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植物多糖缓解结肠炎的作用机制与构效关系研究进展

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摘要: 基于植物多糖在结肠炎干预中的研究基础, 综述近年来植物多糖抗结肠炎作用机制及构效关系的研究进展。植物多糖可通过调控 NLRP3 炎症小体、NF- κ B、MAPK、NRF2/HO-1、PI3K/Akt、JAK/STAT 等信号通路, 重塑肠道菌群, 促进短链脂肪酸等代谢物生成, 并增强肠黏膜屏障功能, 从而缓解实验性结肠炎。植物多糖的抗结肠炎活性与其相对分子质量、单糖组成、主链结构、空间构象、糖醛酸含量、结构修饰等密切相关, 但不同研究在原料来源、提取纯化、结构表征、动物模型条件等方面存在较大差异, 尚难以建立具有普适性的构效规律。目前相关研究仍以细胞和动物实验为主, 临床研究不足。未来应加强同源多糖的结构分级、定向修饰与构象调控研究, 结合多组学、肠道菌群代谢和屏障功能评价解析作用链条, 并依据标准化方案开展临床研究, 以推动植物多糖在功能性食品及结肠炎防治中的转化应用。

关键词: 植物多糖; 结肠炎; 作用机制; 肠道菌群; 构效关系

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0 引言

结肠炎是一类以结肠组织炎症为主要特征的疾病, 组织学上可见浆细胞、嗜酸性粒细胞、巨噬细胞、中性粒细胞等多种炎症细胞浸润^[1]。结肠炎的病因主要包括遗传易感性、环境因素、肠黏膜屏障损伤、肠道菌群失调、免疫反应失衡等。流行病学资料显示, 自 20 世纪 90 年代以来, 结肠炎在多个新兴工业化国家及西方国家的发病率快速上升, 呈现明显的全球化趋势^[2-5]。按病因及临床类型, 结肠炎主

要包括炎症性肠病相关结肠炎、感染性结肠炎、缺血性结肠炎、假膜性结肠炎等。临床上, 患者常以腹痛、腹泻、黏液脓血便等症状为主要表现^[6]。目前结肠炎的常用治疗药物包括氨基水杨酸盐、糖皮质激素、硫嘌呤类免疫抑制剂、生物制剂等^[7]。然而, 这些药物在临床应用中仍受到不良反应、疗效个体差异、长期用药风险等因素的限制^[8]。因此, 越来越多研究者开始关注天然产物在结肠炎防治中的潜在应用, 以实现在发挥干预作用的同时降低不良反应风险^[9]。

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多糖是一种由 10 个以上单糖经糖苷键连接而成的高分子碳水化合物,其相对分子质量从数万到数百万不等^[10]。多糖因具有调节免疫、抗炎、抗氧化、抗肿瘤、调节血糖、抗病毒、抗凝血等多种生物学活性^[11],在食品、医药等领域展现出较好的应用前景。与动物、真菌及藻类多糖相比,植物多糖来源广泛、可持续性较强、整体毒性较低,在食品和药物开发中有更好的安全性和应用可行性。近年来,针对植物多糖在调控炎症反应、肠道菌群稳态、肠黏膜屏障等方面的研究不断深入,鉴于现有相关研究主要来自葡聚糖硫酸钠(Dextran Sulfate Sodium,DSS)或 2,4,6-三硝基苯磺酸(2,4,6-Trinitrobenzenesulfonic Acid,TNBS)诱导的实验性结肠炎模型,研究内容更多涉及炎症性肠病尤其是溃疡性结肠炎相关机制^[10],本文重点围绕实验性结肠炎相关研究成果,从作用机制与构效关系两条主线梳理植物多糖缓解结肠炎研究进展,并分析现有研究的共性规律与局限,以期为植物多糖的功能评价、结构优化及后续转化研究提供参考。

1 植物多糖缓解结肠炎的作用机制

1.1 关键信号通路

近年来,植物多糖缓解结肠炎的潜在作用机制受到广泛关注,其涉及的关键信号通路主要包括 NOD 样受体家族含热蛋白结构域蛋白 3 (NLR Family Pyrin Domain Containing 3,NLRP3)及核因子 κ B (Nuclear Factor Kappa-light-chain-enhancer of Activated B Cells,NF- κ B)、丝裂原活化蛋白激酶 (Mitogen-activated Protein Kinase,MAPK)、核因子 E2 相关因子 2/血红素加氧酶-1(Nuclear Factor Erythroid 2-related Factor 2/Heme Oxygenase-1,NRF2/HO-1)、磷脂酰肌醇 3-激酶/蛋白激酶 B (Phosphoinositide 3-kinase/Protein Kinase B,PI3K/Akt)、Janus 激酶/信号转导及转录激活因子 (Janus Kinase/Signal Transducer and Activator of Transcription,JAK/STAT)等信号通路(见图 1)。

1.1.1 NLRP3 炎症小体 NLRP3 炎症小体是先天免疫系统中重要的炎症信号平台,主要由 NLRP3 蛋白、ASC 及 caspase-1 共同组装而成,可介导 caspase-1

活化,促进促炎细胞因子的成熟与释放^[12-13]。当细胞受到离子稳态失衡、溶酶体或线粒体损伤、活性氧(Reactive Oxygen Species,ROS)升高、 Ca^{2+} 信号改变等刺激时,可诱导 NLRP3 寡聚化并招募含 CARD 结构域的凋亡相关斑点样蛋白(Apoptosis-associated Speck-like Protein Containing a CARD,ASC),进而募集 pro-caspase-1 并促使其自剪切活化,最终组装成具有活性的 NLRP3 炎症小体。^[14-15]。因此,NLRP3 炎症小体异常活化被认为与炎症性肠病的发生发展密切相关,且已有研究^[16]观察到其在 DSS 诱导的结肠炎模型中可被显著激活。H. Li 等^[17]从猴头菇子实体中提取的多糖对 C57BL/6J 小鼠结肠炎具有显著的抗炎和抗氧化作用,这可能与该多糖抑制 NLRP3 炎症小体相关蛋白的活化有关。类似地,从中国土茯苓中提取的果胶多糖在 BALB/c 小鼠结肠组织中可降低 NLRP3、ASC、caspase-1 及 IL-1 β 的表达水平^[18]。此外,果胶多糖还可通过抑制 galectin-3 活性,降低 NLRP3 炎症小体的活化水平,从而降低 IL-1 β 的释放^[19]。由此可见,NLRP3 炎症小体是结肠炎炎症反应的重要调控节点。

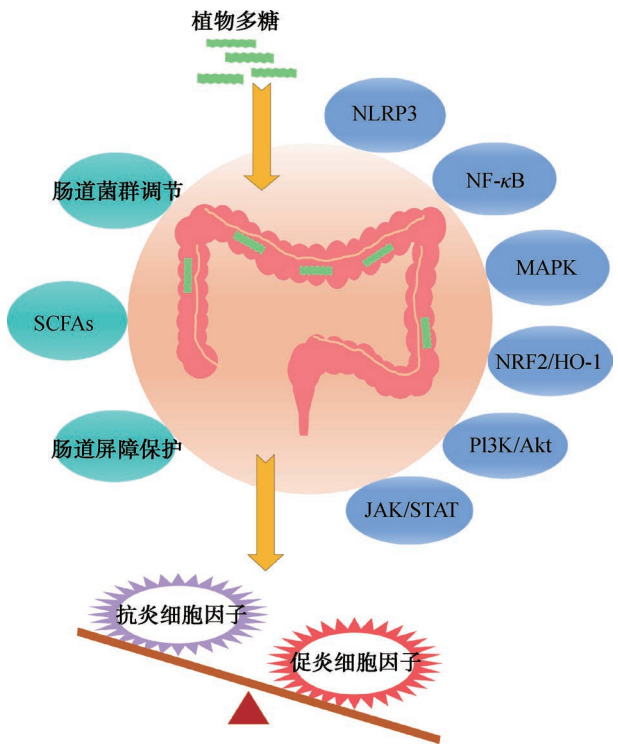


图 1 植物多糖缓解结肠炎的作用机制
Fig. 1 Mechanisms of action of plant polysaccharides in alleviating colitis

