膳食营养与炎症性肠病相关性的研究进展

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摘要 炎症性肠病(IBD)的发病率在全球范围内不断上升,流行病学以及临床研究发现,饮食影响着 IBD 的发病和疾病治疗,饮食模式西方化是导致 IBD 发病率上升的重要因素之一。合理膳食营养对个体健康至关重要,是减少机体炎症,保持免疫系统正常运行的基本因素之一,在疾病的预防和治疗中发挥着重要作用。目前 IBD 临床治疗以药物治疗为主,主要目的在于诱导和维持缓解。近年来临床研究发现,对 IBD 患者进行饮食干预可有效缓解 IBD 症状和预防疾病复发。饮食干预因具有副作用小、安全、经济等优势而受到越来越多研究人员和临床医生的关注。本文综述膳食成分对 IBD 发病和疾病治疗的影响,以及膳食营养成分和肠内营养在 IBD 治疗中的作用的最新研究进展,建议 IBD 患者进行营养风险筛查和专业饮食咨询,鼓励 IBD 患者注重日常饮食管理,培养科学健康的饮食习惯。同时针对目前的饮食干预研究存在的局限性,提出 IBD 饮食干预未来的研究方向,为 IBD 的临床营养干预和饮食干预研究提供参考。

关键词 炎症性肠病；膳食营养；饮食干预
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1 炎症性肠病概述

炎症性肠病 (Inflammatory bowel disease, IBD),包括溃疡性结肠炎(Ulcerative colitis,UC)和克罗恩病(Crohn's disease,CD),是一种慢性、复发性、可缓解的炎症性肠道疾病,以腹痛、腹泻和体质量减轻为主要特征[1-2]。炎症性肠病影响着数百万人,近几十年来,其发病率在全球一直呈上升趋势[3]。在中国,随着经济水平的发展,人们的生活方式发生巨大变化,IBD 的发病率也不断升高。流行病学数据显示,2019 年,我国 IBD 的流行率、发病率、死亡率已分别达到 47 例/10 万人、3 例/10 万人、0.3 例/10 万人[4]。从医院质量检测系统(HQMS) 收集的中国 IBD 患者的住院数据显示,2018 年与 IBD 相关的住院费用已达到 30 亿元[5]。中国作为人口最多的发展中国家,IBD 患病率增加将导致疾病经济负担的大幅增加，以及随之产生的患者生活质量降低、精神压力增大等问题。

虽然 IBD 的确切病因仍不清楚，但普遍认为由多种因素造成,如遗传易感性、肠道菌群紊乱、免疫反应失调、环境[6]等因素。炎症性肠病流行病学变化及其在新兴工业化国家发病率的增加表明，环境变化是诱发遗传易感人群肠道炎症的重要因素之一[1,7-9]。与 IBD 病因相关的主要环境因素包括饮食、压力、空气污染、抗生素和吸烟等[10-11]。饮食作为一个可变的环境风险因子，对 IBD 疾病发生和严重程度有着重要影响。全球 IBD 发病率上升可部分归因于新兴工业化国家的膳食模式偏向于西方化以及摄入过多的加工食品[12-13]。一项饮食质量与 IBD 患者肠道炎症和胃肠道症状关系的调查研究表明,IBD 患者的饮食质量与疾病特征存在显著相关性[14]。

饮食可以调节肠道微生物群并影响免疫系统[15],近十几年来,人们对饮食干预治疗 IBD 的临床应用越来越感兴趣。相关研究已表明,饮食干预以及饮食结合药物治疗是诱导 IBD 缓解的有效方法。本文结合国内外最新研究进展,综述了膳食成分对 IBD 的影响以及 IBD 治疗中的饮食干预策略,为 IBD 的临床营养干预提供参考。

2 膳食成分对炎症性肠病发病的影响

流行病学研究已表明，工业化之前的饮食模式到西方饮食模式的演变与 IBD 发病率的增加密切相关[16-18]。工业化之前的饮食以鱼、蛋、食草动物瘦肉制品、全谷物、水果和蔬菜为主,而现代西方饮食的特点是过度摄入动物源性蛋白质、精制谷

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物、氢化脂肪和加工食品等，水果和蔬菜的摄入减少[12]。饮食模式的改变导致营养的质量和比例发生变化，引起了肠道生态失调，进而引发肠道炎症（图 1）。临床试验表明，采用排除性饮食（减少精制糖、饱和脂肪、乳化剂、红肉和超加工肉类）的 IBD 患者可以更好维持临床缓解，并改善肠道炎症反应[19]。

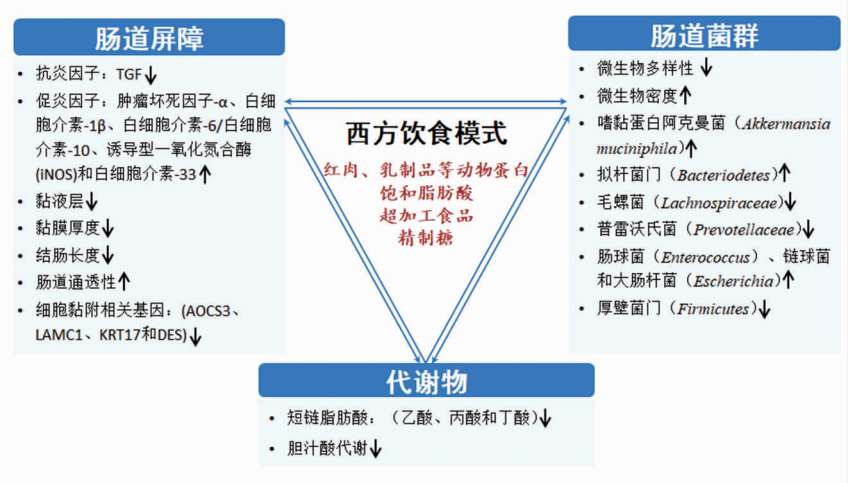

图 1　西方饮食模式对炎性肠病的影响

Fig.1　Influence of Western dietary patterns on inflammatory bowel disease

2.1　膳食蛋白质对 IBD 的影响

西方饮食模式的特点之一是摄入大量红肉、加工肉制品、乳制品作为膳食蛋白质来源。一项来自欧洲的人群队列研究对蛋白质（总蛋白、动物蛋白、植物蛋白）食物来源与 IBD 风险之间的关系进行分析，发现动物蛋白摄入量与 IBD 风险相关，过多食用肉类尤其是红肉与溃疡性结肠炎的发生相关[20]。膳食蛋白质的食用量和来源已被证实会影响肠道菌群的组成，进而对肠道造成影响[21-22]。一项饮食与肠道菌群、结肠炎相关性的动物试验研究发现，与低膳食蛋白摄入量（6%）相比，高膳食蛋白（41%酪蛋白）小鼠的粪便微生物多样性降低，菌门相对丰度增加，厚壁菌门相对丰度减少，肠道微生物密度增加[23]。高膳食蛋白可能通过降低微生物多样性和肠道屏障功能，导致肠道微生物密度增加，增加抗原负荷，提高微生物入侵附近组织的可能性或增加致病代谢物的产生，从而加剧葡聚糖硫酸钠（Dextran Sulfate Sodium，DSS）诱导的肠道损伤。此外也有研究表明，高蛋白（酪蛋白）饮食通过与肠道菌群之间的相互作用增加了肠道炎症的敏感性，这种促炎作用不依赖于适应性免疫，而是由激活的 Ly-6Chi 单核细胞和先天免疫促炎调节驱动[22]。

2.2　膳食脂肪对 IBD 的影响

流行病学和试验研究表明，高脂饮食可引发 IBD[24-26]。高脂饮食中的饱和脂肪酸会诱发机体的炎症反应[27-28]。在一项对成人 IBD 患者随访研究发现，炎症性肠病患者疾病活动度与肝脏脂肪含量增加相关[29]。另一项针对 UC 缓解期患者的交叉研究发现，低脂饮食可降低粪便样本中的炎症标志物，减少患者肠道生态失调，有益于缓解期的 UC 患者[30]。动物实验研究表明，高脂饮食诱导以肠道菌群失调为特征的 IBD 前期状态[31-32]。同时高脂饮食会对代谢和肝脏炎症产生不利影响，并加重结肠炎[33]。此外，有研究表明，摄入过多富含 omega-6 多不饱和脂肪酸的食物（豆油、花生油、菜籽油、豆油等）或 omega-6/omega-3 摄入比例过高会增加溃疡性结肠炎发生的风险[34-37]。

