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小肠脂质吸收与健康：高脂膳食挑战下的运动改善作用*

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摘要 小肠作为膳食脂质进入体内后消化吸收的“首道关卡”，在脂质代谢中扮演着枢纽角色。长期HFD导致小肠脂质超载，绒毛结构异常增生，诱发肠道脂质吸收与代谢紊乱。长期规律运动可增强肠黏膜屏障功能、降低肠道炎症、增加肠道菌群多样性并改善肠道脂代谢紊乱，调节小肠脂质吸收过程。本文综述了健康状态下的小肠脂质吸收过程、高脂膳食对小肠脂质吸收的影响与机制、长期规律运动对小肠脂质吸收的改善作用、过度运动产生的脂质吸收问题，以及运动改善小肠结构与脂质吸收功能的潜在机制，从能量代谢、肠道屏障功能、脑-肠轴、肠道菌群稳态等角度展开分析。通过关注运动对脂代谢紊乱的肠道影响与机制，为运动防治肥胖与脂代谢相关领域科学研究提供新的研究视角与思路，同时为制定科学健身与减脂方案提供重要理论参考，为推动运动健康领域的发展与实践应用提供重要支持。

关键词 脂质吸收，小肠，健康，高脂膳食，运动
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《肥胖症诊疗指南（2024年版）》指出，我国18岁及以上居民的超重和肥胖率分别为34.3%和16.4%，且呈上升趋势。超重/肥胖所带来的健康问题形势严峻[1]。面对日益严峻的健康挑战，国家卫生健康委员会（National Health Commission，NHC）于2024年提出启动为期3年的“体重管理年”专项行动，通过倡导科学饮食和积极参与运动，做到重视体重管理，构建全社会的慢病防控体系。现代生活方式中肥胖形成的两个重要原因是高脂膳食和身体活动不足。高脂膳食会导致肠道菌群代谢紊乱与脂代谢异常，加剧肥胖的进程[2]。小肠在脂质代谢中扮演重要角色：参与膳食脂质的消化、吸收、转运与重合成，小肠结构与功能的完整性直接影响着全身能量代谢平衡[3]。高脂膳食导致的机体胰岛素抵抗、肠道炎症以及菌群紊乱可进一步诱发小肠脂质吸收与代谢紊乱，促使肥胖等慢性代谢性疾病的发生[4-5]。长期高脂膳食还可导致肠道脂质超载，小肠绒毛结构异常增生、紧密连接蛋白表达下调及脂质转运功能紊乱，从而打破肠道脂质吸收与代谢平衡，促进脂质异位沉积[6-8]。运动在改善脂代谢紊乱中具有重要作用：进行规律运动可增强胰岛素敏感性，促进骨骼肌脂肪酸β氧化，同时抑制脂肪合成限速酶活性，从而有效调控脂质动态平衡，减少脂肪过度堆积[9-12]。研究发现，长期规律运动还可调节肠道菌群多样性、降低肠道炎症、增强肠黏膜屏障功能并改善肠道脂代谢紊乱，包括调节小肠脂质吸收过程、改善小肠脂质异常转运以及抑制小肠上皮细胞的脂质重合成[13-16]。然而，不同运动对小肠脂质吸收功能的具体影响及作用机制尚未完全明确。本文综述了高脂膳食与规律运动如中低强度有氧运动、高强度间歇运动、抗阻运动在小肠脂质吸收中的作用及运动改善小肠结构与脂质吸收功能的潜在机制。通过总结规律运动防治肥胖与脂代谢紊乱的肠道影响与机制，为运动与脂代谢相关领域科学研究提供新的研

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究视角与思路，同时为科学健身与减脂提供理论参考。

1　健康状态下膳食脂质的小肠吸收

人体每日摄入的膳食脂质95%被小肠吸收，其中空肠是脂质的主要吸收场所[17-18]。小肠上皮细胞刷状缘膜通过表达不同的转运蛋白，与脂质结合完成脂质的摄取与吸收[19]。其中，膳食甘油三酯（triacylglycerol，TAG）首先在肠腔内被胰脂肪酶水解成游离脂肪酸（free fatty acids，FFA）和甘油，小分子脂肪酸可通过被动扩散直接进入上皮细胞，大分子脂肪酸通过与细胞膜上的白细胞分化抗原36（cluster of differentiation 36，CD36）和脂肪酸转运蛋白4（fatty acid transport protein，FATP4）结合进入上皮细胞完成吸收[20-21]。大约80%的脂肪酸进入内质网并在30 min内迅速重新合成TAG，并包装成乳糜微粒（chylomicron，CM），经淋巴系统进入血液循环再输送至全身，还有一部分脂质可直接在上皮细胞形成细胞质脂滴（cytoplasmic lipid droplet，LCD），可用于储存能量，供上皮细胞营养和能量消耗以及维持小肠脂质吸收与代谢的动态平衡[22]。胆固醇吸收的首要步骤是与胆汁酸形成混合胶束，随后运送至肠上皮的刷状缘膜[23]，由尼曼-匹克C1样蛋白（niemann Pick C1 like 1，NPC1L1）介导胆固醇进入肠细胞后，胆固醇分子会在内质网上经乙酰辅酶A酰基转移酶2（acetyl-CoA acetyltransferase 2，ACAT2）酯化形成胆固醇酯，组装成CM通过淋巴系统进入血液循环，最终被肝脏或其他组织摄取[24-25]。膳食脂质在肠道中的分子吸收过程如图1所示。

