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在线全文

肠黏膜屏障在2型糖尿病中的作用机制及中医药干预研究进展*

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【摘要】 肠黏膜屏障与2型糖尿病（type 2 diabetes mellitus, T2DM）的发生发展存在密切联系，通过调节机体代谢稳态、免疫应答及微生物-宿主互作等机制，在维持机体内环境稳态和抵御病原体侵袭方面发挥重要作用。近年研究证实，肠黏膜屏障失调可能构成T2DM发病的关键环节。本文系统综述了肠黏膜屏障损伤参与T2DM发病的机制与现代医学的干预策略进展，对中医药干预肠黏膜屏障调控T2DM的最新研究进行探讨与总结，目前肠黏膜屏障损伤与T2DM发病的因果关系机制尚未完全阐明，且中西医协同调控机制缺乏系统性研究。中西医结合治疗T2DM可以从中医辨证论治、药物属性平衡以及多靶点治疗等多方面提供启示和依据。未来应充分发挥中西医结合治疗T2DM的优势，围绕肠黏膜屏障展开深入研究，为临床应用提供理论依据。

【关键词】 肠黏膜屏障 糖尿病，2型 中医药 肠道菌群 胰岛素抵抗

Role of Intestinal Mucosal Barrier in Type 2 Diabetes Mellitus and Research Progress in Chinese Medicine Interventions

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[Abstract] The intestinal mucosal barrier is closely associated with the occurrence and development of type 2 diabetes mellitus (T2DM). It plays an important role in maintaining the homeostasis of the internal environment and resisting pathogenic invasion by regulating the metabolic homeostasis, immune response, and microbial-host interactions in the human body. According to recent studies, intestinal mucosal barrier dysregulation may constitute a key link in the pathogenesis of T2DM. Herein, we systematically reviewed the mechanisms by which intestinal mucosal barrier damage contributes to the onset of T2DM and the latest advances in modern medical intervention strategies. We also discussed and summarized the latest research findings on using traditional Chinese medicine (TCM) to regulate the intestinal mucosal barrier in the management of T2DM. Currently, the causal relationship and mechanisms linking intestinal mucosal barrier damage to the onset of T2DM remain unclear, and systematic research on the synergistic regulatory mechanisms of approaches integrating TCM and Western medicine is lacking. The approach to manage T2DM through the integration of TCM and Western medicine can provide insights and basis from multiple aspects, such as TCM syndrome differentiation and treatment, balancing drug properties, and multi-targeted therapy. In the future, we should fully leverage the advantages of integrated TCM and Western medicine in treating T2DM and conduct in-depth research focused on the intestinal mucosal barrier, thereby informing evidence-based clinical applications.

[Key words] Intestinal mucosal barrier Diabetes mellitus, type 2 Medicine, Chinese traditional Gastrointestinal microbiome Insulin resistance

2型糖尿病（type 2 diabetes mellitus, T2DM）是以胰岛素调控葡萄糖代谢能力的下降〔胰岛素抵抗（insulin resistance, IR）〕伴胰岛β细胞功能缺陷所导致的胰岛素分泌相对减少为病理生理学特征的疾病[1]。国际糖尿病联盟（IDF）第十版糖尿病地图集统计数据显示[2]，中国已成

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为糖尿病患者数量最多的国家,过去十年间成人患病率增幅达56%,预测2045年将突破1.74亿人,成为亟待解决的公共卫生问题。肠黏膜屏障是控制宿主-微生物组之间串扰的关键结构,能够通过动态调节选择性通透功能,在维持营养物质吸收与防止致病性细菌渗透及异位的双重稳态中发挥关键作用。病理损伤机制涉及肠道菌群紊乱、紧密连接蛋白表达异常及黏液层结构破坏、免疫失衡,有研究显示肠屏障通透性增加与功能障碍会损害肠道中葡萄糖的吸收,并允许细菌成分如脂多糖(lipopolysaccharide, LPS)等诱发代谢炎症,最终加剧IR[3]。基于上述病理关联,针对肠黏膜屏障结构与功能的修复性干预,已成为调控T2DM代谢失衡的重要策略。