2.3　精制糖对 IBD 的影响

精制糖已广泛应用于现代食品加工，成为我们饮食的一部分。日常食用的饼干、面包、糕点、软饮料、冰淇淋、果酒等，通常富含大量的糖。饮食调查发现，过多摄入含糖饮料会导致 IBD 风险以及 IBD 患者住院率、急诊率、炎症发生率增高[38-39]。

IBD 患者相对于健康人群通常会习惯摄入更多的含糖饮料或食物[40-41]。此外,近期大规模流行病学研究表明,含糖饮料的摄入增加了早发性结直肠癌的风险[42-44]。

尽管大量的数据表明使用过量的糖会对肠道健康造成有害影响,但关于高糖摄入与 IBD 发病机制的关系尚未完全阐明。动物实验研究发现,高糖饮食显著改变肠道微生物群组成,特别是黏液降解菌嗜黏蛋白阿克曼菌(*Akkermansia muciniphila*)和脆弱拟杆菌(*Bacteroides fragilis*)的丰度增加,细菌来源的黏液溶解酶的富集导致了结肠黏液层的侵蚀[45-46]。肠道黏液层物理分离肠道上层细胞和肠道微生物,高糖摄入降低了黏液层的厚度,使肠道细菌接近上皮层,引起肠道炎症。此外,高糖饮食也降低了产生短链脂肪酸(Short-chain fatty acid, SCFAs)的毛螺菌科(Lachnospiraceae)、普雷沃氏菌科(Prevotellaceae)的丰度[47]。SCFAs 可调节肠道免疫和屏障功能、提供细胞能量等,在维持肠道稳态方面发挥着重要作用。

2.4 食品添加剂对 IBD 的影响

食品添加剂通常存在于各种加工食品中,尤其是超加工食品。越来越多的科学证据表明,摄入过多的超加工食品,会加剧 IBD 发生风险。一项超 10 万人数据的人群队列研究表明,长期摄入超加工食品(加工肉类、果酱、薯片、软饮料、饼干、果汁饮料、精制甜点等)与 IBD 的发病风险呈正相关,相比于每天摄入少于一份的超加工食品的人群,每天摄入 1~4 份超加工食品的人群患病风险增加 67%,每天摄入 5 份以上的人群患病风险增加 82%[13]。Chassaing 等[48]的一项随机、双盲临床研究表明,每日食用含有 15 g 乳化剂(羧甲基纤维素)的食品,会对人体肠道菌群和肠道屏障造成损害,从而增加慢性炎症疾病发生的风险。Bhattacharyya 等[49]的一项随机、双盲多中心临床试验评估了缓解期溃疡性结肠炎患者摄入卡拉胶饮食与疾病复发的相关性,研究发现,每日摄入 200 mg 的卡拉胶促进了溃疡性结肠炎缓解期患者的早期复发,且检测显示炎症标志物白细胞介素-6 和粪便钙卫蛋白水平升高。另外,在一些动物实验模型中发现,食品着色剂诱惑红、日落黄、二氧化钛以及甜味剂三氯蔗糖等均可诱发或加重小鼠肠道炎症[50-53]。

3 IBD 管理中的营养建议

IBD 患者通常存在较高的营养不良发生风险。动物及临床研究发现,膳食营养素(优质动植物蛋白质、维生素 D、膳食纤维等)在 IBD 发生和治疗中起着重要作用,对 IBD 患者进行饮食干预或临床营养管理可提高机体营养状态,降低炎症指标、维持肠道屏障、改善临床结局(图 2)。

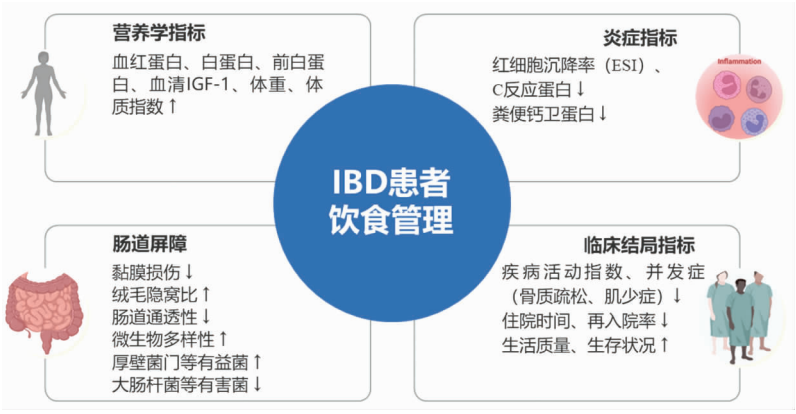

图 2 IBD 患者饮食管理对疾病治疗的作用

Fig.2 The role of dietary management in disease management in patients with IBD

3.1 优质动植物蛋白质

乳清蛋白和大豆蛋白作为优质动植物蛋白代表,具有调节免疫、减轻炎症反应、抗氧化等作用[54-57]。动物实验研究发现,将膳食中的酪蛋白替换成大豆蛋白,可显著减少肠道中炎症细胞因子,增加肠道黏膜厚度,降低试验性 IBD 的严重程度[58-60];

将膳食中的酪蛋白替换或部分替换成乳清蛋白，可增加肠道黏蛋白的表达，调节肠道菌群组成，减轻肠道黏膜损伤[61-62]。Tunc 等[63]用醋酸诱导大鼠溃疡性结肠炎，采用治疗剂量的乳清蛋白(2.39 g/kg)经直肠给药 7 d，乳清蛋白显著降低了大鼠炎症标志物激活蛋白-1(AP-1)、环氧合酶-2(COX-2)、白细胞介素-6、白细胞介素-10、核因子-κB(NF-κB)、肿瘤坏死因子-α 水平，并上调血红素加氧酶-1(HO-1)、Nrf2 表达，推断乳清蛋白通过调节 Nrf2/HO-1 和 NF-κB 通路，对醋酸诱导的结肠炎产生抗炎作用。Benjamin 等[64]的临床研究发现，CD 患者每日补充一定量的浓缩乳清蛋白可降低肠道炎症标志物，改善肠道通透性和肠道形态。不过另一项相似的研究只观察到了 CD 患者身体质量的改善[65]。在对结肠癌患者围手术期、放化疗期间的营养干预研究发现，补充一定量的乳清蛋白，可显著改善患者的身体质量、营养状况，加速身体康复[66-67]。此外，一些研究表明，以大豆蛋白和乳清蛋白为配比的双蛋白可显著提高机体抗氧化能力，增加肠道菌群多样性，提高肠道屏障功能，改善肌肉质量，抑制炎症反应，促进免疫重建[54,68-72]。一项膳食摄入鱼类与 IBD 风险的 meta 分析显示，日常食用鱼类作为蛋白质来源，可降低 CD 发病率[73]。因此，日常饮食中，IBD 患者可每日摄入一定量的乳清蛋白、大豆蛋白、双蛋白以及鱼肉等优质动植物蛋白作为膳食蛋白来源，以提高机体免疫功能，增强抵御疾病的能力。表 1 列出了优质动植物蛋白对 IBD 改善作用的动物和临床试验研究。