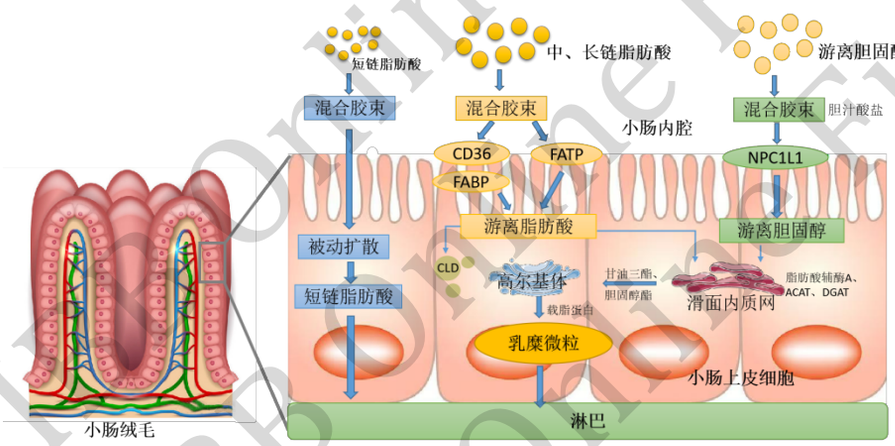

Fig. 1　Schematic diagram of molecular absorption process of dietary lipids in the intestine

图1　膳食脂质在肠道中的分子吸收过程示意图

CD36：白细胞分化抗原36（cluster of differentiation 36）；FATP：脂肪酸转运蛋白4（fatty acid transport protein）；FABP：脂肪酸结合蛋白（fatty acid-binding protein）；NPC1L1：尼曼-匹克C1样蛋白（niemann Pick C1 like 1）；CLD：细胞质脂滴（cytoplasmic lipid droplet）；ACAT：乙酰辅酶A酰基转移酶2（acetyl-CoA acetyltransferase 2）；DGAT：二酰甘油酰基转移酶（diacylglycerolacyltransferase）。

2　高脂膳食对小肠结构与脂质吸收的影响与机制

2.1　高脂膳食导致小肠结构改变与脂质过量吸收

长期高脂膳食可导致小肠脂质超载与过量吸收，造成脂质异位沉积与肥胖。首先，高脂膳食可加速小肠病理性结构重塑，引起小肠长度变长、变重、绒毛过度伸长、微绒毛变长，上皮吸收细胞数量增加，进一步扩大脂质吸收面积，引起小肠脂质超载[26-29]。肠碱性磷酸酶（intestinal alkaline phosphatase，IAP）参与脂肪酸吸收、肠道屏障保护以及肠道微生物群结构平衡[30]。有研究发现，当机体处于营养过剩或长期高脂膳食状态时，小肠绒毛适应性伸长，伴随肠道IAP过表达与脂质过量吸收[31]。绒毛蛋白1（villin-1，VIL-1）与埃兹蛋白（Ezrin）是肠上皮细胞的刷状缘微绒毛的主要组成成分。经过长期高脂膳食后，小肠微绒毛变长，VIL-1、Ezrin表达上调，形成“超吸收型”肠黏膜，进一步导致脂质吸收增加[32]。另外，长期高脂膳食导致小肠脂质摄取蛋白CD36及脂肪酸结合蛋白（fatty acid-binding protein，FABP）、胆固醇摄取蛋白NPC1L1过表达，进一步导致小肠脂质超载与过量吸收，加剧肥胖发生进程[33-34]。

2.2　高脂膳食导致脂质吸收异常的潜在机制

高脂膳食诱导的脂质吸收异常与小肠上皮细胞过度增殖有关。有研究表明，高脂膳食激活PPAR-Wnt/β-catenin信号轴，促进隐窝干细胞增殖，同步激活Notch信号通路，定向诱导干细胞分化为肠吸收细胞（非杯状细胞），造成绒毛上皮细胞亚型比例失调[35]。高脂膳食还可导致肠道炎症，通过激活核因子κB（nuclear factor-κB，NF-κB）炎症通路以及增加肿瘤坏死因子α（tumor necrosis factor-α，TNF-α）表达进一步损害肠道屏障功能，增加肠道通透性，导致大分子脂质与有害物质进入血液循环，造成脂质吸收功能紊乱与脂质累积[36-37]。炎症通路的激活与屏障损伤干预小肠上皮脂质吸收过程，通过促进脂质摄取与转运相关蛋白如固醇调节元件结合蛋白1c（sterol regulatory element binding protein 1c，SREBP-1c）、ATP结合盒转运体A1（ATP-binding cassette transporter A1，ABCA1）表达，造成脂质合成与吸收紊乱[38-40]。

高脂膳食通过改变肠道菌群结构影响小肠脂质吸收。高脂饮食诱导厚壁菌门/拟杆菌门比值升高，短链脂肪酸（short-chain fatty acids，SCFAs）相关的益生菌（如乳杆菌属、普拉梭菌）丰度降低，伴随乙酸、丁酸等SCFAs水平降低，而促炎菌（如脱硫弧菌属）比例升高[41-42]。SCFAs可通过激活AMP依赖的蛋白激酶（adenosine 5'-monophosphate（AMP）-activated protein kinase，AMPK）信号通路抑制肝脏脂肪合成并增强肠道屏障功能[43]。此外，高脂饮食增加革兰氏阴性菌比例，导致脂多糖（lipopolysaccharide，LPS）释放增多，通过TLR4/NF-κB通路激活肠道及全身炎症反应，进一步破坏脂代谢稳态[44]。肠道微生物群可控制肠道内分泌信号的传导，影响肠道细胞中脂肪酸的转运[45]。在高脂膳食诱导下的脂代谢紊乱模型中，补充益生菌后AMPK信号通路与胆固醇代谢通路激活，以调控脂肪的合成和分解[46]。在高脂饮食大鼠中使用雷公藤红素影响肠道菌群的分布后，脂质不能正常通过转运蛋白被小肠绒毛吸收，从而验证了肠道菌群在脂质吸收中发挥的重要作用[47]。高脂膳食对小肠结构与脂质吸收的影响与机制如图2所示。

