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Sweet Potatoes and Colitis - Bioactive Compounds, Mechanisms, and Therapeutic Potential

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ABSTRACT: Ulcerative colitis, a relapsing and remitting disease, is experiencing increasing incidence and prevalence worldwide. Sweet potatoes, a rich source of bioactive components such as polysaccharides, polyphenols, and anthocyanins, have been found to balance the gut microbiota, enhance the intestinal barrier, and promote short-chain fatty acid metabolism, which benefits colitis treatment. However, there is no comprehensive review summarizing the treatment of colitis with sweet potatoes and their active components. Therefore, this review summarizes the colitis treatment effects and potential regulatory mechanisms of various sweet potatoes and their active components. We found that sweet potatoes, particularly purple-fleshed and yellow-fleshed sweet potatoes, exhibit anti-colitis effects. Their active components—such as polysaccharides, polyphenols, and triterpenoid glycoside I—can exert more pronounced therapeutic effects by regulating intestinal microbiota and signaling pathways including NF-κB, Nrf2, and NLRP3. This study provides theoretical insights for promoting the application of sweet potatoes in the functional food field or as an adjunct therapy for colitis.

Keywords: Sweet potato, Colitis, Gut microbiota, Polysaccharides, Polyphenols

1. Introduction

Colitis, including Crohn's disease (CD) and ulcerative colitis (UC), is a non-specific, non-infectious chronic inflammatory condition of the gastrointestinal tract [1]. Previously, colitis was mainly prevalent in regions like Europe and the United States, but in recent years, it has begun to spread in newly industrialized countries in Asia, South America, and Africa due to the adoption of Western diets, increased stress, and poor

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lifestyle habits [2]. The incidence and prevalence of colitis continue to rise globally, and the prevalence of colitis will increase by 2.5 times from 2020 to 2035 [3]. Moreover, with the increasing use of biotherapies and other novel drugs, the medical burden of IBD among patients with CD and UC has increased significantly [4]. Colitis can lead to severe consequences such as loss of appetite, fatigue, diarrhea, bloody stools, and colon ulcers [5]. However, targeted treatments such as 5-aminosalicylic acid and mesalazine are expensive, have side effects, and generally result in poor patient compliance [6]. Therefore, an increasing number of scholars are using dietary interventions to combat colitis, and the number of such studies has been growing in recent years (0).

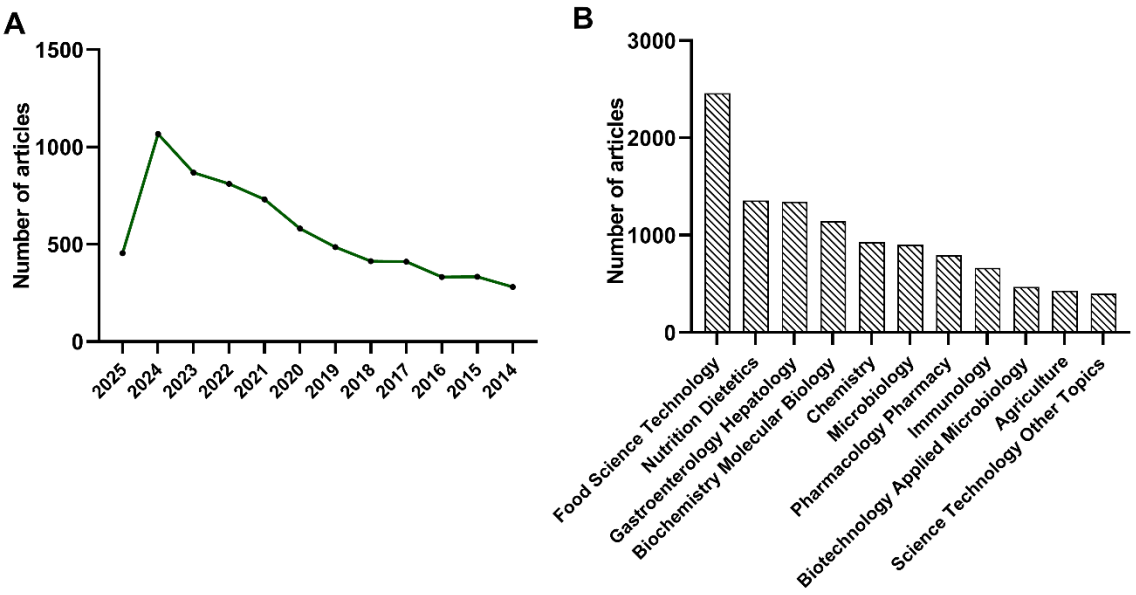


Fig.1 Statistics on food and colitis in web of science based on publication year (A) and research direction (B)

Sweet potato (*Ipomoea batatas* [L.] Lam) is a widely cultivated tuber crop, highly valued for its high yield, drought resistance, and adaptability to different climates and agricultural systems. Both its roots and leaves are edible, making it one of the best foods for addressing nutrition, food security, and poverty issues [7]. Furthermore, sweet potato is a major staple food in South America, Asia, and Africa, containing a wide range of nutrients such as calcium, iron, magnesium, vitamins A and C, polysaccharides, and polyphenols, giving it high nutritional value [8, 9]. Depending on the color, sweet potatoes are mainly divided into white, yellow, and purple types [10], and their main active components vary; for example, orange-fleshed sweet potatoes are particularly rich in β-carotene, while purple-fleshed sweet potatoes contain high levels of acylated anthocyanins and other phenolic substances. In recent years, the anti-colitis effects of active components in sweet potatoes have attracted widespread attention, and scholars have explored the alleviating effects of purple sweet potato polysaccharides, sweet potato resistant starch (RS), purple sweet potato anthocyanins, and yellow sweet potato polysaccharides on colitis symptoms. These natural compounds have the advantages of good efficacy, high patient compliance, high safety, and suitability for high-dose administration, while providing additional health benefits besides anti-inflammation, such as balancing the gut microbiota and controlling weight. Considering the excellent anti-colitis ability of sweet potatoes and the attention of

academic researchers, it is necessary to write a review summarizing the anti-colitis activity and mechanisms of sweet potato polysaccharides.

Therefore, this review discusses the therapeutic potential and mechanisms of sweet potatoes and their active components in fighting colitis, with particular attention to the anti-colitis activity of sweet potato polysaccharides, polyphenols, and anthocyanins. This review provides a theoretical basis for expanding the application of sweet potatoes in the food and pharmaceutical fields.

2. Methods

The following electronic databases were searched from the beginning to February 2025: PubMed, Web of Science, Google Scholar, and CNKI. The keywords of sweet potato and/or colitis were used truncated with other relevant topic terms, such as *Ipomoea batatas*, purple sweet potato, orange sweet potato, polysaccharide, polyphenols, anthocyanin, trifostigmanoside I, Crohn's disease, ulcerative colitis, inflammatory bowel disease, inflammation, bioactive compounds, bioavailability, action mechanism, gut microbiota, intestinal barrier. In addition, we also found some literature from references as a supplement.

3. Colitis: Definition, Treatment and Symptoms

3.1 Definition and treatment of Colitis

The colon is part of the digestive system and its main function is to absorb water and nutrients, form feces, and expel them from the body. However, when an organism suffers from colitis, the integrity of the colon is disrupted and its function is impaired. Colitis, including UC and CD, is a condition in which inflammation occurs in the colon (also known as the large intestine), usually manifesting as swelling, ulceration, and pain in the colon, or even blood in the stool [1]. The distinction between UC and CD lies in the fact that the former is a recurrent, non-penetrating inflammatory disease confined to the colon, while the latter is a recurrent, penetrating inflammatory disease of the gastrointestinal mucosa [5]. The etiology of colitis involves multiple factors, such as the genetic dimension related to family aggregation, twin disease aggregation, and genetic susceptibility and the environmental factors related to time, geography, seasonal variations, and lifestyle [11]. Moreover, psychological stress has been reported to promote the development of colitis [12]. Therefore, the relationship between phenomena such as adverse life events, chronic stress and depression and colitis has become increasingly worthy of investigation in recent years [13].

The treatment modalities for colitis encompass a diverse range of approaches, such as pharmacological interventions, immunomodulators, biologics, small molecule therapies, surgical treatments, as well as monoclonal therapies, cellular therapies, and new treatments such as exosome therapies. However, there are certain drawbacks in various treatment approaches: for example, pharmacological interventions carry serious side effects [14-17], surgical treatment has absolute or relative indications [18], and there is little evidence on the effectiveness of newer therapies, which also need to be validated in animal and clinical studies before proceeding to clinical use. Compared with conventional treatments, dietary interventions are free of toxic side

effects and, to some extent, have high patient compliance, and have been shown to reduce inflammation or lower the risk of relapse.

Despite the tremendous progress made in the modern management of colitis with the introduction of new effective drugs, the adoption of more strict testing methods, and the use of better therapeutic strategies, there are still many unmet needs [19]. For example, the diagnosis of UC is based on a combination of clinical, inflammatory, endoscopic, and histologic parameters. There is still no gold standard, and remission rates for UC are far from optimal [20]. Additionally, perinuclear antineutrophil cytoplasmic antibodies are the most specific serum biomarkers, but have low sensitivity and are not recommended for diagnosis or therapeutic management [21, 22]. In contrast, fecal calprotectin (FC)—a fecal biomarker that correlates with the number of neutrophils infiltrating the intestinal mucosa, is much more sensitive than serum biomarkers but has low specificity, which limits its differentiation from other diseases [23].

3.2 Symptoms of Colitis

3.2.1 Microbiota Dysbiosis and Decreased Production of Short-Chain Fatty Acids

The human gut microbiota consists of an ecological community including bacteria, yeasts, viruses, and parasites, producing nearly 100 trillion microorganisms [24]. The microbiota in a healthy gut is predominantly composed of Firmicutes and Bacteroidetes, accounting for approximately 90% of the microflora. These microorganisms typically affect several important gut functions, including nutrient absorption, mucosal barrier strengthening, xenobiotic metabolism, angiogenesis, and postnatal intestinal maturation [25, 26]. When there are abnormal changes in the internal and external environments, such as disease states, altered dietary structure, chronic hyperglycemia, and medication intake, changes in the structure and composition of the intestinal flora occur—these are preconditions [27, 28]. Short-chain fatty acids (SCFAs) represent the primary end products of bacterial metabolism in the human colon, primarily consisting of acetate, propionate, and butyrate. Butyrate is the preferred energy substrate for colonic epithelial cells and plays a crucial role in maintaining gut homeostasis and enhancing intestinal barrier function via balancing gut microbiota and regulating tight junction proteins [29]. Propionate mainly contributes to the maintenance of glycemic homeostasis, improves insulin sensitivity, and regulates appetite and energy balance [30]. Acetate not only participates in the regulation of appetite and human fat, cholesterol, and ketone body metabolism, but also serves as a major SCFA in the maintenance of the intestinal pH environment [31]. Treatment with SCFAs, especially butyrate, has been reported to reduce intestinal inflammation, inhibit the activation of the NF-κB signaling pathway, enhance the expression of anti-inflammatory cytokines such as IL-10, and modulate the immune system [32].

Compared to healthy subjects, patients with colitis exhibit a reduction in the diversity of the gut microbiome [33, 34], including a decrease in the abundance of beneficial microorganisms (such as Firmicutes) [35, 36], suppression of butyrate-producing bacteria[33, 37-39], and an increase in the proportion of pathogenic bacteria (such as certain species in Bacteroidetes) [34, 40, 41]. These alterations lead to diminished SCFA metabolism and compromised intestinal barrier function, thereby weakening anti-inflammatory responses,

impairing immune defense mechanisms, and accelerating disease progression. The impaired intestinal barrier further facilitates the translocation of endotoxins and bacteria into the intestinal wall, activating the immune system and stimulating the production of inflammatory cytokines. Ultimately, this cascade triggers a severe inflammatory storm, perpetuating a vicious cycle in colitis. In recent years, the gut microbiota has been recognized as a vital "organ" of the human body, and an increasing number of studies have linked this microenvironment to gastrointestinal disorders [42].

3.2.2 Changes in inflammatory markers

Inflammatory markers, usually examined using blood, fecal, and urine samples, are widely used to assess the state of intestinal inflammation and monitor disease progression. Serum C-reactive protein (CRP) and FC show the best accuracy and are among the best studied non-invasive biomarkers of inflammation in colitis [43]. Acute phase reactants in colitis, such as hemoglobin, platelet count, sedimentation, and CRP, rise rapidly in the early stages of disease progression but lack accuracy in identifying mucosal inflammation [44]. In addition, levels of inflammatory mediators such as soluble TNF receptors and IL-6 are elevated in patients with active CD, while soluble IL-1 receptor II concentrations show a negative correlation with CRP [45].

