中国科学: 生命科学 2024 年 第 54 卷 第 12 期: 2427 ~ 2437
SCIENTIA SINICA Vitae lifecn.scichina.com
《中国科学》杂志社 SCIENCE CHINA PRESS

评 述

乳酸菌调控宿主脂肪代谢的研究进展

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收稿日期: 2023-10-09; 接受日期: 2024-01-29; 网络版发表日期: 2024-05-10

摘要 脂肪代谢紊乱是代谢综合征的主要诱发因素之一. 在脂肪代谢紊乱的动物模型或临床肥胖人群中, 肠道微生物, 尤其是乳酸菌属的多样性和组成发生显著性变化. 乳酸菌作为一种常见的益生菌, 也被广泛报道能够影响到宿主脂肪代谢. 基于此, 本文探讨乳酸菌与脂肪代谢的关系, 分析其代谢产物短链脂肪酸、胆汁酸、胞外多糖和亚油酸调控脂肪代谢的潜在分子机制, 并阐述同属乳酸菌和不同复合乳酸菌之间的作用差异, 以期明确乳酸菌调控宿主脂肪代谢的机制, 旨在为食源性途径干预脂肪代谢紊乱所引起的代谢性疾病研究提供思路和借鉴.

关键词 乳酸菌, 脂肪代谢, 代谢产物

哺乳动物胃肠道内定植大量益生菌[1], 其广泛参与肠道内营养物质的消化和吸收, 对宿主生理代谢起到重要的调节作用[2]. 肠道益生菌群主要包括乳酸菌、芽孢杆菌、双歧杆菌和消化链球菌等, 其中健康成年人肠道中乳酸菌数量通常在10^6~10^8(CFU/mL)[3]. 乳酸菌在胃肠道内分布广泛, 目前已经鉴定出200多种, 较为常见的有植物乳杆菌、清酒乳杆菌、鼠李糖乳杆菌、嗜酸乳杆菌、短乳杆菌、约氏乳杆菌和德氏乳杆菌等[4]. 研究表明, 脂肪代谢紊乱伴随着肠道乳酸菌群失衡, 而通过补充乳酸菌可以改善脂肪代谢状态[5~8], 提示肠道乳酸菌在维持宿主脂肪代谢稳态中意义重大. 乳酸菌调控宿主脂肪代谢的作用主要包括胆汁酸、短链脂肪酸、乳酸和胞外多糖等代谢途径. 此外, 乳酸菌能够参与宿主分解自身不能消化的乳糖、纤维、大分子蛋白和部分脂肪, 其代谢产物也能作为信号分子参与宿主脂肪代谢[9]. 因此, 本文从乳酸菌调控宿主脂肪代谢的角度, 阐述乳酸菌与宿主脂肪代谢之间的关系, 总结乳酸菌调控宿主脂肪代谢的分子机制, 以期为乳酸菌在实践中科学应用提供参考.

1 乳酸菌和脂肪代谢

肠道菌群变化是宿主脂肪代谢紊乱的重要标志, 大量的研究已经证实并阐明乳酸菌与代谢疾病之间的初步关系. Crovesy等人[10]在高脂日粮诱导肥胖(high-fat diet, HFD)小鼠模型中发现肠道乳酸菌相对丰度显著降低, 其中植物乳杆菌和副干酪乳杆菌与肥胖呈负相关. 而Million等人[11]在肥胖患者粪便中发现, 肠道加氏乳杆菌和罗伊氏乳杆菌与肥胖指数呈正相关, 补充加氏乳杆菌和罗伊氏乳杆菌DMC2016能够加速肥胖[12~14]. 但也有研究报道罗伊氏乳杆菌J1能够抑制脂肪合成[15], 而植物乳杆菌、罗伊氏乳杆菌和发酵乳杆

引用格式: 陈忠, 马杰, 夏嗣廷, 等. 乳酸菌调控宿主脂肪代谢的研究进展. 中国科学: 生命科学, 2024, 54: 2427–2437
Chen Z, Ma J, Xia S T, et al. Advances in the regulation of host fat metabolism by Lactobacillus (in Chinese). Sci Sin Vitae, 2024, 54: 2427–2437, doi: 10.1360/SSV-2023-0232

菌等复合乳酸菌能激活AMPK信号通路，减少脂肪合成[16]，提示乳酸菌在宿主脂肪代谢紊乱中的作用存在种属差异. 综上可知，脂肪代谢紊乱会影响宿主肠道乳酸菌的组成，而补充乳酸菌也能够调控宿主脂肪代谢，其作用与菌属密切相关. 目前大量的研究聚焦于减少脂肪沉积的乳酸菌株的筛选，表1综述不同乳酸菌改善宿主肥胖的效果，以期为探讨乳酸菌在临床和动物模型上改善脂肪代谢提供预防和治疗参考.