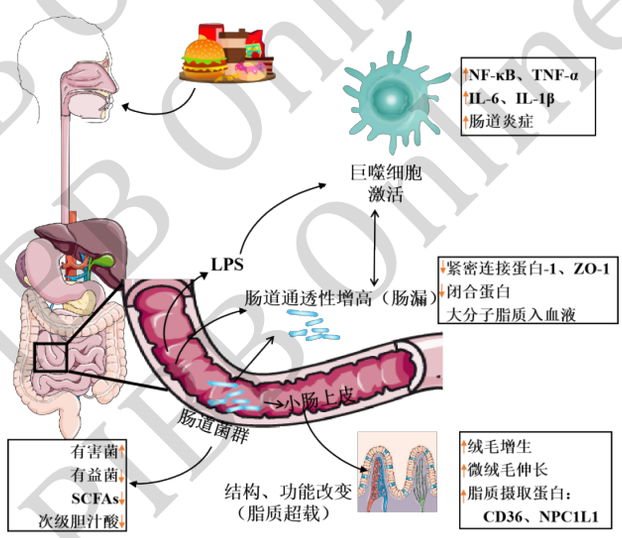

Fig. 2　The effect and mechanism of high-fat diet on small intestine structure and lipid absorption

图2　高脂膳食对小肠结构与脂质吸收的影响与机制

LPS：脂多糖（lipopolysaccharide）；NF-κB：核因子κB（nuclear factor-κB）；TNF-α：肿瘤坏死因子α（tumor necrosis factor-α）；IL-6：白介素-6（interleukin-6）；IL-1β：白介素-1β（interleukin-1β）；ZO-1：闭锁小带蛋白1（Zonula occludens protein 1）；SCFAs：短链脂肪酸（Short-chain fatty acids）；CD36：白细胞分化抗原36（cluster of differentiation 36）；NPC1L1：尼曼-匹克C1样蛋白（niemann Pick C1 like 1）。

3　运动对小肠脂质吸收的影响

运动在改善小肠脂代谢紊乱中有重要作用。长期规律运动可改善由高脂膳食造成的小肠脂质吸收功能紊乱[48-49]。不同运动形式、运动负荷对小肠脂质吸收的影响不同。进行适宜运动有助于肠道屏障的修复、肠道微环境的改善，以及对抗脂质吸收功能紊乱。目前，运动对小肠脂质吸收相关研究不

足，且观点不一。中低强度有氧运动改善小肠结构与脂质吸收功能，促进机体脂代谢与健康；抗阻运动与高强度间歇运动可改善肠道屏障与功能，对小肠脂质吸收影响的研究相对缺乏；剧烈运动、过度运动则引起肠道不适，导致肠道结构破坏以及相关症状出现，影响脂质正常吸收功能[50]。不同运动对小肠结构与脂质吸收功能的影响如图3所示。

3.1 中低强度有氧运动对小肠脂质吸收的影响

长期中低强度的有氧运动，如步行、慢跑和骑行等，可优化小肠脂质摄取与转运[51]。载脂蛋白B48（apolipoprotein B48，Apo B48）是乳糜微粒形成的核心组分，在膳食脂质的运输和代谢过程中至关重要。研究发现，经过长期中低强度运动可降低降低餐后血脂、空腹Apo B48浓度，抑制高脂诱发的小肠脂质吸收功能紊乱[10]。高脂膳食大鼠经过长期中等强度持续运动（moderate-intensity continuous training，MICT）后，小肠屏障功能增强，绒毛变短，脂质吸收表面积降低，脂肪吸收过量得到抑制，体重降低[52-55]。也有观点认为长期有氧运动使小肠绒毛变长，原因可能是运动改善肠道血流和营养供应，促进绒毛的生长和修复[56]。有氧运动对小肠绒毛表面积影响的研究目前尚无统一定论，可能与研究对象、运动形式、运动强度、测量方式不同有关。CD36与FATP在小肠脂肪酸跨膜转运与能量代谢中发挥重要作用[57]。有氧运动可通过激活AMPK信号促进CD36表达，提升脂质氧化利用效率[58]。另外，长期有氧运动可降低空肠ATP结合盒转运体G5/8（adenosine triphosphate-binding cassette transporter G5/8，ABCG5/8）、NPC1L1表达，增加ABCA1基因表达增加，维持肠道胆固醇和胆汁酸的稳态[14，59]。有氧运动可通过优化小肠结构与脂质吸收功能，维持小肠脂质吸收功能稳态，抑制脂质过量吸收，进一步维持脂质摄取与利用的动态平衡。

