Int. J. Sci. R. Tech., 2025 2(11)
A Multidisciplinary peer-reviewed Journal
www.ijsrtjournal.com [ISSN: 2394-7063]

Original Article

Herbal Plants in the Management of Dysbiosis: A Review of Mechanisms, Efficacy, and Therapeutic Potential

Sohail Shaikh*, Pooja Rasal, Huzaifa Patel, Parth Khandelwal, Sanabil Shaikh, Saniya Shaikh

SND college of pharmacy, Babhulgaon, yeola -423401

ABSTRACT

Dysbiosis, an imbalance in the gut microbiota, is implicated in a wide array of diseases, including gastrointestinal disorders (e.g., IBD, IBS), metabolic conditions (e.g., obesity, type 2 diabetes), neurological issues (e.g., mood disorders, neurodegeneration), and immune-related ailments (e.g., autoimmune diseases, cancer). This review explores the mechanisms, efficacy, and therapeutic potential of herbal plants in managing dysbiosis, highlighting their advantages over conventional treatments like antibiotics, which often cause prolonged microbial perturbations. The gut microbiota, dominated by Firmicutes and Bacteroidetes, supports digestion, immune regulation, and pathogen defense. Dysbiosis arises from factors such as diet, stress, and medications, leading to reduced microbial diversity, pathogen overgrowth, and impaired barrier function. Herbal plants offer multimodal actions: prebiotic effects (e.g., polysaccharides promoting SCFAs), selective antimicrobial activity (e.g., berberine inhibiting pathogens while sparing commensals), gut barrier enhancement (e.g., curcumin upregulating tight-junction proteins), and immunomodulation (e.g., polyphenols reducing inflammation). Specific herbs like Panax ginseng, Allium sativum, Zingiber officinale, and others demonstrate anti-dysbiotic properties in preclinical and clinical studies, improving microbiota profiles and symptoms in conditions like IBS and colitis. Polyherbal formulations, such as Triphala (Ayurveda) and Gegen Qinlian Decoction (TCM), leverage synergies for enhanced efficacy, reduced resistance, and dose-sparing. However, challenges include safety risks (e.g., herb-drug interactions, toxicity), standardization issues, and regulatory inconsistencies. Current evidence from in vitro, animal, and early human trials shows promise, but gaps in large-scale RCTs and microbiome standardization persist. Future directions emphasize integration into functional foods, personalized therapies, and rigorous research for evidence-based use in gut health management.

Keywords: Dysbiosis, Gut microbiota, Herbal plants, Prebiotic effects, antimicrobial selectivity, Polyherbal formulations, Safety challenges, Therapeutic potential

INTRODUCTION

Over the past ten years, there has been a significant increase in the interest in human microbiota, particularly in gut microbiota. Even if there are many studies showing that changes in the microbiota makeup are linked to a variety of illnesses, the notion of a "healthy gut microbiota" is still ambiguous [1]. One of the largest interfaces (250–400 mm) between the host, environmental variables, and antigens in the human body is represented by the human gastrointestinal (GI) tract [2]. The term Gut Microbiota refers to all microbes that reside in the digestive system, including viruses, archaea, protists, fungi, and bacteria. Among the many advantages that the microbiota provides to the host are metabolism of nutrients and digestion, synthesis of vitamins, control of the immune system, defense against pathogens, maintenance of gut health and integrity, and metabolic functions. Cancer and chronic illnesses, weight and metabolism, brain and mental health, and systemic health [3]. The over 100 bacterial phyla that have been identified are the subject of this study. The adult human gut microbiota is primarily composed of the Bacteroidetes and Firmicutes phyla, with lesser amounts of Proteobacteria, Verrucomicrobia, Actinobacteria, Fusobacteria, and Cyanobacteria [1]. Dysbiosis is a sign of an unbalanced microbial ecosystem in which the "good" bacteria are unable to effectively control the "bad" ones, and a list of related illnesses. These illnesses are often complicated in terms of both pathogenesis and consequences, and the

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

intestinal microbiota increases daily. The Dysbiosis of Gut Microbiota (DOGMA) was recently discovered to be responsible for all three aspects of the syndrome, which includes hyper-androgenism (acne, hirsutism), anovulation/menstrual irregularity, and hyper-androgenism. the formation of several tiny cysts in the ovaries [4]. It is known that a change in the gut microbiota away from a healthy or normal state (i.e., eubiosis) causes disruption. Dysbiosis [5] is a term for it. Its clinical significance is due to its connection to a wide variety of illnesses and its impact on the course of health and disease. The human microbiome, sometimes known as a "virtual organ," is essential to preserving general health by assisting with digestion, vitamin production, and modulation. Dysbiosis is caused by a variety of factors, including genetic abnormalities, stress, diet, alcohol intake, and infection [3]. The immune system is responsible for defending the body against pathogens. and drugs, among other things [6]. The effects of antimicrobials on dysbiosis have been discussed in a number of studies, and the length of time that microbiota disturbances last seems to vary between antimicrobials for, Some medications (e.g., tetracyclines, macrolides, and sulfonamides) have a shorter disturbance duration [7], while others last for months (e.g., cephalosporins) or years (e.g., fluoroquinolones, clindamycin). Dysbiosis upsets the equilibrium between beneficial and harmful gut bacteria, which can result in neurological, metabolic, and gastrointestinal problems. The direct effects of dysbiosis are on gut health because the microbiota is crucial for digestion, barrier integrity, and immune regulation [8]. Dysbiosis has a direct impact on gut health because the microbiota is essential for these processes. for immunity regulation, barrier integrity, and digestion. The gut microbiota communicates with the brain via neural, immune, and metabolic pathways [9]. The usual course of action for treating dysbiosis is to use conventional methods. although it includes antibiotics, probiotics, and dietary changes, these are severely constrained in comparison to the possible applications of medicinal herbs [10]. Medicinal herbs have demonstrated maybe because of its potential to change the gut flora by encouraging beneficial bacteria (a prebiotic effect). Giving phytochemicals and polyphenols that block pathogens without killing beneficial microbes. Anti-inflammatory and boosting the gut barrier function and providing antioxidant effects. Examples:

1) Berberine (from Berberis spp.) - has gut-modulating and antibacterial effects.
2) Curcumin (from turmeric) – promotes microbiome diversity and has anti-inflammatory properties.
3) Polyphenols in green tea encourage the growth of Bifidobacterium and Lactobacillus [11].

2. Gut Microbiota and Dysbiosis

The gut microbiota, also known as the gut flora or gut microbiome, is a complicated and ever-changing community made up of trillions of microorganisms. that are mainly found in the gastrointestinal (GI) system. Viruses (which make up more than 99% of the microbes), archaea, fungi, bacteria (which predominate), and viruses are all members of this community. protozoa, and bacteriophages) are examples of the microbiota. Its disturbance (dysbiosis) is associated with illnesses such as, and it interacts symbiotically with the host, affecting overall health. Inflammatory bowel illness, diabetes, obesity, and even neurological disorders. The microbiota is formed at birth and changes with time, becoming adult-like by the age of around Age 2–3. It has a high degree of functional redundancy, which means that various microbial communities can carry out similar functions, but individual profiles are unique and frequently vary more between individuals. than over time in a single individual [12, 13] Gut microbiota: The gut has the most diverse and concentrated microbiota, which is made up of bacteria that are mostly from the Firmicutes, Bacteroidetes, actinobacteria, and proteobacteria phyla. For immunological control, metabolism, and digestion, this society is crucial. Age, health, and diet all have an impact on the composition, which varies significantly from one individual to the next (Table no. 1 provides all the information).

Dysbiosis

Significant human illnesses, such as inflammatory bowel disease, obesity, allergies, and autoimmune and auto inflammatory diseases, have been associated with dysbiosis. All three elements of the syndrome of hyper-androgenism (acne, hirsutism), anovulation/ menstrual irregularity, and the Dysbiosis of Gut

Microbiota (DOGMA) have been shown to be caused by it. the formation of several little cysts on the ovaries [4]. Lifestyle also appears to have a significant impact, and even patients with type 2 diabetes showed a moderate amount of gut microbial dysbiosis. The diet in Western nations, which is characterized by increased intake of red meat, animal fat, and excessive sugar, is a contributing factor to the chronic illnesses that affect over 50% of the adult population. Furthermore, foods low in fiber might be crucial in influencing the microbiota of the human gut. Additionally, studies have indicated that the Western diet causes dysbiosis and promotes endotoxemia, probably as a result of intestinal permeability and barrier function degradation [14, 15].

Dysbiosis mechanism

The intricate link between gut microbiota dysbiosis and the onset of cardiovascular illnesses (CVDs) is demonstrated by the dysbiosis mechanism. Negative effects of dysbiosis include elevated vascular inflammation, gut barrier dysfunction, and systemic inflammation brought about by decreased microbial diversity and an increase in pro-inflammatory bacteria. Changes in important microbial metabolites, such as increased trimethylamine-N-oxide (TMAO) and decreased short-chain fatty acids (SCFAs), which are both heavily linked to cardiovascular pathophysiology, mediate these conditions. Through processes like endothelial malfunction, plaque development, and disease progression, such imbalances increase the risk of cardiovascular diseases, which are manifested as heart failure, hypertension, myocardial infarction, and atherosclerosis**.** Importantly, the therapeutic potential of natural compounds, including flavonoids, omega-3 fatty acids, resveratrol, curcumin, and marine-derived bioactive, which can help in targeting gut dysbiosis by restoring microbiota balance and enhancing therapeutic efficacy. Thus, modulating gut microbiota through natural bioactive emerges as a promising strategy for reducing cardiovascular disease burden [16].

Table 1. The major bacterial phyla, their approximate proportions in healthy adults, key genera, and example species

Phylum (Alternative Name)	Approximate Proportion	Key General	Example Species	Notes
1) Firmicutes (Bacillota)	40-60%	Clostridium, Faecalibacterium, Ruminococcus, Eubacterium, Lactobacillus, Pepto coccus, Pepto streptococcus	Faecalibacterium parasitize (most common in adults), Ruminococcus bromic (resistant starch degrader), Clostridium sporogeneses (tryptophan metabolizer), Lactobacillus plantarum	Dominant in fermentation; increases in elderly; anti-inflammatory roles.
2) Bacteroidetes (Bacteroidota)	30-50%	Bacteroides, Prevotella	Bacteroides thetaiotaomicron (polysaccharide degrader), Bacteroides fragilis	Major in colon; aids fiber digestion; higher in high-fiber diets (e.g., rural African populations).
3) Actinobacteria (Actinomycetal)	5-10%	Bifidobacterium	Bifidobacterium adolescent's, Bifidobacterium longum	Vitamin producers (e.g., folate, B12); higher in breast-fed infants.
4) Actinobacteria (Actinomycetal)	1-5%	Escherichia	Escherichia coli	Includes some facultative anaerobes; can increase in dysbiosis.
5) Verrucomicrobia	<1%	Ackerman Nia	Akarnania municipia	Mucin degrader; linked to metabolic health and reduced inflammation.

