

母亲肥胖与子代肥胖关系的研究进展

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摘要: 近年来, 儿童肥胖患病率持续攀升, 对人群健康构成严重威胁。妇幼健康是全民健康的基石, 而生命早期作为与妇幼健康紧密关联的特殊时期, 亦是儿童生长发育的机遇窗口期, 对全生命周期健康管理至关重要。研究表明, 母亲在妊娠前、妊娠期及产后的肥胖状态会显著增加子代肥胖风险, 但不同时期的作用重点及发生发展机制仍不够清晰。本文综述了母亲不同生理阶段肥胖与子代肥胖的关联, 深入探讨了遗传因素、代谢重编程等潜在影响机制, 并总结了相关干预措施, 强调妊娠前-妊娠期-产后全周期防控的重要性, 旨在从生命早期源头深入解析肥胖成因并探索防控策略, 对推动我国儿童肥胖防治、促进妇幼健康具有重要意义。

关键词: 母亲肥胖; 儿童肥胖; 母婴; 生命早期

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Progress on the relationship linking maternal obesity to offspring obesity

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Abstract: In recent years, the rising incidence of childhood obesity has become a significant public health challenge. Maternal and child health (MCH) is the cornerstone of public health for all. Early life, as a critical period closely linked to MCH, is also a window of opportunity for child growth and development, making it crucial for life-cycle health management. Research indicates that maternal obesity status before pregnancy, during pregnancy, and postpartum significantly increases the risk of offspring obesity. However, the specific impacts of these different periods and the underlying developmental mechanisms are not yet fully understood. This paper reviews the association between maternal obesity at different physiological stages and offspring obesity. It further explores potential mechanisms, such as genetic factors and metabolic reprogramming, and summarizes relevant interventions. Emphasizing the importance of full-cycle prevention and control spanning the preconception, pregnancy, and postpartum periods, this review aims to better understand and prevent early-life obesity. This is crucial for reducing the prevalence of childhood obesity and promoting maternal and child health in China.

Keywords: maternal obesity; childhood obesity; mother and child; early life

肥胖是全球性公共卫生挑战, 在儿童群体中尤为突出。数据显示, 2021 年全球 5~14 岁肥胖儿童约有 9 310 万, 预计未来 5 年仍将持续增长^[1]。为应对此趋势, 国家卫健委于 2024 年 6 月联合 16 个部门启动“体重管理年”活动, 推动建立全人群、全生命周期的体重管理理念, 标志着我国肥胖防控进入新阶段。

根据“健康与疾病的发育起源”理论, 生命早期 1 000 天(即胎儿期至生后 2 岁)是生长发育的关键

窗口, 此阶段若暴露于母体代谢异常等不良环境中, 将显著增加成年后肥胖等慢性病风险。作为子代生命最初的微环境, 母代健康状况对子代远期健康有着深远影响。当前育龄期女性肥胖率不断上升, 子代面临的不良健康结局风险进一步加剧^[2]。

母婴肥胖代际传播涉及妊娠前、妊娠期和产后全周期, 构成“代谢记忆-宫内编程-环境暴露”的连续性过程: 妊娠前母体代谢异常通过卵母细胞表观遗传改变等奠定子代肥胖易感基础; 妊娠期通过表

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观遗传重编程、胎盘代谢紊乱加剧代谢风险;产后母乳成分改变、肠道菌群传递及喂养行为进一步巩固肥胖表型。本文突破单阶段研究局限,系统整合三个关键时期,深入阐述母婴肥胖代际传播的影响机制,旨从生命早期入手,为防治儿童超重肥胖、促进妇幼健康发展提供理论依据及实践指导。