3.2 高强度间歇运动对小肠脂质吸收的影响

高强度间歇运动（high-intensity interval training，HIIT）因其独特的强度变化策略，可有效激发机体代谢潜能，在减肥领域得到广泛应用，尤其在减少内脏脂肪堆积方面成效显著[60]。过往研究表明，HIIT不仅能够调节肠道菌群的平衡，维持肠道生态稳定，还不会对肠道的正常结构造成损伤[61]。从脂肪吸收角度，HIIT对小肠脂质吸收功能的影响尚未有明确的研究结论。但有研究指出，运动可促进胃肠蠕动，减少脂肪在肠道内的停留时间，从而加快脂质代谢并减少体内脂质堆积[62]。小肠转运时间会影响脂质和餐后热量吸收。高脂膳食会增加能量密度，增加肠道转运时间，而运动可刺激肠道蠕动，使小肠转运功能增强[63]。有研究表明[64]，运动强度与食物在小肠转运时间成反比，较高强度的运动加速小肠排空，减少脂质在肠腔停留时间，加快脂质代谢，减少脂质在体内堆积。高强度间歇运动还可诱导脂肪酸合成酶乳酰化，从而抑制脂质合成，维持细胞内脂肪酸代谢平衡[65]。这种有益变化可进一步调控小肠脂质摄取与合成，影响脂质吸收。

3.3 抗阻运动对小肠脂质吸收的影响

抗阻运动可提高骨骼肌对游离脂肪酸的利用率，同时对脂质代谢产生积极影响。研究发现，持续8周的抗阻训练可使非酒精性脂肪肝患者的肝脏脂质含量下降，并改善血糖水平和胰岛素敏感性[66]。目前，有大量研究对抗阻运动改善脂代谢紊乱的效果进行研究[67-69]，但抗阻运动影响肠道脂质吸收的研究相对缺乏，且研究手段较为单一，从已有研究来看，进行抗阻运动可有效降低餐后血脂；在餐前进行1 h的抗阻运动可明显降低糖尿病前期男性的餐后血脂水平以及CM水平[70]，提示抗阻训练后肠道脂质吸收可能受到了抑制。肠道屏障功能与通透性的改变直接影响脂质吸收效率。有研究表明，进行长期抗阻运动可改善肠道通透性，修复肠道屏障功能[71-72]；但也有研究显示，重复4组10次70%单次重复最大负荷（one-repetition maximum，1RM）负荷的抗阻运动会增加受过阻力训练成年人的胃肠道症状，如恶心、呕吐与胃肠不适，使肠道损伤标志物——肠脂肪酸结合蛋白（intestinal fatty acid binding protein，I-FABP）水平升高、肠道通透性指标——乳果糖-鼠李糖（L/R）比值升高[73]。提示，一次较高强度的抗阻训练可能导致肠道屏障暂时性受损与通透性增加，但长期抗阻训练使肠道产生适应性变化，增强肠道屏障并对肠道健康产生益处。

3.4 过度运动对小肠脂质吸收的影响

适量运动对小肠结构产生有益改变，但过度运动、超负荷运动则会破坏肠道结构，影响小肠正常吸收功能。肠道是对运动反应较为敏感的器官，不同运动强度与运动方式对小肠结构影响不同。运动与肠道健康和免疫呈现“J曲线”的影响模式，即适度运动对肠道通透性和炎症有积极改善作用，而高强度持续运动或过度运动则对肠道免疫与正常功

能产生有害影响[74]。研究发现，一次高强度运动后，小肠出现短暂功能障碍，肠道通透性增加，小肠绒毛间距出现变化，脂质吸收功能受到影响[75]。还有研究报道，长时间高强度运动后，小鼠小肠绒毛出现萎缩、变短等现象，可能是由于过度运动引发的氧化应激和炎症反应，损伤小肠绒毛的上皮细胞，破坏绒毛正常结构，从而影响脂质等营养素的吸收效率[76]。一项人体研究发现，跑步者在高强度运动后血液I-FABP水平增加，肠道通透性发生改变[77]。过度运动破坏肠道菌群结构，增加炎症风险，从而进一步对脂质吸收产生负面影响。过度训练还会导致机体免疫力下降、能量代谢紊乱及肠道通透性增加，这些影响会导致脂质吸收功能异常，进一步危害机体健康[78]。

3.5　竞技运动员的小肠脂质吸收

竞技运动员的小肠与普通人不同，其小肠绒毛长度较短，且更易出现肠道健康问题与营养吸收不良[79-80]。乳糜泻以小肠绒毛缩短，小肠脂质吸收障碍为主要特征。运动员患乳糜泻的概率高于普通人，营养吸收功能较差[81]。耐力性项目的运动员胃肠道问题发生率较高，尤其是超耐力运动项目，如马拉松、铁人三项等[82]。研究发现，超级马拉松运动员和铁人三项运动员进行高强度持久运动后促炎因子、肌肉及肠道细胞损伤水平的标志物水平升高[83]。另外，在高强度训练期间，耐力运动员患胃肠道疾病的几率较高，对整体竞技运动表现与健康状况产生不良影响[84]。进行高强度对抗性运动，如橄榄球比赛后，运动员肠通透性增加[85]。运动员小肠脂质吸收与营养问题直接影响着运动员的训练效果与运动后恢复，因此一直备受关注。有研究针对不同项目运动员在比赛前进行肠胃适应性训练，逐渐增加碳水化合物摄入量以改善肠道耐受性[86]。在赛前赛后补充植物乳杆菌（*Lactobacillus plantarum*）等益生菌或益生元可帮助运动员修复肠道健康，提升竞技运动表现[84]。