Types of Dysbiosis

Dysbiosis refers to an imbalance in the microbial communities (microbiome) within the body, most commonly in the gut but also in other areas like the skin, mouth, or vagina.

1. Loss of Beneficial Microorganisms (Type 1 Dysbiosis)

This involves a reduction or depletion of keystone microbial species that play protective, anti-inflammatory, or supportive roles in maintaining ecosystem balance. Beneficial bacteria, such as certain strains of *Bifidobacterium* or *Lactobacillus*, help ferment dietary fibers into short-chain fatty acids (SCFAs) like butyrate, which nourish gut cells and regulate immune responses. This can lead to impaired barrier function (e.g., "leaky gut") and increased susceptibility to infections or autoimmune conditions [17].

2. Expansion of Potentially Harmful Microorganisms (Type 2 Dysbiosis)

there is an overgrowth or dominance of pathobionts—microbes that are normally present in low numbers but become harmful when they expand excessively. Examples include opportunistic pathogens like Clostridium difficile, Escherichia coli variants, or fungi such as Candida [18].

3. Loss of Microbial Diversity (Type 3 Dysbiosis)

This category describes a general reduction in the overall richness and evenness of the microbiome, leading to a less resilient ecosystem. Healthy microbiomes typically feature high alpha-diversity (variety within a single site), which provides functional redundancy and stability against perturbations. Causes include long-term antibiotic exposure, high-fat/low-fiber diets, or aging. loss of beneficial microbes may pave the way for pathobiont expansion. Dysbiosis can also be site-specific, such as oral dysbiosis (linked to periodontal disease) or vaginal dysbiosis (associated with bacterial vaginosis [19].

- **Impact on Human Health:**

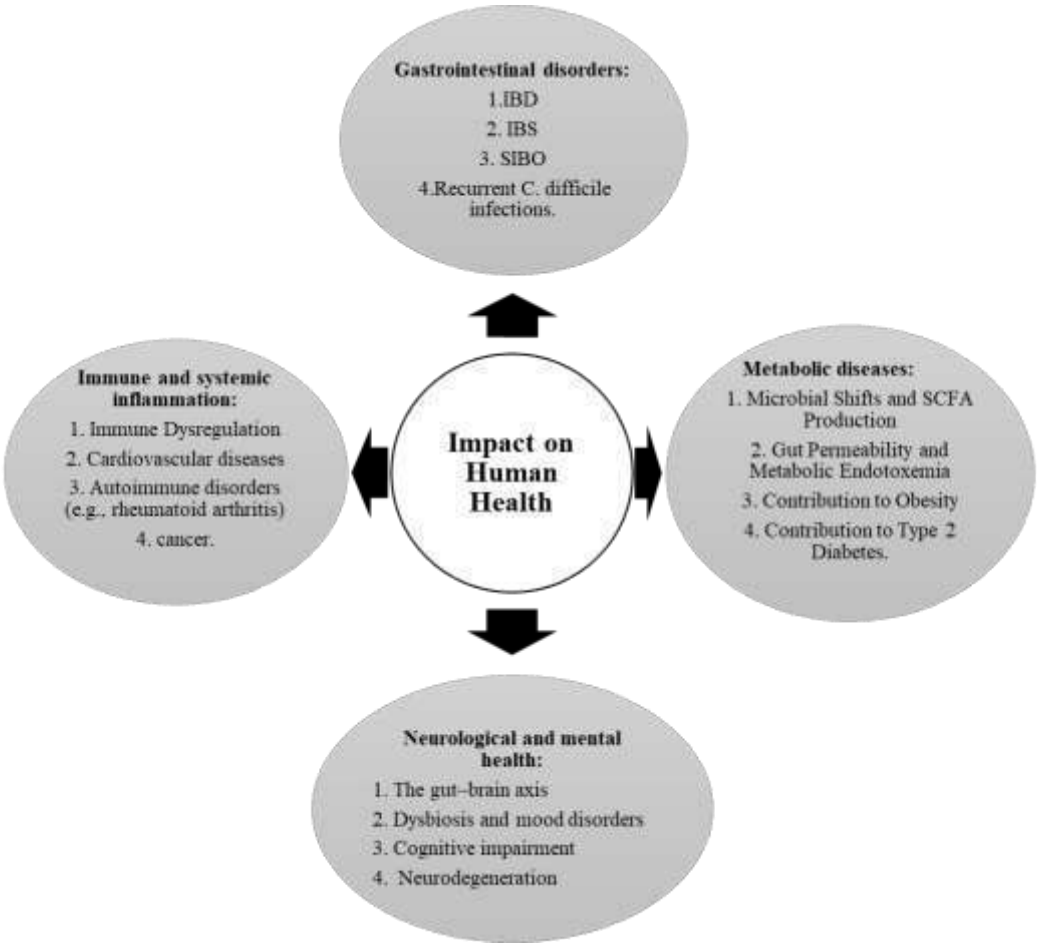

Figure 1: Impact on Human Health

➢ **Gastrointestinal disorders:** dysbiosis is implicated in IBD, IBS, SIBO, and recurrent C. difficile infections [20].

- **Inflammatory Bowel Disease (IBD)**

IBD, encompassing Crohn's disease and ulcerative colitis, is characterized by chronic inflammation of the gastrointestinal tract. Dysbiosis in IBD is marked by a reduction in microbial diversity and shifts in the composition of the gut microbiota. Specifically:

✓ Reduced Beneficial Bacteria.
✓ Increased Pathogenic Bacteria.
✓ Functional Changes [20].

- **Irritable Bowel Syndrome (IBS)**

IBS is a functional gastrointestinal disorder characterized by abdominal pain, bloating, and altered bowel habits. Dysbiosis in IBS is less severe than in IBD but still significant:

- Microbial Imbalance.
- Symptom Correlation.
- Post-Infectious IBS [20].

- **Small Intestinal Bacterial Overgrowth (SIBO)**

SIBO involves excessive bacterial growth in the small intestine, leading to symptoms like bloating, diarrhea, and malabsorption. Dysbiosis in SIBO is characterized by:

✓ Overgrowth of Specific Bacteria.
✓ Altered Microbial Location.
✓ Immune and Structural Effects [20].

- **Recurrent *Clostridium difficile* Infections**

Recurrent *C. difficile* infections (CDI) occur when the gut microbiota fails to resist colonization by *C. difficile*, a gram-positive, spore-forming bacterium. Dysbiosis is a key risk factor:

✓ Loss of Colonization Resistance.
✓ Metabolic Disruption.
✓ Recurrence Mechanism [20].

➢ **Metabolic diseases:** microbial shifts affecting SCFA production and gut permeability drive endotoxemia and insulin resistance—contributing to obesity, metabolic syndrome, and type 2 diabetes [21].

- **Microbial Shifts and SCFA Production**

The gut microbiota produces SCFAs (e.g., acetate, propionate, butyrate) through the fermentation of dietary fibers. These metabolites play critical roles in energy metabolism, gut barrier integrity, and immune regulation. The study by Cani et al. (2007) provides insights into how microbial dysbiosis disrupts SCFA production:

✓ Reduced SCFA-Producing Bacteria.
✓ Impact on Metabolism. [21].

- **Gut Permeability and Metabolic Endotoxemia**

Metabolic endotoxemia refers to elevated levels of LPS, a component of gram-negative bacterial cell walls, in the blood. Cani et al. (2007) demonstrate that dysbiosis increases gut permeability, driving endotoxemia:

✓ Mechanism.
✓ Consequences.
✓ Experimental Evidence. [21].

- **Contribution to Obesity**

Obesity is closely linked to microbial shifts and endotoxemia:

- **Adipose Tissue Inflammation**: LPS-driven inflammation increases macrophage infiltration into adipose tissue, promoting the release of pro-inflammatory cytokines (e.g., TNF-α, IL-6). This impairs adipocyte function and promotes fat storage.

- **Energy Harvest**: Dysbiotic microbiota may enhance energy extraction from food, increasing caloric availability and contributing to weight gain. Cani et al. noted that high-fat diet-fed mice had altered microbial profiles that correlated with increased fat mass.

- **Animal Models**: The study used mice fed a high-fat diet to show that microbial changes preceded obesity, suggesting a causal role. Antibiotic treatment or prebiotics (e.g., oligofructose) that

restored beneficial bacteria reduced endotoxemia and fat gain. [21].

- **Contribution to Metabolic Syndrome**

Metabolic syndrome, characterized by obesity, insulin resistance, dyslipidemia, and hypertension, is exacerbated by microbial dysbiosis:

- ✓ Inflammation and Lipid Metabolism.
- ✓ Gut-Liver Axis.
- ✓ Therapeutic Potential. [21].

- **Contribution to Type 2 Diabetes**

Type 2 diabetes (T2D) is driven by insulin resistance, which is closely tied to microbial dysbiosis:

- ✓ **Insulin Resistance**: LPS-induced inflammation impairs insulin signaling by activating pathways like JNK and IKKβ, which inhibit insulin receptor substrates. Cani et al. found that mice with metabolic endotoxemia exhibited fasting hyperglycemia and reduced insulin sensitivity.

- ✓ **Glucose Homeostasis**: Reduced SCFA production impairs GLP-1 (glucagon-like peptide-1) secretion, a hormone that enhances insulin release and glucose tolerance. Dysbiosis thus disrupts glucose homeostasis.

- ✓ **Human Relevance**: While the study primarily used mouse models, it suggests that similar mechanisms operate in humans, as high-fat diets are associated with microbial shifts and elevated LPS in T2D patients. [21].

- ➢ **Neurological and mental health:** via the gut–brain axis, dysbiosis has been linked to mood disorders, cognitive impairment, and neurodegeneration [22].

- **The Gut–Brain Axis: An Overview**

The gut–brain axis is a bidirectional communication network between the gut microbiota and the central nervous system (CNS), involving neural, immune, and endocrine pathways. Dysbiosis, an imbalance in gut microbial composition, disrupts this communication, contributing to neurological and psychiatric disorders. Cryan et al. (2019) describe the key mechanisms:

- ✓ Neural Pathways**.**
- ✓ Immune Modulation**.**
- ✓ Endocrine Signals**.**
- ✓ Metabolite Production. [22].