T2DM属于中医"消渴"范畴,一般认为T2DM前期归属中医"脾瘅",其核心病机可溯源至中焦枢机失运,《灵枢·本藏》有言"脾脆则善病消瘅易伤",证实T2DM的发生多责之于脾虚[4]。《素问·奇病论》述:"此五气之溢也,名曰脾瘅……此人必数食甘美而多肥也。肥者令人内热,甘者令人中满,故其气上溢,转为消渴",表明"脾瘅"是"消渴"的前期阶段,若未及时进行有效干预,则导致消渴病的发生。《医学入门·脏腑条分》云:"脾与小肠相通",脾脏功能失调可通过"脏腑络属"机制连及肠道,导致清浊不分,水湿潴留,加重机体消化吸收与代谢功能障碍[5]。现代研究表明,中医药干预通过多维度调控机制实现肠黏膜屏障的修复,可通过调控肠道菌群组成及结构、强化紧密连接蛋白的表达水平、促进黏液层的恢复再生、对肠道免疫进行合理调节等[6-7],改善糖脂代谢异常[8]、缓解IR[9]、调节相关炎症介质水平[10],遏制T2DM进展。深入剖析肠黏膜屏障损伤在T2DM的病理机制,综合梳理中医药在治疗T2DM肠黏膜屏障功能障碍方面的最新研究,有助于为中医药在治疗T2DM领域提供参考与未来研究方向,促进传统医学与现代医学的融合创新。

1 肠黏膜屏障参与T2DM发生发展

T2DM的发病机制呈现多因素交互作用的特征。肠黏膜屏障是由生物屏障、化学屏障、物理屏障、免疫屏障构成的复杂体系,参与并调节机体的物质和能量代谢进程。

1.1 菌群失调

肠道微生物群是一个复杂的适应系统,以动态和非线性方式相互作用,实现系统级的突现特性[11],在代谢多种降糖药物方面发挥增强疗效或降低副作用的作用,使其成为糖尿病病理生理学中的核心参与者[12]。肠道核心菌群分为"病生功能群"与"基石功能群"[11],在T2DM状态下二者平衡性失调。"病生功能群"数量较低时,可以保持免疫系统的警觉性,若过度富集,则可能触发代谢炎症,促进代谢性内毒素血症[13],推动T2DM的IR发展[14]。肠道大肠杆菌会导致糖耐量受损,增加的内毒素诱导慢性低度炎症,加剧高脂饮食引起的肥胖和IR[13]。厚壁菌门和拟杆菌门比值(即F/B比值)常被视作衡量肠道微生物群失调与否的关键指标之一。T2DM患者F/B比值高于健康受试者,提示T2DM状态下微生物的多样性及稳定性遭到破坏[15]。肠道内革兰氏阴性菌增多,会大量分泌具有显著内毒性的LPS,利用肠道增加的通透性扩散至体循环,识别并激活Toll样受体4(TLR4)及其下游信号通路如核因子kappa-B(NF-κB),诱导白细胞介素-6(IL-6)以及肿瘤坏死因子-α(TNF-α)等促炎因子的表达,协同放大全身炎症反应,使得胰岛素信号传导受阻,削弱机体对胰岛素的反应性[16]。

"基石功能群"以短链脂肪酸(short-chainfatty acids, SCFAs)产生菌为主,能够优化肠道环境,抑制条件致病菌的过度生长,通过参与多种途径增强葡萄糖代谢能力,抑制糖异生,促进胰岛素分泌,降低IR。T2DM受试者中产丁酸菌*Roseburia intestinalis*和*Faecalibacterium prausnitzii*丰度明显低于对照组[17]。丁酸盐可通过抑制被认为参与糖异生信号传导的组蛋白脱乙酰酶(HDAC),激活胰高血糖素样肽-1(GLP-1)受体基因(*GLP-1R*),强烈诱导肠道L细胞GLP-1分泌[18]。丁酸钠治疗可以通过抑制大鼠胰岛中的β细胞关键转录因子和功能基因来间接增强胰岛素分泌,增强的β细胞功能归因于KATP通道减少和胰岛素基因转录增加,这是由于启动子中占据H3K27bu而引起的胰岛素基因转录升高[19]。此外,SCFAs中丙酸盐特异性激活G蛋白偶联受体41(GPR41)和43(GPR43),增加小鼠循环瘦素水平。GPR41能够发挥抑制食欲作用,其作用机制在于调节瘦素分泌、减少脂质堆积和提升能量消耗,最终促使体质量减轻。GPR43通过调节蛋白激酶C、磷酸酶和张力蛋白同源物通路抑制胰岛素信号传导来减少脂质积累[20]。

