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Ulcerative Colitis and Herbal Medicine: Pathophysiological Mechanisms, Preclinical Evidence, Clinical Evidence, and Therapeutic Opportunities

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ABSTRACT

Ulcerative colitis (UC) is a chronic inflammatory disease of the colon that usually starts in the rectum and is caused by genetic vulnerability, poor epithelial barrier function, microbial dysbiosis, and dysregulated immunological responses. Current pharmacological treatments relieve symptoms but are commonly linked with adverse effects, incomplete remission, and relapse, driving growing interest in herbal therapy as a multi-targeted, safer therapeutic option.This review synthesizes preclinical and clinical evidence on plant species with anti-colitic potential, such as Vitis vinifera, Cocos nucifera, Arctium lappa, Pimpinella candolleana, Ziziphus jujuba, Juglans regia, Terminalia chebula, Cordia dichotoma, and others, focusing on their mechanisms of action and translational relevance. Most species had preclinical investigations, but only a handful had clinical data.These plants exert anti-colitic effects via regulating nuclear factor kappa B, cyclooxygenase-2, tumor necrosis factor-alpha, interleukin-6, interleukin-13, and oxidative stress pathways. Flavonoids, polyphenols, terpenoids, and polysaccharides are bioactive ingredients that repair antioxidant defenses, promote mucosal healing, and regulate the gut flora. Preliminary clinical data and commercial herbal preparations provide additional support for translational potential.Herbal interventions offer a holistic approach to UC by addressing inflammation, oxidative stress, and microbial imbalance; however, clinical validation, extract standardization, and safety testing remain essential before mainstream integration.

Introduction

Ulcerative colitis (UC) is a chronic and idiopathic inflammatory bowel disease (IBD) marked by superficial inflammation of the mucosa that extends from the rectum to the upper parts of the colon. The disease

typically has a relapsing and remitting pattern and presents with symptoms like abdominal pain, bloody diarrhea, and, in severe instances, systemic issues including anemia, weight loss, and fever[1].

Standard treatment approaches include 5-aminosalicylic acids (5-ASA), corticosteroids, immunomodulators, and biologic therapies, with colectomy available as a last-resort option. Despite progress in treatment options, challenges such as drug-related side effects, diminished response to medications, and recurring symptoms after stopping treatment continue to pose significant problems[2]

In recent years, there has been a significant increase in interest in complementary and alternative medicine (CAM), especially herbal medicine. According to the World Health Organization, approximately 80–88% of the world's population depends on plant-derived treatments for their primary health care needs. Herbal remedies are particularly popular in the context of ulcerative colitis because of their properties, such as antioxidant, anti-inflammatory, immunomodulatory, and microbiota-modulating effects, along with a lower toxicity compared to traditional medications.Previous reviews have typically concentrated on individual herbs or broad gastrointestinal issues. In contrast, this review compiles mechanistic, preclinical, clinical, and commercialization data on several herbal treatments specifically for ulcerative colitis. By combining insights from phytopharmacology with contemporary drug development perspectives, we intend to underscore the therapeutic potential, challenges, and opportunities for transitioning herbal remedies into the management of ulcerative colitis.

2. Search Strategy

This review systematically compiled and analyzed the literature on herbal therapies for ulcerative colitis therapy and prevention. A search of ScienceDirect, Web of Science, Scopus, PubMed, and Google Scholar (2020-2026) using keywords such as UC prophylaxis, phytochemistry, natural products, and traditional medicines yielded ~100 articles, of which 95 were shortlisted for relevance to herbal therapeutics in UC management.

3. Etiology

Ulcerative colitis has a multifaceted etiology that includes immunological, mitochondrial, microbial, environmental, genetic, and epithelial aspects. Over 200 IBD sites have been found using GWAS, including UC-specific variations such as HLA-DRB1*0103, ADCY7, HNF4A, and CDH1, which are connected to immune modulation and barrier integrity [3] Environmental-related factors include s6moking , antibiotic exposure, urban lifestyle, soft drink intake, and appendectomy[4] Mucosal inflammation is caused by gut microbiota dysbiosis, which is characterized by increased Enterobacteriaceae/Fusobacteriaceae and decreased Ruminococcaceae/Lachnospiraceae. CDH1 mutations have been associated to epithelial barrier abnormalities that enhance permeability and antigen uptake[5] Immune dysregulation promotes Th17 cells, resulting in cytokine production via the IL-23/IL-17 axis [6]. Furthermore, epithelial energy metabolism is impaired by mitochondrial dysfunction, which compromises barrier integrity and sustains inflammation[7].