1.1.2 NF- κ B 信号通路 NF- κ B 是由 Rel 家族成员构成的转录因子复合体,主要包括 RelA (p65)、RelB、c-Rel、NF- κ B1 (p50/p105) 和 NF- κ B2 (p52/p100) 等^[20]。NF- κ B 的活化主要分为经典途径和非经典途径两种,经典途径多与急性炎症反应的放大相关,而非经典途径主要参与免疫细胞分化及慢性免疫调控过程^[21]。鉴于其在黏膜炎症放大与肠屏障损伤中的核心作用,NF- κ B 被视为结肠炎干预的重要潜在靶点。

在 DSS 诱导的结肠炎小鼠模型中,有研究^[22]报道黄芪多糖可通过抑制 TLR4/MyD88/NF- κ B 信号通路发挥保护作用,减轻炎症反应并维持肠道屏障功能。此外,NF- κ B 可促进 NLRP3、pro-IL-1 β 等炎症相关基因转录,为 NLRP3 炎症小体激活提供启动信号,从而在炎症发生与调控中发挥重要作用。黄芩多糖可通过抑制 NF- κ B、NLRP3 炎症小体相关活性缓解 DSS 诱导的实验性结肠炎^[23];山楂多糖亦可通过抑制 NF- κ B 活化并下调结肠组织促炎细胞因子表达而改善结肠炎表型^[24]。

1.1.3 MAPK 信号通路 MAPK 是一类可被多种外界刺激激活的丝氨酸/苏氨酸蛋白激酶家族,主要亚家族包括细胞外信号调节激酶(ERK)、c-Jun 氨基末端激酶(JNK)和 p38 MAPK^[25]。MAPK 信号通路通常由三级蛋白激酶级联构成,即 MAPKKK、MAPKK 和 MAPK。受到外界刺激后,上述级联依次发生磷酸化并被激活,进而促使活化的 MAPK 转位进入细胞核或在细胞质/质膜相关区域发挥作用,从而调控下游靶基因转录及相关蛋白表达,影响细胞增殖、分化、凋亡等生物学过程^[26]。此外,MAPK 还在先天免疫信号过程中发挥重要作用,且与炎症性疾病的发生发展及干预密切相关^[27]。已有研究^[28]表明,结肠炎的发生发展与 MAPK 信号通路密切相关,免疫细胞中 MAPK 信号通路的异常激活可加剧炎症反应并增加结肠炎发生风险。

多种植物多糖可通过调控 MAPK 信号通路改善结肠炎。例如,从黄心甘薯中分离获得的新型水溶性多糖可促进短链脂肪酸(Short-chain Fatty Acids,SCFAs)的产生,继而激活肠上皮细胞 G 蛋白偶联受体,并促进 MEK/ERK1/2 信号通路活化,从

而改善结肠缩短等病理表型^[29]。值得注意的是,ERK1/2 在不同细胞类型及不同炎症微环境下对结肠炎可能呈现“双刃剑”效应,即其既可能发挥黏膜保护作用,也可能在特定条件下促进炎症反应的发生发展^[30]。SCFAs 受体 GPR41/43 可参与肠上皮屏障及免疫反应调控,其生物学效应具有细胞类型和微环境依赖性^[31]。在肠上皮细胞中,GPR41/43 介导的信号可影响 ERK1/2 活性,进而调节细胞增殖、凋亡和屏障稳态。此外,植物多糖对结肠炎的干预机制并不局限于 MAPK 通路本身,其作用还可能通过与其他信号通路的交叉调控而实现,其中 MAPK 与 NF- κ B 之间的交叉调控较为常见。MAPK 家族 p38、JNK 与 ERK 既可协同介导 NF- κ B 信号通路活化,也可参与 NF- κ B 的下游转录调控^[32]。例如,南瓜多糖及其代谢产物 5-羟基吡啶乙酸可通过激活 PPAR γ 并抑制 MAPK/NF- κ B 信号通路,缓解 DSS 诱导的小鼠结肠损伤及过度炎症反应^[33]。同样,慈菇多糖也可通过调节 MAPK/NF- κ B 信号通路改善结肠炎症状^[34]。

1.1.4 NRF2/HO-1 信号通路 NRF2 是一种可响应多种应激刺激的关键转录因子。当细胞遭受氧化应激或其他有害刺激时,NRF2 从细胞质转位进入细胞核,并与抗氧化反应元件结合,启动多种抗氧化及细胞保护相关基因的转录,从而降低 ROS 介导的氧化损伤^[35]。HO-1 作为典型的氧化应激诱导酶,其表达受 NRF2 正向调控。因此,NRF2/HO-1 信号通路不仅可抵御氧化应激,还能够抑制促炎因子产生并调控多条炎症相关信号通路,在炎症性疾病干预中发挥重要作用^[36-37]。近年来,这一通路已成为氧化应激相关疾病研究的重要信号轴。

在 DSS 诱导的小鼠结肠炎模型中,肠道中紧密连接蛋白 ZO-1 与 occludin 的表达常显著下降,导致肠屏障功能受损。研究^[38]显示,芦荟多糖干预可显著提高 SCFAs 水平,并上调 NRF2/HO-1 信号通路相关表达,从而促进 ZO-1 与 occludin 的表达,改善屏障损伤。此外,蒲公英根多糖同样可通过激活 NRF2/HO-1 通路保护 DSS 诱导的结肠炎小鼠肠道屏障,其对 IEC-6 细胞的体外保护作用亦得到验证^[39]。上述结果表明,SCFAs 代谢调节与 NRF2/

HO-1 信号通路激活可能共同参与肠屏障保护过程。

1.1.5 PI3K/Akt 信号通路 PI3K 是一类可受细胞内外多种刺激激活的脂质激酶,在细胞生长、增殖、代谢、迁移、凋亡、免疫应答等过程中发挥重要作用^[40]。PI3K 被激活后可催化生成磷脂酰肌醇-3,4,5-三磷酸,从而在质膜上招募并促进 AGC 家族 Akt 的激活,主要表现为 Akt 在 Thr308 与 Ser473 位点的磷酸化增强。Akt 作为 PI3K 的关键下游效应分子,参与多种生长与代谢相关过程的调控^[41]。已有研究^[42]表明,PI3K/Akt 通路失衡与结肠黏膜损伤、肠屏障功能障碍及炎症反应加剧密切相关。因此,植物多糖改善结肠炎的作用可能部分通过调控 PI3K/Akt 信号通路实现。

X. B. Lin 等^[43]研究发现,桃胶多糖能显著增加结肠炎小鼠的结肠长度,促进 RAW264.7 和 Caco-2 细胞增殖,其作用机制与 PI3K/Akt 通路被激活相关。Z. Zhang 等^[44]通过口服葛根多糖(*Pueraria lobata* Polysaccharide, PLP)治疗 DSS 诱导的结肠炎小鼠,并对 Caco-2 细胞进行体外研究,发现 PLP 可通过抑制 PI3K/Akt 信号通路缓解结肠炎并保护上皮细胞。此外,另一项研究^[45]表明,*Persicaria hydropiper* 来源的中性多糖可调节 Akt/PI3K/mTOR 和 MAPK 信号通路,从而改善 LPS 诱导的肠道屏障损伤。

1.1.6 JAK/STAT 信号通路 STAT 蛋白是一类位于细胞质内的转录因子家族,主要包括 STAT1—4、STAT5A、STAT5B 和 STAT6,广泛参与细胞增殖、分化、凋亡、免疫调控等多种生物学过程。当 STAT 信号发生缺陷或异常持续激活时,往往会诱发多种疾病^[46]。其中,STAT3 是 IL-6 介导急性期反应的重要信号分子,可调控炎症相关细胞的功能,并与 NF- κ B 通路存在交叉调控,进而影响炎症反应的强度与持续时间。

