能参与母体脂肪代谢调控或能量调节, 孕中期是碘营养影响孕期体重的重要阶段, 属于潜在的暴露窗口期。

1.4 孕期碘营养对妊娠期血脂异常的影响

妊娠期血脂异常指妊娠期女性血清胆固醇和/或三酰甘油水平升高。LI 等^[8]研究结果表明, 孕中期碘过量可显著提高孕妇总胆固醇、低密度脂蛋白胆固醇和高密度脂蛋白胆固醇水平, 提示孕期碘营养可能参与血脂的稳态调节。糖类、脂质及蛋白质代谢可相互调控, 血脂异常还与 2 型糖尿病发病及血糖波动密切相关^[17]。但目前关于孕期碘营养与妊娠期血脂异常关系的研究较少, 仍需进一步研究验证。

1.5 孕期碘营养对妊娠期代谢综合征的影响

妊娠期代谢综合征 (gestational metabolic syndrome, GMS) 是妊娠期特殊的代谢性疾病类型, 会增加不良妊娠结局和远期母子心血管代谢风险。目前国内外对于 GMS 的诊断多沿用成人代谢综合征诊断标准, 可能导致 GMS 患病率被高估^[18]。已有研究表明成年非妊娠期女性碘营养与代谢综合征存在关联, 甲状腺功能正常的成年非妊娠期女性尿碘浓度 $<100\ \mu\text{g/L}$ 显著增加代谢综合征发生风险^[19]。该结果为探索孕期碘营养与 GMS 发生风险间的潜在关联提供了初步线索。孕期碘营养对母体代谢调节有重要作用, 妊娠期女性处于碘需求量激增、生理性胰岛素抵抗及代谢负荷加重的特殊状态, 应加强关注孕期碘营养对 GMS 的影响。

1.6 碘与环境毒物的交互作用对妊娠期代谢的影响

近年来, 研究发现环境毒物 (如重金属和内分泌干扰物等) 与机体代谢密切相关, 已成为新的代谢性疾病危险因素。高碘暴露可增强汞和多环芳烃对三碘甲状腺原氨酸 (triiodothyronine, T_3) 水平的抑制作用, 可能因为高碘暴露与环境因素对脱碘酶活性的影响具有协同效应^[20-21]。脱碘酶是在甲状腺和外周组织中将甲状腺素转化为 T_3 的催化酶, 广泛分布于甲状腺、胎盘、肝脏和肾脏等组织中, 在调节脂质代谢和胰岛素敏感性等方面具有重要作用。目前尚无研究报道碘与环境因素的交互作用对妊娠期代谢性疾病的影响, 但上述证据提示两者复合暴露可能具有协同健康危害, 从而促进代谢性疾病的发生。

2 碘影响妊娠期代谢性疾病的机制

2.1 碘通过氧化应激影响代谢

氧化应激在代谢性疾病的发病机制中至关重要, 碘可能通过调节氧化应激从而影响机体代谢。碘可以

通过 Kelch 样环氧氯丙烷相关蛋白 1 (Kelch-like ECH-associated protein1, Keap1) -核因子 E2 相关因子 2 (nuclear factor erythroid 2-related factor 2, Nrf2) /抗氧化反应元件 (antioxidant response element, ARE) 信号通路发挥抗氧化功能。体外研究表明, Keap1 的碘化可导致 Nrf2 释放并易位至细胞核, 与核内小 Maf 蛋白形成异二聚体, 随后与 ARE 结合并触发抗氧化酶的表达^[22]。动物研究也验证了这一机制, 糖尿病小鼠摄入适量的碘补充剂后, 丙二醛水平显著降低, Nrf2 和抗氧化酶表达水平显著升高^[23]。一项评估碘补充剂对链脲佐菌素诱导的糖尿病模型的预防和治疗效果研究结果显示, 分子碘补充剂能够减少组织的氧化损伤^[24]。对雄性大鼠进行碘化钾重复干预的代谢组学研究结果显示, 在甲状腺内, 相较于甲状腺激素途径, 与儿茶酚胺代谢相关的酪氨酸代谢所受的影响更大, 同时伴随氧化应激和炎症相关反应的外周代谢变化^[25]。另有研究表明, 高碘摄入量的怀孕大鼠 Krüppel 样因子 9 表达上调会抑制硫氧还蛋白还原酶 2 (thioredoxin reductase 2, Txnrd2) 转录^[26]。Txnrd2 是一种关键的抗氧化酶, 这种变化可能导致活性氧在心肌细胞中积累, 引起收缩压升高和心脏功能亢进。此外, 碘缺乏与细胞膜脂质更严重的氧化损伤密切相关, 补碘可减少胎盘细胞中的脂质过氧化水平, 减少氧化应激^[27]。

2.2 碘通过肠道菌群影响代谢

研究发现肠道菌群的丰度与 GDM 和子痫前期等妊娠期代谢性疾病发生风险相关, 其中厚壁菌门的丰度与 GDM 的发生风险呈正相关^[28], 拟杆菌门的丰度与子痫前期的发生风险呈正相关^[29]。而碘与肠道菌群丰度有关。补碘可以使雌性小鼠的多种肠道菌群丰度显著增加, 包括肠道穆尔巴赫菌、异普雷沃菌属和肠球菌属, 上述菌群是厚壁菌门和拟杆菌门的重要成员^[30]。因此, 碘可能通过调控肠道菌群进而影响妊娠期代谢状态, 但具体作用机制尚不明确, 有待进一步深入研究。

3 小 结

孕期碘缺乏、碘超足量或碘过量均可能增加妊娠期代谢性疾病的发生风险, 可导致 GDM、妊娠高血压及子痫前期、孕期过度增重和妊娠期血脂异常的发生。孕期碘营养与妊娠期代谢性疾病关系的暴露窗口期, 以及该关联是否受到不同碘营养评估方法的影响尚未得到充分研究。孕期碘营养影响妊娠期代谢性疾病的生物学机制尚不明确, 目前认为其潜在机制与氧

化应激、肠道菌群丰度有关。建议进一步开展相关研究, 揭示孕期碘营养影响妊娠期代谢性疾病的潜在作用机制。

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