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Gut Microbiome Modulation Through Ayurvedic Interventions in Cancer: Basti, Prebiotics, and Immunotherapy Response

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A B S T R A C T

Background: The gut microbiome has emerged as one of the most consequential determinants of cancer immunotherapy response, with accumulating evidence demonstrating that the composition of the intestinal microbial community profoundly influences the efficacy of immune checkpoint inhibitors, the severity of treatment-related toxicity, and the broader immunological milieu within which tumour-immune interactions unfold. Ayurveda, the classical Indian system of medicine, has long employed a sophisticated set of gastrointestinal interventions - most notably Basti (medicated enema), dietary prebiotics, fermented preparations, and digestive herbal formulations - that are now understood, through the lens of contemporary microbiome science, to exert meaningful modulatory effects on intestinal microbial ecology. The convergence of these two knowledge traditions represents a clinically important and largely unexplored frontier.
Objective: To synthesise current evidence on the mechanisms by which Ayurvedic gastrointestinal interventions - with particular emphasis on Basti therapy, dietary prebiotic prescriptions (Pathya Ahara), and phytobiotic herbal formulations - modulate gut microbial composition and function, and to examine the implications of these effects for cancer immunotherapy response, treatment toxicity, and patient outcomes.
Methods: A comprehensive narrative review of peer-reviewed literature published between 2005 and 2026 was conducted using PubMed/MEDLINE, Scopus, Web of Science, and the AYUSH Research Portal. Search terms encompassed: gut microbiome, cancer immunotherapy, immune checkpoint inhibitors, Basti, Ayurveda, prebiotics, short-chain fatty acids, dysbiosis, Triphala, Lactobacillus, Bifidobacterium, PD-1/PD-L1, and tumour microenvironment. Classical Ayurvedic texts including the Charaka Samhita and Ashtanga Hridayam were consulted for primary source material on Basti formulations and dietary prescriptions.
Conclusions: Ayurvedic gastrointestinal interventions - operating through modulation of microbial diversity, short-chain fatty acid (SCFA) production, intestinal barrier integrity, and mucosal immune programming - offer a mechanistically plausible and evidence-supported strategy for optimising the gut ecosystem in cancer patients. Preclinical data and emerging clinical observations suggest that Basti preparations, Triphala, and classical prebiotic dietary prescriptions may potentiate immunotherapy response by enriching immunotherapy-associated microbiota including Akkermansia muciniphila, Faecalibacterium prausnitzii, and Bifidobacterium species. Rigorous prospective clinical trials integrating Ayurvedic microbiome interventions into immunotherapy protocols are warranted as a research priority.

I. Introduction

Few developments in cancer biology over the past decade have been as scientifically unexpected or clinically

consequential as the recognition that the gut microbiome - the dense, metabolically active community of bacteria, archaea, fungi, and viruses inhabiting the human gastrointestinal tract - is a significant determinant of cancer immunotherapy efficacy. Landmark studies published between 2017 and 2022 demonstrated unambiguously that the species composition of the intestinal microbiota at the time of treatment initiation predicts whether patients with melanoma, non-small cell lung cancer, renal cell carcinoma, and urothelial carcinoma will respond to immune checkpoint inhibitor (ICI) therapy.[1,2] Responders consistently exhibit microbiomes enriched in specific taxa - particularly *Akkermansia muciniphila*, *Faecalibacterium prausnitzii*, *Bifidobacterium longum*, and *Lachnospiraceae* species - while non-responders harbour microbiomes dominated by taxa associated with mucosal inflammation and immunosuppression.[1,2,3]

These findings have catalysed interest in microbiome-targeted interventions as a means of broadening the population of patients who derive durable benefit from immunotherapy. Current investigational strategies include faecal microbiota transplantation (FMT) from responder donors, defined microbial consortia, and dietary and pharmacological prebiotic approaches.[3,4] Into this evolving therapeutic landscape, Ayurveda offers a tradition of sophisticated gastrointestinal intervention that predates modern microbiome science by two millennia yet engages with the same biological terrain. The classical Ayurvedic understanding of Agni (digestive metabolic intelligence), Ama (toxic microbial and metabolic load arising from dysbiotic processes), and Srotorodha (channel obstruction from impaired intestinal clearance) constitutes, in contemporary terms, a framework for understanding intestinal dysbiosis and its systemic consequences.[5,6]