JAK/STAT 通路是经典的细胞因子信号转导通路,主要由细胞膜受体、JAK 及 STAT 蛋白组成。细胞因子与受体结合后激活 JAK,继而介导 STAT 蛋白磷酸化,磷酸化 STAT 能形成二聚体并转位进入细胞核,从而调控靶基因表达,实现细胞因子信号传递^[28]。因此,阐明 JAK/STAT 通路的异常调控有助

于解析疾病机制并寻找潜在干预靶点。

植物多糖缓解结肠炎与 JAK/STAT 通路的调节密切相关。例如,在 DSS 诱导的小鼠结肠炎模型中可见明显炎症损伤,而人参多糖可下调磷酸化 JAK2、STAT1 及 NLRP3 炎症小体相关蛋白的表达,抑制 JAK/STAT/NLRP3 通路的过度激活,减少促炎细胞因子分泌,从而改善结肠炎症状^[47]。此外,Y. F. Chen 等^[48]研究发现,白桦茸多糖可通过抑制 JAK/STAT 通路被激活,调节 Th1、Th2、Th17 与 Treg 细胞的平衡,表现出缓解结肠炎的潜力。进一步研究^[49]表明,JAK/STAT 通路在结肠炎相关免疫信号调控中发挥重要作用,其异常激活可导致免疫细胞亚群失衡并促进炎症反应,进而参与结肠炎的发生发展。

综上所述,植物多糖可通过多条信号通路发挥抗结肠炎作用,其总体效应主要体现在抑制炎症级联反应、缓解氧化应激、调节免疫反应等方面。

1.2 肠道菌群重塑

肠道菌群功能紊乱被认为是结肠炎发生发展的重要因素之一,这一过程往往伴随菌群多样性下降,进而削弱肠道屏障完整性与免疫稳态维持能力。从宿主健康效应角度看,部分肠道菌群成员可表现为潜在有益菌或条件致病菌,但其作用具有菌株及宿主状态依赖性^[50]。部分益生菌可对溃疡性结肠炎起到保护作用。例如,酵母菌、短双歧杆菌(*Bifidobacterium breve*)和乳酸杆菌(*Lactobacillus*)可增强肠道屏障并促进屏障相关蛋白表达^[51-54]。同时,长双歧杆菌(*Bifidobacterium longum*)也可通过抑制 TLR4/NF- κ B 信号通路减少炎症反应^[55]。相反,潜在致病菌如具核梭杆菌(*Fusobacterium nucleatum*)可破坏肠道屏障^[56],屎肠球菌(*Enterococcus faecium*)可诱导炎症因子产生^[57]。因此,调节肠道菌群作用是植物多糖缓解结肠炎的重要途径之一。

C. Nie 等^[58]研究发现,苦瓜多糖(*Momordica charantia* Polysaccharide, MCP)可增加肠道中 *Parabacteroides*、*Allobaculum* 等益生菌的丰度,从而缓解 DSS 诱导的结肠炎。此外,MCP 还可通过抑制 LPS/TLR4/NF- κ B 与 MAPK 信号通路发挥抗炎效果。佛手柑多糖则可减少 *Alistipes* 和 *Bacteroides* 的丰度,增

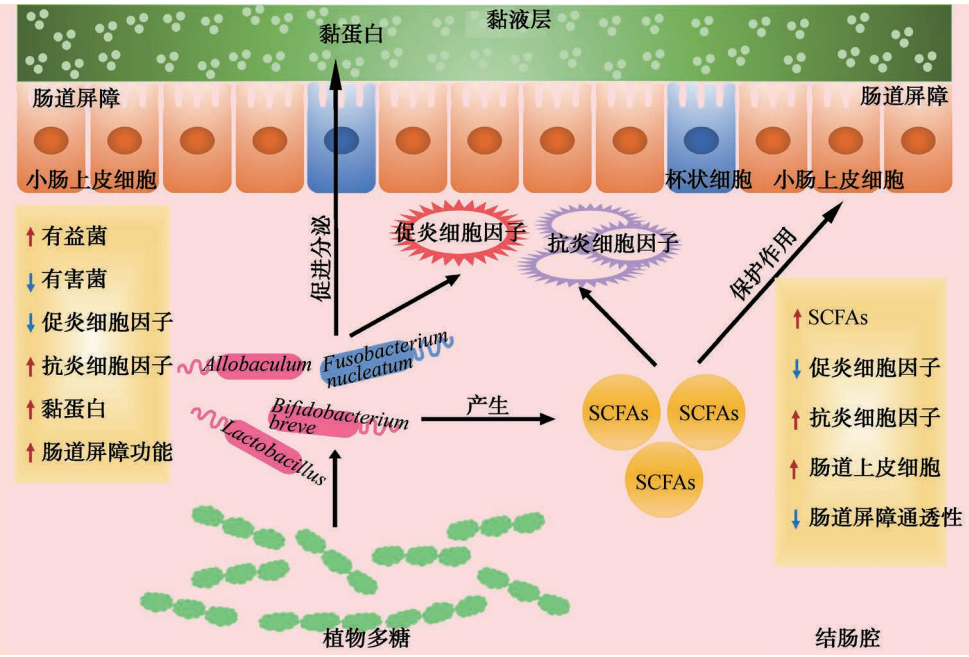


图 2 植物多糖通过调节肠道菌群/SCFAs 缓解结肠炎的机制示意图

Fig. 2 Schematic illustration of the mechanisms by which plant polysaccharides alleviate colitis by modulating the gut microbiota/SCFAs

加 *Butyrivibrio*、*Bifidobacterium*、*Lachnoclostridium* 和 *Romboutsia* 的丰度,促进 SCFAs 生成,显示出较好的抗结肠炎作用^[59]。

在 2,4,6-三硝基苯磺酸(2,4,6-Trinitrobenzene Sulfonic Acid, TNBS)诱导的结肠炎小鼠模型中,荞麦多糖可降低 *Pasteurellaceae* 和 *Oscillospiraceae* 的丰度,并促进 SCFAs 生成;其抗炎作用同样与 NF- κ B 和 MAPK 信号通路的抑制相关^[60]。总体而言,在结肠炎发生发展过程中,炎症反应、信号通路调控、肠道菌群变化与 SCFAs 代谢之间存在紧密的相互作用。

1.3 SCFAs 代谢调节

SCFAs 是肠道菌群发酵膳食成分,尤其是膳食纤维/多糖产生的一类有机酸,在维持肠道稳态与黏膜屏障功能中发挥重要作用,并与溃疡性结肠炎的发生发展密切相关。SCFAs 主要包括乙酸、丙酸和丁酸。其中,乙酸具有来源广、稳定性较好且毒性较低等特点,在维持肠道屏障完整性及发挥对肠上皮细胞的抗炎保护作用方面具有重要意义^[61]。丙酸可促进上皮细胞迁移并有助于创伤愈合,从而在一定程度上改善结肠炎相关损伤。相比之下,丁酸在结肠腔内含量相对较高,已被证实具有抗炎及潜在抗肿瘤作用^[62]。

除作为代谢产物外,SCFAs 还可作为信号分子,与 G 蛋白偶联受体(G Protein-coupled Receptor, GPCR)结合并触发下游级联反应,进而通过调节免疫功能来缓解肠道炎症^[63]。由于植物多糖在人体内难以被直接消化吸收,其中相当一部分可经肠道微生物代谢与发酵生成 SCFAs,因此“多糖—肠道菌群—SCFAs”被认为是其改善肠道炎症的重要机制之一^[64]。例如,HAW1-2 可增加 *Faecalibaculum*、*Alistipes*、*Odoribacter* 等主要 SCFAs 产生菌的丰度,并提高 DSS 诱导结肠炎小鼠肠道中 SCFAs 的水平。此外,四方藤多糖可显著提升小鼠粪便中乙酸、丁酸和异戊酸含量,并激活 GPR41/43 相关信号,增强肠道免疫调控能力,从而发挥抗炎效果^[65]。