表 1 乳酸菌改善宿主肥胖的作用

Table 1 Effect of *Lactobacillus* on ameliorating host obesity

菌株	研究对象	结果	参考文献
加氏乳杆菌持续12周	超重和肥胖人群	改善体重、内脏脂肪堆积和腰围	[17]
副干酪乳杆菌F19或亚麻籽粘液持续6周	绝经后的肥胖妇女	调控微生物菌群, 改善胰岛素敏感性	[18]
益生菌酸奶持续12周	超重或肥胖人群	改善胰岛素抵抗, 总胆固醇和低密度脂蛋白水平显著降低	[19]
鼠李糖乳杆菌DSMZ21690, 嗜酸乳杆菌ATCCB3208, 双歧杆菌ATCCSD6576, 乳双岐杆菌DSMZ32269混合菌株持续12周	肥胖儿童和成人	改善非酒精性脂肪肝, 总胆固醇、低密度脂蛋白和腰围水平显著降低	[20]
乳双歧杆菌和乳酸乳球菌CECT8145持续12周	腹部肥胖人群	减少BMI、腰围和腰高比	[21]
罗伊氏乳杆菌263持续8周	HFD小鼠	提高耗氧量、抗氧化和葡萄糖以及脂质代谢相关基因	[22]
嗜酸乳杆菌、乳酸芽孢杆菌、副干酪乳杆菌和鼠李糖乳杆菌的混合菌株	HFD小鼠	缓解体重的增加和空腹血糖、胰岛素、甘油三酯、炎症标志物、瘦素和趋化素的水平	[23]
鼠李糖乳杆菌LRa05持续8周	高脂饮食的C57BL/6J雄性小鼠	改善体重、血脂和肝细胞脂质, 促进肝脏碳水化合物和能量代谢的水平, 并抑制血糖和肝脏葡萄糖的含量	[24]
发酵乳杆菌LF-CQPC05	高脂饮食小鼠	附睾脂, 肾周脂, ALT, AST, 总胆固醇, 甘油三酯, LDL-C, PPAR-γ和C/EBP-α的水平显著降低, 并提高肝脏和附睾脂中脂蛋白脂肪酶, PPAR-α, CYP7A1和CPT1AmRNA表达水平.	[25]
鼠李糖乳杆菌	高脂肪/高糖饮食加上间歇性缺氧暴露小鼠	缓解脂肪含量、能量消耗不平衡、葡萄糖不耐受、胰岛素抵抗、肝脏脂肪变性和肝损伤以及显著降低脂肪炎症和脂肪细胞的大小, 并增加肝脏FGF21 mRNA表达和循环FGF21蛋白水平	[26]
植物乳杆菌LP3持续8周	SD小鼠	总胆固醇、甘油三酯和LDL-C水平显著降低, 调节拟杆菌门和厚壁菌门的比例、脂肪酸、类固醇和胆汁酸的合成以及亚油酸、亚麻酸和花生四烯酸的代谢	[27,28]
干酪乳杆菌YBJ02持续6周	高脂饮食小鼠	胆固醇、甘油三酯和低密度脂蛋白显著降低. 并抑制PPARγ, CEBP/α和SREBP-1c的表达	[29]
植物乳杆菌FRT10持续8周	高脂饮食小鼠	体重、脂肪重量、肝脏三酰甘油和丙氨酸转氨酶浓度显著降低. 乳酸菌、双歧杆菌和阿克曼氏菌丰度显著增加.	[30]
戊糖乳杆菌GSSK2和植物乳杆菌GS26A持续12周	高脂饮食小鼠	改善糖耐受、氧化应激、脂肪、结肠、和肝脏结构等基因靶点	[31]
清酒乳杆菌ADM14持续10周	高脂饮食小鼠	显著降低附睾脂、总胆固醇和葡萄糖的水平以及附睾脂肪垫相关基因的表达.	[32]
植物乳杆菌KC28持续12周	高脂饮食的C57BL/6J雄性小鼠	肝脏中PGC1-α和CPT1-α上调, 脂肪组织中ACOX-1, PPAR-γ和FAS下调	[33]
罗伊氏乳杆菌HI120	高脂饮食的C57BL/6肥胖小鼠	血清胆固醇水平随着NPC1L1, SREBP-2和HMG-CR表达的降低而降低, 并且HI120介导的亚油酸(LA)转化为共轭亚油酸(CLA)	[34]
干酪乳乳杆菌、鼠李糖乳杆菌、保利加亚乳杆菌	肥胖和术后人群	改善维生素D的水平、炎症因子、血脂和血糖指数	[35]