4. Epidemiology

UC epidemiology has evolved significantly over the past 20 years: incidence has steadied or reduced in high-income countries, while it has grown sharply in low and middle income countries, with sustained increases in certain industrialized nations [8]. Most cases of this illness occur in people between the ages of 30 and 40[9]. UC incidence is highest [10] The greatest incidences of ulcerative colitis are found in Australia (17.4 per 100,000), Canada (19.2 per 100,000), and Northern Europe (24.3 per 100,000) [11][12].The highest

prevalence rates are seen in the United States (214 per 100,000), Canada (248 per 100,000), and Europe (505 per 100,000) [13] [14]. UC prevalence varies across Europe, with Western and Northern countries showing higher rates than Eastern Europe [15].

5. Pathophysiology of Ulcerative Colitis

5.1 Epithelial barrier

In UC, the epithelial barrier which is normally protected by a mucin layer, antimicrobial peptides, and immune cell isolation from microbes is impaired, resulting in decreased Mucin-2 synthesis and sulphation [16].Damage to the epithelial barrier increases permeability, most likely due to a disruption in tight junction control[17]. Barrier loss allows luminal antigens to enter, though whether this precedes UC or results from chronic inflammation is unclear; certain human β-defensins are upregulated in UC [18].

5.2 Commensal microflora

The intestinal immune system balances tolerance to commensals and food antigens with defence against pathogens[19].According to human research, intestinal inflammation development, intensity, and phenotype are all influenced by enteric microbiota[20]. UC arises from a disrupted balance between mucosal immunity and gut microflora, causing an abnormal immune response to harmless bacteria[21].

5.3 Antigen recognition

Antigens stimulate innate immunity through macrophages and dendritic cells that sample luminal microorganisms through epithelial dendrites [22]. In UC, increased, highly stimulatory dendritic cells in the lamina propria stimulate adaptive immunity, with their numbers corresponding with disease activity, implying a crucial role in beginning and maintaining inflammation [23].

Dendritic cells protect the epithelium and battle infection by using pattern-recognition receptors such as TLRs and NODs [24]. TLR polymorphisms influence immunological tolerance and infection susceptibility [25]. NF-κB regulates intestinal inflammation by promoting pro-inflammatory and cell-survival pathways in macrophages and T cells, while protecting epithelial cells [26] [27].

5.4 Dysregulation of immunological responses

NKT cells activate an unusual Th2 response in UC, which disrupts the regulatory/effector T-cell balance. They accumulate in the inflamed colon, releasing IL-4 and IL-13, which enhances NKT activity via positive feedback, causing tissue damage and driving disease pathogenesis [28]. Mutations that cause loss of function of the IL-10 receptor lead to severe UC [29].

5.5 Leucocyte recruitment

In ulcerative colitis, chemoattractants like CXCL8 recruit circulating leukocytes to the inflamed mucosa, amplifying inflammation[30]. Proinflammatory cytokines encourage leukocyte infiltration and maintain inflammation by increasing adhesion molecules such as MAdCAM-1 on mucosal endothelium[31].The pathogenesis is diagrammatically represented in Figure 1.