- **Dysbiosis and Mood Disorders**

Dysbiosis is strongly implicated in mood disorders such as depression and anxiety:

- ✓ **Microbial Shifts**: Reduced microbial diversity and altered abundance of specific taxa (e.g., decreased *Bifidobacterium* and *Lactobacillus*, increased *Clostridium* species) are observed in patients with depression and anxiety. These shifts correlate with symptom severity.

- ✓ **Mechanisms**:

- ○ **SCFA Reduction**: Lower SCFA production (e.g., butyrate) impairs anti-inflammatory pathways and reduces neuroprotection, contributing to depressive symptoms.

- ○ **Neurotransmitter Dysregulation**: Dysbiosis affects the production of serotonin (95% of which is produced in the gut) and GABA, altering mood regulation.

- ○ **HPA Axis Dysregulation**: Dysbiosis increases HPA axis activity, elevating cortisol levels and exacerbating stress-related disorders. [22].

- **Dysbiosis and Cognitive Impairment**

Cognitive impairment, including deficits in memory and executive function, is linked to dysbiosis:

- ✓ **Microbial Changes**: Reduced diversity and shifts in microbial composition (e.g., lower *Akkermansia* and *Bacteroides*) are associated with cognitive decline in conditions like mild cognitive impairment (MCI).

- ✓ **Mechanisms**:

- ○ **Neuroinflammation**: Dysbiosis increases gut permeability, allowing lipopolysaccharides (LPS) and pro-inflammatory cytokines to enter the

bloodstream, triggering neuroinflammation that impairs cognitive function.

- **Amyloid Production**: Gut microbes may influence amyloid-beta production, a hallmark of Alzheimer's-like pathology, via inflammatory pathways.

- **Metabolite Effects**: Reduced SCFA levels impair synaptic plasticity and hippocampal function, critical for learning and memory. [22].

- **Dysbiosis and Neurodegeneration**

Neurodegenerative disorders, such as Alzheimer's disease (AD) and Parkinson's disease (PD), are increasingly associated with gut dysbiosis:

✓ **Microbial Changes**: AD patients show reduced *Bifidobacterium* and increased *Bacteroides*, while PD patients exhibit lower *Prevotella* and higher *Enterobacteriaceae*, correlating with disease severity.

✓ **Mechanisms**:

- **Neuroinflammation**: Dysbiosis promotes systemic inflammation, which exacerbates neuroinflammation and accelerates neurodegeneration. For example, LPS from gram-negative bacteria activates microglia, contributing to neuronal damage.

- **Protein Aggregation**: In PD, dysbiosis may enhance alpha-synuclein misfolding via gut inflammation, with misfolded proteins propagating to the brain via the vagus nerve.

- **Metabolic Disruption**: Altered microbial metabolites (e.g., reduced SCFAs) impair energy metabolism in neurons, contributing to neurodegeneration. [22].

➢ **Immune and systemic inflammation:** chronic imbalance contributes to cardiovascular diseases, autoimmune disorders (e.g., rheumatoid arthritis), and cancer via pro-inflammatory signaling and altered metabolite profiles [23].

- **Dysbiosis and Immune Dysregulation**

Dysbiosis, defined as an imbalance in the gut microbial community, disrupts the delicate balance between the microbiota and the host immune system. Levy et al. (2017) describe how dysbiosis leads to chronic immune activation and systemic inflammation:

✓ Immune Homeostasis Disruption.
✓ Pro-Inflammatory Signaling**.**
✓ Altered Metabolite Profiles**.** [23].

- **Dysbiosis and Cardiovascular Diseases**

Chronic dysbiosis contributes to cardiovascular diseases (CVDs) such as atherosclerosis and hypertension through systemic inflammation and metabolic changes:

✓ **Mechanisms**:

- **Trimethylamine N-Oxide (TMAO)**: Dysbiosis increases the production of TMA by gut microbes, which is converted to TMAO in the liver. TMAO promotes atherosclerosis by enhancing foam cell formation and platelet activation, increasing the risk of thrombosis.

- **Systemic Inflammation**: Elevated LPS levels from dysbiotic microbiota induce endothelial dysfunction and vascular inflammation, key drivers of atherosclerosis.

- **SCFA Reduction**: Decreased SCFA production impairs blood pressure regulation and vascular health, as SCFAs normally promote vasodilation via G-protein-coupled receptors (GPR41, GPR43).

- **Dysbiosis and Autoimmune Disorders (e.g., Rheumatoid Arthritis)**

Dysbiosis is implicated in autoimmune disorders like rheumatoid arthritis (RA) by promoting immune dysregulation and inflammation:

✓ **Mechanisms**:

- **Th17/Treg Imbalance**: Dysbiosis increases Th17 cells, which produce pro-inflammatory cytokines (e.g., IL-17), while reducing Tregs, leading to loss of immune tolerance. This

imbalance drives autoantibody production and joint inflammation in RA.

- **Molecular Mimicry**: Certain dysbiotic bacteria (e.g., *Prevotella copri*) produce peptides that mimic host antigens, triggering autoimmune responses.

- **Gut Barrier Dysfunction**: Increased gut permeability due to dysbiosis allows microbial components to enter the bloodstream, activating systemic immune responses that target joints.

- **Dysbiosis and Cancer**

Dysbiosis contributes to cancer development, particularly colorectal cancer (CRC) and other inflammation-associated cancers, through pro-inflammatory signaling and altered metabolites:

✓ **Mechanisms**:

- **Chronic Inflammation**: Dysbiosis-induced inflammation, driven by LPS and pro-inflammatory cytokines, creates a tumor-promoting microenvironment. For example, elevated IL-6 and TNF-α levels enhance cancer cell proliferation and survival.

- **Genotoxin Production**: Certain dysbiotic bacteria (e.g., *Fusobacterium nucleatum*, *Escherichia coli*) produce genotoxins that damage DNA, increasing the risk of CRC.

- **Metabolite Dysregulation**: Reduced SCFA production impairs anti-tumor immunity, as SCFAs normally enhance immune surveillance by promoting Treg and cytotoxic T-cell activity. Conversely, dysbiosis increases secondary bile acids, which are carcinogenic.

3. Role of Herbal Plants in Gut Health

- **General properties of medicinal herbs relevant to the gut**

Many herbal plants contain polyphenols, alkaloids, terpenoids, and polysaccharides that act through complementary modes:

(i) prebiotic-like fermentation by commensals to yield beneficial metabolites (e.g., SCFAs).
(ii) selective antimicrobial and quorum-modulating actions against pathobionts.
(iii) barrier-protective effects via tight-junction support and mucus enhancement.
(iv) immunomodulation that reduces pro-inflammatory signaling and promotes regulatory pathways. Recent reviews synthesize these multimodal, "ecological" actions of herbal medicines on the microbiome and host physiology. [24,25,26].

- **Mechanisms by which herbs modulate the gut microbiota**

1) Prebiotic effects

Many botanicals supply non-digestible polysaccharides (e.g., inulin-type fructans from chicory/garlic, ginseng and mushroom polysaccharides) that enrich SCFA-producing taxa and influence immune and metabolic outputs. Systematic and experimental work shows inulin and related fibers increase butyrate and improve elements of barrier and metabolic function, though responses can be individual-specific. Plant and mushroom polysaccharides similarly shape communities and immune readouts via SCFAs[27]. Polyphenols—poorly absorbed in the small intestine—reach the colon where microbes catabolize them; resulting phenolic metabolites and shifts in taxa (e.g., Akkermansia, bifidobacteria) underlie their prebiotic-like activity. Reviews describe polyphenols as "duplibiotics," combining prebiotic promotion of commensals with direct suppression of pathobionts [25].

2) Antimicrobial selectivity (sparing commensals)

Botanical compounds (e.g., berberine, catechins, essential-oil phenolics) can inhibit bile-acid–modifying or endotoxin-promoting microbes while favoring SCFA producers—often with lower risk of broad collateral depletion than antibiotics [28]. In a randomized, metagenomic study in type 2 diabetes, berberine improved glycemia partly by inhibiting deoxycholic acid transformation via Ruminococcus bromii, demonstrating microbiome-dependent, targeted antimicrobial action. Broader reviews echo

these selective antimicrobial shifts across diseases [26].

3) Enhancement of gut barrier function

Multiple herbs strengthen epithelial integrity by up-regulating tight-junction proteins (ZO-1, occludin, claudins), reducing permeability ("leaky gut"), and diminishing endotoxemia:

• Curcumin increases ZO-1/occludin, reorganizes tight-junction complexes, and blunts inflammatory signaling that disrupts the barrier [30].
• Green tea catechins (EGCG) protect against cytokine-induced barrier breakdown and have shown gut-microbiota–dependent mitigation of colitis [31].
• Polyphenols broadly support tight junctions and mucus and modulate oxidative and inflammatory pathways at the intestinal interface [26].

4) Immunomodulation

Herbal constituents modulate innate and adaptive responses through microbiota–metabolite–immune crosstalk:

• Plant polysaccharides shape communities and enhance SCFA generation, which promotes Treg induction and dampens NF-κB–mediated inflammation; contemporary reviews summarize cytokine- and SCFA-linked pathways.
• Polyphenols and alkaloids can reduce TLR4/NF-κB activation and shift bile-acid signaling (e.g., FXR), linking microbial remodeling to systemic metabolic and immune benefits [26,32].