Fecal markers can mainly be categorized as leukocytes, serum proteins, or leukocyte products. They offer the advantage of non-invasively detecting localized intestinal inflammation with specificity. Among these, FC and lactoferrin are closely associated with mucosal inflammation. Their concentrations remain stable in healthy individuals, while they are markedly elevated in the fecal samples of patients with colitis and also correlated with the severity of inflammation [46]. For instance, the upper limit for FC is 10 mg/L [47], whereas in patients with CD and UC recurrence, the value of this index rises to approximately 50 mg/L, indicating a 13-fold increased risk of recurrence (sensitivity of 90% and specificity of 83%). The concentration of lactoferrin in healthy individuals is 1.45 ± 0.4 μg/g fecal weight. In contrast, the mean fecal lactoferrin concentration is 440 ± 128 μg/g in patients with CD and 1125 ± 498 μg/g in patients with UC. This biomarker effectively distinguishes inflammatory bowel disease from irritable bowel syndrome with 100% specificity [48].

Urine biomarkers such as prostaglandins [49] and leukotriene metabolites [50] have been studied, but none of them has been widely validated and none of them is commonly used in the clinic. Overall, these biomarkers are closely associated with disease status and inflammation to some extent, providing valuable insights into the pathophysiology of the disease. In the future, multivariate modeling could be employed to identify biomarkers, thereby improving the diagnosis and treatment of colitis and promoting personalized and precise therapies.

3.2.3 Changes in Intestinal Barrier

"Intestinal barrier" is a complex morphological and functional mechanism mainly composed of intestinal microbiota barrier, physical barrier, chemical barrier, and immune barrier, which collectively maintain a stable and balanced state of the body [51, 52]. The intestinal physical barrier consists of intestinal epithelial cells and tight junctions (TJ) between them, and contains claudin-2, zonula occludens-1 (ZO-1) and occludin,

which regulate the selective passage of small molecules [53]. The intestinal chemical barrier is mainly formed by the mucus layer, where mucins (including MUC2 and MUC3) play a crucial role in preventing toxic and harmful substances from reaching the surface of the intestinal epithelium [54]. The intestinal mucosal immune barrier includes gut-associated lymphoid tissue (GALT) and diffuse lymphocytes, which serve as sites of induction and activation of the immune response and as effector sites of intestinal mucosal immunity, respectively. Patients with colitis typically exhibit reduced expression or excessive degradation of TJ proteins and mucins, accompanied by compromised intestinal immune barrier function, thinning of the mucus layer, and decreased viscosity. These pathological alterations facilitate the colonization of pathogenic bacterial in the gut [55]. Furthermore, the fecal microbiota is known to contribute to mucus layer production and the restoration of intestinal mucosal barrier function, but colitis patients demonstrate increased abundance of pathogenic bacteria alongside reduced beneficial microbes, which further exacerbates mucus layer thinning. Collectively, these changes promote increased intestinal permeability, bacterial translocation, and systemic exposure to harmful bacterial metabolites (e.g., endotoxins, SCFAs, ammonia, and amines). This cascade subsequently worsens intestinal barrier dysfunction and amplifies systemic inflammatory responses, establishing a vicious cycle of inflammation [56]. However, there is currently a paucity of comprehensive studies investigating the interplay between gut microbiota dysbiosis, disruption of inflammatory markers, and impairment of intestinal barrier integrity in patients with colitis. Furthermore, few therapeutic studies have simultaneously monitored the coordinated changes in these three critical parameters during colitis treatment, leaving a significant gap in our understanding of their integrated role in disease progression and therapeutic response.

4. Bioavailability of Sweet Potato Compounds in the Human Gut

Bioavailability refers to the proportion of an active ingredient that is released from its matrix during gastrointestinal digestion, enabling it to exert biological activity at target organs [57]. Sweet potato, a globally significant staple crop, is not only rich in essential nutrients such as carbohydrates, vitamins, and minerals but also contains diverse bioactive compounds, including polysaccharides, polyphenols, anthocyanins, and carotenoids [58]. The bioavailability of these bioactive compounds in the human body—including absorption, metabolism, and stability—directly determines the extent to which their health benefits are realized [59, 60].

4.1 Absorption and metabolism of sweet potato polysaccharides in the human intestine

Sweet potato polysaccharide compounds possess large molecular weights and complex structures, which typically prevent direct absorption in the small intestine. Only a small portion is degraded into smaller fragments. *In vivo* studies using fluorescently labeled polysaccharides indicate that only a limited amount of these polysaccharides, after degradation in the small intestine, are absorbed by intestinal epithelial cells into the bloodstream, ultimately reaching organs such as the liver, spleen, and kidneys [60]. The majority of undigested sweet potato polysaccharides are transported directly to the cecum and colon. There, sweet potato polysaccharide chains are degraded by glycosidases, hydrolases, and esterases secreted by the gut microbiota, ultimately yielding monosaccharides and oligosaccharides. These small molecules can be absorbed by the

intestine or further fermented to produce metabolites. Multiple *in vitro* simulated digestion studies have revealed that sweet potato polysaccharides cannot be digested by gastrointestinal fluids during the digestive process. However, the sweet potato polysaccharide structure undergoes changes, exhibiting sweet potato polysaccharide degradation, indicating that the gut microbiota is gradually metabolizing and utilizing sweet potato polysaccharides. Furthermore, the SCFA content significantly increased in multiple groups of sweet potato polysaccharide samples, further regulating the proportion of gut microbiota to achieve the effect of preventing and treating colitis [61, 62]. However, current research on the digestive properties of sweet potato polysaccharides remains limited. Future studies may utilize fluorescent labeling techniques or *in vitro* gastrointestinal models for further investigation.

Notably, the structural characteristics of sweet potato polysaccharides significantly influence their absorption and utilization efficiency. For instance, sweet potato polysaccharides with smaller molecular weights and lower branching degrees are more readily degraded and utilized by the gut microbiota [63]. Furthermore, the fermentation of sweet potato polysaccharides is a dynamic, multi-stage process, with different sweet potato polysaccharide types being degraded by distinct microbial communities in various intestinal segments [64, 65]. Generally, fermentable sweet potato polysaccharides (e.g., RS) undergo rapid fermentation primarily in the proximal colon, while non-fermentable sweet potato polysaccharides (e.g., cellulose) reach the distal colon for slow fermentation. Compared to cellulose, the structure and fermentation characteristics of RS confer higher bioavailability and more pronounced physiological effects in the treatment of colitis [66]. Therefore, sweet potatoes with a higher proportion of fermentable polysaccharides often exhibit better bioavailability, which also translates to superior anti-colitis activity.

4.2 Absorption and metabolism of sweet potato polyphenols in the human gut

Most polyphenolic compounds exist in sweet potatoes in bound forms (e.g., esterified, glycosylated, or polymerized), which remain relatively stable in the oral cavity and stomach. These bound sweet potato polyphenols are primarily released in the small intestine through enzymatic hydrolysis or microbial action [67]. Studies indicate that the absorption rate of sweet potato polyphenols in the small intestine is relatively low, with only 5-10% of small-molecule polyphenols (such as ferulic acid, dihydroxycinnamic acid, and dihydroferulic acid) able to pass through intestinal epithelial cells into the bloodstream [59]. The remaining 90–95% of unabsorbed sweet potato polyphenols continue to travel down to the large intestine [68]. These unabsorbed polyphenols are converted into various small-molecule secondary metabolites, such as 3-hydroxyphenylpropionic acid, hippuric acid, phenylpropionic acid, pyrrole acid, tyrosol, and hydroxytyrosol, through hydrolysis reactions (e.g., deglycosylation and deesterification), cleavage reactions (e.g., C-ring cleavage), and reduction reactions (e.g., double bond reduction) under the action of enriched gut microorganisms such as *Lactobacillus* and *Bifidobacteria*. These polyphenol metabolites can regulate immune cell differentiation and play an important role in colitis protection [69]. For example, protocatechuic acid demonstrates the ability to inhibit M1 macrophage polarization and promote M2 macrophage polarization. Hydroxytyrosol and tyrosol also exhibit inhibitory effects on the gene expression of M1

macrophage markers. Therefore, the higher the microbial utilization rate of polyphenols in the large intestine stage, the more beneficial metabolites are produced, and the greater the potential for alleviating colitis.

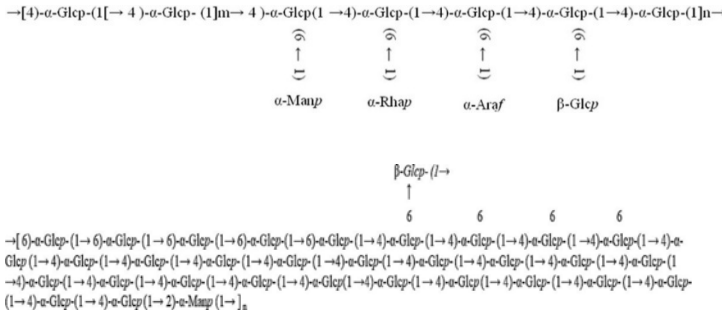
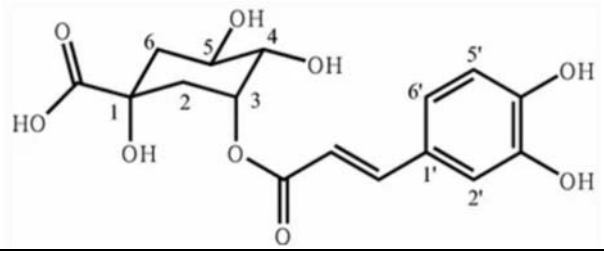
4.3 Stability of sweet potato polysaccharides and polyphenols in the human gut

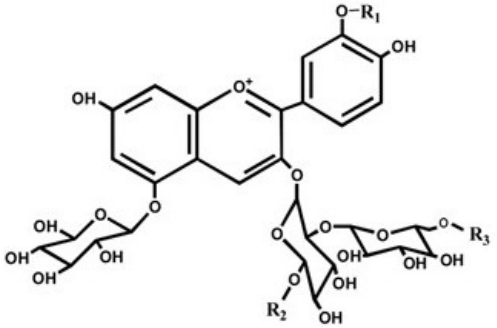
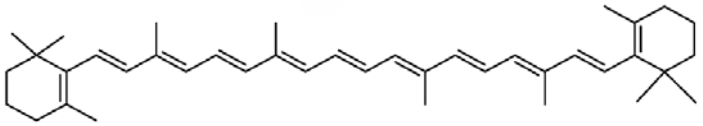
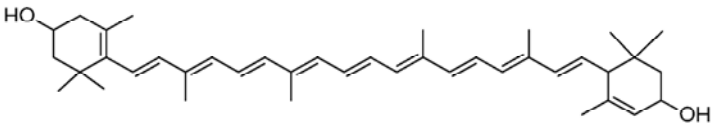
Beyond structural characteristics such as molecular weight, monosaccharide composition, glycosidic bond type, and acylation modifications—which significantly influence their stability in the gut—processing methods and changes in the gastrointestinal environment also affect the stability of sweet potato polyphenols and polysaccharides within the human digestive tract. For instance, thermal processing—one of the most common sweet potato preparation methods—can disrupt cell wall structures, releasing bound polyphenols and polysaccharides to enhance their bioavailability. Conversely, it may also degrade heat-sensitive compounds [70, 71]. For instance, boiling and baking result in significant losses of vitamin C and certain anthocyanins in sweet potatoes, whereas steaming and microwave heating better preserve these heat-sensitive compounds. The acidic environment in the oral cavity and stomach (pH 1.5–3.5) has a relatively minor impact on the stability of sweet potato polyphenols and polysaccharides, with most sweet potato polyphenols and polysaccharides remaining relatively stable in the stomach. Conversely, the alkaline environment (pH 6.5–7.5) and digestive enzymes (e.g., amylase, protease, lipase) in the small intestine significantly affect sweet potato polyphenols and polysaccharides stability [67]. Higher stability means more active components can reach the intestines and exert their effects, thereby enhancing anti-colitis activity. Future research should further explore the absorption, metabolism, and stability mechanisms of sweet potato polysaccharides, polyphenols, and specific active components in the human gut, and develop processing technologies and delivery systems that improve the utilization of sweet potato's active components.