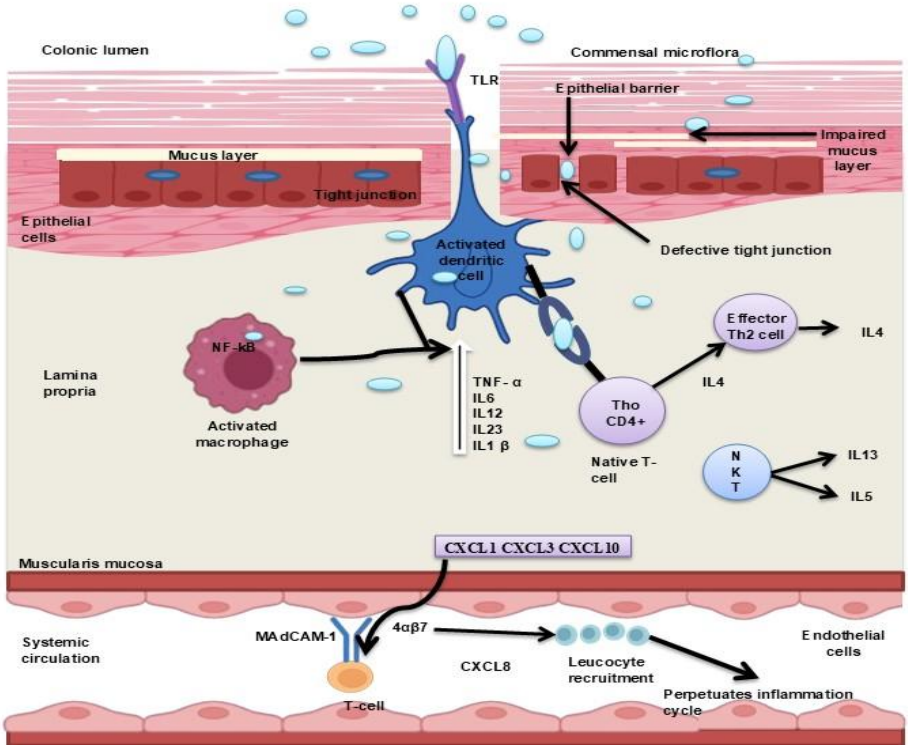

Figure 1: Schematic representation of the immunopathogenesis of ulcerative colitis showing epithelial barrier dysfunction, immune cell activation, and cytokine-mediated inflammation cycle

6. Herbal Remedies for Ulcerative Colitis

According to WHO, CAM use has continuously increased over the world, with approximately 88% of people using it to improve their health or treat/prevent acute and chronic illnesses [32]. Herbal medicine is the most commonly used complementary and alternative medicine modality for chronic illnesses such as colitis, and it is valued for its perceived safety and efficacy, which is equivalent to pharmaceutical drugs. Its benefits include anti-inflammatory (TNF-α, IL-1β, IL-6 regulation), antioxidant, and immunomodulatory qualities. It also has fewer adverse effects and can regulate gut flora and increase intestinal barrier integrity [33].

6.1 *Vitis vinifera*

Acetic acid-induced colitis in rats caused colonic inflammation, oedema, necrosis, and leukocyte infiltration, as well as increased MPO and MDA (oxidative markers) [34]. BGSE and BGSO dose-dependently reduced ulcer index and colitis severity, with best results at BGSE 200 mg/kg and BGSO 8 mL/kg; MPO fell in most groups, while MDA reduction was significant mainly at higher doses. Their phenolic compounds (proanthocyanidins, flavonoids, resveratrol, quercetin, catechin, etc.) provide antioxidant, anti-inflammatory, and antimicrobial effects by suppressing TNF-α, IL-6, IL-1, IL-8, NF-κB, COX-2, and iNOS [35][36]. Antimicrobial action may also aid in managing pathogenic microbes that aggravate ulcerative colitis [37]. Both extracts show similar bioavailability and colonic delivery potential [34].

6.2 *Cocos nucifera*

Various studies evaluated the protective effect of Virgin Coconut Oil (VCO) in DSS-induced ulcerative colitis in mice. VCO exhibited protective effects by reducing IL-[38][39], TNF-α[40][41] increasing IL-10, and improving Disease Activity Index (DAI) and Mouse Colitis Histology Index (MCHI) according to the other findings, VCO acts as a potential anti-inflammatory and antioxidant agent, playing a vital role in the management of ulcerative colitis[40].