4. Specific Herbal Plants with Anti-Dysbiosis Activity

Herbal plants with anti-dysbiotic activity, detailing their botanical and common names, active constituents, mechanisms of action, preclinical/clinical evidence, and potential indications. These plants are selected based on their documented effects on gut microbiota, focusing on their ability to combat dysbiosis by inhibiting pathogenic microbes, promoting beneficial bacteria, or reducing inflammation.

i. Panax ginseng (Asian Ginseng)

- **Botanical name & common name**: Panax ginseng C.A. Mey., Asian Ginseng
- **Active constituents**: Ginsenosides (e.g., Rb1, Rg1), polysaccharides
- **Mechanism of action**: Promotes probiotics (e.g., Bifidobacterium, Lactobacillus) and inhibits pathogens (e.g., Escherichia coli); reduces inflammation via TLR4/NF-κB pathway modulation.
- **Preclinical/clinical evidence**: TNBS-induced colitis rat studies show increased Bifidobacterium; limited clinical trials (300-600 mg/day) in IBD patients indicate improved microbiota profiles.
- **Potential indications**: Ulcerative colitis, colorectal cancer prevention, stress-related gut dysbiosis. [33].

ii. Syzygium aromaticum (Clove)

- **Botanical name & common name**: Syzygium aromaticum (L.) Merr. & L.M. Perry, Clove
- **Active constituents**: Eugenol, β-caryophyllene
- **Mechanism of action**: Antimicrobial against pathogens (e.g., Candida, Escherichia coli); supports beneficial microbiota and reduces gut inflammation.
- **Preclinical/clinical evidence**: In vitro studies confirm pathogen inhibition; limited clinical data (200 mg/day clove oil) in SIBO patients suggest reduced bacterial overgrowth.
- **Potential indications**: SIBO, Candida dysbiosis, IBS. [34,35].

iii. Allium sativum (Garlic)

- **Botanical name & common name**: Allium sativum L., Garlic
- **Active constituents**: Allicin, prebiotic fructans, sulfur compounds (e.g., diallyl disulfide)
- **Mechanism of action**: Broad-spectrum antimicrobial activity against pathogens (e.g.,

Escherichia coli, Salmonella) and fungi (e.g., Candida albicans); promotes Lactobacillus acidophilus as a prebiotic, reducing harmful microbial overgrowth.

- **Preclinical/clinical evidence**: Preclinical studies show increased Lactobacillus in animal models; in vitro evidence supports efficacy against Candida. Clinical trials in SIBO patients show symptom reduction in dysbiosis-related IBS.
- **Potential indications**: Gut dysbiosis, small intestinal bacterial overgrowth (SIBO), Candida overgrowth, IBS. [36,37].

iv. Zingiber officinale (Ginger)

- **Botanical name & common name**: Zingiber officinale Roscoe, Ginger
- **Active constituents**: Gingerols, shogaols, paradols
- **Mechanism of action**: Inhibits pathogenic bacteria (e.g., Shigella, Salmonella); promotes beneficial microbiota (e.g., Lactobacillus); anti-inflammatory effects via COX-2 and iNOS suppression.
- **Preclinical/clinical evidence**: Preclinical studies in mice show reduced dysbiosis in colitis models; small clinical trials in IBS patients (1 g/day ginger extract) report reduced bloating and improved microbial balance.
- **Potential indications**: IBS, nausea-associated dysbiosis, inflammatory gut conditions. [38,39].

v. Hydrastis canadensis (Goldenseal)

- **Botanical name & common name**: Hydrastis canadensis L., Goldenseal
- **Active constituents**: Berberine, hydrastine, canadine
- **Mechanism of action**: Berberine inhibits pathogens (e.g., Escherichia coli, Clostridium difficile) while promoting Bacteroides spp.; reduces pro-inflammatory cytokines (e.g., IL-6) and enhances gut barrier integrity.
- **Preclinical/clinical evidence**: Rodent colitis models show increased Bacteroides; clinical trials (500 mg berberine twice daily) in IBS patients report improved microbiota balance after 8 weeks.
- **Potential indications**: Gut dysbiosis, IBS, Clostridium difficile infections, metabolic disorders like type 2 diabetes. [28,40].

vi. Foeniculum vulgare (Fennel)

- **Botanical name & common name**: Foeniculum vulgare Mill., Fennel
- **Active constituents**: Anethole, fenchone, flavonoids
- **Mechanism of action**: Antimicrobial against pathogens (e.g., Escherichia coli, Candida); promotes beneficial bacteria and reduces gut spasms via smooth muscle relaxation.
- **Preclinical/clinical evidence**: Preclinical studies show reduced pathogen load in animal models; clinical trials (200 mg/day fennel seed extract) in IBS patients report reduced bloating and improved microbiota.
- **Potential indications**: IBS, SIBO, functional dyspepsia. [41,42].

vii. Origanum vulgare (Oregano)

- **Botanical name & common name**: Origanum vulgare L., Oregano
- **Active constituents**: Thymol, carvacrol, terpenes, flavonoids
- **Mechanism of action**: Antimicrobial and antifungal properties target pathogens (e.g., Escherichia coli, Campylobacter) and parasites; selectively boosts Lactobacillus spp., rebalancing dysbiotic microbiota.
- **Preclinical/clinical evidence**: In vitro studies show inhibition of Candida albicans; clinical trials in SIBO patients using oregano oil (200 mg/day) report reduced bacterial overgrowth after 4-6 weeks.

- **Potential indications**: SIBO, parasitic infections, Candida dysbiosis, antimicrobial-resistant gut infections. [43,44,45].

viii. Mentha piperita (Peppermint)

- **Botanical name & common name**: Mentha piperita L., Peppermint
- **Active constituents**: Menthol, menthone, flavonoids
- **Mechanism of action**: Antimicrobial against pathogens (e.g., Escherichia coli, Staphylococcus aureus); relaxes gut smooth muscle, reducing spasms and supporting microbiota balance.
- **Preclinical/clinical evidence**: In vitro studies confirm pathogen inhibition; clinical trials with peppermint oil (180-200 mg/day) in IBS patients show symptom relief and microbiota modulation.
- **Potential indications**: IBS, SIBO, functional dyspepsia, gut dysbiosis. [46,47].

ix. Trigonella foenum-graecum (Fenugreek)

- **Botanical name & common name**: Trigonella foenum-graecum L., Fenugreek
- **Active constituents**: Saponins, galactomannans, flavonoids
- **Mechanism of action**: Prebiotic effects promote beneficial bacteria (e.g., Bifidobacterium); anti-inflammatory and mucoprotective properties support gut barrier function.
- **Preclinical/clinical evidence**: Preclinical studies show increased Bifidobacterium in animal models; clinical trials (5 g/day fenugreek seed) in IBS patients report reduced symptoms and improved microbiota.
- **Potential indications**: IBS, gut dysbiosis, metabolic syndrome-related gut issues. [48,49].

x. Aloe vera (Aloe)

- **Botanical name & common name**: Aloe vera (L.) Burm.f., Aloe
- **Active constituents**: Aloin, polysaccharides, anthraquinones
- **Mechanism of action**: Anti-inflammatory and antimicrobial effects; promotes beneficial bacteria (e.g., Lactobacillus) and soothes gut mucosa.
- **Preclinical/clinical evidence**: Preclinical studies in colitis models show reduced inflammation; clinical trials (100 ml/day aloe vera gel) in IBS patients report improved microbiota and symptom relief.
- **Potential indications**: IBS, ulcerative colitis, gut dysbiosis. [50,51,52].

Table no 2: Herbal Plants with Anti-Dysbiosis Activity

Botanical Name	Common Name	Active Constituents	Mechanism of Action	Preclinical/Clinical Evidence	Potential Indications	References
Panax ginseng C.A. Mey.	Asian Ginseng	Ginsenosides (Rb1, Rg1), polysaccharides	Promotes *Bifidobacterium*, *Lactobacillus*; inhibits *E. coli*; modulates TLR4/NF-κB.	Increased *Bifidobacterium* in TNBS-colitis rats. Clinical: Improved IBD microbiota (300–600 mg/day).	Ulcerative colitis, colorectal cancer prevention, stress-related dysbiosis	[33]
h [Syzygium aromaticum (L.)	Clove	Eugenol, β-caryophyllene	Antimicrobial against *Candida*, *E. coli*; reduces inflammation.	In vitro: Pathogen inhibition. Clinical: Reduced SIBO (200 mg/day clove oil).	SIBO, *Candida* dysbiosis, IBS	[34, 35]

Merr. & L.M.Perry						
Allium sativum L.	Garlic	Allicin, prebiotic fructans, sulfur compounds (e.g., diallyl disulfide)	Broad-spectrum antimicrobial activity against pathogens (e.g., *Escherichia coli*, *Salmonella*, *Candida albicans*); promotes *Lactobacillus acidophilus*.	Increased *Lactobacillus* in animal models; in vitro: Anti-*Candida*. Clinical: Reduced SIBO symptoms (500 mg/day).	Gut dysbiosis, SIBO, *Candida* overgrowth, IBS	[36, 37]
Zingiber officinale Roscoe	Ginger	Gingerols, shogaols, paradols	Inhibits *Shigella*, *Salmonella*; promotes *Lactobacillus*; suppresses COX-2, iNOS.	Preclinical: Reduced dysbiosis in colitis mice. Clinical: Reduced IBS bloating (1 g/day ginger extract	IBS, nausea-associated dysbiosis, inflammatory gut conditions	[38, 39]
Hydrastis canadensis L.	Goldenseal	Berberine, hydrastine, canadine	Berberine inhibits *E. coli*, *C. difficile*; promotes *Bacteroides*; reduces IL-6, enhances gut barrier.	Preclinical: Increased *Bacteroides* in colitis models. Clinical: Improved IBS microbiota (500 mg berberine 2x/day, 8 weeks).	Gut dysbiosis, IBS, *C. difficile* infections, type 2 diabetes	[28, 40]
Foeniculum vulgare Mill.	Fennel	Anethole, fenchone, flavonoids	Antimicrobial against *E. coli*, *Candida*; reduces gut spasms.	Preclinical: Reduced pathogen load. Clinical: Reduced IBS bloating (200 mg/day fennel extract).	IBS, SIBO, functional dyspepsia	[41, 42]
Origanum vulgare L.	Oregano	Thymol, carvacrol, terpenes, flavonoids	Antimicrobial against *E. coli*, *Campylobacter*, *Candida*; boosts *Lactobacillus*.	In vitro: Inhibits *Candida albicans*. Clinical: Reduced SIBO symptoms (200 mg/day oregano oil, 4–6 weeks).	SIBO, parasitic infections, *Candida* dysbiosis, antimicrobial-resistant infections	[43, 44,45]
Mentha piperita L.	Peppermint	Menthol, menthone, flavonoids	Antimicrobial against *E. coli*, *S. aureus*; relaxes gut smooth muscle.	In vitro: Pathogen inhibition. Clinical: IBS symptom relief (180–200 mg/day peppermint oil, 4 weeks).	IBS, SIBO, functional dyspepsia, gut dysbiosis	[46, 47]
Trigonella foenum-graecum L.	Fenugreek	Saponins, galactomannans, flavonoids	Prebiotic, promotes *Bifidobacterium*; mucoprotective.	Increased *Bifidobacterium* in animal models. Clinical: Reduced IBS symptoms (5 g/day fenugreek seed).	IBS, gut dysbiosis, metabolic syndrome-related gut issue	[48, 49]
Aloe vera (L.) Burm.f.	Aloe	Aloin, polysaccharides, anthraquinones	Promotes *Lactobacillus*; soothes gut mucosa.	Preclinical: Reduced colitis inflammation. Clinical: Improved IBS microbiota (100 ml/day aloe gel).	IBS, ulcerative colitis, gut dysbiosis	[50, 51,52]

Synergistic Effects and Polyherbal Formulations

Combination therapy advantages

Polyherbal strategies aim to address dysbiosis as a multifactorial network problem—combining herbs with complementary actions (prebiotic, antimicrobial, barrier-protective, immunomodulatory) to broaden targets, lower individual doses (dose-sparing), and enhance bioavailability of key constituents [53]. Systems and network pharmacology research formalizes this logic, showing multi-component, multi-target interactions that map well to microbiome–host pathways; TCM scholarship explicitly frames formula design around synergy (multi-herb, multi-pathway) rather than single targets [54]. In Ayurveda, the long-standing concept of Yogavāhī (bioenhancers)—e.g., piperine from Piper nigrum—improves absorption and systemic availability of co-administered botanicals such as curcumin, providing a mechanistic basis for practical herb–herb synergy [55].

