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综述

妊娠期肥胖母体肠道菌群对子代的影响及潜在机制研究进展

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［摘要］妊娠期肥胖女性的数量逐年增加，是全球面临的严峻挑战之一。母体妊娠期肥胖不仅会导致母体自身妊娠糖尿病、高血压、早产、流产等，也严重影响子代生后生长发育，增加子代肥胖和患其他代谢疾病的风险。母体妊娠期肠道菌群变化在妊娠和分娩过程中起重要作用，且肠道菌群的代谢产物也与肥胖的代谢调节密切相关。该文综述妊娠期肥胖母体肠道菌群对子代影响的潜在机制，旨在探索和深化对母体肠道菌群影响子代机制的认识，为临床干预提供研究靶点和思路。［中国当代儿科杂志，2026，28（5）：624-628］

［关键词］妊娠期肥胖；肠道菌群；子代肥胖；炎症；激素

Research progress on the effects of maternal gut microbiota in gestational obesity on offspring and potential mechanisms

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Abstract: The number of women with gestational obesity has been increasing year by year and is a serious global challenge. Maternal obesity during pregnancy not only leads to gestational diabetes, hypertension, preterm birth, and miscarriage, but also adversely affects offspring growth and development after birth by increasing the risk of offspring obesity and other metabolic diseases. Changes in the maternal gut microbiota during pregnancy and delivery play important roles, and microbiota-derived metabolites are closely related to the metabolic regulation of obesity. This review summarizes the potential mechanisms by which the maternal gut microbiota in gestational obesity influences offspring, aiming to deepen understanding of how maternal gut microbiota affects offspring and to provide research targets and ideas for clinical intervention.

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Key words: Gestational obesity; Gut microbiota; Offspring obesity; Inflammation; Hormone

妊娠期女性超重或肥胖是全球面临的日益严重的健康问题。中国人群的研究数据表明，18~49岁育龄期女性中，超过1/3处于超重或肥胖状态，且在过去十年中呈上升趋势[1]。妊娠期肥胖不仅影响母体自身，可能导致妊娠糖尿病（gestational diabetes mellitus, GDM）、高血压、抑郁、早产，甚至流产、死产等不良后果；同时也增加子代肥胖和患代谢疾病的风险，严重影响妇幼健康，对儿童生长发育产生深远影响[2]。肠道菌群作为机体代谢活动和免疫调节的重要因素，通过参与妊娠期母体营养物质的代谢，调节母体免疫反应和炎症状态，进一步影响子代的生长发育与健康。调整肠道菌群的组成可减轻妊娠期母体肥胖的负面影响[3]。因此，探讨肠道菌群如何介导妊娠期母体肥胖对子代健康的影响机制，有助于制定特异性的干预策略，进一步改善母亲和子代的健康状况，促进子代更好的生长发育。

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1 妊娠期肥胖女性肠道菌群特点

1.1 肠道菌群

肠道菌群在女性妊娠期间随妊娠时长波动和重塑：妊娠早期，母体肠道菌群中丁酸盐产生菌（butyrate-producing bacteria, BPB）占重要组成部分；妊娠中后期，母体肠道菌群结构发生重大改变，细菌多样性降低，BPB丰度显著降低[4]。肥胖会加剧上述变化，妊娠期肥胖母体的肠道菌群多样性显著低于正常体重母体[5]；肥胖且合并GDM的母体中，布劳特氏菌属（*Blautia*）、厌氧棒状菌属（*Anaerostipes*）等菌属差异显著，并与口服葡萄糖耐量试验1 h和2 h血糖水平显著相关。孕早期体重过度增加的母体肠道菌群中，粪杆菌属（*Faecalibacterium*）、臭杆菌属（*Odoribacter*）、克里斯滕森菌科（*Christensenellaceae*）、毛螺菌科（*Lachnospiraceae*）等BPB丰度显著降低[6]。

Gilley等[7]报道超重母亲分娩的婴儿生后6个月的肠道菌群组成显著区别于非超重母亲分娩的婴儿的肠道菌群，且根据菌群多样性和组成可预测婴儿发育后期的肥胖程度。肥胖母亲分娩的婴儿生后6个月时的肠道菌群中，粪杆菌属的丰度显著降低[8]，这可导致新生儿出生体重增加，在妊娠期肥胖的啮齿类动物中也有类似的变化[6]。

1.2 肠道菌群的代谢产物

除肠道菌群本身外，短链脂肪酸（short-chain fatty acid, SCFA）是肠道菌群分解膳食纤维产生的代谢产物，是结肠细胞的主要能量来源[9]，可提供人体所需能量的10%左右。常见与肥胖相关的SCFA是乙酸、丙酸和丁酸3种。丁酸在妊娠期肥胖母体内的浓度显著降低[10]。妊娠期体重指数增加的母体分娩的新生儿粪便中丁酸盐含量明显减少[11]。以上研究充分说明，妊娠期肥胖母体及其子代的肠道菌群紊乱，并且丁酸盐含量减少。