6.3 *Arctium lappa*

Burdock fructo-oligosaccharide (BFO) shown strong protective benefits against DSS-induced UC in rats, increasing colon index and preserving intestinal integrity at high doses (500 mg/kg/day) [42]. It accelerated recovery, prevented weight loss, reduced DAI scores, and restored colon length [43][44] Protected barrier by increasing goblet cells and decreasing crypt damage [45][46] and lowered oxidative stress by suppressing MPO activity and ROS while reducing spleen enlargement [42]. BFO also downregulated IL-1β, IL-6, TNF-α, iNOS, COX-2, and MCP-1 while maintaining IL-10 [47]. Overall, BFO effectively reduced the severity of UC through barrier protection, oxidative stress reduction, immune modulation, and cytokine regulation, highlighting its potential as a therapeutic or functional food for colitis management[42].

6.4 *Pimpinella candolleana*

In acetic acid-induced UC rat model, Pimpinella candolleana (PC) extract improved body weight, DAI scores, colon/spleen/liver indices, and mucosal integrity, with histology revealing reduced inflammation [48]. UPLC-Q-TOF-MS identified 570 metabolites (flavonoids, coumarins, terpenoids) with anti-inflammatory/antioxidant activity [49],and PC regulated IL-2 and IL-6 in a dose-dependent manner [48]. Network pharmacology and docking revealed 96 UC-related targets, including quercetin, luteolin, and nobiletin substantially binding to NF-κB-related proteins (IKBKB, CHUK, JUN, and TNF-α) [50]. Experiments inhibiting the NF-κB pathway can alleviate colitis by downregulating these proteins [51]. Overall, PC demonstrated multi-component, multi-target, and multi-pathway anti-inflammatory effects, providing scientific evidence for its quality control and clinical application in UC[48].

6.5 *Ziziphus jujube*

The hydroalcoholic extract Ziziphus jujuba, given orally (1500, 3000 mg/kg) or via enema (20%, 40% gel), improved acetic acid-induced UC in rats by increasing SOD and GPx activity, decreasing IL-1, MDA, and MPO, and promoting dose-dependent histopathological healing, with enema outperforming oral administration [52]**.** This effect by polyphenols (gallic acid, catechins, caffeic acid, chlorogenic acid, cinnamic acid, coumarins), which reduce oxidative stress, inhibit neutrophil/mast cell migration, and suppress inflammatory mediators via inhibition of arachidonic acid pathway enzymes (phospholipase, COX) [53] [52]. Overall, the findings suggest *Z. jujuba* extract may serve as a promising therapeutic agent for UC, promoting mucosal healing while blocking oxidative and inflammatory pathway[52].

6.6 *Juglans regia*

Juglone alleviated DSS-induced UC in mice through various routes [54]. It pro-inflammatory cytokines (IL-1β, IL-6, IL-12, IL-23, TNF-α), but had uncertain effects on anti-inflammatory IL-10 and TGF-β. It also reduced oxidative stress by reducing Ucp2 and NF-κB (p-p65) and boosting Nrf2 signaling to restore antioxidant defenses [54][55]. JUG also altered gut microbiota by reducing pathogenic Escherichia coli and Clostridium sensu stricto 1 and increasing beneficial Lachnospiraceae_NK4A136_group, hence promoting SCFA-mediated intestinal health. JUG protects against colitis through inflammatory pathway modulation, antioxidant improvement, and gut microbiota balance, indicating its potential as a new UC therapy [54][56].

6.7 *Terminalia chebula*

Terminalia chebula (TCEA) was evaluated in DSS-induced UC mice, and its components gallic acid and ellagic acid inhibited LPS-induced NO, IL-6, and PGE-2 in RAW264.7 cells. TCEA reduced disease activity index, diarrhea, blood in stool, colon shortening, and histological damage while restoring goblet cell function and lowering oxidative stress [57]. It the TLR4/MyD88/NF-κB pathway, leading to reduced production of pro-inflammatory cytokines [58][59], and revealed that TCEA restored Bacteroidota levels, inhibited Proteobacteria proliferation, and promoted gut microbial diversity towards a healthier state [57].