1.4 肠黏膜屏障保护

除炎症反应、肠道菌群失衡及 SCFAs 代谢异常外,肠道屏障受损同样是结肠炎的重要病理特征之一。肠道屏障在维持肠腔内外环境稳态、协调促炎因子与抗炎因子平衡方面发挥关键作用^[66]。其中,肠上皮细胞之间的紧密连接(Tight Junctions, TJ)是肠道屏障的核心结构,可在提高屏障密封性的同时降低通透性,从而限制肠腔内有害物质跨越上皮细胞进入黏膜层,进而减轻炎症反应^[67]。已有研

究^[68]表明,SCFAs与TJ蛋白表达及屏障稳定性密切相关,可促进屏障功能恢复。植物多糖由于结构多样、难以被直接消化吸收,常在肠道中被微生物降解发酵,其代谢过程及产物与屏障修复之间存在紧密联系。

目前,已有多种植物多糖被报道可通过上调TJ蛋白、抑制上皮细胞凋亡或增强黏液层屏障等方式改善屏障损伤。例如,姜多糖(*Ginger Polysaccharides*, GP)可恢复肠上皮细胞中ZO-1与occludin的表达水平,减轻上皮细胞凋亡,促进屏障修复并缓解炎症反应;同时,GP还具有调节肠道菌群的作用^[69]。姬松茸多糖同样可上调ZO-1与occludin蛋白表达,并显著提高MUC2蛋白水平^[70]。MUC2作为由杯状细胞分泌并覆盖于上皮细胞表面的凝胶样黏蛋白,是黏液层的重要组成部分,可形成抵御细菌黏附与入侵的第一道屏障^[71]。银耳多糖(*Tremella Fuciformis Polysaccharides*, TFP)可减少DSS诱导小鼠结肠溃疡面积,减轻黏膜炎症细胞浸润,并上调GPA33、ZO-1、occludin、MUC2及Clca1等与屏障相关的蛋白表达;在LPS诱导的Caco-2细胞屏障损伤模型中,TFP亦表现出类似的保护作用^[72]。

2 植物多糖抗结肠炎活性的构效关系

植物多糖的生物活性受多层级结构特征共同影响,构效关系是其定向开发和质量控制的重要基础。现有研究主要从相对分子质量、单糖组成、主链结构、空间构象、糖醛酸含量、结构修饰等方面讨论其抗结肠炎活性。但需要注意的是,不同来源多糖在提取纯化方法、结构复杂度、给药剂量及实验模型等方面差异较大,因此跨来源比较所得结论更多体现相关性,尚不能直接视为普适的因果性构效规律。

2.1 相对分子质量

植物多糖的抗结肠炎活性可能与其相对分子质量有关。以白蘑菇多糖(*Agaricus bisporus Polysaccharide*, ABP)为例,与高相对分子质量组分ABP-1相比,低相对分子质量组分ABP-2在DSS诱导的结肠炎小鼠中显示出更明显的干预效果,它不仅能明显改善结肠组织病理损伤,还可促进肠道屏障修复,下调IL-6、IFN- γ 等炎症因子表达,优化肠道菌群结

构,增加益生菌并减少有害菌^[73]。类似结果也见于黄芪多糖研究^[74]中,与高相对分子质量组分APS2相比,低相对分子质量组分APS1能显著改善小鼠体重减轻和结肠缩短,减少结肠病变。此外,相较于高相对分子质量猴头菇多糖(*Hericium erinaceus Polysaccharide*, HEP)组分HEP-70,低相对分子质量组分HEP-30和HEP-50对结肠炎的改善作用更显著^[75]。因此,较低相对分子质量的植物多糖可能具有更好的抗结肠炎活性。

2.2 单糖组成

单糖组成是影响植物多糖抗结肠炎活性的重要结构基础之一,不同单糖及其组合可能通过不同途径影响抗结肠炎活性。以半乳糖为主要单糖的蒸制黄精多糖和黑牛肝菌多糖,可减轻结肠炎小鼠体内的炎症和肠黏膜损伤^[76-77]。含有葡萄糖和甘露糖的含硒茶多糖可显著上调TJ蛋白occludin、claudin-1和ZO-1的表达,同时降低炎症因子水平,并减少有害菌丰度、增加有益菌丰度,从而改善肠道微环境^[78]。此外,由葡萄糖与半乳糖共同组成的薯蓣多糖可通过缓解炎症和氧化应激,减轻肠黏膜结构损伤并促进组织修复^[79]。这些结果表明,单糖组成可能参与调节植物多糖的抗结肠炎活性,但仍需结合糖苷键类型、支化程度、空间构象等结构信息综合判断。

2.3 主链结构

在植物多糖缓解结肠炎的研究中,以D-半乳糖或 β -D-葡萄糖为主的主链结构研究较多,此类多糖对DSS诱导的结肠炎具有一定缓解作用。例如,桃胶多糖属于AG II型半乳聚糖,其主链为1,6- β -D-半乳糖,可显著抑制结肠炎小鼠体内的炎症因子分泌^[80]。类似地,蒸制黄精多糖的主链为1,4- α -D-半乳糖,同样在结肠炎模型中表现出一定的缓解作用^[76]。以 β -D-葡萄糖为主链的植物多糖也具有类似的抗结肠炎效应,如竹笋多糖和山药多糖的主链为1,4- β -D-葡萄糖,二者均可通过抑制炎症反应、促进屏障修复和调节肠道菌群组成改善结肠炎相关指标。Y. Q. Sun等^[81]分离出一种主链由1,3- β -D-葡萄糖和1,6- α -D-半乳糖组成的植物多糖,研究发现该多糖可显著缓解DSS诱导的小鼠结肠炎症状。总之,植物多糖的主链结构可能影响其空间构

象,进而影响其在肠道中的作用效果。

2.4 空间构象

植物多糖具有不同的空间构象,如线性、球形、聚集成团、网络结构等,这些结构可影响植物多糖在肠道中的存在形态,进而影响其抗结肠炎活性。有研究^[82]表明,部分构象相对简单的植物多糖显示出良好的抗结肠炎活性,如以线性 1,4-β-D-甘露糖为主链的铁皮石斛多糖可抑制结肠炎小鼠体内的炎症因子表达、恢复菌群多样性并促进 SCFAs 产生。黄心甘薯多糖也属于线性植物多糖,能缓解结肠病理损伤并改善结肠缩短现象^[29]。以线性 1,4-α-半乳糖醛酸为主链的诺丽果多糖可增加黏液分泌并保护肠黏膜屏障^[83]。呈球形分子结构的人参多糖 (Ginseng Polysaccharides, GPs) 可通过增加有益菌丰度、调节色氨酸代谢来改善结肠炎症状^[84]。桃胶多糖在 0.2 mol/L NaCl 溶液中呈现柔性链卷曲的构象,给药后可降低结肠炎小鼠体内的促炎细胞因子水平并改善结肠氧化损伤^[80]。南瓜多糖呈不规则块状并形成分支或交联网络结构,其可通过抑制 TLR4/NF-κB 和 MAPK 通路减轻 DSS 诱导的结肠炎症状^[85]。总之,植物多糖的空间构象可能影响其在肠道中的作用方式,进而影响其抗结肠炎活性。

2.5 糖醛酸含量

糖醛酸是部分酸性多糖的重要组成单元,其含量与植物多糖的自由基清除能力及抗氧化活性密切相关^[86]。富含糖醛酸的多糖可能具有减轻炎症、保护肠黏膜、调节肠道菌群等作用。例如,南板蓝根来源的水溶性多糖 (Baphicacanthis cusiae Polysaccharide, BCP) 中 BCP-1 和 BCP-2 含有不同比例的糖醛酸,虽然二者均可缓解结肠炎症状,但在体内的治疗效果存在一定差异。糖醛酸含量更高的 BCP-2 在抗炎和抗氧化活性方面优于 BCP-1,说明植物多糖的抗结肠炎活性可能与糖醛酸含量相关^[87]。酸枣仁中提取的 3 种酸性多糖均富含糖醛酸,其中,糖醛酸含量最高的组分对肠上皮屏障保护作用最为显著^[88]。桑椹果实酶辅助提取的 RG-I 型多糖以糖醛酸为主要成分,能够促进 *Bacteroides thetaiotaomicron* 生长,可从微生态层面改善肠道稳态^[89]。综上所述,糖醛酸含量较高的酸性植物多糖可能具有较