1 妊娠前肥胖与子代肥胖的关联及影响机制

母亲妊娠前的健康状况对母婴结局具有深远影响。基于育龄夫妇平均受孕周期及妊娠前健康管理的临床意义,通常将受孕前 3 个月身体质量指数(body mass index, BMI) $\geq 30 \text{ kg/m}^2$ 定义为妊娠前肥胖^[3]。涵盖超 22 万女性的队列研究显示,妊娠前 BMI 每降低 1 kg/m^2 ,妊娠期糖尿病、早产及巨大儿等不良结局风险显著降低^[4]。而母亲妊娠前肥胖则使子代肥胖风险增加约 3 倍^[2],且这种关联性随子代年龄增长持续增强^[5]。通过构建母亲体重轨迹模型进一步发现,与妊娠期或产后体重变化相比,妊娠前高 BMI 对子代儿童期肥胖的影响更为显著^[6]。因此,妊娠前阶段也被视作预防乃至阻断儿童肥胖发展的关键窗口,其影响甚至能跨代传递^[7]。

妊娠前期是生殖健康的重要奠基阶段,妊娠前肥胖的母亲可能通过遗传机制将与能量代谢、脂肪积累和食欲调节相关的肥胖易感基因(如 *INHBE*、*POMC*)传递给子代^[8],还可通过表观遗传修饰调控子代肥胖风险。妊娠前的代谢状态会影响生殖细胞发育,而卵母细胞的发育质量则直接决定胚胎的发育潜能。例如,高脂饮食(high-fat diet, HFD)诱导下,雌鼠卵母细胞中关键调控蛋白 Stella 表达显著下调,干扰卵子发生过程中 DNA 甲基化模式的正常建立,这可能是肥胖表型跨代传递的重要分子机制^[9]。一项出生队列研究发现,母亲妊娠前肥胖显著改变新生儿脐带血甲基化谱,共鉴定出 481 个与母亲 BMI 相关的 CpG 位点,其中 123 个与儿童超重肥胖相关,14 个在母亲 BMI 与子代超重肥胖关联中起显著介导作用^[10]。代谢组学研究则表明,妊娠前超重/肥胖通过特定代谢物影响儿童早期生长,羧基天冬酰胺、4-羟基谷氨酸、黄素腺嘌呤二核苷酸、菜油甾醇和 β -谷甾醇,通过调控各自代谢通路(丙氨酸和天冬氨酸代谢、谷氨酸代谢、核黄素和维生素 A 代谢、雄激素类固醇和甾醇代谢等),实现了这一影响^[11]。

2 妊娠期肥胖与子代肥胖的关联及影响机制

妊娠期肥胖主要受到妊娠前 BMI 及妊娠期体重增长(gestational weight gain, GWG)共同影响。

GWG 推荐范围因妊娠阶段及地域差异有所不同。国际广泛采用 2009 年美国医学研究所(Institute of Medicine, IOM)修订的 GWG 指南^[12]。鉴于与欧美人群体质的差异,国家卫健委 2022 年发布了更适用于中国妇女的《妊娠期妇女体重增长推荐值标准》^[13],根据妊娠前 BMI 对孕妇分类并设定了相应的体重增长推荐范围。将妊娠前 BMI $\geq 28.0 \text{ kg/m}^2$ 定义为肥胖并建议 GWG 控制在 $5 \sim 9 \text{ kg}$ 。GWG 过高会显著增加子代超重肥胖风险,这一结论在大规模队列研究中也得到了证实^[14]。Wrotniak 等^[15]发现,妊娠期体重每增加 1 kg ,子代 7 岁时超重风险上升 3%;若 GWG 超过 IOM 指南推荐范围,该风险将增加 48%。