6.8 *Cordia dichotoma*

Among Cordia dichotoma bark extract fractions evaluated in acetic acid-induced UC, the methanol fraction was most powerful, dramatically lowering pathological lesions, edema, neutrophil infiltration, and colonic MPO/MDA levels [60]. The treatment oxidative stress, and improved mucosal healing by inhibiting macrophage activation, TNF-α transcriptional regulation, and COX inhibition [61]. Overall, fraction restored redox balance and reduced inflammatory mediators, confirming C. dichotoma bark's traditional use and potential as an anti-UC agent [60].

Common models each simulate a different pathology: DSS causes epithelial destruction, barrier disruption, and innate immune activation (acute colitis); TNBS causes haptenization of colonic proteins, resulting in T-cell-mediated, transmural chronic inflammation; and acetic acid causes mucosal ulceration through direct epithelial damage, vascular permeability, and inflammatory infiltration.

Numerous other medicinal plants evaluated in DSS,TNBS and Acetic-acid models as summarized in Table 1 highlight the diverse pharmacological actions of phytoconstituents in modulating disease progression.

Table 1: Phytoconstituents and Mechanistic Insights of Medicinal Plants in Ulcerative Colitis Models

S.No.	Plant name	Major constituent	Model used	Mechanism of action (MOA)	Reference
1	Terminalia bellirica	Gallic acid, ellagic acid	DSS mice	Regulates JAK2/STAT3/IL-6 signaling and lowers oxidative stress	[62]
2	Polygonum cuspidatum	Polydatin	DSS & TNBS mice	Restricts Th17 differentiation and STAT3 phosphorylation.	[63]
3	Tetrastigma hemsleyanum	Orientin, vitexin	DSS mice	Repairs the intestinal barrier and triggers the Keap1/Nrf2 antioxidant pathway.	[64]
4	Pogostemon cablin	Patchouli alcohol	DSS mice	Supresses oxidative stress and intestinal inflammation.	[65]
5	Abelmoschus manihot	Hyperoside, flavonoids	DSS-induced colitis mice	Lowers oxidative stress and inflammatory mediators while protecting the intestinal barrier.	[66]
6	Anethum graveolens (Dill)	Anethole, flavonoids	DSS-induced colitis in Wistar rats	Improves colon histology, lowers oxidative stress and inflammatory mediators, preserves the intestinal barrier, and lowers the disease activity index and inflammatory cytokines.	[67]
7	Plantago major / Plantago spp.	Plantamajoside	DSS mice	Increases intestinal barrier proteins (ZO-1, occludin), modifies gut microbiota, and controls CBS signaling	[68]
8	Butea monosperma	Butrin, isobutrin	In-silico + UC	Targets inflammatory signaling pathways, AKT1, and MAPK1.	[69]

			pathway analysis		
9	Artemisia argyi	Eupatilin, jaceosidin	DSS mouse model	Intestinal inflammation is decreased by immunomodulatory and antioxidant activities.	[70]
10	Tetrastigma hemsleyanum	Flavonoids	DSS mice	Maintains the intestinal epithelial barrier and controls the gut flora.	[71]
11	Pogostemon cablin	Patchouli alcohol	DSS-induced colitis mice	Lowers oxidative damage and intestinal inflammation	[72]
12	Nelumbo nucifera	Quercetin, nuciferine	TNBS rats	Modulation of inflammatory signals using network pharmacology	[73]
13	Punica granatum	Ellagic acid	DSS mice	Inhibits AKR1B1/B3-mediated ROS generation, thereby suppressing NLRP3 inflammasome activation and reducing inflammation	[74]
14	Glycyrrhiza glabra	Glycyrrhiza polysaccharide	DSS mice	Reduces oxidative stress and promotes epithelium healing by suppressing NF-κB-mediated inflammation and altering gut microbiome.	[75]
15	Salvia miltiorrhiza	salvianolic acids + tanshinones	DSS mice	Reduces oxidative stress, inflammation, and intestinal epithelial protection by inhibiting TLR4/PI3K/AKT/mTOR signaling.	[76]
16	Artemisia capillaris	Scoparone	DSS mice	Lowers inflammatory cytokines and oxidative stress	[77]
17	Houttuynia cordata	Quercitrin	DSS mice	Improves the balance of microorganisms and the mucosal barrier	[78]
18	Ginkgo biloba	Bilobalide	DSS mice	Promotes tight-junction proteins and gut microbiota balance decreases pro-inflammatory cytokines, and suppresses AKT/NF-κB p65 and MAPK signaling.	[79]
19	Foeniculum vulgare	Anethole	Acetic acid - mice	Reduces inflammation and oxidative stress	[80]
20	Arnebia euchroma	Shikonin derivatives	DSS rats	Prevents the synthesis of inflammatory mediators	[81]
21	Acacia ferruginea	Pyruvate	DSS mice	Reduces pro-inflammatory cytokines, inhibits TNF-α/NF-κB	[82]