好的抗氧化、黏膜屏障保护及菌群调节作用。

2.6 结构修饰

结构修饰是改变植物多糖生物活性的一种有效方式 (见图 3)。硫酸化、乙酰化及羧甲基化是常见的植物多糖化学修饰手段,可影响其特性和功能^[90]。在结肠炎干预研究中,这些修饰可能通过改变植物多糖的电荷特征、溶解性、分子构象等影响其生物活性。例如,在 DSS 诱导的结肠炎模型中,来源于褐藻 *Ishige okamurae* 的硫酸化多糖可降低结肠组织中促炎细胞因子表达,提高抗炎细胞因子水平,同时调节肠道菌群^[91]。在 TNBS 诱导的结肠炎模型中,来源于海洋红藻 *Hypnea musciformis* 的硫酸化多糖可显著减少结肠组织中中性粒细胞的浸润面积,并减轻肠黏膜的氧化损伤^[92]。此外,羧甲基修饰也能增强植物多糖的抗炎活性。例如,采用超声波提取和 NaOH-氯乙酸方法制备的木瓜多糖 (*Chaenomeles sinensis* Polysaccharides, CSP) 及其羧甲基化衍生物 (CSP-M) 均可通过抑制炎症和氧化应激缓解溃疡性结肠炎,其中 CSP-M 的治疗效果更明显^[93]。由此可知,不同结构修饰形式可能会提高植物多糖的抗结肠炎活性,未来可进一步比较不同修饰方式及修饰程度对其活性的影响。

综上可知,植物多糖的抗结肠炎活性与其相对分子质量、单糖组成、主链结构、空间构象、糖醛酸含量、结构修饰等因素相关。然而,由于原料来源、提

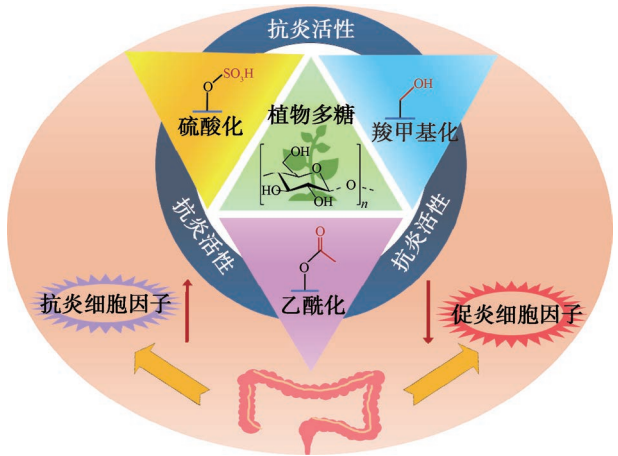


图 3 经硫酸化、羧甲基化及乙酰化修饰的植物多糖缓解结肠炎作用机制示意图

Fig. 3 Schematic illustration of the anti-colitis mechanism of plant polysaccharides modified through sulfation, carboxymethylation and acetylation

取纯化、结构表征和动物模型参数差异较大,尚难以建立结构与活性之间稳定的一一对应关系。后续研究宜优先在同一起来源多糖体系中开展相对分子质量分级、定向修饰或构象调控的对照研究,并结合菌群代谢物、肠屏障指标和炎症信号通路进行多维评价,以验证结构变化与生物活性之间的联系,进一步提炼可重复、可外推的构效规律。

3 结论与展望

本文综述了植物多糖抗结肠炎的研究进展,重点梳理其结构特征、作用机制及结构-活性关系,发现植物多糖对实验性结肠炎具有一定的缓解作用,其机制主要涉及 NLRP3 炎症小体、NF- κ B、MAPK、NRF2/HO-1、PI3K/Akt、JAK/STAT 等信号通路的调节,同时还可通过重塑肠道菌群结构、促进 SCFAs 等微生物代谢产物生成,以及增强肠黏膜屏障功能等多种途径发挥作用。植物多糖的抗结肠炎活性与其相对分子质量、单糖组成、主链结构、空间构象、糖醛酸含量、结构修饰等密切相关,但不同研究之间的原料来源、提取纯化、结构表征和动物模型参数差异较大,因此现阶段更适合将这些结构因素视为潜在影响因素,而非已经确立的普适构效规律。

未来应在同一起来源多糖体系中开展结构分级、定向修饰和构象调控,通过统一实验模型与评价指标验证结构变化与效应变化之间的关系;同时结合宏基因组、代谢组等多组学技术,解析多糖结构—菌群利用—代谢物变化—宿主信号与屏障的作用链条。鉴于现有数据主要来自细胞和动物实验,还应进一步加强安全性、体内行为及高质量临床研究,并关注功能性食品应用中的有效剂量、加工稳定性和质量控制问题,从而提高植物多糖抗结肠炎研究的可重复性与转化价值。

参考文献:

- [1] Wang X J, Huang S W, Zhang M L, et al. Gegen Qinlian decoction activates AhR/IL-22 to repair intestinal barrier by modulating gut microbiota-related tryptophan metabolism in ulcerative colitis mice[J]. *Journal of Ethnopharmacology*, 2023, 302: 115919.
- [2] Kobayashi T, Siegmund B, Le Berre C, et al. Ulcerative colitis[J]. *Nature Reviews Disease Primers*, 2020, 6(1): 74.
- [3] Nakase H, Sato N, Mizuno N, et al. The influence of cytokines on the complex pathology of ulcerative colitis[J]. *Autoimmunity Reviews*, 2022, 21(3): 103017.
- [4] Li J W, Niu C X, Ai H H, et al. TSP50 attenuates DSS-induced colitis by regulating TGF- β signaling mediated maintenance of intestinal mucosal barrier integrity[J]. *Advanced Science*, 2024, 11(11): e2305893.
- [5] Dong D F, Su T X, Chen W, et al. *Clostridioides difficile* aggravates dextran sulfate solution (DSS)-induced colitis by shaping the gut microbiota and promoting neutrophil recruitment[J]. *Gut Microbes*, 2023, 15(1): 2192478.
- [6] Zhao Y X, Luan H F, Gao H, et al. Gegen Qinlian decoction maintains colonic mucosal homeostasis in acute/chronic ulcerative colitis *via* bidirectionally modulating dysregulated Notch signaling[J]. *Phytomedicine*, 2020, 68: 153182.
- [7] Armuzzi A, Liguori G. Quality of life in patients with moderate to severe ulcerative colitis and the impact of treatment: a narrative review[J]. *Digestive and Liver Disease*, 2021, 53(7): 803–808.
- [8] Wisniewski A, Kirchgessner J, Seksik P, et al. Increased incidence of systemic serious viral infections in patients with inflammatory bowel disease associates with active disease and use of thiopurines[J]. *United European Gastroenterology Journal*, 2020, 8(3): 303–313.
- [9] Yuan D, Li C, Huang Q, et al. Current advances in the anti-inflammatory effects and mechanisms of natural polysaccharides[J]. *Critical Reviews in Food Science and Nutrition*, 2023, 63(22): 5890–5910.
- [10] 冀晓龙, 郭建行, 田静源, 等. 植物多糖降解方法及降解产物特性研究进展[J]. *轻工学报*, 2023, 38(3): 55–62.
- [11] Ji X L, Guo J H, Tian J Y, et al. Research progress on degradation methods and product properties of plant polysaccharides[J]. *Journal of Light Industry*, 2023, 38(3): 55–62.
- [12] 冀晓龙, 王治雯, 潘飞兵, 等. 槟榔多糖对 Caco-2 细胞氧化损伤的保护作用[J]. *轻工学报*, 2022, 37(6): 18–24.
- [13] Ji X L, Wang Z W, Pan F B, et al. Protective effect of *Areca catechu* L. polysaccharide on Caco-2 cells oxidative injury[J]. *Journal of Light Industry*, 2022, 37(6): 18–24.
- [14] Xiao T S. Innate immunity and inflammation[J]. *Cellular and Molecular Immunology*, 2017, 14(1): 1–3.
- [15] Fu J N, Wu H. Structural mechanisms of NLRP3 inflammasome assembly and activation[J]. *Annual Review of Immunology*, 2023, 41: 301–316.
- [16] Xian H X, Watari K, Sanchez-Lopez E, et al. Oxidized DNA fragments exit mitochondria *via* mPTP- and VDAC-dependent channels to activate NLRP3 inflammasome and interferon signaling[J]. *Immunity*, 2022, 55(8): 1370–1385. e8.