妊娠期是胎儿器官快速分化的关键窗口期,母体代谢微环境的改变会直接影响胎儿发育。妊娠期肥胖可通过高血糖、胰岛素抵抗和氧化应激等多途径影响子代健康^[16]。GWG 主要包括母体组织、羊水、胎盘和胎儿四部分增重,其中胎儿体重增长占主导,GWG 过高常提示胎儿过度生长^[17]。目前认为,宫内代谢环境可引发胎儿代谢系统重编程,形成代谢紊乱的跨代延续,即“宫内编程机制”^[18],具体包括表观遗传改变(如 *PPAR γ* 基因甲基化)、代谢紊乱(如胰岛素/mTOR 通路抑制)及线粒体损伤(如活性氧积累),这些变化共同扰乱胎儿能量代谢,增加子代肥胖风险^[19]。研究显示,母亲妊娠期营养素摄入与后代摄入模式存在正相关,其中蛋白质与脂肪的代际关联在妊娠阶段更为显著,有力支持了宫内编程机制^[20]。“胎儿营养过剩假说”进一步阐释,母体营养过剩促使过多营养物质经胎盘转移,影响胎儿生长模式、脂肪沉积及下丘脑能量代谢与饮食调控系统的发育,增加肥胖易感性^[21]。此外,妊娠期肥胖还可通过表观遗传-菌群轴调控子代肠道菌群,如引起脐带血短链脂肪酸异常及胎盘代谢物转运障碍,导致新生儿肠道菌群失调(如拟杆菌门减少、厚壁菌门增加)^[22]。

3 产后肥胖与子代肥胖的关联及影响机制

产后肥胖尚无统一定义,通常指分娩后体重持续高于妊娠前水平并维持数月甚至数年。产后 1 年内未恢复妊娠前体重者被视为高危人群,且产后体重滞留易发展为长期肥胖^[23]。产后肥胖具有双重性:一方面,与妊娠前 BMI 及 GWG 密切相关,是产前肥胖状态的延续,持续影响子代发育;另一方面,产后肥胖本身也独立影响子代体重发展,此效应可持续至子代青春期^[24]。Robinson 等^[25]通过多因素回

归分析证实,母亲产后体重与子代超重风险存在剂量-效应关系,产后体重每增加 5kg,子代学龄前期超重风险增加 17%。此外,母乳喂养在产后阶段也发挥着重要作用,既能促进母亲体重恢复,又有助于子代建立健康的体重增长模式^[26]。然而肥胖女性常面临特殊喂养困境,表现出“三低”特征:喂养意愿低、启动率低、持续时间短,更易出现泌乳延迟及乳汁供应不足等问题^[27],这些因素共同加剧了母婴双方的肥胖风险。

产后阶段是生命早期发育的关键延续期,新生儿各系统快速发育,营养供给和微环境建立仍主要依赖于母体,通过母乳喂养、肌肤接触等途径形成独特的母-婴互作体系。产后母乳喂养是预防儿童肥胖的重要保护因素,动物实验显示,延长母乳喂养时间可通过刺激肝脏分泌 FGF21 蛋白,调节下丘脑能量代谢,形成抗肥胖保护机制^[28]。母乳中的生物活性成分有助于调节婴儿肠道菌群稳态和能量代谢,如双歧杆菌等益生菌被发现具有潜在的抗肥胖作用^[29]。但肥胖母亲的哺乳困难常导致过早转为配方奶喂养,这可能通过早期营养过剩增加子代肥胖风险。此外,母亲肥胖状态也会显著改变母乳成分。Isganaitis 等^[30]在肥胖和正常体重母亲的产后乳汁中鉴定出多种差异代谢物,发现母乳中腺嘌呤含量与母体 BMI 及婴儿肥胖、脂肪累积呈正相关;Sabien 等^[31]则发现,肥胖母亲乳汁中单糖和糖醇含量增加,提示母乳代谢组学可能成为预测婴儿肥胖的生物标志物。除生物学因素外,家庭环境同样重要。产后肥胖母亲往往倾向于高能量密度食物,这种饮食偏好通过家庭环境传递给子代,易形成促肥胖的家庭饮食模式^[32]。生物学机制与环境因素的交互作用,共同构成了产后肥胖影响子代健康的多维通路。