5. Colitis Treatment Effects of Sweet Potato

The anti-colitis effects of sweet potatoes and their bioactive components are of great interest (0), particularly polysaccharides, RS, anthocyanins, polyphenol extracts, and trifostigmanoside I. These natural compounds demonstrate individual and synergistic benefits in preventing pathological colonic damage and colon shortening, improving disease activity index (DAI) scores, modulating gut microbiota balance, protecting intestinal barrier integrity, and stimulating SCFA production (Table 1) . The following discussion will provide a comprehensive analysis of both whole sweet potatoes and their specific active constituents in combating colitis.

Table 1 Summary of active components in sweet potatoes

Active Ingredients	Definition	Chemical formula	Content	Biological Function	References
Polysaccharides	Monosaccharides composed of alpha-1,4 glycosidic bonds or beta-1,4 glycosidic bonds in tandem		0.40 g/kg	Gut microbiota regulation, anti-inflammatory, hypoglycemic, antioxidant and anticancer effects	[75, 77-79, 81, 105, 118, 119]
Dietary Fiber	Mainly composed of cell wall polysaccharides (such as cellulose, hemicellulose) and pectin, belonging to plant indigestible carbohydrates, divided into soluble and insoluble fiber	β-d-Xylp-(1→4) β-d-Xylp-(1→3)-α-l-Rhap-(1→2)-α-d-GalpA (1→4)-d-Xylp	110 g/kg	Strongest ABTS and DPPH free radical scavenging ability; modulated gut microbiota and improved gut barrier	[120-123]
Resistant Starch	Starch fractions that resist small intestinal hydrolysis within 120 min but can be fermented in the colon consist primarily of linear α-1,4-D glucans (molecular weight ~1.2 × 105 Da) derived from retro-degradable starches	Consists of D-glucose residues that are covalently linked mainly by α-(1 → 4) glycosidic bonds	401-551 g/kg	Glucose control, improved gut health, lipid regulation, enhanced satiety and micronutrient absorption	[82, 124-126]
Chlorogenic Acid	It is a polyphenol compound consisting of quinic acid and caffeic acid linked by an ester bond		Total polyphenol content: 14.75 g chlorogenic acid equivalent/kg	Inhibited NO production, reduced TNF-α, IL-6, and iNOS levels, and restored intestinal barrier integrity	[104, 127]

Anthocyanins	A flavonoid compound with a C6-C3-C6 basic skeleton and formed by modification of substituents such as hydroxyl and methoxy groups and glycosidation, with structural features that give it unique color and functionality		0.905–10.187 g/kg		
β-Carotene	Fat-soluble carotenoids, precursors of vitamin A		0.038–0.738 g/kg	Antioxidant, anti-inflammatory, anti-mutagenic, anti-tumor, anti-obesity, anti-colitis, anti-diabetic, etc.	[10, 128-131]
Lutein	It is a class of oxygenated carotenoids (e.g., zeaxanthin and its esters) with C40 tetraterpene backbone, β/ε-phytocarotenone ring (hydroxylated at 3 positions) and 9-11 conjugated double bonds, with photoprotective and antioxidant activities		0.15–0.33 mg/kg		
Pg3gal	Anthocyanins, a subclass of flavonoid compounds, are formed by the covalent attachment of a galactose moiety to the C3 hydroxyl group of pelargonidin (an anthocyanidin aglycone) via a β-glycosidic bond	/	/	Inhibiting activation of the NLRP3 inflammsome pathway and balancing intestinal flora	[89]

Pg3gal, pelargonidin-3-galactoside.

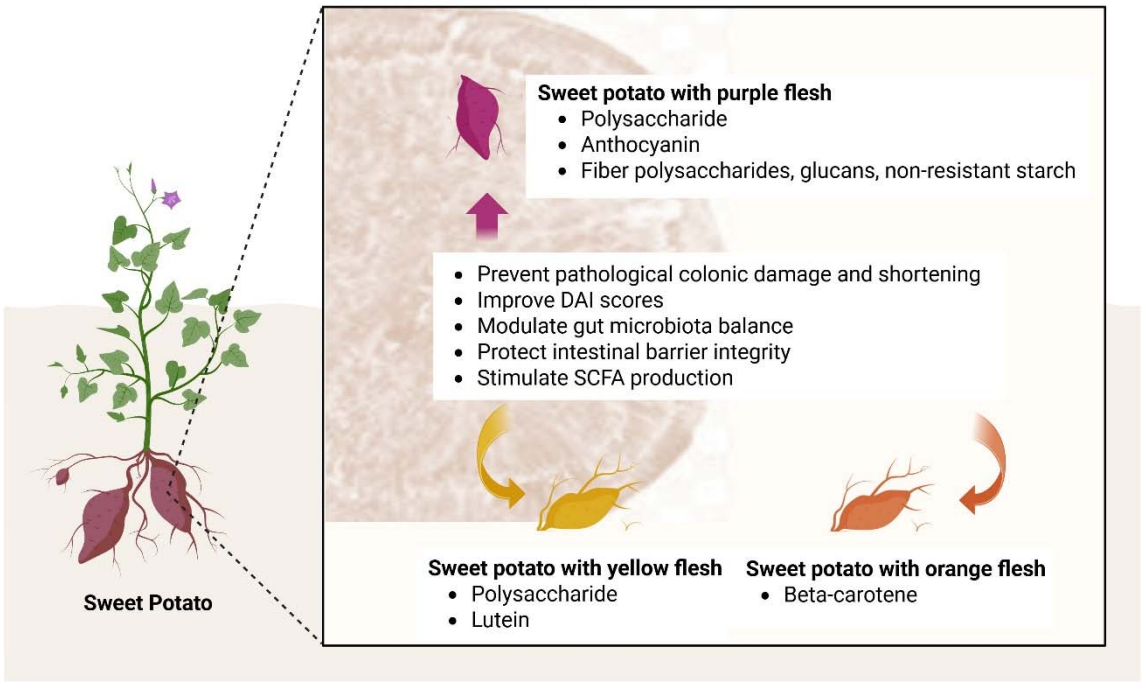


Fig.2 Bioactive components and effects of sweet potato. Sweet potatoes, including purple-, yellow-, and orange-fleshed varieties, are rich in bioactive compounds such as polysaccharides, anthocyanins, and β-carotene. These components alleviate colitis by preventing pathological colonic damage and shortening, improving DAI scores, modulating gut microbiota, maintaining intestinal barrier integrity, and enhancing SCFA production. (Created with BioRender.com)

5.1 Anti-colitis effect of whole sweet potato

Sweet potato is a creeping dicotyledonous plant, exhibiting a range of colors, from white and cream to yellow, orange, and even purple [10]. Nutritionally, sweet potato is rich in carbohydrates, proteins, carotenoids, flavonoids, terpenoids, alkaloids, anthocyanins, and minerals (K, P, Ca, Mg, Fe, Cu) [9]. Among them, orange-fleshed sweet potatoes are particularly rich in β-carotene, while purple-fleshed sweet potatoes contain high levels of acylated anthocyanins and other phenolic substances. These nutrients contribute to a wide array of health benefits, including antioxidant, cardioprotective, antidiabetic, anti-inflammatory, anticancer, antimicrobial, anti-obesity, and immunomodulatory effects. Consuming whole sweet potatoes effectively enhances nutrient bioavailability, improves micronutrient intake (particularly B vitamins and minerals), and enables synergistic interactions among their diverse components. Some scholars have used sweet potatoes—especially purple sweet potatoes—to treat colitis and found that it has a series of anti-inflammatory effects (0), such as improving the gut microbiota, preventing mucosal damage and intestinal barrier damage, inhibiting the secretion of cytokines, and improving mitochondrial function [69, 72]. Sun, Bibi, et al. found that incorporating 10% purple sweet potato powder into the diet of colitis-induced mice alleviated weight loss, improved DAI scores, reduced colon shortening, and mitigated clinical symptoms such as watery diarrhea and bloody stools [73]. Histological analysis confirmed its protective effects on mucosal and crypt architecture. Further research showed that purple sweet potato also participated in immunomodulation, which inhibited the activation of macrophages and down-regulated the levels of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6, and IL-18), and also enhanced intestinal barrier integrity (the expression levels of ZO-1, ZO-2, and occludin

increased). Additionally, Zhu, Bibi, et al. found that daily consumption of feed containing 10% purple sweet potatoes improve the colitis symptoms and the structure of the colonic epithelium in IL-10-deficient mice (a CD model) [72], reduced histopathological scores, and enhanced the intestinal barrier. Purple sweet potatoes can also regulate the gut microbiota of mice, manifested as improved gut microbiota diversity, decreased abundance of Proteobacteria and Deferribacteres, and increased abundance of Firmicutes and Tenericutes. However, the impact of dietary purple sweet potatoes on SCFA production remains unexamined. However, the drawback of these studies is that they did not identify which active components of purple sweet potato played the main role.

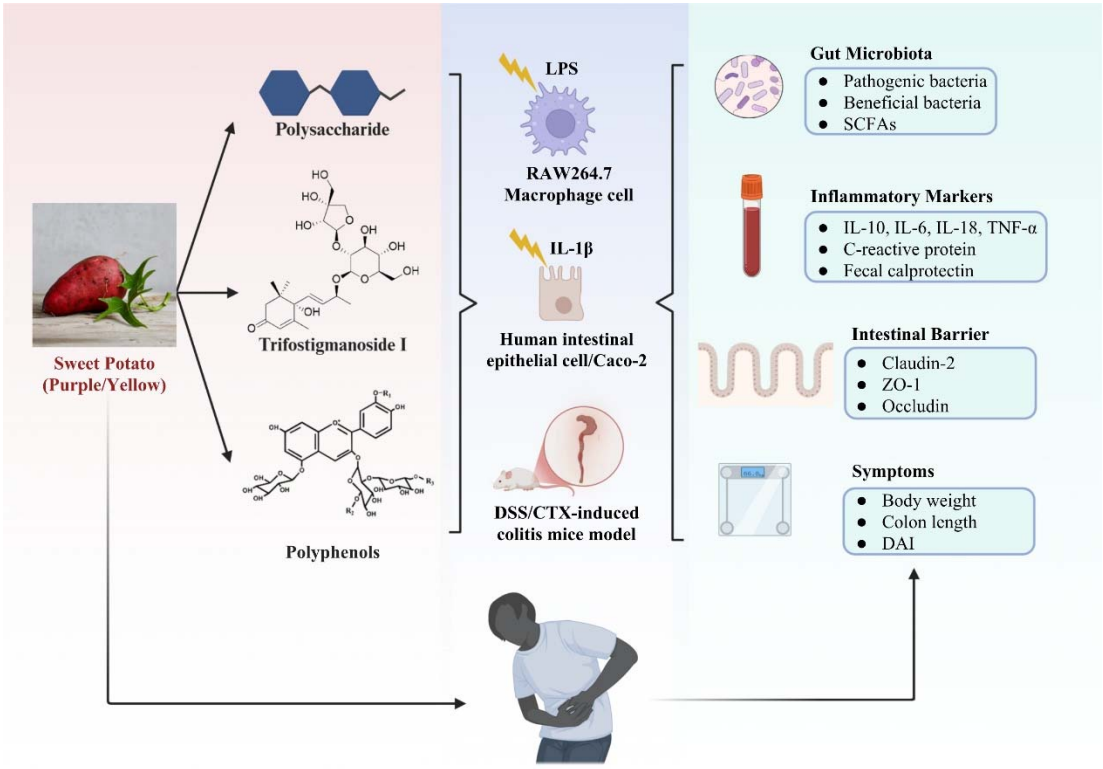


Fig.3 A Unified Framework for the Anti-Colitis Effects of Sweet Potatoes and Their Active Components. (Created with BioRender.com)

Some scholars have also used metabolomics tools to explore the regulatory mechanism of purple sweet potatoes on colitis. Sun, Iniguez, et al. [69]observed that purple sweet potatoes ameliorated colitis through modulation of purine, amino acid, and fatty acid metabolism pathways as well as the malate-aspartate shuttle. Additionally, purple sweet potato inhibited M1 macrophage polarization while promoting M2 polarization, evidenced by reduced COX2 and iNOS (M1 markers) and elevated IL-4 and Fizz1 (M2 markers). Despite these advances, current research disproportionately focuses on purple sweet potatoes, leaving the effects of sweet potato leaves, yellow-fleshed, and orange-fleshed varieties on colitis largely unexplored.