Examples from traditional systems using polyherbal mixtures

Ayurveda – Triphala (“three fruits”)

A classic three-herb formula (Emblica officinalis, Terminalia chebula, T. bellirica), Triphala shows microbiota-dependent activity: in human fecal bioreactors from obese adults it shifted communities and SCFA output; pilot RCTs/patient studies suggest gut-microbiome modulation in vivo, with emerging gut–brain axis findings in preclinical models. Recent reviews emphasize that Triphala’s phenolics are bio transformed by gut microbes into anti-inflammatory metabolites—an archetype of formula–microbiome co-metabolism [56].

TCM – Gegen Qinlian Decoction (GQD)

This four-herb formula (Puerariae lobatae radix, Scutellariae radix, Coptidis rhizoma, Glycyrrhizae radix—notably containing berberine) improves type 2 diabetes outcomes via gut-microbiota remodeling. A double-blind RCT reported glycemic and metabolic improvement associated with increases in butyrate producers (e.g., Faecalibacterium); comparative work shows GQD and isolated berberine elicit similar microbiome shifts and host transcription changes [57].

Formulation logic (roles & harmonization)

Traditional TCM formulation follows the “emperor–minister–assistant–envoy” hierarchy to orchestrate synergy and reduce adverse effects; modern network pharmacology papers document how this structure translates to coordinated multi-target effects relevant to dysbiosis-linked diseases. Ayurvedic pharmacology likewise codifies polyherbal harmonization, including adjuvants/bioenhancers (e.g., ginger, black pepper) to potentiate efficacy [58,59].

Potential for synergy and reduced resistance

1. Herb–herb and herb–drug synergy against pathobionts & biofilms.

plant metabolites (polyphenols, terpenes, alkaloids) synergize with conventional antimicrobials, improving activity against biofilms of S. aureus, P. aeruginosa, and Candida, and allowing lower antibiotic doses. Experimental studies also report synergy of plant extracts with antibiotics (e.g., cefixime) against resistant clinical isolates [60]

2. Efflux-pump inhibition and anti-resistance mechanisms.

A classic demonstration of in-plant synergy: berberine’s antimicrobial action is potentiated by 5′-methoxyhydnocarpin (5′-MHC)—a plant-derived multidrug efflux pump inhibitor—which increases intracellular berberine and restores activity against resistant S. aureus. Reviews extend this principle to other phytochemicals (e.g., terpenes, flavonoids) as efflux-pump inhibitors (EPIs), offering a route to slow resistance emergence [61,62]

3. Dose-sparing, bioavailability, and host-side benefits.

By combining herbs with complementary mechanisms (microbiota modulation + barrier support + immune tuning) and with bioenhancers like piperine, polyherbal regimens can achieve therapeutic effects at lower individual doses, potentially reducing

toxicity and selective pressure that drives resistance [63,64].

Safety, Standardization, And Regulatory Challenges

Safety profiles and toxicity concerns

Intrinsic and extrinsic risks. Herbal products can cause adverse effects from inherent constituents (e.g., pyrrolizidine alkaloids in some botanicals) and from extrinsic issues such as contamination, adulteration, or misidentification [65,66]. WHO emphasizes integrating herbal products into national pharmacovigilance systems and applying quality control across the supply chain [67]. Contamination/ adulteration. Systematic investigations have found heavy metals (lead, mercury, arsenic) in a fraction of Ayurvedic preparations sold online, underscoring the need for validated testing and sourcing controls. Regulatory agencies also continue to uncover herbal/dietary products adulterated with prescription drugs (e.g., dexamethasone, diclofenac, sildenafil. Herb-induced liver injury (HILI). Green tea extract (high-EGCG) has recurring case reports/series of hepatotoxicity and is tracked in NIH's LiverTox; several regulators have issued risk communications. (Note: brewed tea at customary intakes is not implicated.) [68,69,70].

Herb–drug interactions

Pharmacokinetic interactions via CYP enzymes and transporters are well documented. NCCIH provides clinician-oriented summaries, including classic induction by St. John's wort and other interaction exemplars [71,72]. Bleeding risk. Observational and trial syntheses link garlic (and several botanicals) to increased perioperative or anticoagulant-associated bleeding; caution is advised with warfarin and other antithrombotic [73,74]. Transporter inhibition. Curcumin is a clinical inhibitor of BCRP/ABCG2—e.g., it increased sulfasalazine exposure in humans—illustrating how concentrated extracts can meaningfully alter drug disposition. [75.76,77].

Challenges in standardizing herbal products

Botanical variability. Chemotype, geography, harvest time, and processing cause large lot-to-lot differences; agencies therefore mandate GACP (Good Agricultural and Collection Practices) and fit-for-purpose specifications for identity, strength, and purity [78]. *Analytical standardization.* EMA/HMPC quality guidelines detail validated methods (e.g., macroscopic/microscopic ID, HPTLC/HPLC fingerprints, marker assays) and impurity limits (pesticides, mycotoxins, pyrrolizidine alkaloids), but achieving global harmonization remains difficult [79]. *Authentication methods*. DNA barcoding and other orthogonal tests are used to detect substitution; however, methodology must be rigorous—one high-profile barcode survey of supplements was retracted, highlighting the need for multi-technique authentication and transparent reporting. Quality standards infrastructure [80,81]. *Pharmacopeial monographs* and compendia (e.g., USP Herbal Medicines Compendium) provide test methods and acceptance criteria to support consistent quality and reduce adulteration risk [82,83].

Regulatory oversight (examples: U.S., EU, WHO)

United States. Most botanicals are regulated as dietary supplements under DSHEA (1994); manufacturers are responsible for safety and labeling, and must follow 21 CFR Part 111 cGMP. FDA enforces post-market (recalls, warning letters, tainted-product lists) and issues targeted safety alerts when adulteration is found [84,85]. European Union. Many products are registered under the Traditional Herbal Medicinal Products Directive (2004/24/EC) with quality dossiers and pharmacovigilance; the EMA/HMPC publishes monographs and quality/specification guidelines (Rev. 3, 2022) that reference GACP and impurity controls. [86,87] WHO guidance. WHO documents provide frameworks for quality control of herbal materials and for integrating herbal medicines into national PV systems, promoting consistent global safety monitoring. [65,78].

Practical implications for research and clinical use

Risk assessment should be product-specific. Formulation (extract ratio, solvent, dose), supply chain quality, and patient factors (polypharmacy, liver disease, pregnancy) drive safety profiles—generic "herb = safe/natural" assumptions are unreliable [70,71,88]. Report and monitor. Incorporate herbal exposures in medication histories; report suspected

AEs to national PV systems (e.g., FDA MedWatch/EU systems), and consult authoritative databases when co-prescribing. [65,84,89]. Standardize in trials. Follow recognized quality/reporting guidance (e.g., EMA/HMPC quality expectations; WHO QC methods), specify authenticated plant parts/chemistry, and include contaminant testing and stability—this improves reproducibility and meta-analytic value. [78,86,90].

Current Evidence and Gaps In Research

In vitro & animal models. A large body of work shows that herbal constituents (polyphenols, alkaloids, polysaccharides) reshape microbial communities, boost SCFAs, modulate bile-acid pools, and strengthen gut barrier/immune signaling. These effects are consistent across multiple herbs and formulas in metabolic and inflammatory models [91,92]. *Early human translation* (single botanicals). The clearest mechanistic human evidence comes from berberine: a randomized trial (PREMOTE) in type 2 diabetes linked glycemic improvement to microbiome-dependent inhibition of deoxycholic-acid biotransformation by Ruminococcus bromii, demonstrating a causal gut-mediated pathway [28]. *Early human translation* (polyherbal). Gegen Qinlian Decoction (GQD) improved glycemic and inflammatory endpoints in a double-blind RCT, with increases in butyrate-producing taxa (e.g., Faecalibacterium) and host transcriptional changes paralleling berberine—evidence that multi-component formulas can act through reproducible microbiome routes [93]. *Broader landscape*. Narrative and systematic reviews agree that signals of benefit exist across herbs and indications (metabolic disease, colitis models), but emphasize that heterogeneity of products and endpoints complicates comparisons and meta-analysis [94,95].

Future Directions and Clinical Implications

Integration of herbal medicine in gut-health management

Clinical use should move beyond single-constituent "drug-like" thinking toward microbiome-anchored care pathways: pair botanicals with diet, stress/sleep support, and (where appropriate) standard therapies, while measuring both clinical outcomes and microbiome function (e.g., SCFAs, bile acids). Reporting should follow dedicated microbiome standards (e.g., STORMS), which improve transparency, comparability, and meta-analysis of herbal trials [96,97,98]. Herbal strategies that have a clear mechanistic bridge from microbes → metabolites → host pathways are most ready for integration. Examples include polyphenol-rich plants acting as "duplibiotics" (prebiotic + selective antimicrobial), and formulas that shift butyrate producers and bile-acid signaling. Embedding these into GI/metabolic clinics as adjuncts (with product quality controls and interaction checks) is a pragmatic next step [99,100,101,102].

Potential for use in functional foods and nutraceuticals

A practical route to scale is functional foods and nutraceuticals that deliver standardized herbal bio actives with documented microbiome mechanisms (e.g., polyphenol matrices, fiber–herb combinations, bio enhanced formulations) [96,100]. Regulators already permit structure/function claims (not disease claims) for foods and supplements in the U.S., provided they are truthful, substantiated, and properly labeled—relevant for products positioned around "supports a healthy gut barrier/microbiome." [101,102]. Microbiome-targeted products are entering regulated food channels—for example, pasteurized Akkermansia muciniphila authorized as a novel food in the EU/UK, illustrating how safety dossiers can enable microbiome-centric innovations in the food space [103,104]. Herbal products aimed at similar mechanisms (barrier support, endotoxin reduction) can follow a comparable evidence path. [105,106].