				signaling, and protects the integrity of the epithelial barrier by targeting cytosolic phospholipase A2 (cPLA2).	
22	Coptis chinensis	Berberine alkaloids	DSS mice	Enhances the microbiota and gut barrier	[83]
23	Gardenia jasminoides	Geniposide	TNBS rats	Increases antioxidant enzymes while lowering inflammatory mediators and oxidative stress.	[84]
24	Passiflora edulis	Polyphenols	TNBS mice	Enhances the balance of bacteria and antioxidant status	[85]
25	Centella asiatica	Asiaticoside, madecassoside	DSS-induced colitis mice	Enhances intestinal barrier integrity, reduces oxidative stress, and suppresses inflammatory cytokine production	[86]
26	Picralima nitida	akuammine, akuammidine	Acetic acid–induced colitis in rats	Protection of mucosal integrity, inhibition of inflammatory mediators, and antioxidant activity	[87]

Numerous clinical studies have assessed the effectiveness of different medicinal plants in treating ulcerative colitis (UC). Key clinical trials with plant-derived therapies are compiled in Table 2 with an emphasis on the trials' design, dose, patient population, and outcomes.

Table 2: Clinical trials on herbal plants

Plants	**Clinical Trial Design & Dose**	**Patient Population**	**Clinical Outcome**	**Reference**
Curcumin (Curcuma longa)	Double-blind, placebo-controlled, randomized research using mesalamine and bioenhanced curcumin	UC patients (n=69)	Improved clinical and endoscopic remission compared to placebo	[88]
Indigo naturalis	Real-world, multicenter, prospective study; median dose 1.0 g/day	UC patients (n=33)	73% clinical remission and 70% mucosal healing at one year	[89]
Coptis chinensis	Phase IV, randomized, open-label, 900 mg daily (300 mg TID) with 5-ASA	UC patients (n=238)	Improved Baron endoscopic scores and a significantly lower yearly recurrence rate	[90]
Ginger	Ginger supplementation (≈2 g/day) for 12 weeks in	UC patients (n=44)	Evaluating impacts on oxidative stress and inflammatory markers	

	a randomized, double-blind, placebo-controlled research		(TNF-α, hsCRP)	[91]
Boswellia serrata	Randomized experiment comparing sulfasalazine and gum resin at 900 mg/day for six weeks	Chronic colitis patients (n=30)	Remission in 82% of cases (Boswellia) compared to 75% of cases (sulfasalazine); histological and clinical	[92]
Andrographis paniculata	A double-blind, randomized, placebo-controlled study with a 1.8 g/day extract (HMPL-004) over eight weeks	Active UC patients (n=224)	Clinical response: 60% (AP) against 40% (placebo); mesalazine is not inferior.	[93]
Plantago ovata	Randomized, double-blind, placebo-controlled clinical trial; approximately 3.6 g of seed per day for 8 weeks	UC patients (n=61)	Clinical symptoms were better and disease activity was lower than with a placebo.	[94]

A number of commercial Ayurvedic and herbal formulations are available for the treatment of ulcerative colitis, in addition to individual plant-based clinical studies. Important products available in India are included in Table 3 along with their formulations, main herbal ingredients, and purported medicinal benefits.