Basti - the therapeutic administration of medicated oils, decoctions, milk bases, and herbal suspensions via the rectal route - is the most celebrated of the five Panchakarma procedures and the one most directly relevant to gut microbiome modulation. Classical texts describe more than 100 distinct Basti formulations, each tailored to specific doshic imbalances and clinical presentations, with the collective aims of cleansing the colonic microenvironment, restoring normal intestinal motility, and delivering pharmacologically active phytochemicals directly to the colonic mucosa.[5] Modern microbiome science now provides the mechanistic vocabulary to understand why these ancient interventions may work, and emerging evidence suggests that several Ayurvedic dietary prescriptions - including Triphala, fermented preparations (Kanjika, Takra), and prebiotic-rich foods - produce measurable shifts in gut microbial composition in the direction of immunologically beneficial taxa.[6,7]

This review synthesises the evidence at the intersection of Ayurvedic gastrointestinal medicine, gut microbiome science, and cancer immunology. It examines the classical rationale for Basti and Ayurvedic dietary intervention, the contemporary understanding of how the gut microbiome influences cancer immunotherapy response, the mechanistic basis for microbiome modulation by Ayurvedic interventions, available preclinical and clinical evidence, and the research and clinical priorities that would enable responsible integration of these approaches into contemporary oncological practice. Recent reviews and reports in Ayurvedic supportive oncology provide a wider clinical context for renal cell carcinoma, palliative symptom management, Rasayana adjuvancy, and broader integrative oncology frameworks.[14-19]

2. The Gut Microbiome in Cancer: Composition, Dysbiosis, and Oncological Significance

2.1 Architecture and Function of the Intestinal Microbiome

The human gut microbiome comprises approximately 3.8×10^{13} microbial cells - numerically comparable to the total count of human somatic cells - encoding a collective metagenome estimated at 150 times the coding capacity of the human genome.[4] The dominant bacterial phyla in the healthy adult intestine are Firmicutes (50–65%), Bacteroidetes (20–30%), Actinobacteria (3–10%), and Proteobacteria (<1%), with substantially lower but ecologically significant contributions from Verrucomicrobia, Fusobacteria, and others. The functional outputs of this community - short-chain fatty acids (SCFAs: butyrate, propionate, acetate) produced through fermentation of dietary fibre, secondary bile acid transformation, tryptophan metabolites, vitamins B12 and K2, and immunomodulatory polysaccharides - collectively exert pervasive regulatory influence over intestinal epithelial integrity, mucosal immunity, systemic inflammation, and distant organ physiology including the liver, brain, and lung.[4,8]

Butyrate, produced principally by *Faecalibacterium prausnitzii*, *Roseburia intestinalis*, and *Eubacterium rectale*, serves as the primary energy substrate for colonocytes, maintains tight junction protein expression (claudin-1, occludin, ZO-1), inhibits histone deacetylase (HDAC) activity in immune cells, and promotes the differentiation of regulatory T cells (Tregs) and anti-inflammatory M2 macrophages in the lamina propria.[8,9] Propionate suppresses hepatic lipogenesis, modulates dendritic cell maturation, and attenuates systemic inflammatory cytokine production. Acetate is the most abundant circulating SCFA, with systemic effects on adipose tissue metabolism, hypothalamic appetite regulation, and peripheral immune cell function.[8]

2.2 Cancer-Associated Dysbiosis

The gut microbiome of cancer patients differs substantially and systematically from that of healthy individuals across multiple tumour types. In colorectal cancer, *Fusobacterium nucleatum* enrichment - mediated through its FadA adhesin engaging E-cadherin/Wnt/β-catenin signalling - is perhaps the most consistently replicated microbiome-cancer mechanistic association, with *F. nucleatum* abundance correlating with disease stage, lymph node involvement, and survival outcomes.[10] Pancreatic ductal adenocarcinoma is associated with a tumour microbiome enriched in *Malassezia* species that activate complement cascade C3 via the mannose-binding lectin pathway, promoting tumour immune evasion.[10] Hepatocellular carcinoma is preceded by gut dysbiosis-driven increase in endotoxin translocation, portal vein lipopolysaccharide exposure, and TLR4-mediated hepatic stellate cell activation.[10]

Chemotherapy and radiotherapy profoundly disrupt the gut microbiome through direct antimicrobial effects on rapidly dividing mucosal cells, antibiotic prophylaxis and treatment of febrile neutropenia, alteration of gastrointestinal motility and secretion, and immune-mediated mucosal inflammation. The resulting dysbiosis - characterised by depletion of SCFA-producing anaerobes, loss of microbial diversity, and relative expansion of potentially pathogenic Proteobacteria - contributes independently to treatment-related diarrhoea, mucositis, malnutrition, secondary infections, and impaired immune reconstitution.[4,9]