2　乳酸菌对脂肪代谢的调控机制

脂肪代谢紊乱是引起代谢综合征的重要原因之一，通过食源途径调节宿主脂肪代谢成为近年来的研究热点. 乳酸菌能够有效调节宿主脂肪代谢和相关代谢综合征，其作用机制与乳酸菌代谢产物密切相关. 因此，本文进一步探究乳酸菌代谢产物短链脂肪酸、胆汁酸、胞外多糖和亚油酸等调控宿主脂肪代谢的潜在作用和机制(图1和图2).

2.1　短链脂肪酸

短链脂肪酸(Short chain fatty acids, SCFAs)由肠道菌群发酵的多糖和一些宿主难以消化和吸收的碳水化合物产生. 超过95%的SCFAs被肠道快速吸收，转运至肝脏内转化为乙酰辅酶A和丙酰辅酶A，进而参与胆固醇、脂肪酸和糖异生等代谢过程(图2). 其中，植物乳杆菌、清酒乳杆菌和嗜酸乳杆菌均被报道能够产生SCFAs(乙酸、丙酸和丁酸等)[36~38]. 在育肥猪日粮中添加德氏乳杆菌，增加肠道有益菌丰度及乙酸、丙酸和丁酸的浓度，并改善宿主脂肪代谢[39]. Li等人[40]研究发现，在HFD小鼠日粮中添加副干酪乳酸菌，显著增加HFD小鼠肠道中乙酸和丁酸的浓度. 同时，Nguyen等人[38]在肝脏变性小鼠模型中发现补充植物