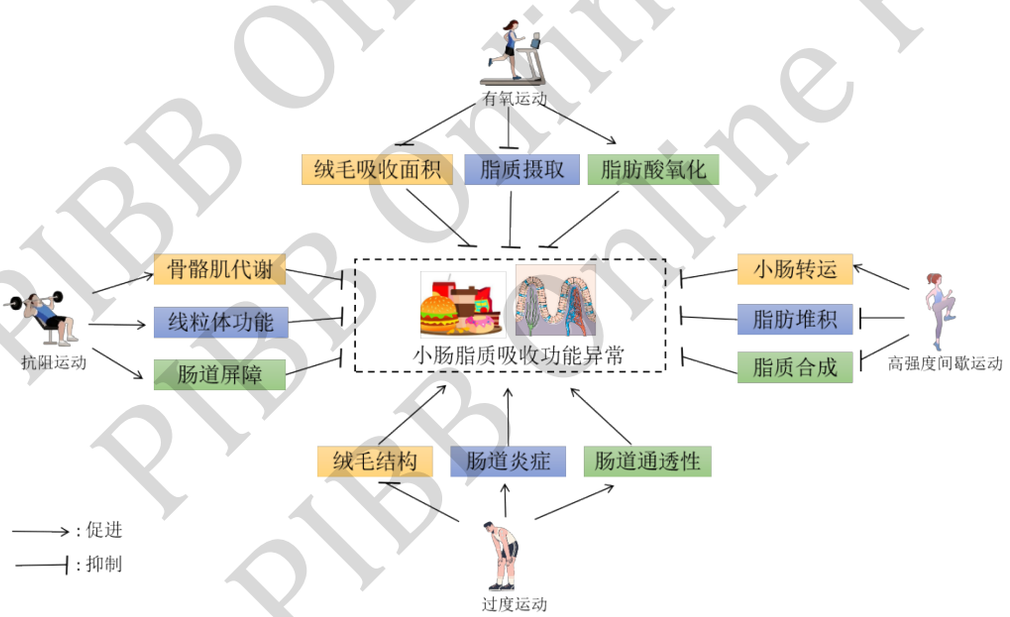

Fig. 3　The effects of different exercises on the structure and lipid absorption function of the small intestine

图3　不同运动对小肠结构与脂质吸收功能的影响

4　运动改善小肠脂质吸收的潜在机制

4.1　运动通过调控骨骼肌能量代谢影响脂质吸收

长期适宜运动可优化骨骼肌能量代谢。研究表明，运动能够激活骨骼肌AMPK/mTORC1信号通路，促进脂质在线粒体中的氧化途径激活，并增强葡萄糖转运能力，这种变化有助于降低肠道脂质堆积，改善肠道微环境[87]。运动还可提升脂肪氧化酶活性，增强脂肪酸线粒体转运限速酶肉碱棕榈酰转移酶1（carnitine palmitoyltransferase1，CPT1）的活性，从而提高β氧化速率，实现脂质的高效分解与消耗[88-89]。PPARs作为核受体转录因子，在脂

质代谢中发挥关键作用。研究发现，有氧运动通过激活PPARα推动脂肪酸β-氧化，增加能量消耗，同时激活PPARγ调节脂肪细胞分化与脂质储存[90]。运动产生的代谢效益还体现在身体成分的改变上。运动通过增加骨骼肌质量提高基础代谢水平，增加静息状态下的能量消耗，这种代谢适应机制与运动过程中的脂质动员形成协同效应，共同促进小肠脂质代谢过程[91]。

4.2 运动参与小肠结构重塑过程

规律运动参与了小肠上皮细胞重塑与再生调控，进一步发挥对脂质吸收功能的调节作用[92-93]。运动介导的鸢尾素（Irisin）可增强肠道类器官的损伤修复能力，在上皮损伤中发挥细胞与线粒体保护功能，减少肠上皮细胞凋亡，该效应与Irisin对Wnt/β -catenin与黏着斑激酶（focal adhesion kinase，FAK）信号通路激活作用有关[94-95]。有氧运动可通过调节细胞凋亡相关蛋白，抑制促凋亡因子如Bax、Caspase家族活性，激活抗凋亡蛋白Bcl-2表达，维持肠道上皮细胞增殖与凋亡的动态平衡[96]。高强度间歇运动通过抑制NF-κB和Notch信号通路转导，纠正高脂膳食诱导的巨噬细胞表型极化异常，并抑制脂肪组织过度增殖[97]。现有研究表明，运动对小肠结构重塑过程提供潜在价值。据此推测，进行适宜运动对小肠绒毛结构与脂质吸收产生的调节效果，极有可能通过细胞增殖与分化相关通路发挥作用，从而部分抵消高脂膳食带来的小肠结构与脂质吸收功能失调的危害。

4.3 运动改善肠道屏障功能

适宜运动通过增加肠道紧密连接蛋白表达，维持肠道屏障完整性，减少脂质过度吸收[98]。肠道屏障是防止有害物质和病原体侵入体内的重要防线，同时也对脂质吸收具有调节作用。紧密连接蛋白是构成肠道上皮细胞紧密连接的重要组成部分，可调节肠道通透性[99]。研究发现，为期12周的中等强度有氧运动可增加肠道上皮细胞中闭合蛋白（occludin）和紧密连接蛋白1（claudin-1）表达，增强肠道屏障功能，防止脂质颗粒与细菌内毒素进入血液循环，降低“肠漏”风险，有助于避免脂质在体内的过度积累，从而改善脂质代谢[100]。运动通过激活SESN2/AMPKα1/HIF-1α通路，保护肠上皮免受高脂膳食损伤，并介导MAPK信号通路调控紧密连接蛋白的转录翻译，进一步增强屏障功能[101-102]。