- [15] Wang F H, Liang Q Q, Ma Y X, et al. Silica nanoparticles induce pyroptosis and cardiac hypertrophy *via* ROS/NLRP3/Caspase-1 pathway [J]. *Free Radical Biology and Medicine*, 2022, 182: 171–181.
- [16] Bauer C, Duewell P, Mayer C, et al. Colitis induced in mice with dextran sulfate sodium (DSS) is mediated by the NLRP3 inflammasome [J]. *Gut*, 2010, 59(9): 1192–1199.
- [17] Li H, Feng J X, Liu C, et al. Polysaccharides from an edible mushroom, *Hericium erinaceus*, alleviate ulcerative colitis in mice by inhibiting the NLRP3 inflammasomes and reestablish intestinal homeostasis [J]. *International Journal of Biological Macromolecules*, 2024, 267: 131251.
- [18] Pan X L, Wang H Y, Zheng Z M, et al. Pectic polysaccharide from *Smilax china* L. ameliorated ulcerative colitis by inhibiting the galectin-3/NLRP3 inflammasome pathway [J]. *Carbohydrate Polymers*, 2022, 277: 118864.
- [19] Simovic Markovic B, Nikolic A, Gazdic M, et al. Galectin-3 plays an important pro-inflammatory role in the induction phase of acute colitis by promoting activation of NLRP3 inflammasome and production of IL-1 β in macrophages [J]. *Journal of Crohn's & Colitis*, 2016, 10(5): 593–606.
- [20] Montaña D E, Hartung S, Wich M, et al. The TLR-NF- κ B axis contributes to the monocytic inflammatory response against a virulent strain of *Lichtheimia corymbifera*, a causative agent of invasive mucormycosis [J]. *Frontiers in Immunology*, 2022, 13: 882921.
- [21] McDaniel D K, Eden K, Ringel V M, et al. Emerging roles for noncanonical NF- κ B signaling in the modulation of inflammatory bowel disease pathobiology [J]. *Inflammatory Bowel Diseases*, 2016, 22(9): 2265–2279.
- [22] Lv (Lyu) J, Zhang Y H, Tian Z Q, et al. *Astragalus* polysaccharides protect against dextran sulfate sodium-induced colitis by inhibiting NF- κ B activation [J]. *International Journal of Biological Macromolecules*, 2017, 98: 723–729.
- [23] Cui L, Wang W, Luo Y, et al. Polysaccharide from *Scutellaria baicalensis* Georgi ameliorates colitis *via* suppressing NF- κ B signaling and NLRP3 inflammasome activation [J]. *International Journal of Biological Macromolecules*, 2019, 132: 393–405.
- [24] Guo C L, Wang Y Q, Zhang S H, et al. *Crataegus pinnatifida* polysaccharide alleviates colitis *via* modulation of gut microbiota and SCFAs metabolism [J]. *International Journal of Biological Macromolecules*, 2021, 181: 357–368.
- [25] Park M Y, Ha S E, Kim H H, et al. Scutellarein inhibits LPS-induced inflammation through NF- κ B/MAPKs signaling pathway in RAW264. 7 cells [J]. *Molecules*, 2022, 27(12): 3782.
- [26] Gomez-Osuna A, Calatrava V, Galvan A, et al. Identification of the MAPK cascade and its relationship with nitrogen metabolism in the green *Alga Chlamydomonas reinhardtii* [J]. *International Journal of Molecular Sciences*, 2020, 21(10): 3417.
- [27] Wen Z Y, Li M Y, Meng J, et al. Genome-wide identification of the MAPK and MAPKK gene families in response to cold stress in *Prunus mume* [J]. *International Journal of Molecular Sciences*, 2023, 24(10): 8829.
- [28] Li C L, Li Z Q, Zheng Y. Natural products targeting the MAPK signaling pathway: potential options for ulcerative colitis treatment [J]. *Frontiers in Pharmacology*, 2025, 16: 1721584.
- [29] Feng X J, Du C, Wang C L. Structural characterization of polysaccharide from yellow sweet potato and ameliorates DSS-induced mice colitis by active GPR41/MEK/ERK 1/2 signaling pathway [J]. *International Journal of Biological Macromolecules*, 2021, 192: 278–288.
- [30] Vukelić I, Detel D, Batičić L, et al. Luteolin ameliorates experimental colitis in mice through ERK-mediated suppression of inflammation, apoptosis and autophagy [J]. *Food and Chemical Toxicology*, 2020, 145: 111680.
- [31] Kim M H, Kang S G, Park J H, et al. Short-chain fatty acids activate GPR41 and GPR43 on intestinal epithelial cells to promote inflammatory responses in mice [J]. *Gastroenterology*, 2013, 145(2): 396–406. e10.
- [32] Han J M, Lee E K, Gong S Y, et al. *Sparassis crispa* exerts anti-inflammatory activity *via* suppression of TLR-mediated NF- κ B and MAPK signaling pathways in LPS-induced RAW264. 7 macrophage cells [J]. *Journal of Ethnopharmacology*, 2019, 231: 10–18.
- [33] Wu M L, Wang Q, Li X D, et al. Gut microbiota-derived 5-hydroxyindoleacetic acid from pumpkin polysaccharides supplementation alleviates colitis *via* MAPKs-PPAR γ /NF- κ B inhibition [J]. *International Journal of Biological Macromolecules*, 2024, 264: 130385.
- [34] Feng Y Q, Chen S M, Song Y T, et al. A novel *Sagittaria sagittifolia* L. polysaccharides mitigate DSS-induced colitis *via* modulation of gut microbiota and MAPK/NF- κ B signaling pathways [J]. *International Journal of Biological Macromolecules*, 2024, 254(Pt 3): 127835.
- [35] Shu L, Fu H J, Pi A W, et al. Protective effect of andrographolide against ulcerative colitis by activating Nrf2/HO-1 mediated antioxidant response [J]. *Frontiers in Pharmacology*, 2024, 15: 1424219.
- [36] Liu M L, Wen H X, Zuo L G, et al. Bryostatin-1 attenuates intestinal ischemia/reperfusion-induced intestinal barrier dysfunction, inflammation, and oxidative stress *via* activation of Nrf2/HO-1 signaling [J]. *FASEB Journal*, 2023, 37(6): e22948.
- [37] Sun Y Y, Zhu H J, Zhao R Y, et al. Remote ischemic conditioning attenuates oxidative stress and inflammation *via* the Nrf2/HO-1 pathway in MCAO mice [J]. *Redox Biology*, 2023, 66: 102852.
- [38] Liu C, Hua H Y, Zhu H K, et al. *Aloe* polysaccharides