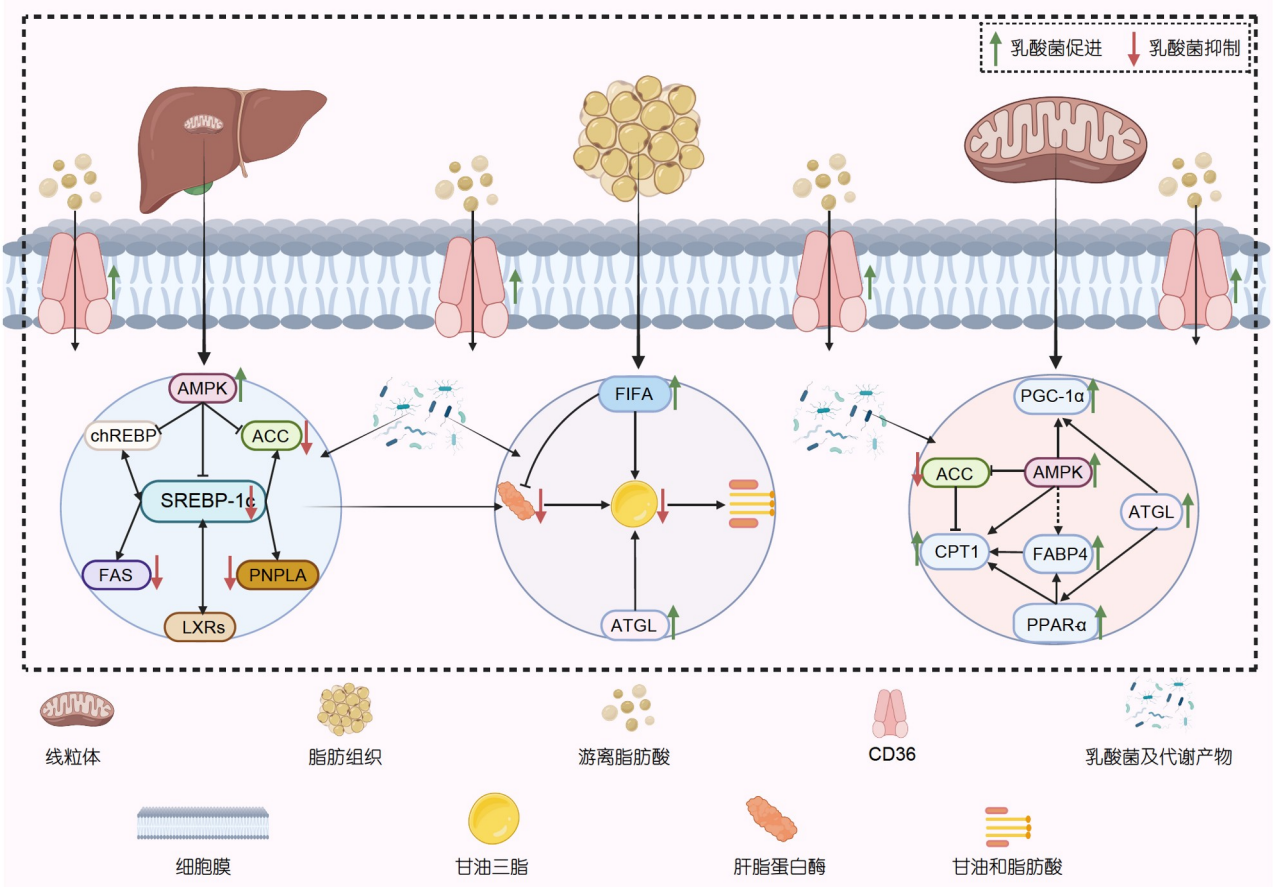

图 1　乳酸菌调控宿主脂肪代谢的机制. SREBP-1c, 胆固醇调节元件结合蛋白1c; FAS, 脂肪酸合成酶; PNPLA3, 脂肪酶样蛋白3; ACC, 乙酰辅酶A羧化酶; AMPK, AMP-活化蛋白激酶; chREBP, 碳水化合物响应元件结合蛋白; LXRs, 肝X受体; CPT1, 肉碱棕榈酰转移酶 1; FABP4, 脂肪酸结合蛋白4; FIFA, 禁食诱导脂肪因子; ATGL, 三脂酰甘油脂肪酶; PPAR-α, 过氧化物酶体增值受体-α; PGC-1α, 过氧化物酶体增殖物激活受体γ共激活因子1α; LPL, 肝脂蛋白酶; TG, 甘油三酯

Figure 1　Proposed mechanisms of *Lactobacillus*-mediated host lipid metabolism. SREBP-1c, sterol regulatory element binding protein-1c; FAS, fatty acid synthase; PNPLA3, patatin-like phospholipase domain-containing protein 3; ACC, acetyl-CoA Carboxylase; AMPK, adenosine monophosphate kinase; chREBP, carbohydrate-responsive element-binding protein; LXRs, liver X receptors; CPT1, carnitine palmitoyltransferase 1; FABP4, fatty acid binding protein 4; FIFA, fasting induced adipose factor; ATGL, adipose triglyceride lipase; PPAR-α, peroxisome proliferator-activated receptor-α; PGC-1α, peroxisome proliferator-activated receptor gamma coactivator 1-α; LPL, lipoprotein lipase; TG, triglycerides