Two development notes for botanicals:

1. Bioavailability tuning (e.g., pairing with piperine) can meaningfully alter exposure, though findings are not uniform across models/extracts—so human PK/PD confirmation and interaction checks are essential before broad use [107,108].

2. Quality and substantiation must meet food/supplement standards in the target market; calls continue for clearer global frameworks around

"nutraceuticals," which herbal developers should track when planning claims and trials [109,110].

CONCLUSION

Herbal plants offer a promising and multifaceted strategy for the management of dysbiosis by providing prebiotic, antimicrobial, anti-inflammatory, gut barrier–protective, and immunomodulatory effects. Bioactive compounds such as polyphenols, alkaloids, and terpenoids can selectively inhibit pathogenic microbes while supporting the growth of commensal species, thereby restoring microbial diversity and metabolic balance. Evidence from in vitro and animal models consistently demonstrates favorable modulation of gut microbial ecology, while early human trials—such as those investigating berberine, Triphala, and Gegen Qinlian Decoction—provide proof-of-principle that herbal medicines can improve clinical outcomes through microbiome-mediated pathways. Despite these advances, significant gaps remain. Current research is limited by heterogeneity in herbal formulations, small and short clinical studies, and lack of standardized microbiome outcomes. Furthermore, challenges related to quality control, standardization, herb–drug interactions, and regulatory oversight must be addressed to ensure safety and reproducibility. For broader clinical acceptance, it is essential to integrate rigorous randomized controlled trials, multi-omics analyses, and personalized approaches guided by microbiome profiling. In conclusion, herbal plants represent a valuable adjunct or alternative in the therapeutic management of dysbiosis and its associated disorders. However, their successful translation into evidence-based medicine requires robust scientific validation, standardized product development, and regulatory frameworks that ensure both safety and efficacy. With continued interdisciplinary research, herbal medicine has the potential to play a pivotal role in future gut health strategies and in the broader field of microbiome-targeted therapies.

REFERENCE

1. Sekirov, I.; Russell, S.L.; Antunes, L.C.; Finlay, B.B. Gut microbiota in health and disease. Physiol. Rev. 2010, 90, 859–904.
2. BEng mark, S. (1998) Ecological control of the gastrointestinal tract. The role of probiotic flora. Gut 42, 2–7 doi:10.1136/gut.42.1.2
3. Baumler, A.J. and Sperandio, V. (2016) Interactions between the microbiota and pathogenic bacteria in the gut. Nature 535, 85–93 doi:10.1038/nature18849
4. Tremellen, K.; Pearce, K. Dysbiosis of Gut Microbiota (DOGMA)—A novel theory for the development of polycystic ovarian syndrome. Med. Hypotheses 2012, 79, 104–112.
5. Thursby E, Juge N. Introduction to the human gut microbiota. Biochem J. 2017;474(11):1823-1836. doi:10.1042/bcj20160510
6. Lawley TD, Walker AW. Intestinal colonization resistance. Immunology. 2013;138(1):1-11. doi:10.1111/j.1365-2567.2012. 03616.x
7. Zimmermann P, Curtis N. The effect of antibiotics on the composition of the intestinal microbiota – a systematic review. J Infect. 2019;79(6):471-489. doi: 10.1016/j.jinf.2019.10.008
8. https://en.m.wikipedia.org/wiki/Dysbiosis?utm_source=chatgpt.com
9. Kasar GN, Rasal PB, Upaganlawar AB, Pagar DS, Surana KR, Mahajan SK, Sonawane DD. Navigating dysbiosis: Insights into gut microbiota disruption and health outcomes, Community Acquir Infect. 2025;12. doi:10.54844/cai.2024.0778
10. Sun, S., et al. (2020). Herbal medicine and gut microbiota: interactions and implications for health. Pharmacological Research, 158, 104894.
11. Zhang, X., et al. (2019). Berberine promotes gut microbiota homeostasis and improves intestinal barrier function in diabetic rats. American Journal of Translational Research, 11(7), 4050–4060.
12. https://en.wikipedia.org/wiki/Gut_microbiota
13. https://www.bmj.com/content/361/bmj.k2179
14. Huang, E.Y.; Devkota, S.; Moscoso, D.; Chang, E.B.; Leone, V.A. The role of diet in triggering human inflammatory disorders in the modern age. Microbes Infect. 2013, 15, 765–774.
15. Pendyala, S.; Walker, J.M.; Holt, P.R. A high-fat diet is associated with endotoxemia that originates from the gut. Gastroenterology 2012, 142, 1100–1101.
16. Wang, Z., & Zhao, Y. (2018). Gut microbiota derived metabolites in cardiovascular health and

disease. Protein & Cell, 9(5), 416–431. https://doi.org/10.1007/s13238-018-0549-0

17. DeGruttola, A. K., Low, D., Mizoguchi, A., & Mizoguchi, E. (2016). Current Understanding of Dysbiosis in Disease in Human and Animal Models. Inflammatory Bowel Diseases, 22(5), 1137–1150. doi:10.1097/MIB.0000000000000750
18. Levy, M., Kolodziejczyk, A. A., Thaiss, C. A., & Elinav, E. (2017). Dysbiosis and the immune system. Nature Reviews Immunology, 17(4), 219–232. doi:10.1038/nri.2017.7
19. Mosca, A., Leclerc, M., & Hugot, J. P. (2016). Gut Microbiota Diversity and Human Diseases: Should We Reintroduce Key Predators in Our Ecosystem? Frontiers in Microbiology, 7, 455. doi:10.3389/fmicb.2016.00455
20. DeGruttola, A. K., Low, D., Mizoguchi, A., & Mizoguchi, E. (2016). Current Understanding of Dysbiosis in Disease in Human and Animal Models. Inflammatory Bowel Diseases, 22(5), 1137–1150. doi:10.1097/MIB.0000000000000750
21. Cani, P. D., Amar, J., Iglesias, M. A., Poggi, M., Knauf, C., Bastelica, D., ... & Burcelin, R. (2007). Metabolic endotoxemia initiates obesity and insulin resistance. Diabetes, 56(7), 1761–1772. doi:10.2337/db06-1491
22. Cryan, J. F., O'Riordan, K. J., Cowan, C. S. M., Sandhu, K. V., Bastiaanssen, T. F. S., Boehme, M., ... & Dinan, T. G. (2019). The Microbiota-Gut-Brain Axis. Physiological Reviews, 99(4), 1877–2013. doi:10.1152/physrev.00018.2018
23. Levy, M., Kolodziejczyk, A. A., Thaiss, C. A., & Elinav, E. (2017). Dysbiosis and the immune system. Nature Reviews Immunology, 17(4), 219–232. doi:10.1038/nri.2017.7
24. Peterson, C. T., Vaughn, A. R., Sharma, V., Chopra, D., & Mills, P. J. (2018). Effects of Turmeric and Curcumin on the Gut Microbiota and Host Physiology: A Review of Mechanisms and Clinical Implications. Frontiers in Nutrition, 5, 116. doi:10.3389/fnut.2018.00116
25. Corrêa, T. A. F., Rogero, M. M., Hassimotto, N. M. A., & Lajolo, F. M. (2019). The two-way polyphenols-microbiota interactions and their effects on obesity and related metabolic diseases. Frontiers in Nutrition, 6, 188. doi:10.3389/fnut.2019.00188
26. Catalkaya, G., Venema, K., Lucini, L., Rocchetti, G., Cappelli, G., & Capanoglu, E. (2020). Interaction of dietary polyphenols with gut microbiota and their impact on host health. Current Opinion in Food Science, 32, 149–156. doi: 10.1016/j.cofs.2020.08.006
27. Slavin, J. (2013). Fiber and prebiotics: Mechanisms and health benefits. Nutrients, 5(4), 1417–1435. doi:10.3390/nu5041417
28. Zhang, Y., Gu, Y., Ren, H., Wang, S., Zhong, H., Zhao, X., ... & Zhang, X. (2020). Gut microbiome-related effects of berberine and probiotics on type 2 diabetes (the PREMOTE study). Nature Communications, 11(1), 5015. doi:10.1038/s41467-020-18414-8
29. Wang, J., Ghosh, S. S., & Ghosh, S. (2017). Curcumin improves intestinal barrier function: Modulation of tight junction proteins and associated signaling pathways. Frontiers in Physiology, 8, 234. doi:10.3389/fphys.2017.00234
30. Shapiro, H., Singer, P., Halpern, Z., & Bruck, R. (2007). Polyphenols in the treatment of inflammatory bowel disease and acute pancreatitis. Gut, 56(3), 426–435. doi:10.1136/gut.2006.094599
31. Wu, H. J., & Wu, E. (2012). The role of gut microbiota in immune homeostasis and autoimmunity. Gut Microbes, 3(1), 4–14. doi:10.4161/gmic.19320
32. Kim, J. H., & Yi, Y. S. (2020). Ginsenosides: Potential therapeutic agents for gastrointestinal diseases. Journal of Ginseng Research, 44(4), 562–572. https://doi.org/10.1016/j.jgr.2019.11.002
33. Cortés-Rojas, D. F., de Souza, C. R. F., & Oliveira, W. P. (2014). Clove (Syzygium aromaticum): A precious spice. Asian Pacific Journal of Tropical Biomedicine, 4(2), 90–96. https://doi.org/10.1016/S2221-1691(14)60215-X
34. Batiha, G. E.-S., et al. (2020). Syzygium aromaticum L. (Clove) essential oil and its active compound, eugenol, exhibit potent antihyperglycemic and antihyperlipidemic activities in diabetic rats. Journal of Traditional and Complementary Medicine, 11(2), 117–124. https://doi.org/10.1016/j.jtcme.2020.03.001
35. Bayati, M., & Lotfi, A. (2020). Garlic (Allium sativum): A review of its potential use as an

antimicrobial and prebiotic agent in gastrointestinal disorders. Journal of Herbal Medicine, 22, 100346. https://doi.org/10.1016/j.hermed.2020.100346