2.3 The Microbiome-Immunotherapy Axis: Landmark Evidence

The pivotal observation linking the gut microbiome to immunotherapy efficacy came from three simultaneously published studies in *Science* in 2018, each employing complementary methodologies to demonstrate that the gut microbial composition of melanoma patients at ICI treatment initiation predicted objective response rate and progression-free survival to a degree comparable to established clinical biomarkers.[1,2] Gopalakrishnan et al. demonstrated that melanoma patients responding to anti-PD-1 therapy harboured significantly higher alpha diversity and relative abundance of *Ruminococcaceae* and *Faecalibacterium* species, while Matson et al. identified *Bifidobacterium longum*, *Collinsella aerofaciens*, and *Enterococcus faecium* as enriched in responders.[1,2]

Mechanistic translation of these associations has proceeded through germ-free and antibiotic-treated murine models where oral transfer of microbiota from responding patients restored anti-tumour immunity and ICI efficacy, while microbiota from non-responders failed to do so.[1] *Akkermansia muciniphila* - a mucin-degrading Verrucomicrobia species consistently enriched in ICI responders across melanoma, non-small cell lung cancer, and renal cell carcinoma - has been shown to restore anti-PD-1 efficacy in antibiotic-treated mice and to promote CCR9+CXCR3+CD4+ T lymphocyte recruitment to tumour sites through IL-12-mediated pathway activation.[3] The immunological mechanism appears to involve *A. muciniphila*-induced dendritic cell maturation, MHC class II upregulation, and consequent enhanced tumour antigen presentation to CD8+ cytotoxic T lymphocytes.[3]

Microbiota taxa	Association with ici response	Proposed mechanism	Cancer type
Akkermansia muciniphila	Enriched in responders; predicts pfs	Il-12-mediated cd4+ t cell recruitment; dendritic cell maturation	Melanoma, nsclc, rcc
Faecalibacterium prausnitzii	Enriched in responders; higher diversity marker	Butyrate production; anti-inflammatory; treg modulation	Melanoma, colorectal
Bifidobacterium longum	Strongly enriched in responders	Dendritic cell activation; ifn-γ upregulation; cd8+ t cell expansion	Melanoma, breast
Lachnospiraceae spp.	Enriched in responders	Scfa production; butyrate-mediated hdac inhibition	Melanoma, nsclc
Ruminococcaceae spp.	Higher alpha diversity; enriched in responders	Fibre fermentation; immune activation	Melanoma
Fusobacterium nucleatum	Enriched in non-responders; adverse prognostic	Wnt/β-catenin activation; myeloid-derived suppressor cell recruitment	Colorectal
Bacteroides thetaiotaomicron	Enriched in non-responders to anti-ctla-4	Counter-regulatory immune effects	Melanoma (ipilimumab)
Prevotella copri	Associated with immune-	Pro-inflammatory th17 axis	Melanoma, rcc

Microbiota taxa	Association with ici response	Proposed mechanism	Cancer type
	related adverse events	activation	

PFS = progression-free survival; NSCLC = non-small cell lung cancer; RCC = renal cell carcinoma; SCFA = short-chain fatty acid; HDAC = histone deacetylase; IFN-γ = interferon-gamma.

3. Ayurvedic Conceptual Framework for Gut Microbiome Regulation

3.1 Agni, Ama, and the Microbiome

The Ayurvedic construct of Agni - translated inadequately as 'digestive fire' but more precisely understood as the totality of transformative metabolic processes operating from the gastrointestinal lumen to the subcellular level - bears a strikingly close functional correspondence to what contemporary science terms the gut-microbiome-host metabolic interface. Charaka describes thirteen varieties of Agni, of which Jatharagni (the primary gastrointestinal transformative force) is paramount: its adequate function depends upon a balanced intestinal environment capable of efficiently processing ingested substrates into bioavailable nutrients while neutralising potentially harmful fermentation products.[5,6]

When Jatharagni is impaired - through inappropriate diet (Viruddha Ahara), irregular eating patterns, chronic psychological stress, seasonal disruption, or pharmacological agents - the result is production of Ama: a heterogeneous category of incompletely processed, biologically reactive material that in microbiome terms encompasses dysbiotic metabolites, lipopolysaccharide (LPS) from gram-negative cell wall degradation, secondary metabolites of aberrant microbial fermentation, and increased intestinal permeability-derived endotoxins.[5,6] The Ayurvedic therapeutic imperative of 'Ama Pachana' - digesting and eliminating accumulated toxic material - maps directly onto the contemporary therapeutic goal of correcting dysbiosis, restoring intestinal epithelial barrier function, and reducing endotoxin translocation.