4 干预措施

母体肥胖的代际影响是从妊娠前代谢异常奠基、妊娠期胎儿编程强化到产后环境暴露巩固的连续性过程,防控策略需覆盖全周期才能有效阻断肥胖的代际传递。

建议将儿童肥胖预防关口前移至母亲妊娠前。肥胖妇女在妊娠前即使轻微减重也可显著改善妊娠结局^[33]。所有备孕女性应接受 BMI 筛查,并根据肥胖程度分层管理及个体化指导。2024 年《柳叶刀》的大型队列研究指出,肥胖孕妇($BMI \geq 30\text{kg/m}^2$)妊娠期低增重或适度减重是安全的,甚至可能对重度肥胖孕妇($BMI \geq 40\text{kg/m}^2$)有益^[34]。饮食与运

动结合是实现有效体重管理的关键。饮食方面,鼓励肥胖女性及其家庭调整膳食结构,健康的饮食模式可积极影响后代饮食习惯;妊娠期注意铁、钙、维生素 D 等关键营养素的补充;产后坚持科学饮食,保证母乳营养的同时控制高热量食物摄入。运动方面,建议妊娠期间进行适量有氧和抗阻训练,如瑜伽、游泳等,以促进新陈代谢^[35]。

目前针对母婴肥胖代际传播的防控体系在科研和实践层面仍存在不足。妊娠前阶段,临床主要依赖母亲 BMI 筛查和普适的饮食运动建议,而基于“健康与疾病的发育起源”理论、旨在改善卵母细胞代谢健康靶向策略尚在探索。动物研究表明,肥胖小鼠补充褪黑素可有效减少活性氧生成、防止减数分裂异常,从而改善母体肥胖所诱导的卵母细胞缺陷,并提高早期胚胎的发育潜力^[36],这为开发针对卵母细胞质量的靶向干预策略提供了潜在分子基础。目前,妊娠期临床管理仍以血糖监测为主,对于母体肥胖相关的慢性炎症微环境失调和表观遗传重编程异常等环节缺乏有效干预手段。但已有动物研究显示,妊娠期膳食补充甲基供体(如叶酸)可调节 DNA 甲基化来影响后代的出生体重和生长轨迹^[37],提示靶向营养干预可能通过表观遗传修饰改善后代表型,但其跨代安全性和长期效应仍需进一步研究。产后管理同样面临挑战,现有策略多集中于提倡母乳喂养,未能针对性应对肥胖母亲乳汁成分异常等具体问题。目前一项临床随机对照研究正对此进行探索,评估产后 8 周有氧运动对乳汁中 3'-唾液酸乳糖等成分的影响,假设运动可能通过提升 3'-唾液酸乳糖水平改善婴儿体重增长曲线^[38],有望为开发针对乳汁成分的非药物干预提供初步的临床依据。

针对“代谢记忆-宫内编程-环境暴露”这一连续性病理过程,未来应开发基于机制、贯穿全生命周期的整合式防控方案。包括但不限于:在孕前阶段探索以改善卵母细胞质量为核心的代谢干预靶点;在妊娠期研发能够特异性调节母体代谢与表观遗传状态的营养或药物策略;在产后制定针对肥胖母亲乳汁特性的个体化喂养支持方案。这些方向亟需更多扎实的临床前及临床研究提供证据支持与转化路径。

综上所述,儿童肥胖的防控关口应前移,甚至可提前至母亲妊娠前,构建包括妊娠前体重管理、妊娠期健康指导以及产后持续监测和支持的综合性干预策略。未来母婴肥胖健康领域的研究应聚焦于多因素整合分析和跨学科合作,进一步深化对其生理和

代谢机制的探究,揭示肥胖代际传播的发病机制和危险因素。此外,随着精准医学的发展,有望利用大数据和人工智能技术开发基于母代特征的子代肥胖及代谢性疾病风险预测模型,将极大提升早期预警与精准干预能力,从而为制定科学的公共健康政策、提升人群整体健康水平提供坚实支撑。

利益冲突 所有作者声明无利益冲突

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