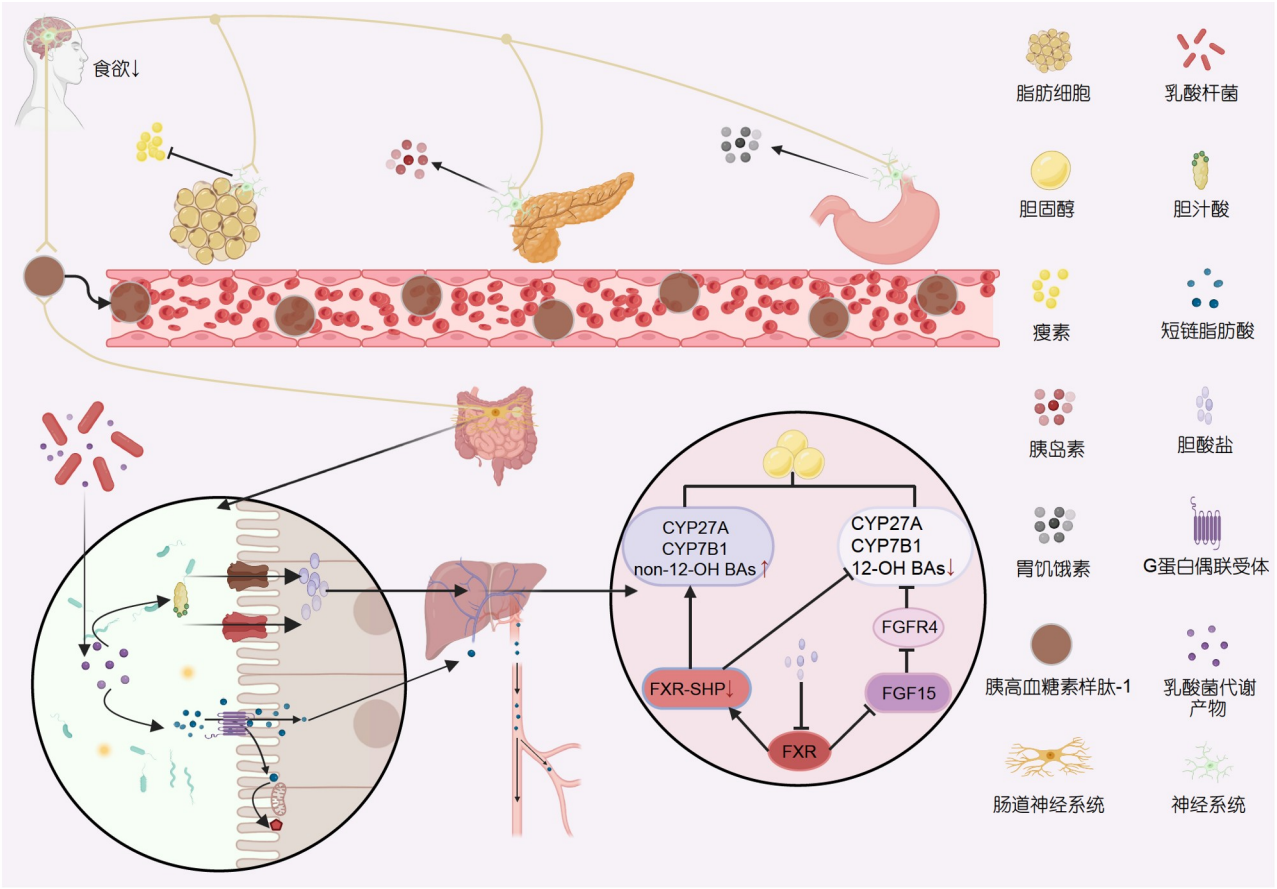

图 2 乳酸菌调控短链脂肪酸和胆汁酸的作用机制
Figure 2 Mechanism by which *Lactobacillus* regulating SCFA and BAs

乳杆菌ZY08显著增加SCFAs水平，同时脂肪吸收合成相关基因*CD36*，*Fasn*，*Dgat1*和*Dgat2*的表达水平显著上调. 研究证实, SCFAs能够作为信号分子激活细胞膜上的G蛋白偶联受体(G protein-coupled receptor, GPR), 进而参与多种脂肪代谢的调控过程. 其中, SCFAs能够通过激活交感神经系统、脂肪组织、胰腺、肠道和其他组织中的GPR41，GPR43和Olfr78(游离脂肪酸受体FFAR家族成员)来调节宿主的能量平衡[41~43]. 此外, 在宿主细胞中, 由于高强度运动或氧气供应不足, 细胞通过无氧代谢途径(乳酸发酵)产生ATP, 这主要发生在供氧不足的肌肉细胞中，但乳酸在细胞内过度蓄积可能导致酸中毒. 相比之下，嗜酸乳杆菌、干酪乳杆菌和植物乳杆菌等乳酸菌通过无氧代谢葡萄糖或其他碳水化合物产生的乳酸[13,44,45]，能够转化为SCFAs，维持肠道酸碱平衡、促进食物消化吸收和调节免疫，进而改善宿主脂肪代谢状态[46~49].

2.2 胆汁酸

胆汁酸(bile acids, BAs)是机体调控能量代谢的重要信号分子，其功能主要涉及肝脏的胆固醇代谢和肠道的脂质吸收. BAs在肝脏内合成有两条途径(图2), 其中经典途径产生12α-羟基化的初级BAs，而替代途径则产生非12α-羟基化的初级BAs，如胆酸盐、脱氧胆酸盐、鹅去氧胆酸、牛磺酸和甘氨酸脱氧胆酸盐、乌索脱氧胆酸盐、石胆酸盐以及去氧胆酸盐等[50]. BAs是微生物和宿主脂肪代谢互作的关键信号分子[51]. Horackova等人[52]研究发现乳酸杆菌含有参与胆汁酸代谢的胆盐水解酶(biliary salt hydrolyase, BSH), 可将从肝脏释放进入肠道的结合型BAs水解为非结合型BAs，其通过促进脂肪乳化和吸收、调节胆固醇代谢以及影响胰岛素敏感性等途径对脂质代谢产生调节作用. 非结合型胆汁酸可以激活细胞核上的法尼醇X