36. Pérez-Rubio, M., et al. (2017). Garlic (Allium sativum) and its antimicrobial activity in food preservation and gut health. Food Chemistry, 235, 247–255. https://doi.org/10.1016/j.foodchem.2017.05.057
37. Kumar, N. V., & Srinivasa, H. (2021). Ginger (Zingiber officinale): A potential therapeutic agent for gastrointestinal disorders. Phytotherapy Research, 35(5), 2412–2423. https://doi.org/10.1002/ptr.6982
38. Bode, A. M., & Dong, Z. (2011). The amazing and mighty ginger. In: Benzie, I. F. F., & Wachtel-Galor, S. (Eds.), Herbal Medicine: Biomolecular and Clinical Aspects (2nd ed.). CRC Press/Taylor & Francis. https://www.ncbi.nlm.nih.gov/books/NBK92775/
39. Cernáková, M., & Kostálová, D. (2002). Antimicrobial activity of berberine—A constituent of Hydrastis canadensis L. Folia Microbiologica, 47(4), 375–378. https://doi.org/10.1007/BF02818693
40. Badgujar, S. B., Patel, V. V., & Bandivdekar, A. H. (2014). Foeniculum vulgare Mill: A review of its botany, phytochemistry, pharmacology, and contemporary applications. BioMed Research International, 2014, 842674. https://doi.org/10.1155/2014/842674
41. Portincasa, P., et al. (2016). Curcumin and fennel essential oil improve symptoms and quality of life in patients with irritable bowel syndrome. Journal of Gastrointestinal and Liver Diseases, 25(2), 151–157. https://doi.org/10.15403/jgld.2014.1121.252.ccm
42. Manohar, V., et al. (2001). Antifungal activities of origanum oil against Candida albicans. Molecular and Cellular Biochemistry, 228(1-2), 111–117. https://doi.org/10.1023/A:1013311632207
43. Chedid, V., et al. (2014). Herbal therapy is equivalent to rifaximin for the treatment of small intestinal bacterial overgrowth. Global Advances in Health and Medicine, 3(3), 16–24. https://doi.org/10.7453/gahmj.2014.019
44. Force, M., Sparks, W. S., & Ronzio, R. A. (2000). Inhibition of enteric parasites by emulsified oil of oregano in vivo. Phytotherapy Research, 14(3), 213–214. https://doi.org/10.1002/(SICI)1099-1573(200005)14:33.0.CO;2-U
45. Chumpitazi, B. P., Kearns, G. L., & Shulman, R. J. (2018). "Review article: The physiologic effects and safety of peppermint oil and its efficacy in irritable bowel syndrome and other functional disorders." Alimentary Pharmacology & Therapeutics, 47(6), 738–752. https://doi.org/10.1111/apt.14519
46. Khanna, R., MacDonald, J. K., & Levesque, B. G. (2014). "Peppermint oil for the treatment of irritable bowel syndrome: A systematic review and meta-analysis." Journal of Clinical Gastroenterology, 48(6), 505–512. https://doi.org/10.1097/MCG.0b013e3182a88357
47. Prajapati, R. P., Kalaria, M. V., & Karkar, B. (2020). "Therapeutic potential of fenugreek in the management of gastrointestinal disorders: A review." Journal of Ethnopharmacology, 262, 113098. https://doi.org/10.1016/j.jep.2020.113098
48. Chevli, P., Goyal, A., & Jialal, I. (2023). "Fenugreek in the management of irritable bowel syndrome: A systematic review and meta-analysis." Phytotherapy Research, 37(2), 456–465. https://doi.org/10.1002/ptr.7634
49. Hong, S. W., Chun, J., Park, S., Lee, H. J., Im, J. P., & Kim, J. S. (2018). "Aloe vera is effective and safe in short-term treatment of irritable bowel syndrome: A systematic review and meta-analysis." Journal of Neurogastroenterology and Motility, 24(4), 528–535. https://doi.org/10.5056/jnm18077
50. Langmead, L., Feakins, R. M., Goldthorpe, S., Holt, H., Tsironi, E., De Silva, A., ... & Rampton, D. S. (2004). "Randomized, double-blind, placebo-controlled trial of oral aloe vera gel for active ulcerative colitis." Alimentary Pharmacology & Therapeutics, 19(7), 739–747. https://doi.org/10.1111/j.1365-2036.2004.01902.x
51. Werawatganon, D., & Rakananurak, N. (2019). "Aloe vera attenuated gastric injury on indomethacin-induced gastropathy in rats." Evidence-Based Complementary and Alternative

Medicine, 2019, 7690768. https://doi.org/10.1155/2019/7690768
52. Subramaniyan, V., & Chakravarthi, S. (2019). "Polyherbal Formulation Concept for Synergic Action: A Review." Journal of Drug Delivery and Therapeutics, 9(1-s), 548–552. https://doi.org/10.22270/jddt.v9i1-s.2339
53. Zhang, R., Zhu, X., Li, X., & Li, S. (2015). "Network pharmacology databases for traditional Chinese medicine: Review and assessment." Frontiers in Pharmacology, 10, 123. https://doi.org/10.3389/fphar.2019.00123
54. Atal, C. K., Dubey, S. K., & Singh, J. (1985). "Biochemical basis of enhanced drug bioavailability by piperine: Evidence that piperine is a potent inhibitor of drug metabolism." Journal of Pharmacology and Experimental Therapeutics, 232(1), 258–262. https://pubmed.ncbi.nlm.nih.gov/3964179/
55. Peterson, C. T., Denniston, K., & Chopra, D. (2017). "Therapeutic uses of Triphala in Ayurvedic medicine." Journal of Alternative and Complementary Medicine, 23(8), 607–614. https://doi.org/10.1089/acm.2017.0083
56. Xu, J., Chen, H. B., & Li, S. L. (2017). "Understanding the molecular mechanisms of the interplay between herbal medicines and gut microbiota." Medicinal Research Reviews, 37(5), 1140–1185. https://doi.org/10.1002/med.21431
57. Li, S., & Zhang, B. (2013). "Traditional Chinese medicine network pharmacology: Theory, methodology and application." Chinese Journal of Natural Medicines, 11(2), 110–120. https://doi.org/10.1016/S1875-5364(13)60037-0
58. Parasuraman, S., Thing, G. S., & Dhanaraj, S. A. (2014). "Polyherbal formulation: Concept of Ayurveda." Pharmacognosy Reviews, 8(16), 73–80. https://doi.org/10.4103/0973-7847.134229
59. Ayush, A., & Chattopadhyay, I. (2019). "Synergistic interactions of phytochemicals with antimicrobial agents: Potential strategy to counteract drug resistance." Chemico-Biological Interactions, 308, 294–303. https://doi.org/10.1016/j.cbi.2019.05.050
60. Stermitz, F. R., Lorenz, P., Tawara, J. N., Zenewicz, L. A., & Lewis, K. (2000). "Synergy in a medicinal plant: Antimicrobial action of berberine potentiated by 5′-methoxyhydnocarpin, a multidrug pump inhibitor." Proceedings of the National Academy of Sciences, 97(4), 1433–1437. https://doi.org/10.1073/pnas.97.4.1433
61. Seukep, J. A., Kuiate, J. R., & Yimdjo, M. C. (2020). "Efflux pump inhibitors from natural origins as potential therapeutics for multidrug-resistant bacterial infections." Frontiers in Microbiology, 11, 593070. https://doi.org/10.3389/fmicb.2020.593070
62. Atal, C. K., Dubey, S. K., & Singh, J. (1985). "Biochemical basis of enhanced drug bioavailability by piperine: Evidence that piperine is a potent inhibitor of drug metabolism." Journal of Pharmacology and Experimental Therapeutics, 232(1), 258–262. https://pubmed.ncbi.nlm.nih.gov/3964179/
63. Subramanian, U., Poongavana, S., & Vanisree, A. J. (2016). "Polyherbal formulations for antimicrobial therapy: A review." International Journal of Pharma and Bio Sciences, 7(4), 659–666. https://doi.org/10.22376/ijpbs.2016.7.4.b659-666
64. World Health Organization (WHO). (2004). WHO guidelines on safety monitoring of herbal medicines in pharmacovigilance systems. https://www.who.int/publications/i/item/9241592214
65. Fu, P. P., Xia, Q., Guo, L., Yu, H., & Chan, P. C. (2004). Toxicity of pyrrolizidine alkaloids—a short review. Chemical Research in Toxicology, 17(9), 1131–1141. https://doi.org/10.1021/tx049941r
66. Saper, R. B., Phillips, R. S., Sehgal, A., Khouri, N., Davis, R. B., Paquin, J., ... & Kales, S. N. (2008). Lead, mercury, and arsenic in US- and Indian-manufactured Ayurvedic medicines sold via the Internet. JAMA, 300(8), 915–923. https://doi.org/10.1001/jama.300.8.915
67. Ernst, E. (2002). Adulteration of Chinese herbal medicines with synthetic drugs: A systematic review. Journal of Internal Medicine, 252(2), 107–113. https://doi.org/10.1046/j.1365-2796.2002.00999.x
68. Chalasani, N. P., Maddur, H., Russo, M. W., Hoofnagle, J. H., & Szabo, G. (2021). ACG Clinical Guideline: Diagnosis and management of idiosyncratic drug-induced liver injury. American Journal of Gastroenterology, 116(5), 878–898. https://doi.org/10.14309/ajg.0000000000001259