5.2 Anti-colitis effect of sweet potato polysaccharide

Polysaccharides, as natural macromolecules, represent one of the most significant bioactive constituents in sweet potatoes. are usually composed of more than ten monosaccharide molecules, which are linked by α- or β-glycosidic bonds, ultimately forming backbones and branches ranging from thousands to millions, as well as various combinations of natural polymers. Sweet potato polysaccharides are biodegradable, non-toxic, and

exhibit broad-spectrum bioactivities including gut microbiota modulation, anti-inflammatory, hypoglycemic, antioxidant, and anticancer effects [74-76]. Water extraction, alkaline extraction, and enzymatic hydrolysis represent three principal methods for obtaining sweet potato polysaccharides, with each technique yielding polysaccharides possessing distinct structural characteristics, bioactivities, and protein contents. Tang, Sun, et al. discovered that in purple sweet potatoes, water-soluble polysaccharides (WSP) contain 72.83% carbohydrates and 1.44% protein; dilute alkali-soluble polysaccharides (DASP) contain 65.74% carbohydrates and 1.33% protein; concentrated alkali-soluble polysaccharides (CASP) contain 69.06% carbohydrates and 1.30% protein. Multiple studies [77-79] indicate that in dextran sulfate sodium (DSS)-induced colitis models, purple sweet potato WSP ameliorated weight loss, colon shortening, and histopathological damage while suppressing pro-inflammatory cytokines (IL-1β, TNF-α, IL-6) and oxidative markers (MDA) and restoring the anti-inflammatory cytokines IL-10 and antioxidants (SOD, T-AOC). Feng, Du, et al. reported that yellow sweet potato polysaccharides similarly exhibited anti-colitis effects consistent with these findings [80]. The former's anti-colitis effects were attributed to its modulation of the Firmicutes/Bacteroidetes (F/B) ratio, suppression of Proteobacteria, and enrichment of beneficial microbes, thereby elevating acetate, propionate, and butyrate levels. The latter effect may enhance SCFA production and suppress inflammatory cytokines via the GPR41/MEK/ERK1/2 pathway. However, comparative studies are needed to determine whether yellow sweet potato polysaccharides outperform their purple counterparts. In addition, the positive changes in colitis are also related to the activation of pathways such as carbohydrate metabolism, amino acid metabolism, glycan biosynthesis and metabolism, cofactor and vitamin metabolism—reflecting the multi-faceted improvement effect of purple sweet potato polysaccharides. Sun et al. found that purple sweet potato polysaccharide inhibited the levels of metabolites such as oleic acid, 17-hydroxyprogesterone, and neocembrene, and increased the levels of metabolites such as equol, 5′-deoxyadenosine, flavanone, and (S)-9-hydroxy-10-undecenoic acid, which was related to the regulation of cutin, suberine and wax biosynthesis, biosynthesis of unsaturated fatty acids, fatty acid biosynthesis, and steroid hormone biosynthesis by purple sweet potato polysaccharide [81]. Correlation analysis established positive associations between pro-inflammatory metabolites and *Rikenella*, *Odoribacter*, *Bacteroides*, *Staphylococcus*, whereas *Bifidobacterium, Gemella*, and *Aerococcus* correlated with anti-inflammatory metabolites. However, currently, there are few studies using metabolomics to explore the mechanism of sweet potato polysaccharides intervening in colitis, and the current research is only focused on purple sweet potato polysaccharides.

Polysaccharides extracted from sweet potatoes through different methods exhibit varying degrees of anti-colitis efficacy. WSP and CASP from sweet potato restored spleen parameters and immune cytokine levels (e.g., IL-2 and IL-6), while DASP enhanced TNF-α, IL-2, and IL-6 levels. All three polysaccharides increased SCFA production [75]. Therefore, polysaccharides obtained by different extraction methods have different anti-colitis effects. In the future, it is advisable to design more appropriate treatment plans according to the symptoms of colitis patients. In addition to identifying the polysaccharide components from sweet potatoes, some scholars have explored the efficacy of special sweet potato polysaccharide components, such

as sweet potato fiber polysaccharides, dextrans, or RS components, in intervening in colitis. Both SRDF and RS improve colon length, histopathology, and enhance intestinal barrier function. The former increases the expression of TJ proteins (ZO-1, occludin, TJ-1), while the latter limits goblet cell loss and intestinal epithelial cell apoptosis [82]. This may attribute to their positive microbiota-modulating effect (increased *Lactobacillus*, *Alloprevotella*, *Bifidobacterium*, and Firmicutes/Bacteroidetes ratio; decreased *Staphylococcus*, *Bacteroides*, and *Akkermansia*) and SCFA-stimulating effects (acetate, propionate, butyrate), as well as modulation of carbohydrate/lipid/amino acid metabolic pathways. However, a shortcoming of this study is that it did not compare the colitis treatment effects of different fiber polysaccharides. Currently, most research on special polysaccharide components—such as RS and dextran—focuses on purple sweet potatoes, and few studies pay attention to the anti-colitis effects of the special components of red sweet potatoes and yellow sweet potatoes. In addition, comparative studies evaluating the anti-colitis activities of distinct polysaccharide components across different sweet potato varieties are essential to identify the most therapeutically potent fractions, thereby facilitating their clinical translation.

5.3 Anti-colitis effect of sweet potato polyphenols

Polyphenols represent a diverse class of bioactive and ubiquitous plant secondary metabolites, offering advantages such as easy accessibility, high reaction specificity, and low toxicity [83]. They are primarily categorized into flavonoids and phenolic acids. The former mainly exist in the tuberous roots of sweet potatoes, where anthocyanins, carotenoids, and lutein are dominant in purple, orange, and yellow varieties, respectively [84]. The latter are predominantly composed of CA and caffeoylquinic acid (CQA) derivatives, which are distributed throughout vegetative tissues, including leaves, petioles, stems, and storage roots. Moreover, polyphenols in sweet potato leaves and flesh both exhibit significant anti-inflammatory effects. A study on sweet potato leaves using an *in vitro* anti-colitis model revealed that polyphenols can inhibit NO production, reduce levels of TNF-α, IL-6, and iNOS, restore intestinal barrier integrity (upregulation of ZO-1, claudin-1, and occludin expression), and enhance the integrity of the Caco-2 cell monolayer. This effect may be related to the inactivation of the NF-κB inflammatory pathway. However, it remains unclear whether the phenolic acids in sweet potato leaves can also exert good anti-colitis effects *in vivo*. A study on sweet potato flesh using a general anti-inflammatory model with macrophages [85] revealed that polyphenols can reduce levels of IL-6, TNF-α, iNOS, and ROS, activate the Nrf2 pathway, and inhibit the activation of the NF-κB pathway. However, this provides only indirect evidence for potential anti-colitis applications. Li et al. incorporated the purple sweet potato polyphenols into the diet of mice and found that as the content of purple sweet potato polyphenols in the diet increased, the symptoms of colitis in mice improved gradually. Specifically, the high-dose polyphenol group [86] exhibited a more significant increase in colon length, significantly reduced levels of IL-6, IL-17, and IL-1β, and showed marked suppression of *Escherichia* and an increase in *Akkermansia* levels. These findings collectively suggest that purple sweet potato polyphenols not only effectively ameliorate colitis but can also be administered at relatively high doses without adverse effects.

Purple sweet potatoes are rich in anthocyanins, the chemical structures of which are mainly composed of cyanidin and paeoniflorin in the forms of monoacylation and diacetylation, and which exhibit high thermal stability and UV resistance. According to Cevallos-Casals and Cisneros-Zevallos, the content of anthocyanin in purple sweet potatoes far exceeds that found in purple carrots, purple corn, and red grapes [87]. Fecal transplant experiments confirmed that purple sweet potato anthocyanins mainly exert anti-colitis effects by regulating the intestinal microbiota. The study by Mu et al. demonstrated that the purple sweet potato anthocyanins regulate the gut microbiome by restoring the proportions of probiotics such as *Bifidobacterium*, *Turicibacter*, and *Lactobacillus*, and by inhibiting the proportions of pathogens such as *Helicobacter* and *Staphylococcus*. This manifested as reduced weight loss in mice, increased ratio of colon weight to length, improved pathological damage, elevated expression of ZO-1 and occludin, and inhibited the pro-inflammatory factors TNF-α and IL-6 [88]. However, the drawback of this study is that it did not detect the components of anthocyanins in purple sweet potato, making it unclear which functional components actually play a role. Additionally, single components from purple sweet potato anthocyanins might exhibit higher anti-colitis activities compared to whole purple sweet potato anthocyanins. For instance, pelargonidin-3-galactoside (Pg3gal) is an effective active ingredient isolated from purple sweet potato anthocyanins. When used at a concentration of 10–50 mg/kg, it significantly improved colitis symptoms [89]. Further research found that Pg3gal can inhibit the activation of the NLRP3 inflammasome pathway and balance the intestinal microbiota (decrease in Firmicutes, Bacteroidetes, and Verrucomicrobia; increase in Proteobacteria and Deferribacteres). In addition, some protein-bound anthocyanin compounds extracted from sweet potato (p-BAC-PSP) also exhibit anti-inflammatory effects [90]. While their anthocyanin content is comparable to that of free anthocyanin compounds (FAC-PSP), FAC-PSP demonstrates stronger anti-colitis effects by more effectively modulating the Nrf2 and NF-κB signaling pathways. However, these findings were obtained using general anti-inflammatory models rather than anti-colitis mouse models, which may lead to differences between the results and the actual anti-colitis outcomes *in vivo*. There may be differences in the anti-colitis effects of polyphenols from purple sweet potato, yellow sweet potato, and sweet potato leaves. However, current research mainly focuses on the anti-colitis activity of the polyphenols and anthocyanins from purple sweet potato, and there is a lack of comparative studies on the anti-colitis effects of polyphenols from various sweet potato sources.

5.4 Anti-colitis effect of Trifostigmanoside I

Trifostigmanoside I, an active compound derived from sweet potatoes with the chemical formula (6S,7E,9R)-6,9-dihydroxymegastigma-4,7-dien-3-one-9-O-β-D-apiofuranosyl-(1→2)-β-D-glucopyranoside, remains poorly characterized in terms of its biological activities [91, 92]. Emerging evidence suggests that trifostigmanoside I isolated from sweet potatoes may aid in the treatment of colitis [91]. In one study, this compound significantly upregulated MUC2 expression through activation of PKCα/β and its downstream effector ERK1/2, ultimately enhancing intestinal barrier function. Nonetheless, the investigation into trifostigmanoside I in this study is insufficient. Consequently, we do not have a clear understanding of other

colitis-related activities of trifostigmanoside I, including its potential to improve DAI scores, alleviate histopathological changes, modulate gut microbiota composition, and stimulate SCFA production. In addition, considering the excellent therapeutic effect of trifostigmanoside I on the colonic mucosa, this compound could be strategically combined with other therapeutic agents to enhance the overall therapeutic effects in colitis.

6. Anti-colitis mechanism of sweet potato

The regulatory effects of sweet potatoes and their bioactive components on colitis have been extensively investigated by researchers (Table 2). And the correspondence between components and key results is shown in 0. The most critical signaling pathways and biological system involved include the gut microbiota, NF-κB pathway, Nrf2 pathway, and NLRP3 inflammasome pathway (0).