Table 3: Marketed Ayurvedic formulations and proprietary herbal products for ulcerative colitis

Brand Name / Product	Company / Origin	Formulation Type	Ingredients	Evaluation
Ulcerative Colitis Care Pack	Planet Ayurveda (India)	Pack of 4 formulations (Capsules + Churna + Tablet)	Arjuna (*Terminalia arjuna*), Pitta Balance (*Praval pishti, Giloysatva, Mukta pishti* etc.), Kutajghan Vati (*Holarrhena antidysenterica*), VatsakadiChurna (*Kutaj, Bilva, Saunf*)	Clinical use in Ayurveda; preclinical, anecdotal patient reports
Hemostop-B Powder	Planet Ayurveda (India)	Herbal powder	Nagkesar, Arjuna bark, Lodhra	Used in UC to control bleeding; Ayurvedic case-based reports
Mimosa-Di Capsules	Planet Ayurveda (India)	Capsules	*Mimosa pudica* (Lajjalu), Kutaj extract	Promotes gut mucosa repair; empirical assessment
Ulcerative	DAD Ayurveda	Herbal Pack	Ulcer Care Tea (*Kutaj,*	Ayurvedic traditional

Colitis Treatment Pack	(India)	(Tea+ Powder)	*Saunf, Bilva*), Live-Care Powder (Liver-support herbs), Brain-Boost Tea	claim; not peer-reviewed
Double-Strength UC Pack	Always Ayurveda (India)	Herbal capsule pack	Coolstrin-B (*Arjuna, Nagkesar*), Motion Stop (*Bilva, Saunf*), Vasaka Capsules (*Adhatodavasica*)	Used to treat bleeding, irritation, and diarrhea (Ayurvedic reports)
UC Treatment Package	Deep Ayurveda (India)	Capsules+ Powder Pack	Kutajghan Vati, Nervocare, Curcumin, Herbal Powder	Ayurvedic practice; empirical claims
Divya Cologrit	Patanjali Ayurveda (India)	Herbal Tablets	*Aegle marmelos*, *Holarrhena antidysenterica*, cumin, fennel, rose, camphor	Marketed for UC; traditional claims
UC Care Range	Yukti Herbs (India)	Capsules assortment	Pitta Balance, Gut Heal, Bleed Savior, Kamdhudha Ras, others	Traditional Ayurvedic blend; proprietary formula

7. Challenges & Prospects

Although research on ulcerative colitis (UC) has progressed significantly, several challenges remain, including unclear etiology, absence of specific biomarkers, variable treatment efficacy, and adverse effects associated with conventional drugs. Herbal medicines are gaining popularity due to their favorable safety profile, multi-target mechanisms of action, and ability to regulate gut microbiota. However, their use is limited by inconsistent standardization, insufficient clinical evidence, and ambiguous regulatory frameworks. Additionally, identifying active compounds and understanding potential drug-herb interactions remain major hurdles. Future research should focus on precision drug delivery systems, microbiome-targeted approaches, integration of herbal medicine with nanotechnology, and the application of advanced tools such as artificial intelligence and network pharmacology. To fully harness the therapeutic potential of herbal treatments in UC, collaborative efforts across disciplines, well-designed multi-center clinical trials, and progress in genetics, stem cell therapies, and strategies to enhance mucosal healing are essential.

8. Conclusion

Multiple factors contribute to the development of ulcerative colitis, including immunological dysregulation, microbial imbalance, genetic predisposition, and epithelial barrier dysfunction. While conventional treatments remain the primary approach for controlling acute inflammation, their long-term use is often limited by side effects and incomplete remission. Traditional medicines, particularly herbal phytotherapeutics, offer a complementary strategy with multi-targeted actions—such as reducing oxidative stress, modulating NF-κB and TLR signalling pathways, regulating Th2/Th17 cytokine responses, and restoring gut microbiota balance. In experimental models of colitis, botanicals like *Vitis vinifera*, *Cocos nucifera*, *Arctium lappa*, *Pimpinella candolleana*, *Ziziphus jujube*, *Juglans regia*, *Terminalia chebula*,*Cordia dichotoma* and several other medicinal plants have demonstrated protective effects. These findings highlight the therapeutic potential of natural substances in promoting mucosal healing and attenuating inflammation in ulcerative colitis.

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