表 1　优质动植物蛋白对 IBD 的改善作用

Table 1　Ameliorative effect of high quality animal and plant proteins on IBD

饮食蛋白来源	干预对象	疾病类型	周期	干预结果	参考文献
动物实验					
普通饮食中的蛋白来源为大豆蛋白(35%)	雄性 SD 大鼠	三硝基苯磺酸(TNBS)	4 周	1.改善了结肠黏膜结构 podoplanin⁺的浸润； 2. 抑制结肠肿瘤坏死因子-α⁺细胞增值、RANKL 表达； 3. 抑制骨蛋白中促炎因子肿瘤坏死因子-a 和白细胞介素-6 表达； 4. 减轻 TNBS 引起的大鼠高破骨细胞表面和骨形成率受抑制	[58]
普通饮食中的蛋白来源为大豆蛋白(20%)	雄性和雌性 SPF SAMP 小鼠	DSS	6 周	1. 粪便髓过氧化物酶(MPO)和 FITC-葡聚糖渗透性评分显著降低； 2. 鹅卵石病变严重程度降低； 3.乳酸杆菌科(*Lactobacillaceae*)和 *Leuconostraceae* 丰度增加； 4. 代谢物谷氨酰胺、丁酸浓度增加，血浆亚油酸浓度降低	[59]
普通饮食中的蛋白来源为大豆蛋白(20%)	C57BL/6 小鼠	DSS	12 d	1. 降低了结肠中黏蛋白 MUC1 和三叶因子 TFF-3 的含量； 2. 抑制 DSS 诱导的结肠长度降低； 3. 降低结肠炎症评分； 4. 降低结肠和盲肠肿瘤坏死因子-a 表达	[60]
饮食中 50%的酪蛋白替换成低温/高温处理的浓缩乳清蛋白(L/HWPC)	雌性 BALB/c 小鼠	DSS	3 周	1. 两种蛋白均可改善小鼠体质量丢失，低温处理浓缩乳清蛋白作用效果更显著； 2. 高温处理浓缩乳清蛋白可改善小鼠结肠黏膜，但存在严重的白细胞浸润；低温处理浓缩乳清蛋白显著降低结肠炎症，并改善了黏膜结果； 3. 低温/高温处理浓缩乳清蛋白均可增加结肠黏蛋白水平；低温处理浓缩乳清蛋白结肠中髓过氧化物酶水平降低； 4. 低温浓缩乳清蛋白下调 Gbp1、Gbp2、Gbp6 和 Cxcl9 的表达	[61]

（续表 1）

饮食蛋白来源	干预对象	疾病类型	周期	干预结果	参考文献
饮食中的酪蛋白替换成乳清蛋白	雌性 Wistar 大鼠	DSS	12 d+7 d	1. 降低了白细胞介素-1β、钙卫蛋白和诱导型一氧化氮合酶表达； 2. 减轻了腹泻和粪便失血的临床症状； 3. 增加了粪便黏蛋白的分泌； 4. 增加了乳酸杆菌和双歧杆菌表达	[62]
普通饮食+2.39 g/kg（4.78）体质量乳清蛋白临床试验	雌性 Wistar 大鼠	醋酸	7 d	1. 降低炎症标志物 AP-1、COX-2、白细胞介素-6、白细胞介素-10、NF-κB 和肿瘤坏死因子-α 水平； 2. 上调 Nrf2 和 HO-1 表达，激活 Nrf2/HO-1 通路	[63]
普通饮食+0.5 g/kg 体质量蛋白质的浓缩乳清蛋白	24~46 周岁	克罗恩病	8 周	1. 肠道通透性和肠道形态显著改善； 2. 绒毛隐窝比显著增加； 3. 炎症标志物（肠道上皮淋巴细胞 IELs）降低	[64]
普通饮食+30 g 乳清蛋白或 24 g 大豆分离蛋白	–	克罗恩病	16 周	1. 乳清蛋白与大豆分离蛋白均可降低肱三头肌皮褶厚度和体脂百分比，增加上臂肌围和矫正上臂肌面积和体瘦百分比； 2.C 反应蛋白与疾病活动指数呈显著正相关，与白蛋白和前白蛋白呈负相关	[65]
普通饮食+13.5 g 高度纯化的乳清蛋白	34~85 周岁	结直肠癌	6 个月	1. 相对于对照组存在 26.3%营养不良风险，乳清蛋白干预组均营养正常； 2. 乳清蛋白组肌少症百分比低于对照组； 3. 乳清蛋白组可降低血液和胃肠道毒性发生率	[66]
19.8 g 乳清蛋白	56~79 周岁	结直肠癌	4+4 周	1. 明显改善患者的步行功能	[67]

3.2 维生素 D

维生素 D 是一种类固醇激素，包括维生素 D_2（角钙化醇）和维生素 D_3（胆钙化醇）两种形式。维生素 D_2 只能从食物中获取，而维生素 D_3 既可以从食物中获取，也可以通过皮肤光照内源性合成[74]。营养缺乏、肠道吸收不良、户外运动减少以及皮质类固醇使用引起的维生素 D 缺乏常见于 IBD 患者中，40%~80%的 IBD 患者存在维生素 D 不足或缺乏的现象[75-77]。临床研究发现，IBD 患者维生素 D 水平与其疾病严重程度显著相关[78-80]，维生素 D 水平越高，IBD 患者炎症程度越低。此外，维生素 D 缺乏的人群患 IBD 风险比非维生素缺乏者高 2 倍[81]。维生素 D 可作为免疫调节剂参与调节有致病风险的先天和适应性免疫反应，减轻炎症反应，维持肠道屏障功能，促进肠道内稳态[82-83]。补充维生素 D 可以降低 IBD 患者的临床复发风险，特别是在临床缓解期的 CD 患者[84-85]。IBD 合并维生素 D 缺乏的患儿每日口服维生素 D_3（2 000 IU/d），IBD 活动评分、炎症标志物水平、住院频率和急诊次数均显著降低[86]。由于维生素 D 对机体免疫系统有一定影响，在 IBD 缓解期和活动性，其不足可能会影响免疫系统的正常运作和肠道黏膜的愈合。日常生活中可注重富含维生素 D 食物的摄入，如鱼肝油、肝脏、海鱼等，另外，通过阳光照射刺激皮肤合成也是维生素 D 的一个主要来源。欧洲肠外肠内营养学会（ESPEN）[87]建议对于疾病活动期 IBD 患者（成人和儿童）和接受类固醇治疗的患者，应监测血清中维生素 D 水平，并在不足时及时补充。

3.3 膳食纤维

随着生活水平的提高，食物精细化、过度加工、动物性食物摄入比例增大导致膳食纤维摄入量严重不足。膳食纤维根据水溶性一般分为可溶性膳食纤维和不溶性膳食纤维。可溶性膳食纤维

包括β-葡聚糖、果胶、低聚果糖等，燕麦、大麦、水果、豆类和大多数根茎类蔬菜都富含可溶性膳食纤维[88]。可溶性膳食纤维易于消化，可在肠道内消化，通过支持肠道微生物群生长以及短链脂肪酸的生成来改善肠道微环境，保持肠道完整性[89]。不溶性膳食纤维包括木质素、纤维素，半纤维素等，主要来源于植物的根、茎、叶、皮中，不易发酵，具有增加大便体积、缓解便秘的作用[88]。一项膳食纤维与 IBD 风险之间关系的 meta 分析表明，膳食纤维摄入量与 CD 风险呈负相关，但与 UC 无关[90]。对 IBD 患者膳食纤维摄入情况进行分析研究发现，IBD 患者摄入的膳食纤维摄入量低于与健康人群，也低于膳食纤维推荐摄入量[91-92]。低纤维饮食会减少 SFCAs 生成，并导致有害代谢物增加，通过黏液层液化增加感染的易感性[93]。目前的膳食指南对 IBD 患者应摄入的膳食纤维数量和类型并不是很明确 [94]。《中国炎症性肠病营养诊疗共识》[95]建议，在 IBD 活动期，尤其是肠道炎症严重而且伴有明显腹痛、腹泻时，或者并发肠道狭窄或者穿透性病变时，宜选择不含或者少含膳食纤维的食物。在 IBD 缓解期，尤其是患者没有腹痛、腹泻时，可考虑补充适量富含膳食纤维的食物。