1.2 黏液稀薄

肠黏液层是肠道上皮细胞表面覆盖的一层保护性凝胶状分泌层,起到化学屏障的作用。肠上皮中的杯状细胞负责控制糖基化和持续制造黏液,与Peneth细胞分泌的抗菌物质如抗菌肽、防御素协同阻挡细菌远离肠上皮的同时,形成抗菌扩散梯度,为菌群提供营养和黏附位点。黏液层与共生菌群存在双向互作,共生菌和病原菌能调节黏液的合成、分泌、厚度和黏度[21]。沙门氏菌触发干扰素-γ受体信号控制杯状细胞释放黏液到肠腔,协调黏膜防御[22]。高度糖基化黏蛋白(mucin2, MUC2)是黏液层最重要的组成部分。菌群代谢物如吲哚-3-乙酸通过芳

烃受体3'-磷酸腺苷5'-磷酸硫酸合成酶2和溶质载体家族35成员B3通路增强肠道MUC2硫酸化，阻止细菌酶促降解黏蛋白聚糖，维持肠道稳态[23]。肠道微生物中*Vibrio cholerae*诱导黏蛋白降解，*Listeria monocytogenes*抑制黏液生成，*Lactobacillus spp*促进MUC2产生与分泌，*Bifidobacterium longum*恢复黏液生长，*Lactobacillus reuteri*增加黏液层厚度[21]。嗜黏蛋白阿克曼菌(*Akkermansia muciniphila*)是调节黏液层的关键细菌，可通过二磷酸腺苷庚糖的释放和α蛋白激酶1/肿瘤坏死因子受体相关因子相互作用的具有叉形头相关结构域的蛋白轴的激活诱导MUC2的表达[24]。此外，该菌可促进INS-1细胞葡萄糖转运蛋白2(glucose transporter 2, GLUT2)和葡萄糖激酶基因的表达[25]。刺激肠道中GLP-1的分泌，促进脂肪组织分化以及葡萄糖的摄取，达到增强胰岛素敏感性、改善IR的作用[26]。

1.3　肠壁受损

完整的肠壁通过肠上皮细胞间连接复合物如紧密连接、黏附连接、桥粒和间隙连接等结合，共同构成了肠黏膜物理屏障。而紧密连接是决定肠黏膜通透性的关键因素，由闭锁蛋白(occludin)、闭合蛋白(claudin)和外周膜蛋白(Zo)等构成，可通过动态调节来控制必需分子的运输和限制有害物质的渗透，当表达降低时，会导致肠道上皮完整性的丧失，自动损害肠道中的葡萄糖吸收，促进疾病的发生和发展[3]。有研究发现，在肥胖与糖尿病小鼠模型中，高血糖状态会异常激活肠上皮细胞的GLUT2依赖性转录重编程，同时改变紧密连接完整性，促使肠道屏障通透性增加[27]。肌球蛋白轻链激酶是紧密连接通透性的关键调节因子，通过刺激肌球蛋白ⅡA(MyoⅡA)的调节轻链磷酸化来进行胰岛素信号传导，同时MyoⅡ是胰岛素反应性葡萄糖转运蛋白4(GLUT4)介导的3T3-L1脂肪细胞葡萄糖摄取所必需的[28]。