- ameliorate acute colitis in mice *via* Nrf2/HO-1 signaling pathway and short-chain fatty acids metabolism[J]. International Journal of Biological Macromolecules, 2021, 185: 804–812.
- [39] Yan S K, Yin L J, Dong R. Inhibition of IEC-6 cell proliferation and the mechanism of ulcerative colitis in C57BL/6 mice by dandelion root polysaccharides[J]. Foods, 2023, 12(20): 3800.
- [40] Krimmer S G, Schlessinger J. Unveiling molecular insights into the mechanism of activation of oncogenic phosphoinositide 3-kinase mutants[J]. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119(50): e2218237119.
- [41] Dornan G L, Burke J E. Molecular mechanisms of human disease mediated by oncogenic and primary immunodeficiency mutations in class IA phosphoinositide 3-kinases[J]. Frontiers in Immunology, 2018, 9: 575.
- [42] Niu S Y, Zhang Y. Targeting the PI3K/Akt pathway for the treatment of ulcerative colitis: integrative regulatory features of traditional Chinese medicine[J]. Frontiers in Pharmacology, 2025, 16: 1620138.
- [43] Lin X B, Lan M Y, Xu C, et al. Peach gum polysaccharides promotes epithelial proliferation to attenuate ulcerative colitis by PI3K/AKT pathway[J]. Journal of Functional Foods, 2023, 107: 105662.
- [44] Zhang Z, Cui Y S, Ouyang H, et al. *Radix Pueraria Lobata* polysaccharide relieved DSS-induced ulcerative colitis through modulating PI3K signaling[J]. Journal of Functional Foods, 2023, 104: 105514.
- [45] Cheng X X, Zhu Y H, Huang J H, et al. A neutral polysaccharide from *Persicaria hydropiper* (L.) Spach ameliorates lipopolysaccharide-induced intestinal barrier injury *via* regulating the gut microbiota and modulating AKT/PI3K/mTOR and MAPK signaling pathways[J]. Journal of Ethnopharmacology, 2024, 320: 117403.
- [46] Long D, Mao C H, Zhang W, et al. Natural products for the treatment of ulcerative colitis: focus on the JAK/STAT pathway[J]. Frontiers in Immunology, 2025, 16: 1538302.
- [47] Li Y G, Yu P, Fu W W, et al. Polysaccharides from *Panax ginseng* C. A. Meyer alleviated DSS-induced IBD by inhibiting JAK2/STAT1/NLPR3 inflammasome signalling pathway in mice[J]. Journal of Functional Foods, 2022, 91: 105013.
- [48] Chen Y F, Zheng J J, Qu C, et al. *Inonotus obliquus* polysaccharide ameliorates dextran sulphate sodium induced colitis involving modulation of Th1/Th2 and Th17/Treg balance[J]. Artificial Cells, Nanomedicine, and Biotechnology, 2019, 47(1): 757–766.
- [49] Fan L N, Qi Y D, Qu S W, et al. *B. adolescentis* ameliorates chronic colitis by regulating Treg/Th2 response and gut microbiota remodeling[J]. Gut Microbes, 2021, 13(1): 1826746.
- [50] Yang Y P, Zheng X J, Wang Y Q, et al. Human fecal microbiota transplantation reduces the susceptibility to dextran sulfate sodium-induced germ-free mouse colitis[J]. Frontiers in Immunology, 2022, 13: 836542.
- [51] Zeng X J, Li X J, Yue Y, et al. Ameliorative effect of *Saccharomyces cerevisiae* JKSP39 on *Fusobacterium nucleatum* and dextran sulfate sodium-induced colitis mouse model[J]. Journal of Agricultural and Food Chemistry, 2022, 70(44): 14179–14192.
- [52] Mu Z Y, Yang Y J, Xia Y J, et al. Probiotic yeast BR14 ameliorates DSS-induced colitis by restoring the gut barrier and adjusting the intestinal microbiota[J]. Food & Function, 2021, 12(18): 8386–8398.
- [53] Li Y Q, Xu H T, Zhou L Y, et al. *Bifidobacterium breve* protects the intestinal epithelium and mitigates inflammation in colitis *via* regulating the gut microbiota-choleic acid pathway[J]. Journal of Agricultural and Food Chemistry, 2024, 72(7): 3572–3583.
- [54] Al-Sadi R, Nighot P, Nighot M, et al. *Lactobacillus acidophilus* induces a strain-specific and toll-like receptor 2-dependent enhancement of intestinal epithelial tight junction barrier and protection against intestinal inflammation[J]. The American Journal of Pathology, 2021, 191(5): 872–884.
- [55] Chen Y, Chen H Q, Ding J H, et al. *Bifidobacterium longum* ameliorates dextran sulfate sodium-induced colitis by producing conjugated linoleic acid, protecting intestinal mechanical barrier, restoring unbalanced gut microbiota, and regulating the toll-like receptor-4/nuclear factor- κ B signaling pathway[J]. Journal of Agricultural and Food Chemistry, 2021, 69(48): 14593–14608.
- [56] Liu L, Liang L P, Yang C H, et al. Extracellular vesicles of *Fusobacterium nucleatum* compromise intestinal barrier through targeting RIPK1-mediated cell death pathway[J]. Gut Microbes, 2021, 13(1): 1902718.
- [57] Seishima J, Iida N, Kitamura K, et al. Gut-derived *Enterococcus faecium* from ulcerative colitis patients promotes colitis in a genetically susceptible mouse host[J]. Genome Biology, 2019, 20(1): 252.
- [58] Nie C, Zhao Y Y, Wang P J, et al. *Momordica charantia* Polysaccharide intervention ameliorates the symptoms of dextran sulfate sodium (DSS)-induced colitis by modulating gut microbiota and inhibiting inflammation[J]. Journal of Functional Foods, 2024, 112: 105970.
- [59] Yang Y R, Zhang Y W, Song J P, et al. Bergamot polysaccharides relieve DSS-induced ulcerative colitis *via* regulating the gut microbiota and metabolites[J]. International Journal of Biological Macromolecules, 2023, 253: 127335.
- [60] Yang J Y, Chen S Y, Wu Y H, et al. Ameliorative effect of