受体(farnesoid X receptor, FXR), 进而调节胆汁酸、脂肪和葡萄糖代谢稳态[53]. 研究发现, 当肝脏FXR信号受到抑制时, 胆固醇7α-羟化酶(cholesterol 7-alpha hydroxylase, CYP7A1)分解代谢胆固醇和胆汁酸生物合成的能力增强, 从而造成循环系统中胆固醇含量下降[54]. Wang等人[55]在HFD饲喂小鼠中补充植物乳杆菌AR133和干酪乳杆菌pWQH01发现, 肝脏中FXR mRNA表达量显著降低, 而CYP7A1、肝脏胆固醇X受体(LXR)和低密度脂蛋白受体(low density lipoprotein receptor, LDLR)表达显著上升, 提示乳酸菌BSH酶活通过胆汁酸循环影响FXR信号调控胆固醇代谢.

2.3　胞外多糖

胞外多糖(extracellular polysaccharides, EPS)具有多样型结构, 可分为同多糖(homopolysaccharides, HoPS)和异多糖(heteropol, HePS). HePS通过乳酸菌胞内液和其他细胞结构产生, 而HoPS通过乳酸菌细胞外的酶产生[56].

研究报道, 短乳杆菌、植物乳杆菌、发酵乳杆菌、鼠李糖乳杆菌和德氏乳杆菌均能够产生HoPS和HePS[57~60]. 其中, HoPS可分为葡聚糖、果聚糖和半乳糖, 分别由D-葡萄糖、D-果糖和D-半乳糖组成[61]. 与HoPS不同, HePS结构更加复杂, 其由多个单糖(D-核糖、D-阿拉伯糖、D-木糖)、己糖(D-葡萄糖、D-半乳糖、D-甘露糖)、N-乙酰化单糖(N-乙酰氨基葡萄糖和N-乙酰氨基半乳糖)和醛酸(D-葡萄糖醛酸、D-半乳糖醛酸)组成[62]. 研究发现, N-乙酰化单糖、醛酸和己糖均能够作为肠道发酵底物来调节肠道菌群以及SCFAs的水平[63]. Yasuo等人[64]研究发现, D-果糖的几何异构体阿洛酮糖促进HFD饲喂小鼠的脂肪酸氧化能力, 并抑制脂肪生成能力. 在体外试验中, 从植物乳杆菌H31中提取的HePs显著降低HepG2细胞中α-淀粉酶的水平, 并显著增加葡萄糖转运蛋白-4(glucose transporter 4, GLUT-4)、蛋白激酶B(protein kinase B, AKT-2)、AMPK和SCFAs的水平, 从而进一步调控脂肪代谢[65]. 同时, 副干酪乳酸杆菌M7提取的EPS能够将脂肪细胞胆固醇水平降低75%[66], 而植物乳杆菌RJF4衍生的EPS能够将脂肪细胞胆固醇水平降低42.24%[67]. Lee等人[68]研究发现, EPS通过与细胞膜上的细菌感受体TLR2结合, TLR2下游蛋白MyD88启动AMPK信号级联反应影响脂肪的合成和分解. 然而, 注射鼠李糖乳杆菌EPS会导致小鼠导致严重的炎症, 这可能是由于注射剂量过高激活小鼠免疫系统引起的[69]. 总之, 不同EPS对脂肪代谢具有特异性, 而同属乳酸菌产生的EPS也具有差异性. 因此, 本文总结不同乳酸菌来源的EPS调节脂肪代谢的试验研究(表2).

2.4　亚油酸及其代谢产物

亚油酸(linoleic acid, LA)是一种十八碳烯酸, 广泛参与机体脂肪代谢. 研究发现, 嗜酸乳杆菌、干酪乳杆菌、植物乳杆菌和鼠李糖乳杆菌等乳酸菌均能够通过发酵作用参与亚油酸的代谢产生多种脂肪酸, 如羟基脂肪酸、共轭脂肪酸、氧化脂肪酸和部分饱和脂肪酸, 参与抗氧化、抗炎、能量代谢和脂肪调控等多种生理过程[82~84]. Kim等人[85]发现, 亚油酸经过氧化、脱氢等酶催化反应产生的10-oxo-12(Z)-octadecenoic acid(KetoA)能够显著激活小鼠中PPARγ, 并刺激脂蛋白酶mRNA表达水平, 从而调节宿主能量代谢. 进一步研究小鼠全身整体能量代谢相关基因发现, KetoA显著上调线粒体不耦联蛋白1(uncoupling protein 1, UCP1)和过氧化物酶体增殖激活受体γ共激活因子-1(peroxisome proliferator-activated receptor gamma coactivator-1, PPARGC1A), 最终改善小鼠能量代谢[85].