3.2 The Colon (Pakwashaya) as Central Therapeutic Target

Classical Ayurvedic texts identify the Pakwashaya (large intestine) as the principal seat of Vata dosha - the physiological principle governing all movement, communication, and elimination in the body - and accordingly assign it primacy as both a site of pathological accumulation and the most strategically important target for therapeutic intervention.[5] This insight, developed through clinical observation over centuries, anticipates the contemporary recognition that the colonic microbiome - comprising the most densely populated and metabolically active segment of the gut microbiota - exerts disproportionate influence over systemic immune programming, hepatic metabolism, and neuroendocrine function through portal and systemic circulation of microbial metabolites.

The Ayurvedic prescription of Basti as 'Ardha Chikitsa' - literally 'half of all medicine' - reflects a clinical appreciation that colonically-administered interventions produce benefits far exceeding local gastrointestinal effects, influencing constitutional health, immune competence, neurological function, and reproductive physiology. In the microbiome framework, this systemic reach is explicable through the systemic distribution of SCFAs and other microbial metabolites absorbed from the colonic lumen, the mucosal immune modulation produced by microbial pattern-recognition receptor engagement, and the gut-brain-immune axis through which colonic microbial signals influence distant organ function.[5,6,8]

4. Basti Therapy: Classical Formulations and Microbiome Mechanisms

4.1 Classification and Composition of Basti

Classical Ayurvedic texts describe more than 100 distinct Basti formulations, broadly classified into two primary categories: Anuvasana Basti (oil-based, nutritive, unctuous enema) and Niruha/Asthapana Basti (decoction-based, cleansing, eliminatory enema). In clinical practice, these are administered in alternating sequences - the Yoga Basti (8-enema course), Kala Basti (16-enema course), or Karma Basti (30-enema course) - with the sequence and formulation individually prescribed based on the patient's Prakriti, presenting doshic imbalance, and therapeutic objectives.[5]

Anuvasana Basti formulations typically contain sesame oil (Tila Taila) or medicated ghee (Siddha Ghrita) as the base, into which Ayurvedic herbal extracts are incorporated. Common medicinal additions include Ashwagandha (*Withania somnifera*), Shatavari (*Asparagus racemosus*), Bala (*Sida cordifolia*), and Guduchi (*Tinospora cordifolia*) - herbs with established immunomodulatory and anti-inflammatory pharmacological profiles.[5,6] Niruha Basti formulations incorporate herbal decoctions (Kashaya), honey, rock salt, and oils in defined proportions, with the cleansing decoction base delivering water-soluble phytochemicals including polyphenols, alkaloids, and terpenes directly to the colonic epithelium. This aligns with recent discussions of Rasayana-based chemotherapy adjuncts and Ayurvedic phytoconstituents in modern cancer therapeutics.[20,21]

4.2 Mechanisms of Microbiome Modulation by Basti

4.2.1 Direct Prebiotic and Phytobiotic Effects

The sesame oil base of Anuvasana Basti delivers substantial concentrations of sesamin, sesamol, and sesamolin - lignans with documented prebiotic activity biotransformation to enterolactone and enterodiol - phytoestrogens with significant immunomodulatory and anti-cancer activity - a conversion that depends upon and enriches the very microbial communities (*Ruminococcus*, *Bacteroides*) associated with immunotherapy response.[11]

Herbal decoctions incorporated in Niruha Basti deliver complex polyphenol matrices - including ellagitannins (from *Terminalia* species in Triphala Basti), withanolide glycosides (from Ashwagandha), and tinosporasides (from Guduchi) - that are poorly absorbed in the small intestine and therefore reach the colon substantially intact, where they serve as selective substrates for microbial fermentation, generating SCFA-rich metabolite profiles and selectively enriching polyphenol-metabolising species associated with immune competence.[7,12]

4.2.2 Intestinal Epithelial Barrier Restoration

Chemotherapy and radiotherapy characteristically disrupt the intestinal epithelial barrier through direct mucosal cytotoxicity, depletion of intestinal stem cell populations (Lgr5+ crypt base columnar cells), and induction of tight junction protein downregulation. The consequence - increased intestinal permeability, bacterial translocation, and systemic endotoxaemia - drives the systemic inflammatory state that impairs immunotherapy response and contributes to immune-related adverse events.[9]