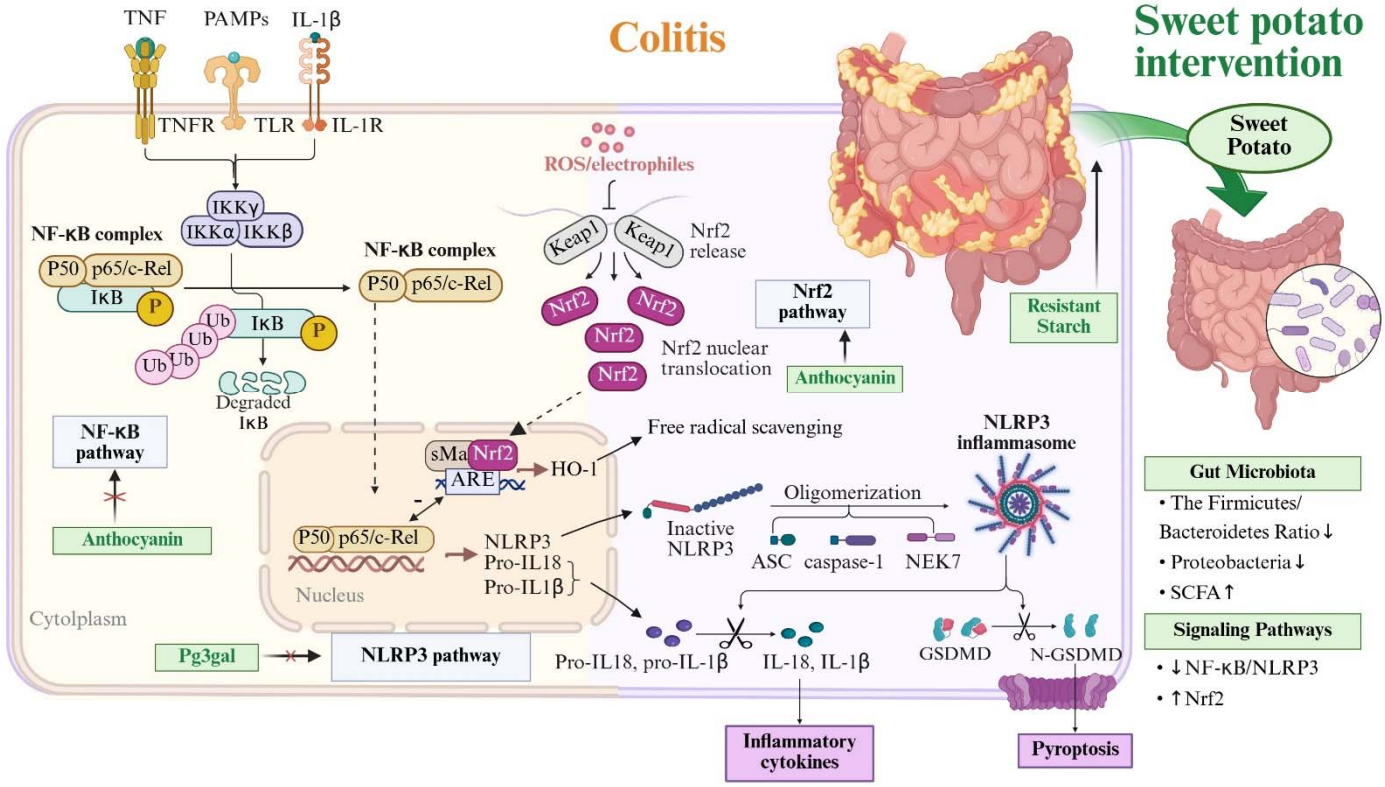


Fig.4 Diagram of the anti-colitis mechanism of sweet potato. The mechanism of inflammatory response in colitis involves inflammatory factors, receptors, NF-κB pathway and NLRP3 inflammasome pathway. In colitis, inflammatory factors such as TNF, PAMPs, and IL-1β activate the IKKα/IKKβ complex through receptors, leading to IκB degradation and release of NF-κB complexes (P50/p65/c-Rel) into the nucleus to promote inflammatory gene expression. Meanwhile, ROS and electrophiles inhibited Nrf2 release, further activating NF-κB complexes and promoting inflammatory gene expression. Inflammatory factors and ROS activated NLRP3 inflammatory vesicles, leading to the maturation and release of IL-18 and IL-1β, which triggered the inflammatory response and focal death. Sweet potato intervenes to alleviate colitis through multiple mechanisms. Anthocyanins in sweet potato reduce the production and release of inflammatory factors, scavenge free radicals, and alleviate oxidative stress by inhibiting the NF-κB/NLRP3 pathway as well as activating the Nrf2 pathway, which ultimately reduces the inflammatory response and jowls and alleviates colitis. Resistant starch in sweet potato can also regulate intestinal microorganisms, increase the production of SCFA, and decrease the Firmicutes/Bacteroidetes ratio and Proteobacteria levels, etc. (Created with BioRender.com)

Table 2 Therapeutic effects and mechanisms of sweet potato active components on colitis

Active ingredient	Composition	Model	Dose	Metric changes	Gut microbiota	SCFAs	Intestinal barrier	Signaling pathways	References
Dietary PP	Total polyphenol content: 14.75 g chlorogenic acid equivalent/kg dry weight	Seven-week-old male IL-10-deficient mice	100 g/kg	Body weight ↑; DAI, IL-1B, IL-6 ↓; improved histopathology	F/B ↑	BA ↑	Goblet cells, Muc2, Muc3, ZO-1 and Occludin ↑	NF-κB pathway ↓	[72]
Dietary PP	/	DSS-induced colitis model in six-week-old male C57 BL/6 J mice	100 g/kg	M2 polarization of macrophages, IL-4 ↑; amino acids, fatty acids, organic acids, carbohydrates, and polyamines, COX2, iNOS ↓	*Bifidobacterium* and *Lactobacillus* ↑	/	/	Amino acids, fatty acids, organic acids, carbohydrates, and polyamines were altered	[69]
Dietary PP Supplement	PP powder = 5.01% water+ 3.54% crude protein+ 2.50% crude ash+ 5.80% crude insoluble fiber+ soluble carbohydrate	DSS-induced colitis model in six-week-old male C57 BL/6 J mice	100 g/kg	Body weight, colon length ↑; mitochondrial protein homeostasis, DAI, TNF-α, IL-1β, IL-6, IL-18, colonic mucosal damage, crypt deformity and inflammation↓; improved histopathology	/	/	ZO-1, Occludin, Claudin-1 ↑	/	[73]
Whole PP	/	DSS-induced colitis model in six-week-old male C57BL/6J mice	100 g/kg	Body weight, colon length ↑; DAI, diarrhea and hemorrhage symptoms, colonic mucosal damage, crypt deformity and inflammation ↓	/	/	/	/	[132]
SRDF	Contains glucose (38.47 %), galactose (21.81 %), arabinose (9.12 %), xylose (5.7 %), and mannose (1.56 %)	High-fat diet induced loss of intestinal integrity model in five-week-old male C57BL/6 J mice	0.27, 0.54 and 1.08 g/kg	Colon length, IL-22 ↑; CRP, IL-6, TNF-α, LPS ↓; crypt architecture normalization and improved submucosal edema	*Bifidobacterium* ↑; F/B ↓	PA, BA ↑	Goblet cells, ZO-1, Occludin, Claudin-1 ↑	M2-mAchR/TLR 2 pathway ↑; NF-κB pathway ↓	[120]

PSPRS	Protein content <1%; characterized by C A-type crystals	DSS-induced colitis model in four-week-old males ICR mice	0.10 and 0.30 g/kg	Body weight, colon length, IL-10, IgA ↑; IL-1β, IL-6, TNF-α, MPO, inflammatory cell infiltration and crypt reduction↓; improved colon histopathology	*Lactobacillus* ↑; *Bacteroides*, *Staphylococcus*, *Akkermansia*, *Jeotgalicoccus* ↓; F/B ↑	AA, PA, BA ↑	Symptoms of cupping disappearance ↓; intestinal barrier ↑	NF-κB pathway ↓	[82]
ASPP	Consists of a backbone of 1,4-α-D-glucopyranosyl residues and a side chain substituted at the O-6 position, which consists of terminal α-d-mannopyranosyl, α-d-rhamnopyranosyl, α-larabinofuranosyl and β-d-glucopyranosyl residues; MW: 180kDa	DSS-induced colitis model in four-week-old female ICR mouse	0.40 g/kg	Immune organ index, colon length ↑; IL-1β, IL-6, and TNF-α ↓; improvement in colon histopathology	*Lactobacillus*, *Roseburia* ↑; *Bacteroides*, *Staphylococcus* ↓; F/B ↑	AA, PA ↑	ZO-1, occluding ↑; goblet cells, MUC2 ↓	NF-κB pathway ↓	[79]
Root WSP	Consists mainly of →4)-α-D-Glcp-(1→ and →4,6)-α-D-Glcp-(1→ ; MW: 103 kDa, Glc: Man = 20.44:1.00	LPS-induced inflammation model of RAW264.7 cells	0.40 g/kg	IL-10, T-AOC, SOD ↑; NO, TNF-α, IL-1β, IL-6 ↓	*Bacteroides*, *Lactobacillus*, *Bifidobacterium*, *Parabacteroides* ↑; *Psychrobacter*, *Staphylococcus* ↓; F/B ↑	AA, PA, BA ↑	/	NF-κB pathway ↓	[78]
YSPP	α-L-Araf-(1→, →5)-α-L-Araf-(1→, →6)-β-D-Galp-(1→, β-D-Glcp-(1→, →3)-α-L-Araf-(1→, →3)-α-L-Rhap-(1→; Rha:Ara:Gal:Glc:Gal A:GlcA= 1.00:2.00:3.63:1.21:1. 17:1.14	DSS-induced colitis model in SPF male C57 BL/6J mice	0.10 and 0.20 g/kg	Body weight, colon length, thymic index, IL-6, IL-1β ↑; DAI, splenic index, IL-10, inflammatory cell infiltration and crypt reduction↓; improved histopathology	/	AA, PA, BA, isobuty ric acid ↑	Goblet cells ↑	PR 41/MEK/ERK 1/2 signaling pathway ↑; NF-κB pathway ↓	[80]

PSPP	Purified homogeneous polysaccharides; MW: 103 kDa and 180 kDa, respectively	DSS-induced colitis model in four-week-old ICR mice	0.40 g/kg	IL-10 ↑; TNF-α, IL-6, IL-1β ↓	*Lactobacillus*, *Bifidobacterium* ↑; *Parasutterella*, *Desulfovibrio* ↓	/	/	Significant changes in 25 metabolites, such as fatty acid biosynthesis, steroid hormone metabolism, and indole derivatives	[81]
WSP DASP CASP	Carbohydrate and protein content: WSP = 72.83% and 1.44%; DASP= 65.74% and 1.33%; CASP= 69.06%+ 1.30%; WSP and CASP have pyran-type structures; CASP has typical glucose residues in the form of pyranose	CTX-treated model in four-week-old female ICR mouse	0.40 g/kg	Splenic index, TNF-α, IL-2, IL-6, TNF-α ↑	*Ruminococcus* and *Oscillospira* ↑; *Sutterella* ↓; F/B ↑	AA, PA, BA ↑	/	/	[75]
WSP	Mainly composed of →4)-α-D-Glcp-(1→ and →4,6)-α-D-Glcp-(1→ ; MW: 103 kDa, Glc: Man = 20.44:1.00	DSS-induced colitis model in four-week-old female ICR mouse	0.40 g/kg	Body weight, colon length, IL-10, T-AOC, SOD ↑; DAI, IL-1β, IL-6, and TNF-α ↓; improved colon histopathology	*Lactobacillus* ↑; F/B ↑	AA, PA, BA ↑	/	/	[77]
PSPP-1 (novel dextran)	The main chain consists of the →4)-α-d-Glcp (1→ glycosyl group and branches at the O-2, O-3 and O-6 positions of the α-d-Glcp (1→ residue)	LPS-induced inflammation model of RAW264.7 cells	0.04, 0.08 and 0.16 g/kg	NO, IL-1β, IL-6, TNF-α, IFN-β ↓	/	/	/	MyD88, MAPK, NF-κB and AP-1 signaling pathways, TRIF-dependent pathway ↑	[103]

PSPP-1	The main chain is α-(1 → 4)-D-glutamyl, branching into 1 → 2, 1 → 3, and 1 → 6 glycosidic bonds	LPS-induced inflammation model of RAW264.7 cells	0.04, 0.08 and 0.16 g/kg	L-4, IL-10 ↑; Cell proliferation, NO, ROS, Ca^{2+}, IL-1β, IL-6, TNF-α ↓	/	/	/	MyD88, MAPK, NF-κB and AP-1 signaling pathways, TRIF-dependent pathway ↑	[105]
WSP DASP CASP	Carbohydrate and protein content: WSP = 72.83% and 1.44%; DASP= 65.74% and 1.33%; CASP= 69.06%+ 1.30%	LPS-induced inflammation model of RAW264.7 cells	0.025, 0.050, 0.100, 0.200, 0.400 and 0.800 g/kg	Cell viability，IL-10 ↑; hagocytosis, NO, TNF-α, IL-1β, IL-6, apoptosis ↓	/	/	/	/	[118]
PSPAE	Five phenolic acid standards: chlorogenic acid, caffeic acid, trans ferulic acid, p-coumaric acid and protocatechuic acid	DSS-induced colitis model in four-week-old male C57BL/6 mice	150 and 250 g/kg	Body weight, colon length ↑; Splenomegaly, liver hypertrophy, IL-6, IL-17, IL-1β, inflammatory cell infiltration and crypt reduction ↓	*Escherichia* and *Akkermansia* ↑	/	Goblet cells ↑	AP-1, NF-kB and MAPK signaling pathways ↓	[86]
PSPAE	Paeoniflorin-3-sophoroside-5-glucoside and anthocyanin-3-sophoroside-5-glucoside, mono- or di-acylated from caffeic acid and p-coumaric acid	DSS-induced colitis model in eight- to ten-week-old female BALB/c mice	0.02 g/kg	Body weight, colon length ↑; IL-1β, IL-6, TNF-α ↓; improved colon histopathology	*Bifidobacterium*, *Ruminococcus* *Lactobacillus* ↑; *Helicobacter*, *Staphylococcus* ↓	AA, PA, BA ↑	ZO-1, occludin, goblet cells, MUC2 ↑	NF-κB, MAPK pathway ↓	[88]
Pg3gal	Derivatives of anthocyanins, molecular structure: Pelargonidin-3-O-sophoroside-6-C-glycoside	DSS-induced colitis model in male C57BL/6J mice	0.025 g/kg	Colon length ↑; DAI, IL-6, IL-1β, TNF-α, cellular apoptosis in intestinal epithelial cells, inflammatory cell infiltration ↓	*Escherichia* ↓; *Duncaniella*, *Akkermansia* and *Muribaculum* ↑; F/B ↑	SCFAs ↑	/	NLRP3 pathway ↓	[89]
p-BAC-PSPP	Total anthocyanin and protein contents were 16.46 ± 3.36 mgC3G/g and 62.50 ± 0.09%, respectively	LPS-induced inflammation model of RAW264.7 cells	0.0125, 0.0250, 0.0500, 0.1000 and 0.2000 g/kg	iNOS, TNF-α, NO, TNF-α, ROS accumulation ↓	/	/	/	Nrf2/HO-1 ↑; MAPKs, NF-κB and AP-1 signaling pathways ↓	[110]