3.4　Omega-3

Omega-3 多元不饱和脂肪酸有 3 种主要类型，分别为 α-亚油酸、二十二碳五烯酸(EPA)和二十二碳六烯酸(DHA)。α-亚油酸主要存在于奇亚籽、亚麻籽等植物性食物中，EPA 和 DHA 则主要存在于深海鱼类等动物性食物以及海藻中。流行病学数据表明[96-97]，omega-3 对降低 IBD 风险有潜在的益处，特别是 UC，摄入较高含量的 omega-3 可能会降低疾病的发病率。一些试验研究也表明，补充 omega-3 可以有效治疗或缓解 IBD[98-101]。由于缺乏足够的临床试验支持，ESPEN 不推荐 IBD 患者特别补充 omega-3[87]。不过，鉴于 omega-3 具有的改善肠道菌群多样性、修复肠道功能、调节肠道免疫系统[102-104]等功能，国际炎症性肠病研究组织饮食指南[105]推荐 UC 患者可以从饮食上获取天然来源的 omega-3。

3.5　肠内营养

肠内营养(Enteral nutrition，EN)是用于诱导 IBD 缓解的主要治疗方法之一，尤其是克罗恩病。如表 2 所示，肠内营养制剂可减轻 IBD 患者炎症指标，提高患者整体营养水平，减少手术并发症发生率，改善肠道菌群失调等。肠内营养制剂在诱导儿童克罗恩病临床缓解方面与皮质类固醇治疗效果相当，在诱导黏膜愈合方面优于皮质类固醇治疗[106-108]。考虑到使用皮质类固醇对儿童生长发育的影响，ESPEN 建议将肠内营养制剂作为第一线治疗以诱导缓解儿童和青少年急性活动期克罗恩病[87]。肠内营养制剂对成人克罗恩病同样具有显著改善作用，不过其效果不如皮质类固醇[107]。目前，可用于肠内营养支持的产品主要包括肠内营

表 2　肠内营养制剂在 IBD 患者中的临床应用

Table 2　Clinical application of enteral nutrition in patients with IBD

研究国家/地区	周期	疾病类型	年龄	干预效果	参考文献
加拿大	4~10 周	IBD	7~13 周岁	1.粪便微生物 α-多样性显著增加 2.富集普拉梭菌(*Faecalibacterium prausnitzii*)、产丙酸戴阿李斯特菌 (*Dialister propionicifaciens*) 和粪副拟杆菌(*Parabacteroides merdae*) 3.大肠埃希菌/志贺菌属(*Escherichia/Shigella*)、浑浊戴阿李斯特菌(*Dialister invisus*)和 *Negativibacillus* 显著减少	[109]
加拿大	8 周	CD 和 UC	5~18 周岁	1. 布劳特氏菌属(*Blautia*)、塞利单胞菌属(*Sellimonas*)和来自瘤胃菌科非典型细菌 (*Uncharacterized bacteria from family Ruminococcaceae*)丰度增加 2. 颗粒链菌属(*Granulicatella*)、嗜血杆菌属(*Haemophilus*)和链球菌属(*Streptococcus*)丰度降低	[110]

(续表 2)

研究国家/地区	周期	疾病类型	年龄	干预效果	参考文献
土耳其	8 周	CD 和 UC	10~17.7 周岁	1.CD 临床缓解率高于对照组一倍,UC 临床缓解率高对照组 3 倍; 2.UC 组凝血细胞水平高于对照组	[111]
中国	8 周	CD	6~18 周岁	1. 儿童克罗恩病疾病活动指数(PCDAI)评分和钙卫蛋白水平下降; 2. 微生物组和胆汁酸代谢恢复到正常水平; 3. 厚壁菌门(*Firmicutes phylum*)、黄酮属(*Flavonifractor*)和 *V* 型梭菌(*Clostridium V*)的相对表达量增加	[112]
瑞典	6 周	CD	7.8~16.4 周岁	1.红细胞沉降率(ESR)、C 反应蛋白和粪便钙卫蛋白显著降低; 2. 血红蛋白、白蛋白和体质量显著升高; 3. 促进黏膜愈合	[113]
中国	15~21 周	CD	26~57 周岁	1. 术前血清白蛋白、前白蛋白、血红蛋白水平高于非肠内营养制剂组; 2. 术后并发症发生率降低	[114]
中国	8 周	CD	15~34 周岁	1. ESR、C 反应蛋白和克罗恩病活动指数(CDAI)显著下降; 2. 血清白蛋白水平增加; 3. 厚壁菌门(*Firmicutes*)、瘤胃球菌属(*Ruminococcus*)、毛螺菌科(*Lachnospiraceae*)、厌氧棍状菌属(*Anaerotruncus*)、黄杆菌属(*Flavonifractor*)和新鞘氨醇杆菌属 (*Novosphingobium*)丰度显著增加,变形菌门(*Proteobacteria*)丰度下降	
中国	12 周	CD	20~30 周岁	1.CDAI、C 反应蛋白、ESR、血小板显著降低; 2. 白蛋白、血红蛋白升高; 3. 诱导多种与炎症和营养指标有关的多种脂类物质和种类改变; 4. 促进黏膜愈合	[115]
新西兰	8 周	CD	16~38 周岁	1. 炎症标志物 C 反应蛋白和粪便钙卫蛋白水平下降; 2. 营养标志物血清 IGF-1 和白蛋白升高	[116]
澳大利亚	6 周	CD	20~74 周岁	1. 改善主观幸福指数; 2. C 反应蛋白降低,血清白蛋白增加,体质量增加; 3. 减少手术干预以及术后并发症	[117]

养制剂和特殊医学用途配方食品两类，我国还尚未有专门针对 IBD 患者的肠内营养制剂，特殊医学用途配方食品产品类别虽包括炎症性肠病全营养配方食品,由于此类别产品需要经过临床试验,临床试验周期长，此类配方食品尚未见到注册申报信息。

4 总结与展望

研究表明，合理膳食营养在 IBD 的病因和治疗中起着重要作用。膳食营养关系着个体的营养状态,对个体健康至关重要。IBD 患者由于进食减少、消化吸收障碍、药物副作用等因素的影响,通常面临营养不良的风险。营养不良往往造成 IBD 患者复发率、并发症发生率、住院频率、住院时间和死亡率增加[118-119]。饮食干预可确保 IBD 患者充分的营养摄入,以改善其营养状况,提高其免疫能力,从而缩短疾病进程和提升临床疗效。科学的饮食干预在部分患者的治疗上优于药物，且是安全的,已被证明是 IBD 管理的有效策略。国际指南上没有为 IBD 患者推荐完整的饮食方案，但建议 IBD 患者应进行营养风险筛查和专业的饮食咨询,以改善营养治疗,提高临床缓解率,并有助于

避免营养不良以及相关并发症[87]。目前国际上有一些流行的针对 IBD 患者的特殊饮食，包括特定碳水饮食(SCD)、无麸质饮食(GFD)、低 FODMAP 饮食、地中海饮食(MED)等[120]。这类饮食主要是通过减少或排除一些可能破坏肠道黏膜或引起肠道菌群失调的特定饮食成分，如加工肉类、精制糖、麸质、食品添加剂等，以改善或缓解肠道症状。相比于肠内营养制剂，这种特殊的饮食策略可提高 IBD 患者长期饮食干预的依从性。IBD 患者的饮食管理除了关注宏量营养素的摄入，还应定期检查微量营养素缺乏情况，尤其是维生素 B_{12}、维生素 D、叶酸、铁、锌、钙等。临床研究发现，IBD 患者由于肠道吸收障碍、饮食受限等易导致微量营养素缺乏。在通过饮食提供营养干预时，除了保证充足的饮食来源的天然膳食营养素的摄入，必要时也可使用膳食营养素补充剂，以确保机体达到正常的营养状态，避免由于营养素缺乏而造成的不良影响。

近年来，由于饮食干预的局限性，如样本量少、依从性差、干预周期短、疾病异质性等，绝大多数饮食干预研究结果属于低质量的证据，难以获得权威临床指南的认可。因此，未来需要更多高质量的随机对照试验（Randomized controlled trial，RCT）数据用于支持饮食干预在 IBD 疾病治疗中的作用。获得 ESPEN 支持的肠内营养制剂由于口感不佳，以及可能引起腹泻、呕吐等并发症的原因，导致 IBD 患者依从性较差，进而影响了治疗效果，使得肠内营养制剂的临床应用受到一定限制。后续针对性开发 IBD 的肠内营养制剂或炎症性肠病全营养配方食品时，需要更多考虑适口性以及有益于肠道健康的配方组成等问题。另外，由于 IBD 患者的高度异质性，未来的饮食干预也可考虑结合个体的临床和生物学指标，制定个体化饮食方案，并通过定期测量相关指标，及时调整相应饮食方案，提供针对 IBD 患者的个性化精准营养干预策略。

参 考 文 献

[1] LEE D, ALBENBERG L, COMPHER C, et al. Diet in the pathogenesis and treatment of inflammatory bowel diseases[J]. Gastroenterology, 2015, 148(6): 1087-1106.