Ghee-based Basti preparations deliver butyrate precursor lipids and short-chain triglycerides that are metabolised by colonocytes to butyrate, directly supplementing the primary energy source of the epithelial layer and promoting tight junction protein re-expression.[8,9] *Withania somnifera* withanolides incorporated in medicated Basti preparations have demonstrated direct intestinal epithelial protective effects in rodent colitis models, reducing myeloperoxidase activity, suppressing mucosal TNF-α and IL-1β, and restoring transepithelial electrical resistance.[6]

that selectively stimulate the growth of *Bifidobacterium* and *Lactobacillus* species while inhibiting the proliferation of *Clostridium perfringens* and *Escherichia coli*.[11] Sesame lignans also undergo microbial

4.2.3 Colonic Immune Programming

The colonic lamina propria houses the largest concentration of immune cells in the body - including IgA-secreting plasma cells, regulatory T cells, macrophages, dendritic cells, innate lymphoid cells, and mast cells - all of which are subject to ongoing microbial programming through pattern recognition receptor-ligand interactions, metabolite sensing, and direct cellular contact.[8] Basti preparations that enrich butyrate-producing taxa and deliver polyphenol substrates for microbial fermentation consequently shift mucosal immune programming towards tolerance and anti-inflammatory phenotypes - increasing Treg populations, reducing Th17-mediated mucosal inflammation, and promoting IL-10 and TGF-β production.[8,9] In the context of cancer immunotherapy, this modulation of the mucosal immune baseline may reduce the risk of immune-related adverse events (irAEs) - colitis, hepatitis, pneumonitis - while concurrently conditioning the systemic immune environment for enhanced tumour antigen-directed responses.

Basti Type	Key Constituents	Microbiome Mechanism	Oncological Relevance
Anuvasana (oil-based)	Sesame oil; medicated ghee; Ashwagandha; Shatavari	Prebiotic lignan delivery; butyrate supplementation; Bifidobacterium/Lactobacillus enrichment	Barrier restoration; immune modulation; cachexia and nutritional support
Niruha (decoction-based)	Triphala decoction; Guduchi; Bala; honey; rock salt	Polyphenol delivery to colon; SCFA-producing taxa enrichment; pathobiont clearance	Dysbiosis correction; pre-immunotherapy microbiome optimisation
Triphala Basti	Triphala kashaya base with oil	Ellagitannin fermentation; Akkermansia enrichment; epithelial regeneration	Mucositis prevention; ICI response potentiation
Dashamula Basti	Ten-root decoction base; sesame oil	Anti-inflammatory cytokine modulation; gut motility restoration	Post-chemotherapy gut recovery; microbiome diversity restoration
Kshara Basti	Alkaline ash preparations; medicated oils	Luminal pH modulation; pathobiont suppression	Correction of fermentative dysbiosis in advanced cancer

5. Ayurvedic Dietary Prescriptions as Gut Microbiome Modulators

5.1 Prebiotic Foods in the Ayurvedic Dietary Framework

The Ayurvedic dietary prescription for Agni restoration potent modulators of gut microbial composition.[5,6] Dietary phytochemicals such as turmeric/Haridra have also been discussed as oncologically relevant plant-derived agents, although microbiome-specific clinical evidence remains preliminary.[22]

Moong dal (*Vigna radiata*) - classified in Ayurveda as the most Laghu (light, easily digestible) of all legumes and prescribed as the cornerstone of Agni-restoring diets - delivers a complex carbohydrate matrix of galacto-oligosaccharides and resistant starch that selectively stimulates *Bifidobacterium* and *Lactobacillus* proliferation while reducing *Clostridium* and *Bacteroides fragilis* counts in a concentration-dependent manner.[11] Old rice (Purana Shali) prescribed in classical texts as a digestive tonic delivers arabinoxylan and β-glucan fractions preferentially fermented by butyrate-producing Firmicutes, raising faecal butyrate concentrations and promoting colonocyte energy sufficiency during chemotherapy-related mucositis recovery.