共轭亚油酸(conjugated linoleic acid, CLA)是一类由亚油酸经过异构反应得到的多种同分异构体的脂肪酸. CLA在结构上具有多个双键, 这些双键的位置和几何异构性质不同, 形成CLA的多种同分异构体[86]. 最常见的CLA异构体包括顺式-9, 反式-11(c9, t11)和顺式-10, 反式-12(c10, t12)等[87]. 研究发现, 乳杆菌属、乳球菌属和链球菌属能够通过代谢亚油酸合成CLA[88,89]. 其中, 罗伊氏乳杆菌、鼠李糖乳杆菌和植物乳杆菌均被报道能够产生CLA, 并进一步参与宿主自由基清除、脂肪代谢调节[90~92]. Lee等人[93]研究报道, 鼠李糖乳杆菌PL60产生的CLA, 通过上调UCP2以及抑制脂肪酸合成酶(ACC和FAS), 表现出在HFD饲喂小鼠中抗肥胖的作用. 研究发现CLA的同分异构体顺式-9和反式-11, 能够显著降低HFD饲喂小鼠中血清甘油三脂的浓度并提高HDL-C的浓度[94].

3　复合乳酸菌调控宿主脂肪代谢的应用

相较于单一乳酸菌, 复合乳酸菌在调控宿主脂肪

表 2 乳酸菌代谢产物—胞外多糖改善宿主肥胖的作用

Table 2 Effect of *Lactobacillus*-EPS on ameliorating host obesity

来源菌株	试验对象	作用	参考文献
鼠李糖乳杆菌GG	高脂日粮肥胖小鼠	肝脏中TAG和胆固醇以及血清TAG显著降低	[69]
粘膜乳杆菌A1	高脂日粮肥胖小鼠	调节肠道微生物, 并显著降低血清、肝脏和主动脉窦中的脂质蓄积	[70]
植物乳杆菌L-14	高脂日粮肥胖小鼠	AMPK、AKT和P-AMPKα水平显著上升, PPARγ, C/EBPα, ACC, SREBP-1和FAS显著降低	[68]
鼠李糖乳杆菌GG	C57BL/6J小鼠	皮下、性腺、血清和肝脏的TAG水平、肝脏胆固醇、PPARγ, AP2, FAS, SCD1, LPL和DGAT1显著降低	[71]
植物乳杆菌JY039	高脂日粮肥胖小鼠	调节肠道微生物平衡, PYY, GLP-1显著上升. 促炎因子IL-6, TNF-α和抗炎因子IL-10来减少炎症	[72]
发酵乳杆菌FTDC8312	BALB/C小鼠	调节肠道菌群平衡	[73]
鼠李糖乳杆菌GG	斑马鱼	改善试验组的肠道微生物失调, 激活HIFα. 减少肝脂变性	[74]
植物乳杆菌x1	糖尿病小鼠	改善小鼠抗氧化能力、细胞因子和胰腺损伤, 调节肠道菌群, 并显著增加SCFAs和BSH	[75]
发酵乳杆菌LF2	BALB/c小鼠	粪便中SCFAs水平显著增加, 肠道中有益菌相对丰度显著增加	[76]
干酪乳杆菌LC-XCAL	高脂日粮肥胖小鼠	显著降低肝脂甘油三酯、胆固醇和脂肪垫	[77]
布氏乳杆菌TCP.016	肝损伤小鼠	降低血清、LPS和细胞因子的水平, 显著提高超氧化物歧化酶和谷胱甘肽的活性, 改善肠道微生物平衡, 抑制致病菌, 并增加益生菌的相对丰度	[78]
清酒乳杆菌ADM14	C57BL/6J小鼠	显著降低血液中胆固醇水平, 改善炎症	[32]
植物乳杆菌JLK0142 EPS	无菌小鼠	改善肠道免疫球蛋白A(IgA)和血清细胞因子IL-2和TNF-α水平	[79]
植物乳杆菌LRCC5310	BALB/c小鼠	提高抗氧化活性和α淀粉抑制活性	[80]
植物乳杆菌H31	HepG2细胞	降低胰岛素抵抗HepG2细胞α-淀粉酶活性, 并上调GLUT-4, AKT-2, AMPK基因表达	[65]
保加利亚乳杆菌	C3H/HeJ小鼠	EPS通过myd88介导的INF-γ, IL-12, IL-18等细胞因子激活NK细胞	[81]