因此，妊娠期肥胖女性的肠道菌群组成及代谢产物失衡，对子代菌群和代谢产生影响。挖掘关键菌群的丰度及其功能变化，对于制定干预策略以降低妊娠期肥胖对母婴健康的影响非常重要。

2 妊娠期肥胖母体肠道菌群改变对子代的影响机制

2.1 肠道菌群在母婴间的垂直传播

胎盘和羊水并非完全无菌环境，宫内存在特定的微生物群落，对子代初始肠道菌群的形成具有重要作用。研究发现，胎盘和羊水中的微生物与母体的肠道菌群存在相似性，提示胎儿在子宫内就可能直接受到母体菌群的影响[12]。此外，母体肠道菌群通过分泌细胞外囊泡与胎儿进行相互作用，这可能在胎儿免疫系统的初步构建中起到关键作用[13]。

分娩方式在胎儿肠道菌群的建立中起至关重要的作用。阴道分娩的婴儿通常会通过接触母体的阴道菌群和肠道菌群获得初始的微生物群落[14]，而剖宫产出生的婴儿由于缺乏与母体微生物的直接接触，往往表现出肠道菌群的多样性降低等特点[15]。妊娠期肥胖母体肠道菌群发生显著改变，可通过上述垂直传播方式影响子代菌群组成及免疫构建。

2.2 肠道菌群通过引起系统性炎症影响胎盘

肥胖人群肠道屏障的完整性受损，以及肠上皮中紧密连接蛋白（如ZO-1、Occludin和Claudin）的表达显著减少，会导致肠道通透性增加，细菌脂多糖可通过受损的肠道屏障进入血液循环，进而引发系统性炎症[16]。研究表明，妊娠期肥胖与胎盘及母体血浆中促炎细胞因子［如白细胞介素（interleukin, IL）-6、IL-1β、IL-8和单核细胞趋化蛋白-1］水平升高有关[17]。动物实验证实，肥胖母鼠在妊娠后期免疫细胞发生变化，表现为脾脏中T细胞亚群增加，辅助性T细胞1和辅助性T细胞17比例显著升高，通过分泌干扰素-γ和IL-17A等细胞因子增强炎症反应[18]。母体的炎症状态会增加胎儿生长受限的发生风险[19]，同时胎盘的炎症状态可能影响胎盘的结构和功能，导致胎盘的营养物质转运能力下降，减少脂质向胎儿的运输，其中包括胎儿大脑发育所必需的长链多不饱和脂肪酸，最终影响胎儿的神经发育[20]。

2.3 肠道菌群通过SCFA调控激素分泌

肠道是重要的内分泌器官之一，肠内分泌细胞可分泌20余种激素。其中酪酪肽（也叫肽YY）和胰高血糖素样肽-1（glucagon-like peptide-1, GLP-1）涉及调节食物摄入和胰岛素分泌，在肥胖研究中具有重要意义[21]。肽YY可诱导饱腹感，从而减少热量摄入。GLP-1是胰高血糖素原合成的多肽，由胰高血糖素或胰高血糖素原基因编码，是目前肥胖治疗的热门靶点且与肠道菌群变化密切相关[22]。分泌肽YY和GLP-1的肠内分泌细胞亚群被称为L细胞。已证实L细胞表面表达SCFA的受体G蛋白

偶联受体（G protein-coupled receptor, GRP）41 和 GPR43[23]。研究表明，过度妊娠增重与脐带血中肽 YY 水平呈负相关[24]。一方面，SCFA 可通过调节肠道菌群组成，影响妊娠期肥胖母体的食欲。另一方面，在动物模型中，通过胎儿交感神经组织、胃肠道和胰腺中的 GPR41 和 GPR43 来检测母体肠道微生物的代谢产物 SCFA[25]。另有研究表明，对肥胖孕鼠给予根皮苷干预，可通过激活 SCFA-GPR43 通路促进 GLP-1 分泌，改善子代因妊娠期母体肥胖导致的代谢紊乱[26]，提示 SCFA 可能通过调控激素分泌影响胎儿的代谢过程[27]。此外，SCFA 与胰岛素敏感性受损和 GDM 有极大的关联性[28]，妊娠期肥胖导致母体患 GDM 的风险增加，GDM 母体的肠道菌群和 SCFA 通过调节胰岛素分泌改变胎盘的结构和功能，影响胎儿的代谢调控[29]。