[2] LLOYD-PRICE J, ARZE C, ANANTHAKRISHNAN AN, et al. Multi-omics of the gut microbial ecosystem in inflammatory bowel diseases[J]. Nature, 2019, 569(7758): 655-662.

[3] KAPLAN G G, WINDSOR J W. The four epidemiological stages in the global evolution ofinflammatory bowel disease[J]. Nat Rev Gastroenterol Hepatol, 2021, 18(1): 56-66.

[4] ZHANG Y, LIU J, HAN X, et al. Long-term trends in the burden of inflammatory bowel disease in China over three decades: A joinpoint regression and age-period-cohort analysis based on GBD 2019 [J]. Front Public Health, 2022, 7(10): 994619.

[5] HE Y M, MAO R, YUAN G, et al. The hospitalization burden of inflammatory bowel disease in China: a nationwide study from 2013 to 2018[J]. Therap Adv Gastroenterol, 2022, 13(15): 1-14.

[6] ANANTHAKRISHNAN AN. Environmental risk factors for inflammatory bowel diseases[J]. Dig Dis Sci, 2015, 60(2): 290-298.

[7] NG S C, SHI H Y, HAMIDI N, et al. Worldwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies [J]. Lancet, 2017, 390 (10114): 2769-2778.

[8] NG S C, SHI H Y, HAMIDI N, et al. Worldwide incidence andprevalence of inflammatory bowel disease in the 21st century: a systematic review of populationbased studies [J]. Lancet, 2018, 390 (10114): 2769-2778.

[9] SHOUVAL D S, RUFO P A. The role of environmental factors in the pathogenesis of inflammatory bowel diseases[J]. JAMA Pediatr, 2017, 171(10): 999-1005.

[10] ANANTHAKRISHNAN A N. Epidemiology and risk factors for IBD[J]. Nature Reviews Gastroenterology & Hepatology, 2015, 12(4): 205-217.

[11] ANANTHAKRISHNAN A N, BERNSTEIN C N, ILIOPOULOS D, et al. Environmental triggers in IBD: A review of progress and evidence[J]. Nat Rev Gastroenterol Hepatol, 2018, 15(1): 39-49.

[12] SASSON A N, INGRAM R J M, ZHANG Z, et al. The role of precision nutrition in the modulation of microbial composition and function in people with

inflammatory bowel disease[J]. Lancet Gastroenterol Hepatol, 2021, 6(9): 754-769.

[13] NARULA N, WONG E C L, DEHGHAN M, et al. Association of ultra-processed food intake with risk of inflammatory bowel disease: prospective cohort study[J]. BMJ, 2021, 14(374): n1554.

[14] DE GRAAF M C G, SPOOREN C E G M, HENDRIX E M B, et al. Diet quality and dietary inflammatory index in dutch inflammatory bowel disease and irritable bowel syndrome patients[J]. Nutrients, 2022, 14(9): 1945.

[15] WASTYK H C, FRAGIADAKIS G K, PERELMAN D, et al. Gut-microbiota-targeted diets modulate human immune status[J]. Cell, 2021, 184(16): 4137-4153.

[16] HAMMER T, LOPHAVEN S N, NIELSEN K R, et al. Dietary risk factors for inflammatory bowel diseases in a high-risk population: Results from the Faroese IBD study[J]. United European Gastroenterology Journal, 2019, 7(7): 924-932.

[17] MOKHTAR N M, NAWAWI K N M, VERASINGAM J, et al. A four-decade analysis of the incidence trends, sociodemographic and clinical characteristics of inflammatory bowel disease patients at single tertiary centre, Kuala Lumpur, Malaysia[J]. BMC Public Health, 2019, 19(Suppl 4): 550.

[18] ANANTHAKRISHNAN A N. Epidemiology and risk factors for IBD[J]. Nat Rev Gastroenterol Hepatol, 2015, 12(4): 205-217.

[19] NITESCU M, ISTRATESCU D, PREDA C M, et al. Role of an exclusion diet (reduced disaccharides, saturated fats, emulsifiers, red and ultraprocessed meats) in maintaining the remission of chronic inflammatory bowel diseases in adults[J]. Medicina (Kaunas), 2023, 59(2): 329.

[20] DONG C, MAHAMAT-SALEH Y, RACINE A, et al. OP17 Protein intakes and risk of inflammatory bowel disease in the European Prospective Investigation into Cancer and Nutrition cohort (EPIC-IBD)[J]. Journal of Crohn's and Colitis, 2020, 14(Supplement_1): S015.

[21] TAN J, NI D, TAITZ J, et al. Dietary protein increases T cell independent sIgA production through changes in gut microbiota-derived extracellular vesicles[J]. Nature Communications, 2022, 13(1): 4336.

[22] KOSTOVCIKOVA K, COUFAL S, GALANOVA N, et al. Diet rich in animal protein promotes pro-inflammatory macrophage response and exacerbates colitis in mice[J]. Front Immunol, 2019, 10: 919.

[23] LLEWELLYN, SEAN R, BRITTON, et al. Interactions between diet and the intestinal microbiota alter intestinal permeability and colitis severity in mice[J]. Gastroenterology, 2018, 154(4): 1037-1046.

[24] KHALILI H, CHAN S S M, LOCHHEAD P, et al. The role of diet in the aetiopathogenesis of inflammatory bowel disease[J]. Nat Rev Gastroenterol Hepatol, 2018, 15(9): 525-535.

[25] ARNONE D, VALLIER M, HERGALANT S , et al. Long-term overconsumption of fat and sugar causes a partially reversible pre-inflammatory bowel disease state[J]. Frontiers in nutrition, 2021, 8: 758518.

[26] BARNES E L, NESTOR M, ONYEWADUME L, et al. High dietary intake of specific fatty acids increases risk of flares in patients with ulcerative colitis in remission during treatment with aminosalicylates[J]. Clin Gastroenterol Hepatol, 2017, 15(9): 1390-1396.

[27] ROCHA D M, CALDAS A P, OLIVEIRA L L, et al. Saturated fatty acids trigger TLR4-mediated inflammatory response[J]. Atherosclerosis, 2016, 244: 211-215.

[28] MALESZA I J, MALESZA M, WALKOWIAK J, et al. High-fat, western-style diet, systemic inflammation, and gut microbiota: A narrative review[J]. Cells, 2021, 10(11): 3164.

[29] VAN LINGEN E, TUSHUIZEN M E, STEENHUIS M E J, et al. Disease activity in inflammatory bowel disease patients is associated with increased liver fat content and liver fibrosis during follow-up[J]. Int J Colorectal Dis, 2022, 37(2): 349-356.

[30] FRITSCH J, GARCES L, QUINTERO M A, et al. Low-fat, high-fiber diet reduces markers of inflammation and dysbiosis and improves quality of life in patients with ulcerative colitis[J]. Clin Gastroenterol Hepatol, 2021, 19(6): 1189-1199.

[31] ARNONE D, VALLIER M, HERGALANT S, et al. Long-term overconsumption of fat and sugar causes a partially reversible pre-inflammatory bowel disease state[J]. Front Nutr, 2021, 18(8): 758518.