1.4　免疫失衡

肠道免疫屏障是由肠相关淋巴组织、淋巴细胞以及分泌的免疫效应分子如分泌型免疫球蛋白A(secretory immunoglobulin A, sIgA)共同构成。sIgA能够包被细菌，形成抗原复合物，刺激肠道黏液分泌，阻止有害细菌黏附肠黏膜，进入肠壁。肥胖和IR诱发的慢性亚临床炎症会致使胰岛素敏感性下降和胰岛β细胞功能缺陷，引发肠黏膜免疫中的巨噬细胞浸润、T细胞与B细胞发生改变、浆细胞分泌免疫球蛋白A(immunoglobulin A, IgA)等发生紊乱，提示肠道免疫平衡参与T2DM的发生和发展。sIgA的产生依赖于微生物群和特定微生物，并能驱动整体免疫反应的程度[29]。在T2DM患者中，调节性B细胞水平的升高与IR严重程度相关，同时与野生型小鼠相比，B细胞缺陷小鼠显示出葡萄糖耐量和胰岛素敏感性的改善[30]。高脂饮食会改变肠内IgA^+免疫细胞的活动，并通过IgA水平的降低来加剧IR，破坏体内糖平衡。肥胖$IgA^{-/-}$小鼠的葡萄糖耐量和胰岛素敏感性恶化，肠道通透性增加，不能将IgA转运至肠腔中的聚合Ig受体(pIgR)，可能与细菌侵蚀恶化和促炎细胞因子介导的上皮紧密连接破坏有关[31]。pIgR的激活通过MEK/ERK信号通路调节IgA转运来维持肠道细菌稳态，并上调紧密连接蛋白以稳定屏障功能[32]。用抗生素消除高脂饮食喂养的$IgA^{-/-}$小鼠的肠道微生物群可以改善葡萄糖稳态，这提示IgA缺乏期间，肠道微生物的改变很可能是肠道炎症和疾病恶化的机制[31]。Th17和Treg作为$CD4^+$ T细胞的亚群，Th17/Treg细胞激活和迁移在肠道微生物和宿主代谢中发挥调节葡萄糖和脂质代谢作用[33]。

2　现代医学调控肠黏膜屏障干预T2DM的策略

靶向肠黏膜屏障的干预措施是极具潜力的T2DM治疗方法，现代医学研究聚焦在益生菌益生元、粪菌移植(FMT)、促进菌群定植等方面以调节肠道稳态来预防或治疗T2DM。选择性地补充适合的益生菌或促进益生菌生长或富集的益生元可改善人体肠道菌群的紊乱，如预防性补充益生菌副干酪乳杆菌L14通过改善氧化应激、IR、炎症、血脂异常和保护β细胞，同时缓解肠道菌群失调，对T2DM大鼠显示出极好的预防作用[34]。健康供体中存在维持人体健康所需的关键功能菌群，FMT能够补充重要细菌进而纠正机体菌群失调。如FMT联合或不联合二甲双胍通过定植供体来源的微生物群可显著改善T2DM患者的胰岛素抵抗和体质量指数以及肠道微生物群落[35]。此外，利用膳食纤维选择性富集的肠道细菌可缓解T2DM，肠道菌群通过代谢膳食纤维产生的短链脂肪酸，可以保护肠黏膜屏障，防止内毒素入血，还可介导菌群对血糖稳态的影响，促进胰岛素分泌，改善T2DM的预后[36]。口服聚乙二醇包裹的共生细菌可强化其在肠道黏液层中的移动性和穿透能力，并优先定位和滞留，促进IgA的生成和抑制病原菌入侵，刺激黏液和紧密连接蛋白的分泌，通过改善葡萄糖耐受和IR来缓解脂肪组织的增生，减轻炎症环境，有效保护肠黏膜屏障和预防糖尿病的发生[37]。

3　中医药调控肠黏膜屏障干预T2DM的作用机制

T2DM因个体差异、病程阶段或证候类型不同，常采

用不同的治法与方药。中药具有不同的四气五味、药性归经与功效，在修复T2DM肠黏膜屏障的复杂病理过程中，通过“性味配伍-靶点协同”以多层次调控、系统调控肠道菌群和修复肠道物理屏障完整性，调节肠道免疫应答，多靶点协同，最终实现肠黏膜稳态的恢复，契合中医“同病异治”的现代系统生物学内涵(图1)。