SPLPAs	Isochlorogenic acid A, isochlorogenic acid B, isochlorogenic acid C, chlorogenic acid, cryptochlorogenic acid, neochlorogenic acid, caffeic acid	IL-1β-induced monolayer cell barrier damage model of Caco-2	0.0002 g/kg	NO, IL-6, TNF-α ↓	/	/	ZO-1, Occludin, Claudin-1 ↑	NF-κB pathway, MAPK pathway ↓	[104]
Tubers Extract	/	LPS-induced inflammation model of RAW264.7 cells	0.001, 0.010 and 0.100 g/kg	IL-6, Tnf, iNOS ↓	/	/	/	Nrf2 pathway ↑; NF-κB pathway ↓	[85]
Trifostigmanoside I	C24H38O12	IL-1β induced monolayer barrier loss model of LS174T and Caco-2 cells	/	/	/	/	MUC2, ZO-1, Occludin ↑	PKCα/β-ERK1/2 signaling pathway ↑	[91]

Note: ↑ Increase the measurement parameters; ↓ reduction of measurement parameters. PP, purple potato; LPS, lipopolysaccharides; PSPP, purple sweet potato polysaccharide; DSS, dextran sulfate sodium; WSP, water-soluble polysaccharide; DASP, dilute alkali-soluble polysaccharide; CASP, concentrated alkali-soluble polysaccharide; CTX, cyclophosphamide; F/B, Firmicutes/Bacteroidetes ratio; ASPP alkali-soluble polysaccharide; AA, acetic acid; PA, propionic acid; BA, butyric acid; PSPRS, purple sweet potato resistant starch; SRDF, sweet potato residue dietary fiber; YSPP, yellow sweet potato polysaccharide; p-BAC-PSP, protein-bound anthocyanin compounds from purple sweet potato; PSPAE, purple sweet potato anthocyanin extract; Pg3gal, pelargonidin-3-galactoside; SPLPAs, sweet potato leaf phenolic acids; MW: Molecular weight; DAI, disease activity index; Nrf2, nuclear factor erythroid 2-related factor 2; NF-κB, nuclear factor kappa-B; Glc, glucose; Man, mannose; Rha, rhamnose; Ara, arabinose; Gal, galactose; GalA, galacturonic acid; GlcA, glucuronic acid.

Table 3 Correlation between sweet potato active ingredients and key results

Active ingredient	Symptoms (e.g. body weight, colon length, DAI)	Inflammatory factor (e.g. TNF-α, IL-1β, IL-6, IL-10)	Gut microbiota (e.g. Firmicutes, Bacteroidetes)	SCFAs	Intestinal barrier (e.g. ZO-1, Occludin, Claudin-1)
Whloe purple potato	√	√	√	√	√
Polysaccharides	√	√	√	√	√
Polysaccharides-1	×	√	×	×	×
Yellow sweet potato polysaccharide	√	√	×	√	√
anthocyanin extract	√	√	√	√	√
P3gal	√	√	√	√	×
Protein-bound anthocyanin compounds	×	√	×	×	×
leaf phenolic acids	×	√	×	×	√
Tubers Extract	×	√	×	×	×
Trifostigmanoside I	×	×	×	×	√

6.1 Mechanisms of gut microbiota regulation by sweet potato

The human gut microbiota comprises trillions of microorganisms, most of which are bacteria and viruses and are considered non-pathogenic. Current estimates suggest that the number of gut bacteria reaches approximately 10^{14}, including at least 1000 different species and genera, with anaerobic microorganisms constituting the dominant populations and classifiable into six major phyla: Firmicutes, Bacteroidetes, Actinobacteria, Fusobacteria, Proteobacteria, and Verrucomicrobia [93]. The gut microbiota works synergistically with the host's defense and immune systems to prevent pathogen colonization and invasion; it also plays a role in mitigating various chronic diseases and adverse conditions, such as diabetes, cardiovascular disease, colitis, hepatitis, neuritis, fatigue, and aging. Furthermore, the gut microbiota performs essential metabolic functions: it is involved in the regulation of unsaturated fatty acid biosynthesis, fatty acid biosynthesis, steroid hormone biosynthesis, and metabolic pathways of purines, amino acids, and fatty acids, while facilitating the extraction of energy and nutrients from food, the production of SCFAs and polyphenol metabolites, and the absorption of amino acids [69, 81]. Alterations in the microbiota can result from exposure to various environmental factors, including diet, toxins, drugs, and pathogens. Microbial dysbiosis is particularly pronounced in colitis patients, characterized by reduced microbial diversity, an increase in pathogenic bacteria, and a significant decrease in beneficial bacteria—especially those that produce high levels of SCFAs. A Polish study [94] indicates that in samples from ulcerative colitis patients, the genera *Escherichia-Shigella*, *Peptostreptococcus*, *Bacillus*, *Veillonella* were significantly enriched, while beneficial bacteria such as *Akkermansia*, *Faecalibacterium* and *Bifidobacterium*, and butyrate-producing bacteria like *Faecalibacterium*, *Blautia*, and *Ruminococcus* were significantly depleted. Overall, beneficial bacteria—including *Bifidobacterium*, *Lactobacillus*, and *Enterococcus faecium*—can ferment sweet potato polysaccharides and metabolize sweet potato polyphenols, thereby stimulating the accumulation of SCFAs and the production of beneficial secondary metabolites. This plays a crucial role in promoting intestinal metabolism and preventing colitis. In contrast, an increased proportion of pathogenic bacteria exacerbated colitis, with microorganisms such as *Enterobacteriaceae*, *Streptococcus bovis*, *Staphylococcus*, *Helicobacter*, *E. coli* identified as factors contributing to colitis deterioration. Sweet potatoes and their active compounds contribute to maintaining gut microbiota balance, ultimately ameliorating colitis [95].

Several sweet potatoes varieties and their active compounds have been shown to increase gut microbiota diversity. For example, Zhu et al. found that dietary purple sweet potato [72], Sun, Chen, et al., Sun et al. and Gou et al. found that purple sweet potato polysaccharides [77, 79, 81], and Wang et al. found that purple sweet potato RS—all demonstrated the ability to reverse the decrease in the F/B ratio observed in DSS-treated groups, while also significantly reducing Proteobacteria abundance [82]. Proteobacteria are known to contain numerous human pathogens, such as *Brucella*, *Bordetella*, *Neisseria*, *Shigella*, *Salmonella*, *Yersinia*, *Escherichia*, *Rickettsia*, and *Helicobacter*. These pathogens have been shown to increase *Nos2* expression and nitrate production, further inducing inflammatory bowel disease [96]. A decreased F/B ratio is common in both

CD and UC patients. Firmicutes extract energy from food more efficiently than Bacteroidetes, thereby promoting the activation of energy metabolism pathways (e.g., carbohydrate and fatty acid metabolism) and the production of butyrate. Butyrate contributes to higher anti-inflammatory activity, better absorption of nutrients by intestinal epithelial cells, and enhanced colitis recovery. Firmicutes is associated with an increased risk of CD relapse and elevated inflammation levels when its proportion is reduced [97, 98]. Therefore, the regulation of Proteobacteria proportion and the F/B ratio by purple sweet potatoes and their active substances reduces the abundance of harmful bacteria, inhibits the production of pro-inflammatory substances (e.g., nitrate and *Nos2*), and effectively stimulates butyrate secretion and intestinal nutrient absorption—collectively promoting colitis recovery. In particular, purple sweet potatoes and their bioactive components demonstrate selective modulatory effects on gut microbiota composition, particularly enhancing beneficial bacteria (e.g., *Bifidobacterium*, *Lactobacillus*) while suppressing potentially harmful species (e.g., *Mucispirillum*, *Clostridium*). For instance, Zhu et al. found that dietary purple sweet potatoes upregulated *Bifidobacterium*, *Acetobacteroides*, *Acetatifactor*, and *Rikenella*, while downregulating *Mucispirillum*, *Clostridium*, *Bacteroides*, *Subdoligranulum*, *Romboutsia*, and *Parasutterella* [72]; purple sweet potato polysaccharide led to an increase in *Norank-f-Lachnospiraceae*, *Lactobacillus*, and *Roseburia*, and a decrease in *Bacteroides* and *Staphylococcus* [79]; purple sweet potato RS promoted the abundance of butyrate-producing bacteria such as *Alloprevotella* and *Bifidobacterium*, while inhibiting colitis-inducing strains such as *Alistipes*, *Desulfovibrio*, *Staphylococcus*, and *Jeotgalicoccus* [82]. Overall, although different bioactive components exhibit varying microbial modulation effects, they consistently improve the beneficial-to-pathogenic microbial ratio in the gut ecosystem.

However, some studies support that sweet potatoes and their active components reduce the F/B ratio. For example, purple sweet potato polysaccharides can restore the imbalanced ratio of higher Firmicutes (64.73%) and lower Bacteroidetes (26.97%) ratio in the DSS-induced colitis model to a more balanced range: Firmicutes (35.90–47.03%) and Bacteroidetes (45.10-58.30%) [75]. Sun, Gou, et al. found that purple sweet potato WSP could reverse the relatively high Firmicutes and relatively low Bacteroidetes level in the lipopolysaccharides (LPS)-stimulated group, restoring them to levels similar to those in the normal control group [78]. Sweet potato dietary fiber also reduced the F/B ratio, increased gut microbiota diversity, and improved the relative abundance of bacterial families including Lactobacillaceae, Peptostreptococcaceae, Streptococcaceae, Muribaculaceae, Akkermansiaceae, and Rikenellaceae. These changes contribute to enhenced metabolic function of the gut ecosystem and facilitating colitis recovery. In particular, a study by Chen et al. found that Pg3gal, one of the anthocyanin components isolated from purple sweet potato, can simultaneously and positively modulate the abundance of Firmicutes and Bacteroidetes [89]. Specifically, it significantly increased the proportion of *Lactobacillus*, while decreasing the proportion of *Deferribacteres*, *Akkermansia*, and *Escherichia*, thereby restoring gut microbiota balance in mice and leading to significant improvement in colitis. The reasons for the different regulatory effects of sweet potatoes and their active components on the F/B ratio and other bacteria found in different experiments need to be further explored.