[32] YASUZAWA T, NISHI R, ISHITANI S, et al. Effects of enzamin, a microbial product, on alterations

of intestinal microbiota induced by a high-fat diet[J]. Nutrients, 2022, 14(22): 4743.

[33] CHOE A R, KIM S E, LEE H A, et al. Influence of high-fat diet on colitis and liver health in inflammatory bowel disease: A study using murine model of DSS-induced colitis[J]. Journal of Crohn's and Colitis, 2020, 14: S180.

[34] DE SILVA, P S A, OLSEN A, CHRISTENSEN J, et al. An association between dietary arachidonic acid, measured in adipose tissue, and ulcerative colitis[J]. Gastroenterology, 2010, 139(6): 1912-1917.

[35] TJONNELAND A, OVERVAD K, BERGMANN M M, et al. Linoleic acid, a dietary n-6 polyunsaturated fatty acid, and the aetiology of ulcerative colitis: a nested case-control study within a European prospective cohort study[J]. Gut, 2009, 58(12): 1606-1611.

[36] KESHTELI A H, VALCHEVA R, NICKURAK C, et al. Anti-inflammatory diet prevents subclinical colonic inflammation and alters metabolomic profile of ulcerative colitis patients in clinical remission[J]. Nutrients, 2022, 14(16): 3294.

[37] LEWIS J D, ABREU M T. Diet as a trigger or therapy for infammatory bowel diseases[J]. Gastroenterology, 2017, 152(2): 398-414.

[38] AHSAN M, KOUTROUMPAKIS F, RIVERS C R, et al. High sugar-sweetened beverage consumption is associated with increased health care utilization in patients with inflammatory bowel disease: A multi-year, prospective analysis[J]. J Acad Nutr Diet, 2022, 122(8): 1488-1498.

[39] FU T, CHEN H, CHEN X, et al. Sugar-sweetened beverages, artificially sweetened beverages and natural juices and risk of inflammatory bowel disease: a cohort study of 121,490 participants[J]. Aliment Pharmacol Ther, 2022, 56(6): 1018-1029.

[40] RAFFNER B A, JEFFRY K, SARGUN S, et al. Sweets and inflammatory bowel disease: Patients favor artificial sweeteners and diet foods/drinks over table sugar and consume less fruits/vegetables[J]. Inflammatory Bowel Diseases, 2023, 29(11): 1751-1759

[41] YANG X Y, CHEN P F, HE J H. High consumption of sweetened beverages might increase the risk of inflammatory bowel diseases[J]. Clin Gastroenterol Hepatol, 2019, 17(7): 1417-1418.

[42] YUAN C, JOH H K, WANG Q L, et al. Sugar-sweetened beverage and sugar consumption and colorectal cancer incidence and mortality according to anatomic subsite[J]. Am J Clin Nutr, 2022, 115(6): 1481-1489.

[43] HUR J, OTEGBEYE E, JOH H K, et al. Sugar-sweetened beverage intake in adulthood and adolescence and risk of early-onset colorectal cancer among women[J]. Gut, 2021, 70(12): 2330-2336.

[44] JOH H K, LEE D H, HUR J, et al. Simple sugar and sugar-sweetened beverage intake during adolescence and risk of colorectal cancer precursors[J]. Gastroenterology, 2021, 161(1): 128-142.

[45] KHAN S, WALIULLAH S, GODFREY V, et al. Dietary simple sugars alter microbial ecology in the gut and promote colitis in mice[J]. Sci Transl Med, 2020, 12(567): eaay6218.

[46] SHON W J, JUNG M H, KIM Y, et al. Sugar-sweetened beverages exacerbate high-fat diet-induced inflammatory bowel disease by altering the gut microbiome[J]. J Nutr Biochem, 2023, 113: 109254.

[47] LAFFIN M, FEDORAK R, ZALASKY A, et al. A high-sugar diet rapidly enhances susceptibility to colitis via depletion of luminal short-chain fatty acids in mice[J]. Sci Rep, 2019, 9(1): 12294.

[48] CHASSAING B, COMPHER C, BONHOMME B, et al. Randomized controlled-feeding study of dietary emulsifier carboxymethylcellulose reveals detrimental impacts on the gut microbiota and metabolome[J]. Gastroenterology, 2022, 162(3): 743-756.

[49] BHATTACHARYYA S, SHUMARD T, XIE H, et al. A randomized trial of the effects of the no-carrageenan diet on ulcerative colitis disease activity[J]. Nutr Healthy Aging, 2017, 4(2): 181-192.

[50] HE Z, CHEN L, CATALAN-DIBENE J, et al. Food colorants metabolized by commensal bacteria promote colitis in mice with dysregulated expression of interleukin-23[J]. Cell Metab, 2021, 33(7): 1358-1371.

[51] KWON Y H, BANSKOTA S, WANG H, et al. Chronic exposure to synthetic food colorant Allura Red AC promotes susceptibility to experimental colitis via intestinal serotonin in mice[J]. Nat Commun, 2022, 13(1): 7617.

[52] GUO M R, LIU X R, TAN Y W, et al. Sucralose

enhances the susceptibility to dextran sulfate sodium (DSS) induced colitis in mice with changes in gut microbiota[J]. Food Funct, 2021, 12(19): 9380-9390.

[53] RUIZ P A, MORÓN B, BECKER H M, et al. Titanium dioxide nanoparticles exacerbate DSS-induced colitis: role of the NLRP3 inflammasome[J]. Gut, 2017, 66(7): 1216-1224.

[54] WU X L, HOU Q H, ZHAO Z Y, et al. Effects of soy-whey protein nutritional supplementation on hematopoiesis and immune reconstitution in an allogeneic transplanted mice[J]. Nutrients, 2022, 14(15): 3014.

[55] BUMRUNGPERT A, PAVADHGUL P, NUNTHANAWANICH P, et al. Whey protein supplementation improves nutritional status, glutathione levels, and immune function in cancer patients: A randomized, double-blind controlled trial[J]. J Med Food, 2018, 21(6): 612-616.

[56] ROSIGNOLI DA CONCEIÇÃO A, DIAS K A, MICHELIN SANTANA PEREIRA S, et al. Protective effects of whey protein concentrate admixtured of curcumin on metabolic control, inflammation and oxidative stress in Wistar rats submitted to exhaus tive exercise[J]. Br J Nutr, 2022, 127(4): 526-539.

[57] BABAJAFARI S, AKHLAGHI M, MAZLOOMI S M, et al. The effect of isolated soy protein adjunctive with flaxseed oil on markers of inflammation, oxidative stress, acute phase proteins, and wound healing of burn patients; a randomized clinical trial[J]. Burns, 2018, 44(1): 140-149.

[58] METZGER C E, NARAYANAN S A, ZAWIEJA D C, et al. A moderately elevated soy protein diet mitigates inflammatory changes in gut and in bone turnover during chronic TNBS-induced inflammatory bowel disease[J]. Appl Physiol Nutr Metab, 2019, 44(6): 595-605.

[59] RAFFNER BASSON A, GOMEZ-NGUYEN A, LASALLA A, et al. Replacing animal protein with soy-pea protein in an ´american diet´ controls murine crohn disease-like ileitis regardless of firmicutes: Bacteroidetes ratio[J]. J Nutr, 2021, 151(3): 579-590.

[60] JIANG H, PRZYBYSZEWSKI J, MITRA D, et al. Soy protein diet, but not *Lactobacillus rhamnosus* GG, decreases mucin-1, trefoil factor-3, and tumor necrosis factor-α in colon of dextran sodium sulfate-treated C57BL/6 mice[J]. Journal of Nutrition, 2011, 141(7): 1239.

[61] JAYATILAKE S, ARAI K, KUMADA N, et al. The effect of oral intake of low-temperature-processed whey protein concentrate on colitis and gene expression profiles in mice[J]. Foods, 2014, 3(2): 351-368.