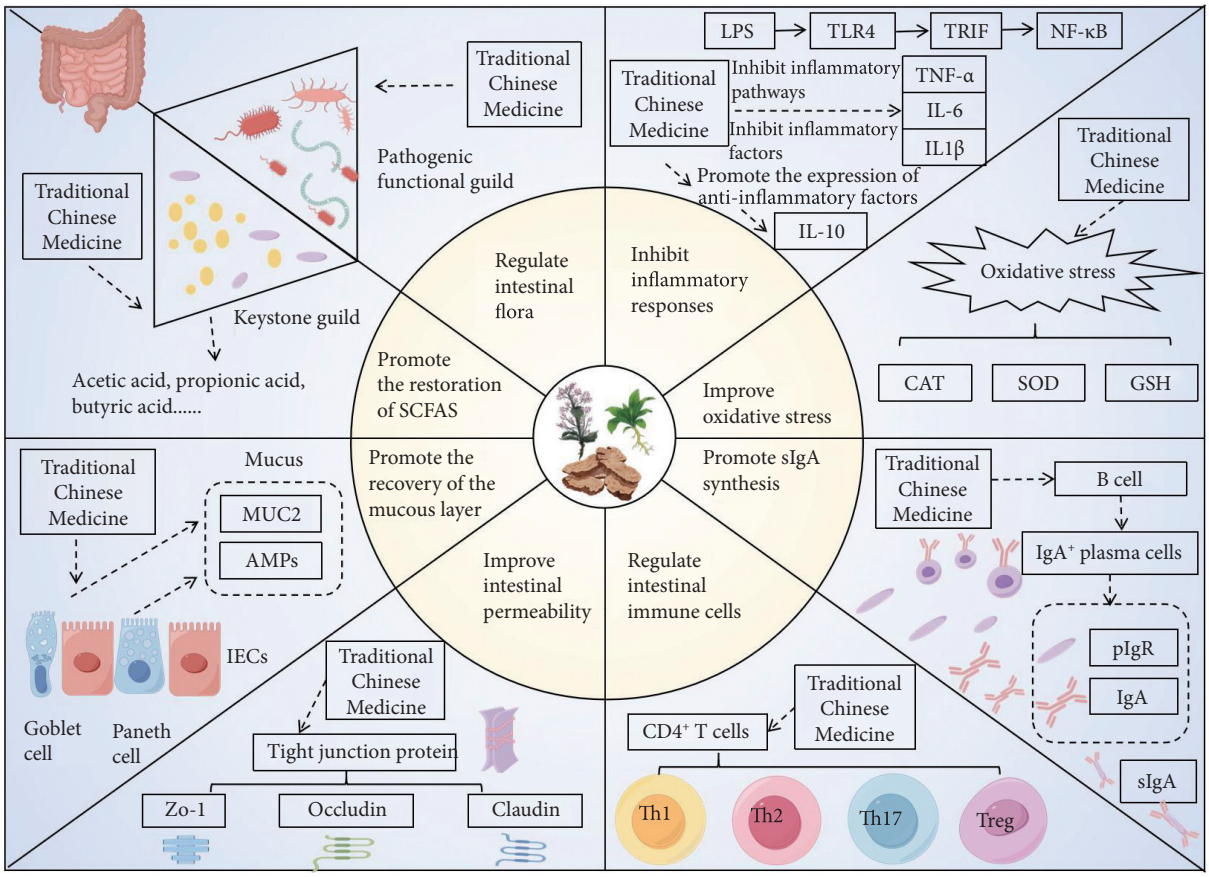

图 1 中药调控肠黏膜屏障干预T2DM

Fig 1 TCM regulates the intestinal mucosal barrier for intervention in T2DM

SCFAs: short-chainfatty acids; LPS: lipopolysaccharide; TLR4: toll-like receptor 4; TRIF: TIR-domain-containing adapter inducing interferon-β; NF-κB: nuclear factor kappa-B; TNF-α: tumor necrosis factor-α; IL-6: interleukin-6; IL-1β: interleukin-1β; IL-10: Interleukin-10; CAT: catalase; SOD: superoxide dismutase; GSH: glutathione; MUC2: mucin 2; AMPs: antimicrobial peptides; IECs: intestinal epithelial cells; Zo-1: zonula occludens-1; pIgR: polymeric immunoglobulin receptor; IgA: immunoglobulin A; sIgA: secretory immunoglobulin A.

3.1 调节肠道菌群及代谢物

3.1.1 中药单体或有效成分

《名医别录·中品》言黄连“止消渴”且“调胃，厚肠”，黄连主要活性成分小檗碱能够降低T2DM大鼠厚壁菌门、脱硫杆菌门相对丰度，升高益生菌Muribaculaceae菌科、瘤胃球菌属__B相对丰度，改善T2DM大鼠的菌群紊乱[38]。活血类中药牛膝的活性成分多糖(ABP)增加了产生SCFAs的细菌丰度，包括*Alloprevotella*，*Bacteroides*，*Prevotellaceae_UCG_001*，*Prevotellaceae_NK3B31_group*和*Akkermansia*，激活了由SCFAs产生菌介导的GLP-1/GLP-1R/环磷酸腺苷/蛋白激酶A/cAMP反应元件结合蛋白/胰岛素通路缓解T2DM[39]。薏苡仁归脾、胃经，能够利水渗湿、健脾止泻，其活性成分多糖(CSP)可通过调节T2DM小鼠的肠道微生物组成，特异性富集产SCFAs菌，激活胰岛素样生长因子1/磷脂酰肌醇-3-激酶/蛋白激酶B信号通路，改善T2DM小鼠糖脂代谢紊乱[40]。