The improvement of colitis by sweet potato powder, polysaccharides, RS, polyphenols, etc. is also reflected in their ability to stimulate the secretion of SCFAs. In the study of Wang et al., the use of sweet potato RS increased the levels of acetate, propionate, and butyrate from 2.32 ± 0.43, 0.27 ± 0.07, and 0.27 ± 0.12 mg/100 g to 3.96 ± 0.39, 0.57 ± 0.11, and 0.74 ± 0.13 mg/100 g, respectively[82]. Similarly, alkali-soluble purple sweet potato polysaccharide, water-soluble purple sweet potato polysaccharide, and yellow sweet potato polysaccharide have also demonstrated significant efficacy in stimulating the production of acetate, propionate, and butyrate. Gut microorganisms can degrade polysaccharides that cannot be digested by gastrointestinal fluids and further synthesize SCFAs. The anti-inflammatory effects of these SCFAs (0) are very complex and are mainly reflected in the following points: 1. SCFAs can promote the synthesis of ZO-1, claudin-1, and occludin and enhance the intestinal barrier; 2. SCFAs can provide energy to epithelial cells and maintain their integrity; 3. SCFAs have direct anti-inflammatory activity and can effectively scavenge inflammatory factors; 4. SCFAs can act on FOXP3 regulatory T cells, producing anti-inflammatory factors TGF-β and IL-10 and inactivating the NF-κB signaling pathway; 5. SCFAs can be recognized by specific SCFA receptors GPRs expressed on intestinal epithelial cells and innate immune cells, such as GPR41, GPR43, and GPR109A, thereby enhancing epithelial barrier function and further inhibiting NF-κB through a calcium-dependent mechanism; 6. SCFAs promote the differentiation of Tregs and reduce the expression of pro-inflammatory cytokines by inhibiting important transcriptional regulators HDACs. These changes ultimately enhance the expression of downstream effectors, including caspase-1, IL-18, and IL-1β, further inhibiting colitis. Feng, Du, and Wang found that feeding 100 mg/kg and 200 mg/kg of yellow sweet potato polysaccharide both led to increased levels of acetic acid, propionic acid, isobutyric acid, and butyric acid, which effectively upregulated GPR41 protein expression, further resulting in the activation of the downstream MEK-ERK pathway (increased expression of ERK1/2, Ras, Raf, and MEK1/2)[80]. These molecular events collectively contributed to the promotion of intestinal epithelial cell proliferation, inhibition of apoptosis, and amelioration of colitis. However, current research on the modulation of SCFAs and their downstream signaling pathways by sweet potatoes or their bioactive components remains limited, and many pathways have not yet been explored.

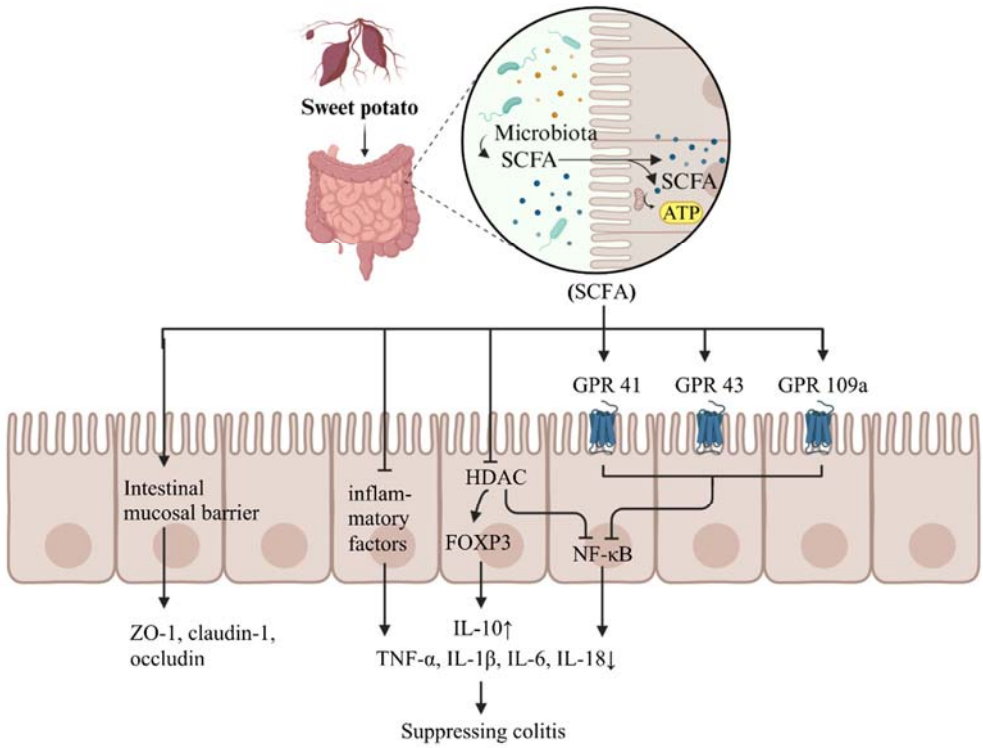


Fig.5 Schematic Diagram of Short-Chain Fatty Acids' Mechanism Against Colitis. (Created with BioRender.com)

Different types of sweet potato polysaccharides also lead to different SCFA regulation effects, which also means that their anti-colitis effects are different. Tang et al. reported that WSP had the best SCFA-promoting effect, with acetic acid, propionic acid, butyric acid, and valeric acid concentrations of 15.03%, 6.22%, 6.65%, and 2.35%, respectively, far higher than the 4.18%, 3.56%, 4.05%, and 0.92% in the colitis group [75]. DASP ranked second, with acetic acid, propionic acid, butyric acid, and valeric acid concentrations of 10.67%, 4.23%, 5.19%, and 1.03%, higher than the performance of purple sweet potato polysaccharide powder and CASP. Therefore, it is necessary to study the extraction methods, physicochemical properties, and structure of purple sweet potato polysaccharide in relation to its ability to promote SCFA secretion.

6.2 Sweet potato inhibits colitis by modulating the NF-κB pathway and its upstream pathway

NF-κB plays a crucial role in regulating cellular responses to diverse stimuli, including immune reactions, inflammation, cell proliferation, and apoptosis. When NF-κB is inactive, the NF-κB p50/p65 complex resides in the cytoplasm, and its nuclear translocation is inhibited by binding to the inhibitor of κB protein (IκB). Consequently, it generally remains in an inactive status under normal conditions. Upon cellular stimulation by factors such as ROS, LPS, and inflammatory factors, a signaling cascade is triggered, ultimately activating IKK. This leads to IκBα phosphorylation and its subsequent dissociation from NF-κB, which is primarily composed of p50 and p65 (RelA). The liberated NF-κB dimer then translocates to the nucleus, where it modulates the transcription of genes involved in immunity and inflammation, including those encoding IL-1β and components of the NLRP3 inflammasome [99-101]. The NF-κB pathway is crucial in both the development and mitigation of colitis. Studies have demonstrated that the NF-κB signaling pathway is activated in colitis patients, as evidenced by increased expression of NF-κB, IKK, and IκBα [102]. Studies

have shown that sweet potato components exert their anti-colitis effects by modulating the NF-κB pathway [85, 103-105].

Both sweet potato leaf and flesh polyphenols exhibit anti-colitis effects by inhibiting the NF-κB pathway [85, 90, 104]. Examples include sweet potato leaf polyphenol extracts rich in chlorogenic acid isomers, sweet potato tuber polyphenol extracts, and anthocyanin-binding proteins. Zhang et al. showed that sweet potato leaf polyphenols downregulated NF-κB-p65, Lamin B1, and IκB-α levels, which improved the inflammatory conditions and membrane permeability in an *in vitro* colitis model [104]. Similarly, *in vivo* colitis models showed that both p-BAC-PSP (anthocyanins and protein contents were 16.46 ± 3.36 mgC3G/g and 62.50 ± 0.09%, respectively) and FAC-PSP (anthocyanins content was 16.82 ± 1.49 mgC3G/g and no protein was detected) suppressed MAPK pathway activation (decreased ERK, p38, and JNK expression) [90]. This further led to reduced activation of downstream NF-κB and AP-1, thereby inhibiting the expression of pro-inflammatory mediators and cytokines (iNOS, NO, TNF-α). The differences in anti-colitis effects between FAC-PSP and p-BAC-PSP were not significant.

Sweet potato polysaccharides can also inhibit colitis by modulating the NF-κB pathway. Two types of purple sweet potato polysaccharides (one composed of an α-(1 → 4)-D-Glc backbone with 1 → 2, 1 → 3, and 1 → 6 glycosidic linkages forming branches, and another is a glucan with a molecular weight of 18.3 kDa) both demonstrated the ability to modulate the inflammatory response, but their regulatory effects differed [103, 105]. Specifically, the LPS-injured group exhibited increased expression of p-IκBα/IκBα/β, p-IκBβ/IκBα/β, p-IκBα/IκBα, and p-NF-κB/NF-κB, while treatment with the first type of purple sweet potato polysaccharide reversed these changes, revealing its protective effect against colitis through NF-κB transactivation. The second type of purple sweet potato polysaccharide activated IκB-α degradation and increased the phosphorylation of NF-κB, IKKα/β, and IκB-α, but the expression of IKKα/β and NF-κB-p65 slightly changed ($P > 0.05$). These results suggest that the glucan can exert immune-enhancing effects by activating the NF-κB pathway in RAW264.7 cells. The differential regulatory effects of purple sweet potato polysaccharides on the NF-κB pathway may be associated with their molecular weight and monosaccharide composition. However, limited research in this area has resulted in unclear relationships between the polysaccharides' structural characteristics (molecular weight, sugar composition, carbon backbone) and their NF-κB modulation efficacy and anti-colitis activity.

Except for direct modulation of the NF-κB signaling cascade, research suggests that sweet potato active components can also inhibit the upstream TLR4/MyD88 pathway, leading to a series of downstream inhibitory events [103, 105]. TLRs are type I transmembrane glycoproteins containing three functional domains, which are tightly associated with processes like innate immunity, inflammation, cell growth, and cancer. Once stimulated, the TIR cytoplasmic domain of TLR4 binds MyD88, thereby recruiting IRAK4. After that, activated IRAK4 dissociates from MyD88, binds to and activates TRAF6, ultimately forming Complex-1. Subsequently, Complex-1 separates from TLR4 to stimulate TAK-1 and TAB 1-3 accumulation and further forms Complex-2. TAK-1 then phosphorylates and activates the IKK complex, leading to IκB protein

phosphorylation, proteasomal degradation, and NF-κB nuclear translocation. Sweet potato polysaccharides can interfere with colitis progression by modulating the TLR4/MyD88/NF-κB pathway. Song et al. stated that purple sweet potato polysaccharide can block the TLR4/MyD88/NF-κB signaling cascade, as shown by decreased TLR2 and TLR4 expression and reduced levels of MyD88, p-IRAK4, IRAK4, TIRAP, IRAK1, p-IRAK1, TRAF6, TAK1, and p-TAK1 [105]. This indicated that sweet potato polysaccharide inhibited activation of the upstream TLR4/MyD88 signaling. As a result, NF-κB was inactivated and the purple sweet potato exhibited excellent anti-inflammatory effects. However, no studies have yet used inhibitors or activators to confirm whether the TLR4/MyD88/NF-κB pathway is the only signaling pathway by which sweet potato active substances exert their anti-colitis effects.

6.3 Sweet potato inhibits colitis by modulating the Nrf2 pathway

Nrf2 (NF-E2-related factor 2), a member of the Cap'n'collar (CNC) transcription factor family, contains 605 amino acids and is classified into seven highly conserved functional domains (Neh1-Neh7) [106]. Keap1, composed of 624 amino acids, is a cysteine-rich protein containing 27 Cys residues in humans. The N-terminal domain of Nrf2 influences its stability and ubiquitination through its negative regulator Keap1, while the Neh5 domain is responsible for its cytoplasmic localization [107]. Under normal conditions, Nrf2 binds to Keap1, which then drives Nrf2 ubiquitination and proteasomal degradation. Under oxidative stress conditions or upon exposure to Nrf2 activators, Nrf2 dissociates from Keap1 and translocates to the nucleus, heterodimerizes with small Maf proteins, and transactivates an ARE battery of genes, ultimately initiating the synthesis of antioxidant enzymes like SOD and GSH, and the transcription of HO-1. HO-1, an inducible isoform and rate-limiting enzyme, catalyzes the degradation of heme into carbon monoxide (CO) and free iron, and the degradation of biliverdin into bilirubin, thereby exerting free radical scavenging and cytoprotective effects [108]. If this pathway is inhibited, antioxidant enzyme levels might be reduced, and ROS cannot be cleared promptly. This triggers the activation of inflammatory pathways, such as NF-κB and NLRP3 signaling, ultimately leading to the release of IL-1β and IL-18 precursors into the extracellular space, further inducing inflammatory diseases [109].