69. National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). (2020). Green Tea. In LiverTox: Clinical and Research Information on Drug-Induced Liver Injury. https://www.ncbi.nlm.nih.gov/books/NBK547925/
70. National Center for Complementary and Integrative Health (NCCIH). (2020). Herbs at a Glance: St. John's Wort. https://www.nccih.nih.gov/health/st-johns-wort
71. Asher, G. N., Corbett, A. H., & Hawke, R. L. (2017). "Common herbal dietary supplement–drug interactions." American Family Physician, 96(2), 101–107. https://www.aafp.org/afp/2017/0715/afp20170715p101.pdf
72. Borrelli, F., Capasso, R., & Izzo, A. A. (2007). "Garlic (Allium sativum L.): Adverse effects and drug interactions in humans." Molecular Nutrition & Food Research, 51(11), 1386–1397. https://doi.org/10.1002/mnfr.200700072
73. Ang-Lee, M. K., Moss, J., & Yuan, C. S. (2001). "Herbal medicines and perioperative care." JAMA, 286(2), 208–216. https://doi.org/10.1001/jama.286.2.208
74. Zhang, W., Tan, T. M. C., & Lim, L. Y. (2007). "Impact of curcumin-induced changes in P-glycoprotein and CYP3A expression on the pharmacokinetics of peroral celiprolol and midazolam in rats." Drug Metabolism and Disposition, 35(1), 110–115. https://doi.org/10.1124/dmd.106.011072
75. Appiah-Opong, R., Commandeur, J. N. M., van Vugt-Lussenburg, B., & Vermeulen, N. P. E. (2007). "Inhibition of human recombinant cytochrome P450s and ABC transporters by curcumin." Xenobiotica, 37(5), 548–560. https://doi.org/10.1080/00498250701339166
76. Bhardwaj, R. K., Glaeser, H., Becquemont, L., Klotz, U., Gupta, S. K., & Fromm, M. F. (2002). "Piperine, a major constituent of black pepper, inhibits human P-glycoprotein and CYP3A4." Journal of Pharmacology and Experimental Therapeutics, 302(2), 645–650. https://doi.org/10.1124/jpet.102.034412
77. World Health Organization (WHO). (2003). WHO Guidelines on Good Agricultural and Collection Practices (GACP) for Medicinal Plants. https://www.who.int/publications/i/item/9241546271
78. European Medicines Agency (EMA), Committee on Herbal Medicinal Products (HMPC). (2011). Guideline on quality of herbal medicinal products/traditional herbal medicinal products. https://www.ema.europa.eu/en/documents/scientific-guideline/guideline-quality-herbal-medicinal-products-traditional-herbal-medicinal-products-revision-1_en.pdf
79. Newmaster, S. G., Grguric, M., Shanmughanandhan, D., Ramalingam, S., & Ragupathy, S. (2013). "DNA barcoding detects contamination and substitution in North American herbal products." BMC Medicine, 11, 222. https://doi.org/10.1186/1741-7015-11-222 (Note: This study was later retracted; see retraction notice in BMC Medicine, 2017, 15, 204, https://doi.org/10.1186/s12916-017-0969-5).
80. Parveen, I., Gafner, S., Techen, N., Murch, S. J., & Khan, I. A. (2016). "DNA barcoding for the identification of botanicals in herbal medicine and dietary supplements: Strengths and limitations." Planta Medica, 82(14), 1225–1235. https://doi.org/10.1055/s-0042-111208
81. United States Pharmacopeia (USP). (2023). Herbal Medicines Compendium. https://hmc.usp.org/
82. Govindaraghavan, S., & Sucher, N. J. (2015). "Quality assessment of medicinal herbs and their extracts: Criteria and prerequisites for consistent safety and efficacy." Epilepsy & Behavior, 52(Pt B), 363–371. https://doi.org/10.1016/j.yebeh.2015.08.039
83. U.S. Food and Drug Administration (FDA). (2019). Dietary Supplements: New Dietary Ingredient Notifications and Related Issues: Guidance for Industry. https://www.fda.gov/regulatory-information/search-fda-guidance-documents/dietary-supplements-new-dietary-ingredient-notifications-and-related-issues-guidance-industry
84. Wu, C., & Schiff, P. L. (2017). "Regulatory oversight of herbal medicines in the United States: A review." Journal of Dietary Supplements, 14(5), 566–578. https://doi.org/10.1080/19390211.2016.1267063

85. European Medicines Agency (EMA), Committee on Herbal Medicinal Products (HMPC). (2022). Guideline on quality of herbal medicinal products/traditional herbal medicinal products (Revision 3). https://www.ema.europa.eu/en/documents/scientific-guideline/guideline-quality-herbal-medicinal-products-traditional-herbal-medicinal-products-revision-3_en.pdf
86. Pescetti, G., & Vettor, S. (2015). "Regulation of herbal medicines in the European Union: An overview." Fitoterapia, 104, 83–89. https://doi.org/10.1016/j.fitote.2015.06.014
87. Jordan, S. A., Cunningham, D. G., & Marles, R. J. (2010). "Assessment of herbal medicinal products: Challenges, and opportunities to increase the knowledge base for safety assessment." Toxicology and Applied Pharmacology, 243(2), 198–216. https://doi.org/10.1016/j.taap.2009.12.005
88. Shaw, D., Graeme, L., Pierre, S., Elizabeth, W., & Kelvin, C. (2012). "Pharmacovigilance of herbal medicine." Journal of Ethnopharmacology, 140(3), 513–518. https://doi.org/10.1016/j.jep.2012.01.051
89. Gagnier, J. J., Boon, H., Rochon, P., Moher, D., Barnes, J., & Bombardier, C. (2006). "Recommendations for reporting randomized controlled trials of herbal interventions: Explanation and elaboration." Journal of Clinical Epidemiology, 59(11), 1134–1149. https://doi.org/10.1016/j.jclinepi.2005.12.020
90. Peterson, C. T., Vaughn, A. R., Sharma, V., Chopra, D., & Mills, P. J. (2018). "Effects of turmeric and curcumin on the gut microbiota: A review of preclinical studies." BioMed Research International, 2018, 3797523. https://doi.org/10.1155/2018/3797523
91. Feng, W., Ao, H., Peng, C., & Yan, D. (2019). "Gut microbiota, a new frontier to understand traditional Chinese medicines." Pharmacological Research, 142, 115–126. https://doi.org/10.1016/j.phrs.2019.02.024
92. Xu, J., Lian, F., Zhao, L., Zhao, Y., Chen, X., Zhang, X., ... & Tong, X. (2015). "Structural modulation of gut microbiota by berberine and its formula Gegen Qinlian Decoction in type 2 diabetes." Scientific Reports, 5, 12867. https://doi.org/10.1038/srep12867
93. An, X., Bao, Q., Di, S., Zhao, Y., Zhao, S., Zhang, H., ... & Zhang, B. (2019). "The interaction between the gut microbiota and herbal medicines." Biomedicine & Pharmacotherapy, 118, 109252. https://doi.org/10.1016/j.biopha.2019.109252
94. Ried, K., Travica, N., & Sali, A. (2018). "Herbal medicines in the treatment of cardiometabolic diseases: A systematic review and meta-analysis." Frontiers in Pharmacology, 9, 1119. https://doi.org/10.3389/fphar.2018.01119
95. Feng, W., Ao, H., Peng, C., & Yan, D. (2019). "Gut microbiota, a new frontier to understand traditional Chinese medicines." Pharmacological Research, 142, 115–126. https://doi.org/10.1016/j.phrs.2019.02.024
96. Pferschy-Wenzig, E. M., & Bauer, R. (2015). "The relevance of pharmacognosy in the context of gut microbiota research." Phytochemistry Reviews, 14(6), 935–951. https://doi.org/10.1007/s11101-015-9429-9
97. Mirzayi, C., Renson, A., Zohra, F., Elsafoury, S., Geistlinger, L., Kasselman, L. J., ... & Waldron, L. (2021). "Reporting guidelines for human microbiome research: The STORMS checklist." Nature Medicine, 27(11), 1885–1892. https://doi.org/10.1038/s41591-021-01552-x
98. An, X., Bao, Q., Di, S., Zhao, Y., Zhao, S., Zhang, H., ... & Zhang, B. (2019). "The interaction between the gut microbiota and herbal medicines." Biomedicine & Pharmacotherapy, 118, 109252. https://doi.org/10.1016/j.biopha.2019.109252
99. Selma, M. V., Espín, J. C., & Tomás-Barberán, F. A. (2009). "Interaction between phenolics and gut microbiota: Role in human health." Journal of Agricultural and Food Chemistry, 57(15), 6485–6501. https://doi.org/10.1021/jf902107d
100. Jordan, S. A., Cunningham, D. G., & Marles, R. J. (2010). "Assessment of herbal medicinal products: Challenges, and opportunities to increase the knowledge base for safety assessment." Toxicology and Applied Pharmacology, 243(2), 198–216. https://doi.org/10.1016/j.taap.2009.12.005
101. Asher, G. N., Corbett, A. H., & Hawke, R. L. (2017). "Common herbal dietary supplement–drug interactions." American Family Physician, 96(2), 101–107.

https://www.aafp.org/afp/2017/0715/afp20170715p101.pdf

102. U.S. Food and Drug Administration (FDA). (2023). Label Claims for Conventional Foods and Dietary Supplements. https://www.fda.gov/food/nutrition-food-labeling-and-critical-foods/label-claims-conventional-foods-and-dietary-supplements
103. Thakkar, S., Anklam, E., Xu, A., Ulberth, F., Li, J., Li, B., ... & Belcher, G. (2020). "Regulatory landscape of dietary supplements and herbal medicines from a global perspective." Regulatory Toxicology and Pharmacology, 114, 104733. https://doi.org/10.1016/j.yrtph.2020.104733
104. European Food Safety Authority (EFSA), Panel on Nutrition, Novel Foods and Food Allergens (NDA). (2021). "Safety of pasteurised Akkermansia muciniphila as a novel food pursuant to Regulation (EU) 2015/2283." EFSA Journal, 19(9), e06780. https://doi.org/10.2903/j.efsa.2021.6780
105. Commission Implementing Regulation (EU) 2022/168. (2022). "Authorising the placing on the market of pasteurised Akkermansia muciniphila as a novel food under Regulation (EU) 2015/2283." Official Journal of the European Union, L 33, 9–11. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:32022R0168
106. Shoba, G., Joy, D., Joseph, T., Majeed, M., Rajendran, R., & Sridhar, P. R. (1998). "Influence of piperine on the pharmacokinetics of curcumin in animals and human volunteers." Planta Medica, 64(4), 353–356. https://doi.org/10.1055/s-2006-957450
107. Atal, C. K., Dubey, S. K., & Singh, J. (1985). "Biochemical basis of enhanced drug bioavailability by piperine: Evidence that piperine is a potent inhibitor of drug metabolism." Journal of Pharmacology and Experimental Therapeutics, 232(1), 258–262. https://pubmed.ncbi.nlm.nih.gov/3964179/
108. World Health Organization (WHO). (2019). WHO Global Report on Traditional and Complementary Medicine 2019. https://www.who.int/publications/i/item/9789241515436
109. Govindaraghavan, S., & Sucher, N. J. (2015). "Quality assessment of medicinal herbs and their extracts: Criteria and prerequisites for consistent safety and efficacy." Epilepsy & Behavior, 52(Pt B), 363–371. https://doi.org/10.1016/j.yebeh.2015.08.039.

HOW TO CITE: Sohail Shaikh*, Pooja Rasal, Huzaifa Patel, Parth Khandelwal, Sanabil Shaikh, Saniya Shaikh, Herbal Plants in the Management of Dysbiosis: A Review of Mechanisms, Efficacy, and Therapeutic Potential, Int. J. Sci. R. Tech., 2025, 2 (11), 410-431. https://doi.org/10.5281/zenodo.17627134