以上提示，母体的肠道菌群及其代谢产物在妊娠期可通过调节激素对代谢的调控，进而对子代的健康产生深远影响。

2.4 肠道菌群参与调控表观遗传

表观遗传学修饰也是重要的分子机制之一。肠道菌群可以产生多种生物代谢物，其中一些可作为表观遗传底物、辅助因子或表观遗传酶活性的调节剂[30]。妊娠期肥胖母体肠道菌群对子代的表观遗传调控主要是通过 DNA 甲基化和组蛋白修饰实现的。肥胖母体肠道菌群代谢产物（如叶酸、胆碱）可能通过参与一碳代谢影响胎儿表观基因组，调控肥胖相关基因（如 *PPARγ*、*leptin*）的甲基化状态[31]。研究表明，母体内叶酸水平充足可能有助于降低与母体肥胖相关的对子代的负面代谢影响[32]。一项动物研究报告表明，在妊娠期给喂食高脂饮食的大鼠补充叶酸可减少雄性子代的肝脂肪变性[33]。低水平的胆碱已被证实会诱发高同型半胱氨酸血症，这已成为 2 型糖尿病的生物标志物[34]。一项队列研究提示，胆碱可缓解 GDM 导致的胎盘脂肪运输过度以及胎儿过度生长的问题[35]。胆碱代谢受肠道菌群影响，母鼠胆碱消耗型肠道细菌会降低甲基供体的可用性，影响胆碱代谢，并改变成年小鼠及其后代的全基因组 DNA 甲基化模式[36]。丁酸作为肠道菌群代谢产物之一，也是组蛋白去乙酰化酶抑制剂，可能通过修饰胎儿染色质结构影响代谢相关基因的表达[37]。

母体肠道菌群在妊娠期肥胖中通过以上 4 种可能的机制影响子代肥胖（图 1），这 4 个机制同时也为临床上减轻妊娠期肥胖以及减少妊娠期肥胖导致的子代肥胖提供干预靶点。

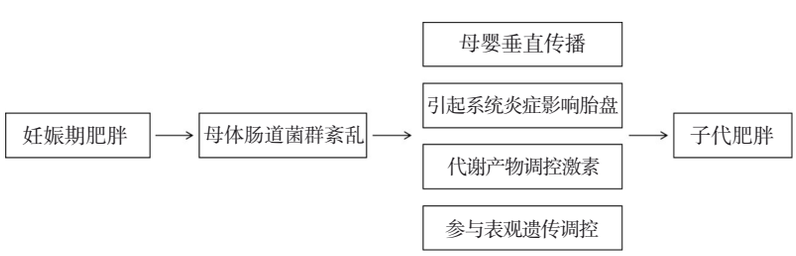


图 1 妊娠期肥胖母体肠道菌群及代谢产物对子代影响的可能机制

3 益生菌和益生元在妊娠期肥胖中的应用

一项基于现有证据的系统综述显示，妊娠期将益生菌和益生元作为补充剂使用，总体而言对孕妇是安全的[38]。一项针对 219 项随机对照试验和 17 项系统评价/荟萃分析的研究表明，益生菌是最常见且有效的干预措施，可减轻 GDM，降低空腹血清胰岛素、糖化血红蛋白水平及胰岛素抵抗[39]。然而，也有研究对 50 例肥胖母体在孕期摄入益生菌后对婴儿生后 9 个月内肠道菌群组成及围生期母婴健康结局的影响进行了观察，结果发现孕期补充益生菌并未改变婴儿的肠道菌群多样性，仅与肥胖相关的柯林斯菌属水平下降[40]。尽管在孕期补充益生菌和益生元可能会对特定的、与肥胖相关的肠道微生物种类产生影响，但目前的证据并未能一致证明益生菌和益生元在减轻母体和子代的体重/体重指数方面的有效性。因此，益生菌和益生元在控制妊娠期母体肥胖和预防代际肥胖方面的作用具有潜力，但尚无定论。目前益生菌和益生元的临床研究尚无统一标准，致使各类研究中所用的益生菌和益生元的作用效果无法定量分析，进而导致研究结果存在差异。

4 总结与展望

母体妊娠期肥胖导致子代肥胖是目前儿童及成年后肥胖的重要原因之一，明确其机制对预防和改善肥胖母体分娩肥胖子代具有重要意义。肠道菌群紊乱在肥胖的致病机制中扮演重要角色，但其在母体妊娠期肥胖导致子代肥胖中的具体机制尚不明确。本文详细综述了肠道菌群在妊娠期肥胖母体对子代影响的潜在机制。可针对性地采用调整饮食、补充益生菌与益生元等肠道菌群调节方式，尽可能降低母体肥胖对子代的不良影响。通过多学科的合作与研究，有望揭示更为复杂的生物机制，从而为母婴健康提供更为精准的指导和支持。

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