Sweet potatoes and their active components are also known for their excellent anti-inflammatory activity through Nrf2 pathway modulation. Examples include yellow sweet potato polyphenols [85], purple sweet potato polysaccharides [103, 105], purple sweet potato protein-bound anthocyanins [110], and sweet potato leaf extract [104]. Notably, some sweet potatoes varieties or sweet potato active components have been shown to inhibit colitis by regulating the Nrf2 pathway. Matsumoto et al. demonstrated that polyphenol-rich light-yellow sweet potato extracts exhibited excellent DPPH radical and ROS scavenging activity and significantly reduced IL-6, TNFα, and iNOS levels [85]. This was linked to activation of the Nrf2 pathway and inhibition of the NF-κB pathway by yellow sweet potato polyphenols. ML385, an Nrf2 inhibitor, blocks Nrf2's ability to bind small Maf proteins and inhibits its activity as a transcription factor. Blocking Nrf2 signaling with ML385 reversed the yellow sweet potato polyphenol-induced decrease in IL-6, while the expression of TNF and iNOS showed no significant change. The expression levels of nuclear Nrf2, NF-κB, and cytoplasmic

HO-1 showed no significant differences between groups treated with or without yellow sweet potato polyphenols, suggesting that NF-κB activity was suppressed by Nrf2 pathway activation. This study highlights the importance of the Nrf2 pathway in anti-inflammatory mechanisms.

In addition to modulating MAPKs, NF-κB, and AP-1 signaling, Jiang et al. found that p-BAC-PSP and FAC-PSP effectively activated the Nrf2 pathway, as evidenced by increased Nrf2 and HO-1 mRNA levels [90]. This antioxidant potential may be because sweet potato anthocyanins can donate hydrogen atoms from hydroxyl groups to unstable and highly reactive free radicals, which can stabilize, neutralize, or transform the free radicals into non-reactive states. However, this study only explored the Nrf2 pathway modulation effects of p-BAC-PSP and FAC-PSP at the mRNA level and did not further verify their regulatory effects at the protein level. Furthermore, the regulatory mechanisms of many sweet potato active components on the Nrf2-NF-κB cascade are still unclear, and it is also unknown which type of sweet potato active component has the greatest efficacy in regulating the Nrf2 signaling pathway and alleviating colitis, requiring more in-depth research to promote clinical applications.

6.4 Sweet potato inhibits colitis by modulating the NLRP3 pathway

The NLRP3 inflammasome, composed of NOD-, LRR- and pyrin domain-containing protein 3, belongs to the NLR protein family and plays a critical role in mediating host immune responses to microbial infections and cellular damage [111]. As the core protein of the NLRP3 inflammasome, NLRP3 contains a central nucleotide-binding and oligomerization (NACHT) domain, which promotes self-oligomerization and possesses ATPase activity. ASC contains two transduction domains: a pyrin domain that can connect to upstream NLRP3 and a caspase recruitment domain (CARD) that can connect to downstream caspase-1 [112]. The C-terminal conserved LRRs domain can regulate NLRP3 activity and prevent inflammasome formation by inhibiting the interaction between NLRP3 protein and ASC. However, when danger signals such as PAMPs, DAMPs, exogenous invaders, or environmental stresses are present, the conformation of the NLRP3 protein transforms into a form that facilitates its binding to ASC. Subsequently, the CARD of ASC binds to the CARD on procaspase-1, promoting NLRP3 inflammasome self-assembly. This further triggers the proteolytic cleavage of dormant procaspase-1 into active caspase-1, which then converts the cytokine precursors pro-IL-1β and pro-IL-18 into mature and biologically active IL-1β and IL-18, respectively. Mature IL-1β is a pro-inflammatory mediator that leads to more severe inflammatory and immune cascades [111, 113]. Therefore, extracting sweet potato active substances that target the NLRP3 inflammasome is essential for alleviating or even curing colitis.

Sweet potato polysaccharides, polyphenols, and anthocyanins all exhibit good NLRP3 inflammasome regulatory effects, which have been confirmed in numerous studies. In one of them, LPS treatment increased the expression of NLRP3, ASC, and Caspase-1/pro-Caspase-1, while purple sweet potato polysaccharide treatment significantly reversed these changes, demonstrating that purple sweet potato polysaccharide can regulate the inflammatory environment by inhibiting inflammasome activation [105]. Activated NF-κB signaling can increase the transcription levels of NLRP3 inflammasome-related components and pro-IL-1β,

and purple sweet potato polysaccharide can also indirectly inhibit NLRP3 inflammasome activation by inhibiting NF-κB signaling, exhibiting a multi-pathway inflammation regulatory mechanism. Furthermore, Pg3gal also inhibited the expression of pyroptosis-related proteins, such as NLRP3, ASC, cleaved-Caspase-1, N-GSDMD, and IL-1β, reduced pro-inflammatory cytokine levels (decreased TNF-α and IL-6), and ultimately exerted anti-colitis effects (improved colon shortening, reduced DAI scores, and improved colon mucosal damage and inflammatory cell infiltration) [89]. However, although this study explored the dual regulatory mechanism of Pg3gal on the gut microbiota and pyroptosis signaling pathways, it did not explore the correlation between Pg3gal, gut microbiota, and pyroptosis signaling pathways. Besides, although there are some studies on sweet potato active components regulating the NLRP3 inflammasome to intervene in colitis, few studies have focused on the regulation of upstream signals of the NLRP3 inflammasome by sweet potato active components, such as Gal-3, miR-124-3p/RBP4, and JAK-STAT.

6.5 Sweet potato inhibits colitis by modulating other pathways

In addition to the mechanisms mentioned above, several other protein signals are involved in the regulation of colitis by sweet potato active components. MUC2 expression in the intestinal barrier is induced by activating PKCα/β and its downstream molecule ERK1/2. Trifostigmanoside I upregulated PKCα/β and ERK1/2 phosphorylation, which further led to mucin production, resulting in improved intestinal barrier function and better colitis recovery [91]. The MAPK/ERK signaling pathway involves ERK, JNK, and p38 MAPK molecules, and its dysregulation can lead to abnormal cellular behaviors, including enhanced cell proliferation, differentiation, migration, senescence, and apoptosis. This is related to many physiological functions in the human body, such as tumors, bone marrow stem cell differentiation, insulin regulation, and inflammation. Studies have shown that the MAPK pathway is highly related to ROS and the NLRP3 inflammasome, and targeted downregulation of p38 MAPK contributes to inhibited assembly and activation of the NLRP3 inflammasome, thereby exhibiting broad anti-inflammatory effects. Purple sweet potato polysaccharide inhibited the LPS-induced MEK1/2-ERK1/2, MEK3/6-P38, and MKK7-JNK cascades, reducing the relative expression of p-MEK1/2 and p-ERK/ERK, p-MEK3/6 and p-P38/P38, and p-MKK7 and p-JNK/JNK. Furthermore, AP-1 signaling, mainly containing of c-Fos and c-Jun, as a downstream signal of MAPK, has the effect of promoting the expression of inflammatory mediators and activating inflammatory storms [105]. Studies have found that LSP can induce an increase in the relative expression of p-c-Jun/c-Fos and p-c-Fos/c-Fos, while purple sweet potato polysaccharide counteracted the above phosphorylation process. Similarly, p-BAC-PSP and FAC-PSP can also inhibit MAPKs signaling and AP-1 translocation (decreased JNK and c-Jun expression), thereby inhibiting the expression of pro-inflammatory mediators and cytokines (TNF-α, NO), and ultimately alleviating colitis progression [90]. However, most current studies have not fully elucidated the regulatory mechanisms of various sweet potato active components on the upstream and downstream of MAPK signaling.

7. Clinical studies on the anti-colitis activity of sweet potato

Sweet potatoes provide essential micronutrients and a wide range of macronutrients, including polysaccharides, polyphenols, anthocyanins, vitamins, and minerals, all of which contribute to colon health. Given that sweet potatoes have shown impressive anti-inflammatory effects in animal studies and cell models, some researchers have explored the anti-colitis effects of sweet potato-based diets in clinical settings. One clinical trial involving three UC patients found that a whole food plant-based diet containing sweet potatoes improved colitis symptoms [114]. In one case, a 35-year-old female showed no inflammation in the sigmoid colon, along with healed ulcers, reduced scar tissue, and diminished redness extending up to 10 cm in the rectum. A second case, a 35-year-old male, experienced weight loss and complete resolution of colitis, while the third, a 41-year-old male, demonstrated relief from colitis, no longer requiring enemas, and a reduced dosage of mesalamine. These findings suggest that a whole food plant-based diet incorporating sweet potatoes has significant benefits for treating colitis. However, since whole food plant-based diets are highly complex and sweet potatoes constitute only a small component, the specific role of sweet potatoes in colitis remains unclear. Another study surveyed 65 patients (57% with CD, 43% with UC) and found that these patients consumed a large amount of inflammatory foods, such as beef (65%) and coffee (60%), but had lower intakes of anti-inflammatory foods, with only small amounts of garlic (75%), olive oil (54%), and sweet potatoes (23%) [115]. This research highlights the importance of developing healthy dietary habits among colitis patients. Overall, more clinical studies are needed to explore the benefits of incorporating sweet potatoes into the diets of colitis patients.

Current clinical trials primarily rely on case studies or questionnaire surveys, with limited research on human interventions and even fewer studies targeting single active components. Such trials are constrained by multiple factors, including the inability to establish placebo-controlled groups, cross-contamination between study groups, and the inclusion of patients receiving drug therapy. In human studies, dietary interventions typically involve complex dietary patterns rather than the intake of a single food component [116]. Consequently, subjects' diets usually include multiple foods and nutrients alongside sweet potatoes, making it difficult to determine whether sweet potatoes exert anti-colitis effects independently or through interactions with other dietary components. Furthermore, due to interindividual variations in food metabolism and the specificity of gut microbiota, there may be significant differences in the absorption and metabolism of active components from sweet potatoes among different individuals. This prevents us from accurately quantifying the amount of the truly effective substance in the consumed sweet potatoes, further complicating the research. Additionally, the aforementioned clinical studies investigated the anti-colitis effects of a whole-food plant-based diet containing sweet potatoes, but failed to identify the specific active components responsible for these effects. Although advanced techniques like high-performance liquid chromatography (HPLC) and mass spectrometry (MS) have been widely applied to identify sweet potato constituents, they suffer from drawbacks such as lengthy analysis times, high solvent consumption, and difficulties in isolating specific compounds [117]. Therefore, future efforts should focus on enhancing the isolation of specific active components from sweet potatoes to identify those with optimal anti-colitis activity in clinical settings,

followed by recruiting subjects to validate their anti-colitis efficacy. On the other hand, specific active components in sweet potatoes, such as trifostigmanoside I, not only exhibit unclear anti-colitis activity in animal studies but also exist at extremely low concentrations. Currently, there are no effective methods for their separation and purification, posing significant challenges to the clinical application of these unique active components.

8. Conclusion

The increasing number of colitis patients is placing a significant burden on global healthcare systems. Research has found that sweet potatoes and their components, such as polysaccharides, polyphenols, anthocyanins, and trifostigmanoside I, have therapeutic effects on colitis through mechanisms involving the gut microbiota, NF-κB, Nrf2, and NLRP3 pathways. These effects manifest as reduced colitis pathological damage, thickened intestinal barrier, balanced beneficial and harmful flora, and stimulated SCFA secretion. However, current research largely focuses on purple sweet potato anthocyanins and polysaccharides. Whether other important sweet potato active substances, such as sweet potato pectin, glucan, and phenolic acids, can also produce good anti-colitis effects remains unclear. In addition, the regulatory mechanisms of some sweet potato active components are not well understood. For example, the connections between signaling pathways such as Nrf2, NLRP3, and AMPK and sweet potato active components and anti-colitis effects have not been fully explored. Finally, the relationship between sweet potato polysaccharide properties and anti-colitis activity is also very important. Whether the molecular weight, monosaccharide composition, α-glycosidic bond to β-glycosidic bond ratio, etc., of sweet potato polysaccharides will affect anti-colitis activity is also unresolved. These limitations restrict the widespread application of sweet potatoes in the field of functional foods or nutritional supplements, and even more so, prevent the acquisition of sweet potato active components for clinical colitis treatment.

Currently, the anti-colitis activity of purple sweet potato polysaccharides and anthocyanins has been basically clarified. In the future, research can proceed along two avenues: first, further exploration of the synergistic mechanisms between polysaccharides and polyphenols in combating colitis, such as whether polysaccharides can promote microbial growth and metabolism, thereby accelerating the beneficial transformation of polyphenols in the gut; second, integrating genomics, metabolomics, and gut microbiome technologies to construct a network regulation model that links sweet potato bioactive components, protein metabolites, and anti-colitis effects. Regarding clinical trials, future studies should consider incorporating more randomized controlled trials to validate the clinical efficacy of purple sweet potato polysaccharides and anthocyanins. Sweet potato active components are safer than other colitis treatment drugs, such as mesalazine and sulfasalazine. Before clarifying the clinical effect, they can first be used as an auxiliary dietary therapy to intervene in colitis to accelerate the patient's recovery.

Conflict of Interest Statement

The authors declare that they have no conflicts of interest.

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