- buckwheat polysaccharides on colitis *via* regulation of the gut microbiota [J]. International Journal of Biological Macromolecules, 2023, 227: 872–883.
- [61] Deleu S, Arnauts K, Deprez L, et al. High acetate concentration protects intestinal barrier and exerts anti-inflammatory effects in organoid-derived epithelial monolayer cultures from patients with ulcerative colitis [J]. International Journal of Molecular Sciences, 2023, 24(1): 768.
- [62] Zhang H Q, Hao Z H, Zhang R Y, et al. *Artemisia argyi* polyphenols Attenuates DSS-induced colitis in mice by regulating the structural composition of gut microbiota [J]. Phytomedicine, 2024, 132: 155897.
- [63] Parada Venegas D, de la Fuente M K, Landskron G, et al. Short chain fatty acids (SCFAs)-mediated gut epithelial and immune regulation and its relevance for inflammatory bowel diseases [J]. Frontiers in Immunology, 2019, 10: 277.
- [64] Bai Y, Zhou X J, Zhao J B, et al. Sources of dietary fiber affect the SCFA production and absorption in the hindgut of growing pigs [J]. Frontiers in Nutrition, 2021, 8: 719935.
- [65] Lin Y, Lv (Lyu) Y S, Mao Z A, et al. Polysaccharides from *Tetrastigma Hemsleyanum* Diels et Gilg ameliorated inflammatory bowel disease by rebuilding the intestinal mucosal barrier and inhibiting inflammation through the SCFA-GPR41/43 signaling pathway [J]. International Journal of Biological Macromolecules, 2023, 250: 126167.
- [66] Zhou R, Huang K W, Chen S M, et al. Zhilining Formula alleviates DSS-induced colitis through suppressing inflammation and gut barrier dysfunction *via* the AHR/NF- κ Bp65 axis [J]. Phytomedicine, 2024, 129: 155571.
- [67] Moonwiriyaakit A, Pathomthongtaweetchai N, Steinhagen P R, et al. Tight junctions: from molecules to gastrointestinal diseases [J]. Tissue Barriers, 2023, 11(2): 2077620.
- [68] Song M S, Zhang Z, Li Y N, et al. Midgut microbiota affects the intestinal barrier by producing short-chain fatty acids in *Apostichopus japonicus* [J]. Frontiers in Microbiology, 2023, 14: 1263731.
- [69] Hao W, Chen Z J, Yuan Q, et al. Ginger polysaccharides relieve ulcerative colitis *via* maintaining intestinal barrier integrity and gut microbiota modulation [J]. International Journal of Biological Macromolecules, 2022, 219: 730–739.
- [70] Ji Z H, He S, Xie W Y, et al. *Agaricus blazei* polysaccharide alleviates DSS-induced colitis in mice by modulating intestinal barrier and remodeling metabolism [J]. Nutrients, 2023, 15(23): 4877.
- [71] Yao D B, Dai W L, Dong M, et al. MUC₂ and related bacterial factors: therapeutic targets for ulcerative colitis [J]. eBioMedicine, 2021, 74: 103751.
- [72] Xiao H Y, Li H L, Wen Y F, et al. *Tremella fuciformis* polysaccharides ameliorated ulcerative colitis *via* inhibiting inflammation and enhancing intestinal epithelial barrier function [J]. International Journal of Biological Macromolecules, 2021, 180: 633–642.
- [73] Liu W, Wang L J, Yuan Q, et al. *Agaricus bisporus* polysaccharides ameliorate ulcerative colitis in mice by modulating gut microbiota and its metabolism [J]. Food & Function, 2024, 15(3): 1191–1207.
- [74] Huo Z Q, Li J X, Li X F, et al. Functional fractions of *Astragalus* polysaccharides as a potential prebiotic to alleviate ulcerative colitis [J]. International Journal of Biological Macromolecules, 2024, 271: 132580.
- [75] Tian B M, Geng Y, Xu T R, et al. Digestive characteristics of *Hericium erinaceus* polysaccharides and their positive effects on fecal microbiota of male and female volunteers during *in vitro* fermentation [J]. Frontiers in Nutrition, 2022, 9: 858585.
- [76] Gong H, Gan X N, Qin B Y, et al. Structural characteristics of steamed *Polygonatum cyrtoneura* polysaccharide and its bioactivity on colitis *via* improving the intestinal barrier and modifying the gut microbiota [J]. Carbohydrate Polymers, 2024, 327: 121669.
- [77] Wei S X, Wang L F, Chen X D, et al. Polysaccharide from *Boletus aereus* ameliorates DSS-induced colitis in mice by regulating the MANF/MUC₂ signaling and gut microbiota [J]. International Journal of Biological Macromolecules, 2024, 266: 131232.
- [78] Zhao Y N, Chen H, Li W T, et al. Selenium-containing tea polysaccharides ameliorate DSS-induced ulcerative colitis *via* enhancing the intestinal barrier and regulating the gut microbiota [J]. International Journal of Biological Macromolecules, 2022, 209: 356–366.
- [79] Zhang Q, Wu G R, Shen S M, et al. Dioscoreae persimilis polysaccharide ameliorates DSS-induced ulcerative colitis in mice through modulation of microbiota composition [J]. Journal of Holistic Integrative Pharmacy, 2023, 4(2): 157–165.
- [80] Wei C Y, Yao L, Zhang Y, et al. Structural characterization of peach gum polysaccharide and its effects on the regulation of DSS-induced acute colitis [J]. International Journal of Biological Macromolecules, 2023, 225: 1224–1234.
- [81] Sun Y Q, Huo J X, Zhong S, et al. Chemical structure and anti-inflammatory activity of a branched polysaccharide isolated from *Phellinus baumii* [J]. Carbohydrate Polymers, 2021, 268: 118214.
- [82] Zhang Y, Wu Z J, Liu J X, et al. Identification of the core active structure of a *Dendrobium officinale* polysaccharide and its protective effect against dextran sulfate sodium-induced colitis *via* alleviating gut microbiota dysbiosis [J]. Food Research International, 2020, 137: 109641.
- [83] Jin M Y, Wang Y X, Yang X B, et al. Structure character-

- ization of a polysaccharide extracted from noni (*Morinda citrifolia* L.) and its protective effect against DSS-induced bowel disease in mice[J]. *Food Hydrocolloids*, 2019, 90: 189–197.
- [84] Wan L, Qian C, Yang C M, et al. Ginseng polysaccharides ameliorate ulcerative colitis *via* regulating gut microbiota and tryptophan metabolism[J]. *International Journal of Biological Macromolecules*, 2024, 265: 130822.
- [85] Huang L L, Sun Q, Li Q H, et al. Screening and characterization of an anti-inflammatory pectic polysaccharide from *Cucurbita moschata* Duch [J]. *International Journal of Biological Macromolecules*, 2024, 264: 130510.
- [86] Zheng L, Ma Y H, Zhang Y J, et al. Increased antioxidant activity and improved structural characterization of sulfuric acid-treated stepwise degraded polysaccharides from *Pholiota nameko* PN-01[J]. *International Journal of Biological Macromolecules*, 2021, 166: 1220–1229.
- [87] Du J L, Wang B L, Chen J H, et al. Extraction, characterization and bioactivities of novel purified polysaccharides from *Baphicacanthis Cusiae* Rhizoma et *Radix*[J]. *International Journal of Biological Macromolecules*, 2016, 93: 879–888.
- [88] Liu Y, Zhang Y Q, Mei N J, et al. Three acidic polysaccharides derived from sour jujube seeds protect intestinal epithelial barrier function in LPS induced Caco-2 cell inflammation model[J]. *International Journal of Biological Macromolecules*, 2023, 240: 124435.
- [89] Li S J, Li M X, Yue H, et al. Structural elucidation of a pectic polysaccharide from *Fructus Mori* and its bioactivity on intestinal bacteria strains[J]. *Carbohydrate Polymers*, 2018, 186: 168–175.
- [90] Li Z W, Du Z M, Wang Y W, et al. Chemical modification, characterization, and activity changes of land plant polysaccharides: a review[J]. *Polymers*, 2022, 14 (19): 4161.
- [91] Qin L, Xu H, Cao J H, et al. Alleviative effects of sulfated polysaccharide from *Ishige okamurae* against DSS-induced ulcerative colitis *via* inhibiting inflammation and modulating gut microbiota[J]. *International Journal of Biological Macromolecules*, 2024, 268: 131915.
- [92] V Brito T, Barros F C N, Silva R O, et al. Sulfated polysaccharide from the marine algae *Hypnea musciformis* inhibits TNBS-induced intestinal damage in rats[J]. *Carbohydrate Polymers*, 2016, 151: 957–964.
- [93] Li C, Jiao Y K, Shen S M, et al. *Chaenomeles sinensis* polysaccharide and its carboxymethylated derivative alleviate dextran sulfate sodium-induced ulcerative colitis *via* suppression of inflammation and oxidative stress[J]. *Bio-medicine & Pharmacotherapy*, 2023, 169: 115941.

Research progress on the mechanisms and structure-activity relationships of plant polysaccharides in alleviating colitis

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Abstract: Based on the accumulating evidence on plant polysaccharides in colitis intervention, this review summarizes recent advances in their anti-colitis mechanisms and structure-activity relationships. Current evidence indicates that plant polysaccharides can ameliorate experimental colitis by modulating signaling pathways involving the NL-RP3 inflammasome, NF- κ B, MAPK, NRF2/HO-1, PI3K/Akt, and JAK/STAT; reshaping the gut microbiota; promoting the production of microbial metabolites such as short-chain fatty acids (SCFAs); and enhancing the intestinal mucosal barrier. Their anti-colitis activities are associated with molecular weight, monosaccharide composition, backbone structure, spatial conformation, uronic acid content, and structural modification. However, substantial heterogeneity in raw materials, extraction and purification procedures, structural characterization, and animal models makes it difficult to establish generalizable structure-activity relationships. Current evidence is still dominated by cell and animal studies, while clinical studies remain limited. Future studies should prioritize structure-guided fractionation, targeted modification, and conformational regulation of homologous polysaccharides; integrate multi-omics with gut microbial metabolism and barrier-function assessment to clarify the underlying mechanisms; and conduct clinical studies according to standardized protocols to promote the translational application of plant polysaccharides in functional foods and in the prevention and treatment of colitis.

Key words: plant polysaccharide; colitis; mechanism of action; gut microbiota; structure-activity relationship

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