4.4 运动降低肠道炎症

运动通过调节抗炎机制、改善炎症因子表达对肠道慢性炎症及相关代谢疾病产生改善的作用[103]。进行规律有氧运动可抑制肠道NF-κB炎症信号转导降低促炎因子白细胞介素-6（interleukin-6，IL-6）、TNF-α的释放，减轻肠道炎症反应[104]。肠道慢性炎症状态会干扰肠道正常的生理功能，影响肠道与全身脂质代谢过程[105]。本研究团队前期实验证明，12周MICT和HIIT抑制肠道NF-κB信号通路，改善高脂膳食大鼠肠道炎症状态，减少高脂膳食大鼠脂质沉积[106]。运动可诱导骨骼肌分泌抗炎因子IL-10，发挥机体抗炎与代谢调节作用[107]，通过降低肠道炎症并与脂代谢相互作用，缓解高脂与肥胖带来的脂毒性危害[108]。炎症降低可减轻高脂膳食诱发的肠道屏障损伤，改善机体脂代谢紊乱[109]。另外，运动通过降低肠道炎症，影响脂质吸收相关蛋白的表达，调控小肠脂质转运与吸收[110]。

4.5 运动调控脑–肠轴神经信号通路影响脂质吸收

运动通过脑-肠轴之间的各种信号交流在调节肠道脂代谢中发挥重要作用。中枢神经系统、自主神经系统下丘脑-垂体-肾上腺轴介导的神经内分泌信号网络参与肠道脂代谢调控[111]。本团队前期研究认为，中枢神经系统产生的代谢性炎症可通过脑干摄食中枢影响全身代谢[112]。脑干-迷走神经-小肠信号通路对炎症反应与脂质吸收具有关键调控作用。迷走神经参与胃肠道的蠕动、消化液分泌和营养物质吸收。2024年发表于《*Nature*》的研究揭示了脑干延髓的迷走神经背核（dorsal nucleus of vagus nerve，DMV）通过γ-氨基丁酸（gamma-aminobutyric acid，GABA）能神经元靶向调节小肠绒毛长度与脂质吸收，该项研究为中枢系统在小肠脂质吸收中的作用提供有力支撑[113]。交感神经则主要在应激和运动状态下发挥作用，调节胃肠道的血流分布和代谢活动。运动时，交感神经兴奋，并释放去甲肾上腺素等神经递质，作用于肠道平滑肌细胞和内分泌细胞，调节肠道的蠕动和激素分泌[114]。在应激状态下，下丘脑-垂体-肾上腺轴激活皮质醇含量增加进一步影响肠道神经元的功能[115]。

运动还可通过诱导神经内分泌肽和肌源因子释放调节脂质代谢稳态。研究表明，进行长期规律运动可促进神经肽Y（neuropeptide Y，NPY）、肽YY（Peptide YY，PYY）、胃饥饿素（ghrelin）、胰

高血糖素样肽1（glucagon-like peptide-1，GLP-1）、5-羟基色氨酸（5-hydroxytryptophan，5-HT）、脑源性神经营养因子（brain-derived neurotrophic factor，BDNF）等分泌，进一步调控食欲与脂质代谢稳态[116-117]。有研究发现，相较于有氧运动，为期12周的抗阻运动抑制超重男性血清饥饿素（ghrelin）水平效果更优，可能与运动强度与儿茶酚胺相关机制有关[118]。BDNF在小肠中不仅参与神经系统的调控，还广泛影响肠道上皮屏障、免疫反应、代谢平衡及肠-脑轴通信。运动后肠道BDNF表达增加，可能通过影响下游PI3K/Akt与NF-κB信号通路抑制高脂膳食与炎症反应介导的上皮细胞过度增殖[119-121]。这些神经内分泌调节机制协同作用，可优化肠道脂质吸收与代谢。

4.6　运动通过改善肠道菌群稳态影响脂质吸收

运动能够通过改善肠道菌群稳态和调节代谢产物，影响脂质吸收和代谢健康。长期规律运动通过促进有益菌（如拟杆菌属、瘤胃球菌属）的生长，增加SCFAs的生成[122-123]。SCFAs不仅为肠上皮细胞提供能量，还通过抑制胆固醇7α-羟化酶（cholesterol 7-alpha hydroxylase，CYP7A1）的活性，减少胆汁酸的合成，促进胆汁酸的重吸收，从而影响脂质的消化和吸收过程[124]。研究发现，肠道菌群代谢物芳香族氨基酸（包括色氨酸、苯丙氨酸和酪氨酸）能够作用于肠道，调节肠道免疫，降低慢性炎症和脂肪吸收，达到预防肥胖的效果[125]。运动可改变肠道微生物群的组成，包括增加有益菌（如拟杆菌属、黄杆菌属、厚壁菌门罗氏菌属和阿克曼菌）的数量，减少有害菌（如普拉粪杆菌属）的数量，从而改善肠道代谢稳态与健康[126-127]。运动还通过调节肠道微生物群影响胆汁酸代谢，增加胆汁酸受体的表达，增加次级胆汁酸生成，进一步影响脂质代谢[128]。空肠微生物群变化通过延缓肠道脂肪吸收阻止高脂肪饮食诱导的小鼠肥胖。某些肠道微生物可以激活PPARγ等转录因子，促进脂肪细胞的分化和脂质的储存。另外，运动能通过调节肠道微生物群影响胰岛素信号通路，从而改善机体脂质代谢[129]。

总之，运动可能通过调控小肠上皮细胞增殖、改善肠道炎症与屏障功能、介导脑-肠轴与肠道菌群及其代谢产物等多方面机制发挥作用，抑制小肠结构紊乱与脂质吸收功能失调，部分抵消高脂带来的肠道健康危害。运动改善小肠脂质吸收的潜在机制如图4所示。