代谢方面更占优势. 研究发现, 在HFD小鼠灌胃复合植物乳杆菌(CECT 7527, 7528和7529)较植物乳杆菌S4-1单独处理, 显著降低血清中胆固醇和低密度脂蛋白的浓度[95]. Yoo等人[96]也报道补充复合乳酸菌(*L. curvatus* HY7601和*L. plantarum* KY1032)显著缓解HFD诱导的脂肪堆积, 并协同抑制脂肪酸合成相关基因. Savcheniuk等人[97]在HFD小鼠补充多种复合乳酸菌与单株乳酸菌进行比较, 复合乳酸菌处理组的肥胖患病率、内脏脂肪组织重量和血清脂质水平显著降低, 提示不同乳酸菌符合使用对脂肪代谢调控机制产生协同作用. Wang等人[98]发现, 复合乳酸菌(鼠李糖乳杆菌LS-8和面包乳杆菌MNo47)对脂肪代谢的调节具有三种潜在机制. (i) 复合乳酸菌通过增加HFD小鼠肠道中SCFAs的水平和有益菌的相对丰度, 并降低内毒素水平和有害菌相对丰度. (ii) 复合乳酸菌能够抑制HFD小鼠促炎因子的表达, 并调节与脂肪代谢相关基因的表达, 改善糖耐量和降低胰岛素抵抗. (iii) 复合乳酸菌调节HFD小鼠血清甘油三酯、总胆固醇和低密度脂蛋白胆固醇的水平, 改善整体脂肪代谢状况. 由此可见, 复合乳酸菌相较于单一乳酸菌在改善宿主脂肪代谢方面效果更加显著. 在进一步深入研究乳酸菌作用时, 亟需开展更多的临床试验, 系统筛选出调控宿主脂肪代谢的菌株组合方式.

4 总结与展望

脂肪代谢紊乱是造成多种代谢综合征疾病的主要因素之一. 乳酸菌在改善和介导宿主脂肪代谢紊乱上效果显著, 本文重点阐述乳酸菌以胆汁酸、短链脂肪酸、胞外多糖、亚油酸和肠道菌群等干预和

调控宿主脂肪代谢的作用. 由于乳酸菌的多样性, 不同乳酸菌对脂肪代谢的分子机制和作用效果存在显著差异. 鉴于当前研究主要关注乳酸菌对代谢性疾病的短期干预效果, 对其与不同个体之间存在的肠道微生态、基因差异和长期摄入乳酸菌的潜在影响尚不明确. 因此, 未来应深入挖掘不同乳酸菌菌株之间的代谢产物效应和机制, 为菌株的筛选和效应验证提供更系统的依据. 同时, 完善对乳酸菌在肠道中的定植机制和长期摄入的潜在影响的研究, 为乳酸菌在临床应用中的安全性提供更充分的数据支持. 此外, 还需要关注不同人群之间的差异, 以及乳酸菌在代谢性疾病治疗中的潜在风险. 这些研究将为深化乳酸菌与脂肪代谢关系的认识、拓展相关临床应用提供科学基础.

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Advances in the regulation of host fat metabolism by *Lactobacillus*

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Lipid metabolism disorder is one of the main factors causing metabolic syndrome. In animal models displaying disrupted fat metabolism or in clinical populations affected by obesity, there are substantial changes in the diversity and composition of gut microbiota, particularly within the genus *Lactobacillus*. As a probiotic, *Lactobacillus* is expected to extensively impact host fat metabolism. Therefore, this study explored the relationship between *Lactobacillus* and fat metabolism. It analyzed the underlying molecular mechanisms by which metabolites of *Lactobacillus* (such as short-chain fatty acids, bile acids, exopolysaccharides, and linoleic acid) regulate fat metabolism. Additionally, the study elaborated on the differences between the same and different *Lactobacillus* complexes to clarify the mechanism of lactic acid bacteria in regulating host fat metabolism. This study aims to provide perspectives and references for controlling food-borne metabolic diseases caused by lipid metabolism disorders.

***Lactobacillus*, Lipid metabolism, metabolite**

doi: 10.1360/SSV-2023-0232