[62] SPRONG R C, SCHONEWILLE A J, RVD MEER. Dietary cheese whey protein protects rats against mild dextran sulfate sodium-induced colitis: role of mucin and microbiota[J]. J Dairy Sci, 2010, 93(4): 1364-1371.

[63] TUNC N, SAHIN A, DEMIREL U, et al. Favourable effects of whey protein on acetic acid-induced ulcerative colitis in a rat model[J]. Arch Med Sci, 2021, 18(6): 1617-1625.

[64] BENJAMIN J, MAKHARIA G, AHUJA V, et al. Glutamine and whey protein improve intestinal permeability and morphology in patients with Crohn´s disease: a randomized controlled trial[J]. Dig Dis Sci, 2012, 57(4): 1000-1012.

[65] MACHADO J F, OYA V, COY C S, et al. Whey and soy protein supplements changes body composition in patients with Crohn´s disease undergoing azathioprine and anti-TNF-alpha therapy[J]. Nutr Hosp, 2015, 31(4): 1603-1610.

[66] MAZZUCA F, ROBERTO M, ARRIVI G, et al. Clinical impact of highly purified, whey proteins in patients affected with colorectal cancer undergoing chemotherapy: Preliminary results of a placebo-controlled study[J]. Integr Cancer Ther, 2019, 18: 1-11.

[67] GILLIS C, LOISELLE S E, FIORE JF J R, et al. Prehabilitation with whey protein supplementation on perioperative functional exercise capacity in patients undergoing colorectal resection for cancer: A pilot double-blinded randomized placebo-controlled trial[J]. J Acad Nutr Diet, 2016, 116(5): 802-812.

[68] REN G X, YI S Q, ZHANG H R, et al. Ingestion of soy-whey blended protein augments sports performance and ameliorates exercise-induced fatigue in a rat exercise model[J]. Food Funct, 2017, 8(2): 670-679.

[69] REN G X, ZHANG J P, LI M H, et al. Gut mi-

crobiota composition influences outcomes of skeletal muscle nutritional intervention via blended protein supplementation in posttransplant patients with hematological malignancies[J]. Clin Nutr, 2021, 40(1): 94-102.

[70] HUANG Y C, ZHANG K, ZHANG L, et al. Dosage of dual-protein nutrition differentially impacts the formation of atherosclerosis in ApoE-/-Mice[J]. Nutrients, 2022, 14(4): 855.

[71] ZHANG J J, ZHANG Q W, LIU H, et al. Soy-whey dual-protein alleviates osteoporosis of ovariectomized rats via regulating bone fat metabolism through gut-liver-bone axis[J]. Nutrition, 2022, 103-104: 111723.

[72] YU Y, ZHANG J, WANG J, et al. Effect of blended protein nutritional support on reducing burn-induced inflammation and organ injury[J]. Nutr Res Pract, 2022, 16(5): 589-603.

[73] MOZAFFARI H, DANESHZAD E, LARIJANI B, et al. Dietary intake of fish, n-3 polyunsaturated fatty acids, and risk of inflam matory bowel disease: a systematic review and meta-analysis of observational studies[J]. Eur J Nutr, 2020, 59(1): 1-17.

[74] TRIANTOS C, AGGELETOPOULOU I, THOMOPOULOS K, et al. Vitamin D-liver disease association: Biological basis and mechanisms of action[J]. Hepatology, 2021, 74(2): 1065-1073.

[75] FATAHI S, ALYAHYAWI N, ALBADAWI N, et al. The association between vitamin D status and inflammatory bowel disease among children and adolescents: A systematic review and meta-analysis[J]. Front Nutr, 2023, 9: 1007725.

[76] HAUSMANN J, KUBESCH A, AMIRI M, et al. Vitamin D deficiency is associated with increased disease activity in patients with inflammatory bowel disease[J]. J Clin Med, 2019, 8(9): 1319.

[77] KIM S, KANG Y, PARK S, et al. Association of vitamin D with inflammatory bowel disease activity in pediatric patients[J]. J Korean Med Sci, 2019, 34(32): e204.

[78] HAM N S, HWANG S W, OH E H, et al. Influence of severe vitamin D deficiency on the clinical course of inflammatory bowel disease[J]. Dig Dis Sci, 2021, 66(2): 587-596.

[79] CENNI S, RUSSO G, DE N A, et al. P169 Vitamin D levels are inversely associated with inflammation in pediatric Inflammatory bowel disease patients[J]. Journal of Crohn's and Colitis, 2023, 17(1): 150.

[80] RASOULI E, SADEGHI N, PARSI A, et al. Relationship between vitamin D deficiency and disease activity in patients with inflammatory bowel disease in Ahvaz, Iran[J]. Clin Exp Gastroenterol, 2020, 13: 419-425.

[81] BAIDOUN F, SARMINI M T, ALKHADRA Y, et al. Association between vitamin D deficiency and inflammatory bowel disease (IBD)[J]. The American Journal of Gastroenterology, 2019, 114: S448.

[82] TRIANTOS C, AGGELETOPOULOU I, MANTZARIS G J, et al. Molecular basis of vitamin D action in inflammatory bowel disease[J]. Autoimmun Rev, 2022, 21(8): 103136.

[83] VERNIA F, VALVANO M, LONGO S, et al. Vitamin D in inflammatory bowel diseases. mechanisms of action and therapeutic implications[J]. Nutrients, 2022, 14(2): 269.

[84] VALVANO M, MAGISTRONI M, CESARO N, et al. Effectiveness of vitamin D supplementation on disease course in inflammatory bowel disease patients: Systematic review with meta-analysis[J]. Inflamm Bowel Dis, 2024, 30(2): 281-291.

[85] INFANTINO C, FRANCAVILLA R, VELLA A, et al. Role of vitamin D in celiac disease and inflammatory bowel diseases[J]. Nutrients, 2022, 14(23): 5154.

[86] EL AMROUSY D, EL ASHRY H, HODEIB H, et al. Vitamin D in children with inflammatory bowel disease: A randomized controlled clinical trial[J]. J Clin Gastroenterol, 2021, 55(9): 815-820.

[87] BISCHOFF S C, BAGER P, ESCHER J, et al. ESPEN guideline on clinical nutrition in inflammatory bowel disease[J]. Clin Nutr, 2023, 42(3): 352-379.

[88] YUSUF K, SAHA S, UMAR S. Health benefits of dietary fiber for the management of inflammatory bowel disease[J]. Biomedicines, 2022, 10(6): 1242.

[89] DAI F J, CHAU C F. Classification and regulatory perspectives of dietary fiber[J]. J Food Drug Anal, 2017, 25(1): 37-42.

[90] MILAJERDI A, EBRAHIMI-DARYANI N, DIELEMAN L A, et al. Association of dietary fiber, fruit, and vegetable consumption with risk of inflammatory

bowel disease: A systematic review and meta-analysis[J]. Adv Nutr, 2021, 12(3): 735-743.

[91] DAY A S, DAVIS R, COSTELLO S P, et al. The adequacy of habitual dietary fiber intake in individuals with inflammatory bowel disease: A systematic review[J]. J Acad Nutr Diet, 2021, 121(4): 688-708.

[92] DAVIS R, DAY A, BARRETT J, et al. Habitual dietary fibre and prebiotic intake is inadequate in patients with inflammatory bowel disease: findings from a multicentre cross-sectional study[J]. J Hum Nutr Diet, 2021, 34(2): 420-428.

[93] JOHANSSON M E, PHILLIPSON M, PETERSSON J, et al. The inner of the two Muc2 mucin-dependent mucus layers in colon is devoid of bacteria[J]. Proc Natl Acad Sci U S A, 2008, 105(39): 15064-15069.

[94] STEPHEN A M, CHAMP M M, CLORAN S J, et al. Dietary fibre in Europe: current state of knowledge on definitions, sources, recommendations, intakes and relationships to health[J]. Nutr Res Rev, 2017, 30(2): 149-190.