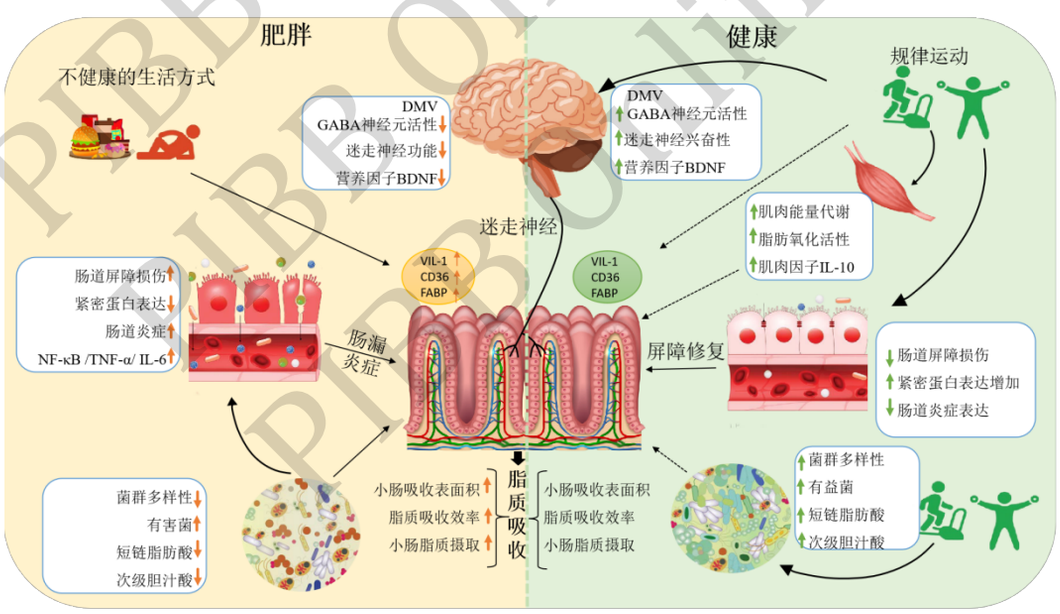

Fig. 4　Potential mechanism by which exercise improves small intestine lipid absorption

图4　运动改善小肠脂质吸收的潜在机制示意图

DMV：迷走神经背核（dorsal motor nucleus of vagus）；GABA：γ-氨基丁酸（gamma-aminobutyric acid）；BDNF：脑源性神经营养因子（brain-derived neurotrophic factor）；NF-κB：核因子κB（nuclear factor-κB）；TNF-α：肿瘤坏死因子α（tumor necrosis factor-α）；IL-6：白介素-6（interleukin-6）；IL-10：白介素-10（interleukin-10）；VIL-1：绒毛蛋白1（villin-1）；CD36：白细胞分化抗原36（cluster of differentiation 36）；NPC1L1：尼曼-匹克C1样蛋白（Niemann Pick C1 like 1）。

5 总结与展望

长期高脂膳食可加速小肠病理性结构重塑、屏障损伤与脂质吸收功能紊乱进程，最终导致小肠膳食脂质过量，加速脂质积累与肥胖发生。而运动可通过抑制上皮细胞凋亡、保护黏膜屏障、降低肠道炎症、改善菌群紊乱与调节小肠脂质吸收来部分抵消高脂带来的危害。不同运动对脂质吸收调节效果不同，中低强度的有氧运动、抗阻运动、高强度间歇运动可改善肠道微环境，促进机体脂质代谢与健康；而剧烈运动、过度运动则引起肠道不适，导致肠道结构破坏以及相关症状出现，影响脂质吸收的正常功能。然而，现阶段关于运动与小肠脂质吸收的研究仍存在诸多局限性与不足：a. 不同运动对小肠脂质吸收相关研究不足，没有直接证据证明不同运动形式对小肠结构、脂质吸收表面积是否有抑制效果；b. 运动与肠道脂质吸收相关研究多为动物研究，人体实验研究较少；c. 人体研究测试手段有限，测试指标多为血液指标，体现肠道功能指标有限；d. 运动改善小肠脂质吸收功能的影响研究不够系统、结果不一致，相关调控机制尚不明晰。

未来研究可关注以下方向：a. 揭示不同运动方式、运动负荷、不同进食与运动时间段动态变化对人体脂质吸收的影响，确定适宜运动强度范围、阈值和形式以保证运动诱导的肠道脂代谢调节有益；b. 考虑利用多种新型技术如胶囊内窥镜、核磁共振技术、同位素标记、营养吸收动态监测、代谢与转录组学等对人体小肠绒毛结构、脂质吸收过程进行监测并展开研究；c. 通过研究运动对脂质吸收的影响，确定小肠脂质吸收相关标志物，为机体脂质吸收、代谢与肥胖指标监测提供有利参考；d. 对运动改善小肠脂质吸收相关机制进行探索，关注肠道菌群、神经内分泌系统与肠道免疫的影响与互作网络，为运动与脂代谢调控、科学减重提供理论与实践参考。

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Small Intestine Lipid Absorption and Health: The Improvement Effect of Exercise Under The Challenge of High-fat Diet*