3.1.2 中药药对

黄芩-黄连性味相须，清热燥湿，厚肠止利，能够使产生SCFAs的细菌如*Bacteroidales S24-7 group_norank*、*Parasutterella*、*Prevotellaceae UCG-001*、*Ruminiclostridium*和*Ruminiclostridium 9*经其组合处理后显著富集，埃希氏菌-志贺氏菌等次生胆汁酸细菌的数量急剧减少，改善T2DM大鼠的糖脂代谢[41]。黄连-肉桂配伍，黄连泻火坚阴、肉桂通肾气，通过调节肠道菌群、增加SCFAs含量，促进胆汁酸中胆酸、鹅去氧胆酸分泌，减少熊去氧胆酸分泌，降低db/db小鼠肠道通透性状态、增强肠道屏障作用，

最终达到对T2DM的防治作用[42-43]。

3.2　抗炎与缓解氧化应激

人参大补元气兼能生津补虚，其化学成分人参皂苷Rg5缓解了糖尿病db/db小鼠高血糖症状，显著降低血清炎症因子TNF-α、IL-6、IL-1β水平及蛋白表达，降低血浆LPS、肝脏TLR4相关炎症蛋白，逆转了结肠肠道菌群失调，F/B比值显著降低，增加拟杆菌门和变形菌门的丰度[44]。《神农本草经·卷一》中强调了铁皮石斛厚肠胃的功效，其复合多糖铁皮石斛多糖（DOP）可提升Occludin、Claudin-1和*Zo-1* mRNA表达水平，通过LPS/TLR4/β-干扰素TIR结构域衔接蛋白/NF-κB轴减少LPS渗漏、抑制血清炎症因子TNF-α、IL-6、IL-1β水平和氧化应激损伤，抑制有害菌*Helicobacter*，促进益生菌*Allobaculum*、*Bifidobacterium*和*Lactobacillus*的增殖，来缓解T2DM小鼠糖脂代谢功能障碍[45]。黄芪补气升阳，助脾健运，其多糖（APP）可通过抑制T2DM小鼠血清氧化应激过氧化氢酶、超氧化物歧化酶、谷胱甘肽水平和促炎因子LPS、TNF-α、IL-6水平，促进抗炎因子IL-10的表达，以88.92%的还原率强烈抑制肠道病原体志贺氏菌，并促进有益菌*Allobaculum*和*Lactobacillus*的生长（分别提高11.72%和30.70%）来保护肠道屏障及改善T2DM小鼠的糖脂代谢紊乱[46-47]。《医学心悟·三消》："治下消者，宜滋其肾"，冬虫夏草补肾益精，其多糖中的纯化级分a（AEPSa），通过增加T2DM小鼠中*Allobaculum*、*Alistipes*，减少肠球菌和*Ruminococcus_torques_group*来重塑肠道菌群，抑制结肠TLR4/NF-κB通路的激活，上调肠道紧密连接蛋白表达，从而改善葡萄糖和血脂代谢[48]。

3.3　调节肠道免疫

泻心汤清热泻火，可清中焦湿热，恢复脾胃运化功能，在抑制结肠上皮细胞中HDAC表达的同时，可显著促进G蛋白偶联受体的表达，进而提高TGF-β和IL-18水平，调节$CD4^+$T细胞分化，维持肠道免疫稳态，恢复肠道屏障功能，缓解T2DM[49]。健脾清化方可通过减少肠组织中免疫细胞Th1、Th2、Th17比例及转录因子T-bet与RORα，增加Treg细胞比例，改善T2DM大鼠胰岛素抵抗[50-51]。MUC2携带共生菌抗原至派氏结（Peyer's patches），激活B细胞分化为sIgA浆细胞。以健脾益气清热为核心的健脾消渴方有效上调T2DM大鼠结肠Zo-1、MUC2、Occludin表达，减轻肠道屏障损伤，改善T2DM大鼠糖脂水平[52]。中医大家施今墨认为糖尿病患者常表现为正气亏虚，故治疗应注意气阴双补[53]，黄芪归脾经，调理脾胃、益气补虚，黄芪多糖能促进空肠中IgA^+细胞的增殖与分化，分泌更多的sIgA，有利于增强肠黏膜免疫功能[54]。龙眼肉润燥生津，其多糖通过改善IgA浆细胞的生成和迁移、上调pIgR的表达，促进sIgA跨上皮转运，从而加强sIgA的肠道分泌[55]。