[95] 中华医学会肠内肠外营养学分会, 中国医药教育协会炎症性肠病专业委员会. 中国炎症性肠病营养诊疗共识[J]. 中华消化病与影像杂志(电子版), 2021, 11(1): 8-15.
Chinese Society for Parenteral and Enteral Nutrition, Inflammatory Bowel Disease Professional Committee of China Medical Education Association. Consensus on nutritional diagnosis and treatment of inflammatory bowel disease in China[J]. Chin J Digest Med Imageol (Electronic Edition), 2021, 11(1): 8-15.

[96] ASTORE C, NAGPAL S, GIBSON G. Mendelian randomization indicates a causal role for Omega-3 fatty acids in inflammatory bowel disease[J]. Int J Mol Sci, 2022, 23(22): 14380.

[97] MOZAFFARI H, DANESHZAD E, LARIJANI B, et al. Dietary intake of fish, n-3 polyunsaturated fatty acids, and risk of inflammatory bowel disease: a systematic review and meta-analysis of observational studies[J]. Eur J Nutr, 2020, 59(1): 1-17.

[98] ROHWER N, CHIU C Y, HUANG D, et al. Omega-3 fatty acids protect from colitis via an Alox15-derived eicosanoid[J]. FASEB J, 2021, 35(4): e21491.

[99] ABHARI M B, AFSHAR P F, ALIMORADZADEH R, et al. Comparing the effect of including omega-3 to treatment regimen in elderly patients with ulcerative colitis with placebo: A randomized clinical tria[J]. Immunopathologia Persa, 2019, 6(1): 1-10.

[100] UCHIYAMA K, NAKAMURA M, ODAHARA S, et al. N-3 polyunsaturated fatty acid diet therapy for patients with inflammatory bowel disease[J]. Inflamm Bowel Dis, 2010, 16(10): 1696-1707.

[101] MIYAGUCHI K, TSUZUKI Y, ICHIKAWA Y, et al. Positive zinc intake and a Japanese diet rich in n-3 fatty acids induces clinical remission in patients with mild active ulcerative colitis: a randomized interventional pilot study[J]. J Clin Biochem Nutr, 2023, 72(1): 82-88.

[102] KOPPELMANN T, POLLAK Y, BEN-SHAHAR Y, et al. The mechanisms of the anti-inflammatory and anti-apoptotic effects of omega-3 polyunsaturated fatty acids during methotrexate-induced intestinal damage in cell line and in a rat model[J]. Nutrients, 2021, 13(3): 888.

[103] FU Y W, WANG Y D, GAO H, et al. Associations among dietary omega-3 polyunsaturated fatty acids, the gut microbiota, and intestinal immunity[J]. Mediators Inflamm, 2021, 2021: 8879227.

[104] DURKIN L A, CHILDS C E, CALDER P C. Omega-3 polyunsaturated fatty acids and the intestinal epithelium-a review[J]. Foods, 2021, 10(1): 199.

[105] LEVINE A, RHODES J M, LINDSAY J O, et al. Dietary guidance from the international organization for the study of inflammatory bowel diseases[J]. Clin Gastroenterol Hepatol, 2020, 18(6): 1381-1392.

[106] PIGNEUR B, RUEMMELE F M. Nutritional interventions for the treatment of IBD: Current evidence and controversies[J]. Therap Adv Gastroenterol, 2019, 12: 1-12.

[107] NARULA N, DHILLON A, ZHANG D, et al. Enteral nutritional therapy for induction of remission in Crohn's disease[J]. Cochrane Database Syst Rev, 2018, 4(4): CD000542.

[108] PIGNEUR B, LEPAGE P, MONDOT S, et al. Mucosal healing and bacterial composition in response to enteral nutrition vs steroid-based induction therapy-a randomised prospective clinical trial in children with Crohn's disease[J]. J Crohn's Colitis, 2019, 13(7): 846-855.

[109] FALCONE E L, HAN Y, KREUZBURG S, et al. Exclusive enteral nutrition induced sustained changes in the microbiota and improved inflammatory bowel disease in a pediatric patient with chronic granulomatous disease[J]. J Allergy Clin Immunol Pract, 2021, 9(2): 1011-1014.

[110] HART L, FARBOD Y, SZAMOSI J C, et al. Effect of exclusive enteral nutrition and corticosteroid induction therapy on the gut microbiota of pediatric patients with inflammatory bowel disease[J]. Nutrients, 2020, 12(6): 1691.

[111] AGIN M, YUCEL A, GUMUS M, et al. The effect of enteral nutrition support rich in TGF-β in the treatment of inflammatory bowel disease in childhood [J]. Medicina (Kaunas), 2019, 55(10): 620.

[112] LV Y, LOU Y, LIU A, et al. The impact of exclusive enteral nutrition on the gut microbiome and bile acid metabolism in pediatric Crohn's disease[J]. Clin Nutr, 2023, 42(2): 116-128.

[113] ROLANDSDOTTER H, JÖNSSON-VIDESÄTER K, L FAGERBERG U, et al. Exclusive enteral nutrition: Clinical effects and changes in mucosal cytokine profile in pediatric new inflammatory bowel disease[J]. Nutrients, 2019, 11(2): 414.

[114] JIANG T Y, JIANG Y M, JIN Q W, et al. Role of perioperative nutritional status and enteral nutrition in predicting and preventing post-operative complications in patients with Crohn's disease [J]. Front Nutr, 2023, 9: 1085037.

[115] YANG Q F, HUANG Z C, DIAO N, et al. Lipidomics reveals significant alterations associated with exclusive enteral nutrition treatment in adult patients with active Crohn's disease[J]. Ann Transl Med, 2022, 10(19): 1062.

[116] WALL C L, GEARRY R B, DAY A S. Treatment of active crohn's disease with exclusive and partial enteral nutrition: A pilot study in adults[J]. Inflamm Intest Dis, 2018, 2(4): 219-227.

[117] LIU J, ANDREWS J M, TARIK S, et al. Benefits of exclusive enteral nutrition in adults with complex active crohn's disease: A case series of 13 consecutive patients[J]. Crohn's & Colitis 360, 2019, 1(3): otz044.

[118] GAJENDRAN M, UMAPATHY C, LOGANATHAN P, et al. Analysis of hospital-based emergency department visits for inflammatory bowel disease in the USA[J]. Dig DisSci, 2016, 61(2): 389-399.

[119] JENSEN G L. Inflammation as the key interface of the medical and nutrition universes: a provocative examination of the future of clinical nutrition and medicine[J]. Jpen J Parenter Enteral Nutr, 2006, 30(5): 453-463.

[120] HE P D, YU L L, TIAN F W, et al. Dietary patterns and gut microbiota: The crucial actors in inflammatory bowel disease[J]. Adv Nutr, 2022, 13(5): 1628-1651.

Research Progress on the Correlation between Dietary Nutrition and Inflammatory Bowel Disease

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Abstract The incidence of inflammatory bowel disease (IBD) is increasing globally. Epidemiological and clinical studies have found that diet affects the onset and treatment of IBD, and westernization of diet pattern is one of the important factors leading to the increase of IBD incidence. Reasonable dietary nutrition is very important for individual health, is one of the basic factors to reduce inflammation and maintain the normal operation of the immune system, and plays an important role in the prevention and treatment of diseases. At present, the clinical treatment of IBD is mainly drug therapy, which is mainly aimed at inducing and maintaining remission. In recent years, clinical studies have found that dietary intervention in patients with IBD can effectively relieve IBD symptoms and prevent disease recurrence. Dietary intervention has attracted more and more attention from researchers and clinicians because of its advantages such as low side effects, safety and economy. This paper summarizes the influence of dietary components on the onset and treatment of IBD, as well as the latest research progress on the role of dietary components and enteral nutrition in the treatment of

IBD, recommends that IBD patients undergo nutritional risk screening and professional dietary consultation, and encourages IBD patients to pay attention to daily diet management and cultivate scientific and healthy eating habits. At the same time, in view of the limitations of the current diet intervention studies, the future research direction of IBD diet intervention was proposed to provide reference for clinical nutrition intervention and diet intervention studies of IBD.

Keywords inflammatory bowel disease; dietary nutrition; dietary intervention