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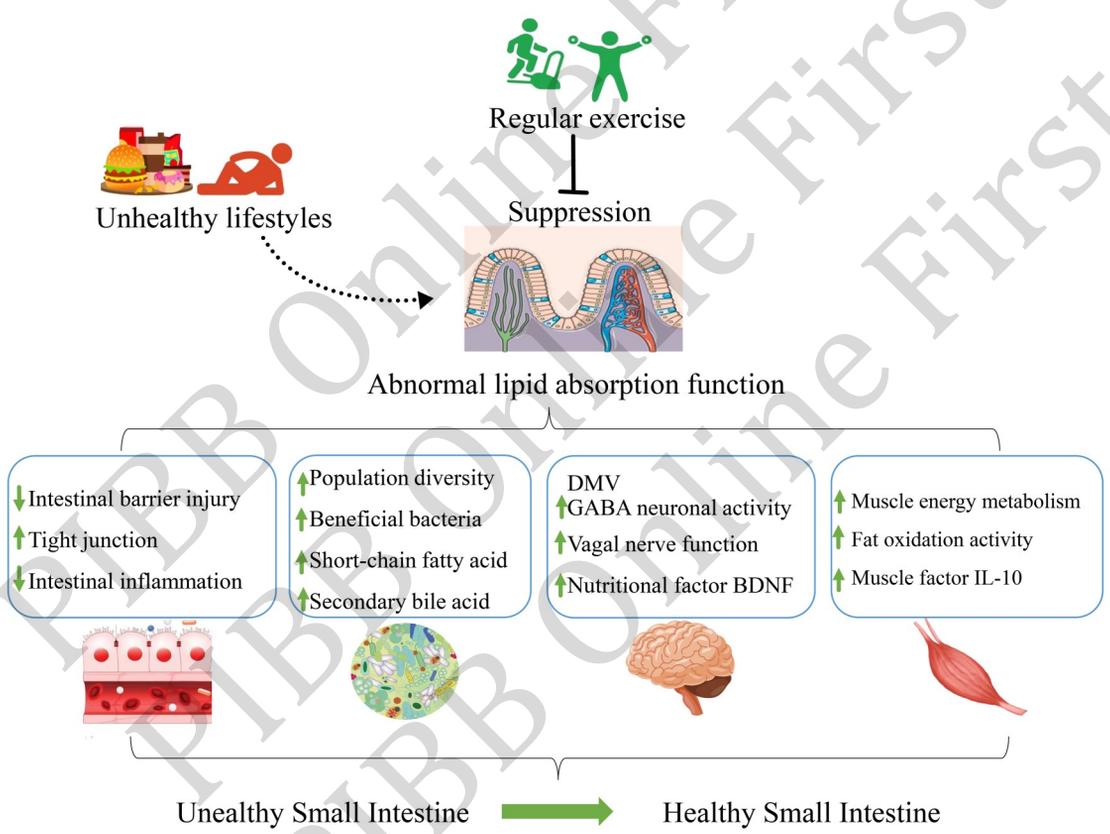
Graphical abstract



Abstract　The two core causes of obesity in modern lifestyle are high-fat diet (HFD) and insufficient physical activity. HFD can lead to disruption of gut microbiota and abnormal lipid metabolism, further exacerbating the process of obesity. The small intestine, as the "first checkpoint" for the digestion and absorption of dietary lipids into the body, plays a pivotal role in lipid metabolism. The small intestine is involved in the digestion, absorption, transport, and synthesis of dietary lipids. The absorption of lipids in the small intestine is a crucial step, as overactive absorption leads to a large amount of lipids entering the bloodstream, which affects the occurrence of obesity. HFD can lead to insulin resistance, disruption of gut microbiota, and inflammatory response in the body, which can further induce lipid absorption and metabolism disorders in the small intestine, thereby promoting the occurrence of chronic metabolic diseases such as obesity. Long term HFD can accelerate pathological structural remodeling and lipid absorption dysfunction of the small intestine: after high-fat diet, the small intestine becomes

longer and heavier, with excessive villi elongation and microvilli elongation, thereby increasing the surface area of lipid absorption and causing lipid overload in the small intestine. In addition, overexpression of small intestine uptake transporters, intestinal mucosal damage induced "intestinal leakage", dysbiosis of intestinal microbiota, ultimately leading to abnormal lipid absorption and chronic inflammation, accelerating lipid accumulation and obesity. Exercise, as one of the important means of simple, economical, and effective proactive health interventions, has always been highly regarded for its role in improving lipid metabolism homeostasis. The effect of exercise on small intestine lipid absorption shows a dose-dependent effect. Moderate to low-intensity aerobic exercise can improve the intestinal microenvironment, regulate the structure and lipid absorption function of the small intestine, promote lipid metabolism and health, while vigorous exercise, excessive exercise, and long-term high-intensity training can cause intestinal discomfort, leading to the destruction of intestinal structure and related symptoms, affecting lipid absorption. Long term regular exercise can regulate the diversity of intestinal microbiota, inhibit inflammatory signal transduction such as NF-κB, enhance intestinal mucosal barrier function, and improve intestinal lipid metabolism disorders, further enhancing the process of small intestinal lipid absorption. Exercise also participates in the remodeling process of small intestinal epithelial cells, regulating epithelial structural homeostasis by activating cell proliferation related pathways such as Wnt/β-catenin. Exercise can regulate the expression of lipid transport proteins CD36, FATP, and NPC1L1, and regulate the function of small intestine lipid absorption. However, the research on the effects of long-term exercise on small intestine structure, villus structure, absorption surface area, and lipid absorption related proteins is not systematic enough, the results are inconsistent, and the relevant mechanisms are not clear. In the future, experimental research can be conducted on the dose-response relationship of different intensities and forms of exercise, exploring the mechanisms of exercise improving small intestine lipid absorption and providing theoretical reference for scientific weight loss. It should be noted that the intestine is an organ that is sensitive to exercise response. How to determine the appropriate range, threshold, and form of exercise intensity to ensure beneficial regulation of intestinal lipid metabolism induced by exercise should become an important research direction in the future.

Key words lipid absorption, small intestine, health, high-fat diet, exercise
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