3.4　多靶点协同效应

复方核心优势在于其整体性与多靶点协同调控机制。肥胖是T2DM的主要风险因素，参苓白术散可治疗脾虚湿盛型肥胖，课题组前期发现其能够逆转肥胖ZDF大鼠胰岛β细胞去分化效果[56]，亦可调整肠道菌群结构，增加产SCFAs的细菌的相对丰度，预测脂肪酸生物合成与代谢、甘油酯代谢、糖酵解/糖异生等代谢通路可能发挥关键作用[57]。肥胖者易积聚痰湿，《石室秘录·肥治法》言："肥人多痰，乃气虚也"，二陈汤能够燥湿化痰、理气和中，课题组前期研究发现，二陈汤可改善经高脂饮食诱导的肥胖大鼠脂质代谢紊乱，调节肠道菌群并促进丁酸的产生，通过乙酰组蛋白3-赖氨酸9促进脂肪酸β-氧化，以改善肥胖相关的脂肪变性[58]。《伤寒论·辨太阳病脉证并治上》："大烦渴不解，脉洪大者，白虎加人参汤主之"，白虎加人参汤主治气分热盛、气阴两伤证，能够降低门水平F/B比值，在属水平上，提升乳酸菌属、*Blautia*和*Anaerostipes*的相对丰度，降低*Allobaculum*、*Candidatus Saccharimonas*和*Ruminococcus*的相对丰度，增加结肠中Zo-1和Occludin的表达，抑制TLR4/NF-κB介导的炎症反应来改善T2DM大鼠的高血糖[59]。当归补血汤补气、生血，能够提高GK大鼠胰岛素敏感性，降低炎症介质的表达，并改善全身氧化应激，同时改善T2DM大鼠肠道中的微生物多样性（如*Romboutsia*、*Firmicutes*和*Bacilli*）与丙氨酸、天冬氨酸和谷氨酸代谢[60]。葛根芩连汤主治大肠传导失司所致的下利，擅于清利胃肠湿热，降低T2DM大鼠血糖和炎性细胞因子水平，提升紧密连接蛋白Occludin、Claudin-1水平，通过增加有益细菌（如粪球菌、双歧杆菌、*Blautia*和*Akkermansia*）来改变肠道微生物群的组成，升高了特定胆汁酸的水平，有助于改善脂质代谢[61-62]。

4　中西医结合治疗T2DM的启示

肠黏膜屏障在T2DM发病机制层面，涉及完整性受损、菌群失调异位、黏液层变薄、紧密连接蛋白破坏、免疫失衡等多因素。现代医学研究通过补充益生菌益生元、FMT、增加黏液层厚度、促进菌群定植等重塑肠道菌群平衡来预防或治疗T2DM。筛选最有效的菌株组合，以及根据不同宿主和疾病基因个体化FMT仍具有挑战[63]。中医药调控肠黏膜屏障治疗T2DM多有成效，为中西医结合治疗T2DM提供策略与启示：①诊疗模式优势互补：通过中医辨证论治调节机体的整体状态，同时利用西医快速控制血糖。②药物属性与个体差异的平衡：不同寒

热虚实属性的药物在治疗T2DM时需要根据患者的具体情况进行选择,在中西医结合治疗中,需要综合考虑药物属性和患者体质,避免单一药物的长期使用对机体造成不良影响。③多靶点治疗的思路:中药的多成分、多靶点特性使其在治疗T2DM时能够从多个方面发挥作用。例如,某些中药成分可能通过改善肠道菌群、调节肠道微环境来间接影响血糖代谢。中西医治疗相结合,可以更全面地控制病情,减少并发症的发生。

综上所述,中西医结合治疗T2DM可以从中医辨证论治、药物属性平衡以及多靶点治疗等多方面提供启示和依据。未来,通过中西医结合优势互补,围绕肠黏膜屏障进行精准施策,为T2DM的诊断、治疗、预后开辟全新路径。

* * *

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