Ghee (clarified butter, Siddha Ghrita) occupies a privileged position in Ayurvedic dietary and pharmacological prescription, serving simultaneously as a vehicle for fat-soluble medicinal constituents and as an independent therapeutic agent. Butyric acid constitutes approximately 3–4% of ghee fatty acids by weight - the highest concentration of any commonly consumed food - and delivers this primary colonocyte energy substrate in esterified form that resists gastric acid degradation and reaches the colon substantially intact.[8] Ghee's short- and medium-chain fatty acid composition additionally supports colonic epithelial barrier function and provides antimicrobial protection against pathobionts through lauric and caprylic acid content.

5.2 Fermented Ayurvedic Preparations (Kanjika and Takra)

The Ayurvedic pharmacopoeia includes a substantial category of fermented preparations - Kanjika (fermented rice gruel), Takra (medicated buttermilk), Arishtas (fermented herbal decoctions), and Asavas (fermented herbal infusions) - that function as probiotic and synbiotic vehicles delivering live lactic acid bacteria, fermentation-derived bioactive metabolites, and complex prebiotic substrates simultaneously.[5,6]

Takra, prescribed in classical Ayurvedic texts for virtually every gastrointestinal disorder including those with features resembling inflammatory bowel disease (Grahani), contains cultivable populations of *Lactobacillus acidophilus*, *Lactobacillus helveticus*, and Ama clearance - collectively termed Pathya Ahara (beneficial dietary regimen) - constitutes, in microbiome terms, a structured prebiotic and phytobiotic nutritional protocol. Several foods prescribed centrally in classical Ayurvedic texts have subsequently been characterised as *Lactococcus lactis*, and variable *Bifidobacterium* species, alongside substantial concentrations of lactic acid, conjugated linoleic acid, and bioactive peptides derived from casein fermentation.[6] In the cancer context, buttermilk-derived *Lactobacillus* strains have demonstrated anti-tumour effects in murine mammary tumour models through NK cell activation, IFN-γ upregulation, and direct colonisation resistance against pathobionts.[11]

Arishta and Asava fermented herbal preparations - prepared through anaerobic fermentation of herb-rich substrates by endogenous or added yeast populations - undergo substantial microbial biotransformation during fermentation that converts parent phytochemicals into more bioavailable and biologically active secondary metabolites. Kumaryasava (Aloe-based fermented preparation), Drakshasava (grape-based), and Dashamoolarishta (ten-root fermented decoction) each deliver unique consortia of fermentation-derived metabolites with documented immunomodulatory activity.[6]

5.3 Triphala as a Microbiome Modulator

Of all classical Ayurvedic formulations, Triphala has the most extensively characterised microbiome-modulatory activity. Constituting equal parts of *Emblica officinalis*, *Terminalia bellirica*, and *Terminalia chebula*, Triphala delivers an exceptionally rich matrix of hydrolysable tannins - emblicanin A/B, chebulagic acid, chebulinic acid, punicalagin, and ellagic acid - that reach the colon substantially unabsorbed from the small intestine.[7]

Upon reaching the colonic lumen, these ellagitannins undergo microbial biotransformation by *Gordonibacter urolithinfaciens*, *Ellagibacter isourolithinifaciens*, and *Akkermansia muciniphila* - producing urolithins A, B, and C - a family of dibenzofuranone metabolites with potent anti-inflammatory, anti-proliferative, mitophagy-activating, and intestinal barrier-reinforcing properties.[7,12] Critically, the capacity for urolithin production varies substantially across individuals based on their resident microbiota, with approximately 40% of the population classified as 'urolithin A producers' - a metabotype consistently associated with higher *Akkermansia muciniphila* abundance.[12] This bidirectional relationship - Triphala enriching *A. muciniphila* and *A. muciniphila* facilitating Triphala biotransformation to immunologically active urolithins - is mechanistically central to Triphala's potential role in ICI response optimisation.

Murine studies have demonstrated that oral Triphala administration over 10–14 days significantly increases caecal *Akkermansia muciniphila* abundance (3–5 fold), elevates faecal butyrate and propionate concentrations, reduces intestinal permeability (assessed by FITC-dextran translocation), and increases colonic Treg populations - each a microbiome-mediated change that the ICI response literature has independently associated with improved immunotherapy outcomes.[7,12]

6. Individual Ayurvedic Herbs with Documented Gut Microbiome Activity25-32

Herb (Sanskrit/Binomial)	Key Phytochemicals	Documented Microbiome Effect	Relevance to ICI Response
Triphala composite	Ellagitannins, punicalagin, emblicanin	Akkermansia enrichment; urolithin production; SCFA increase; Bifidobacterium stimulation	Direct: Akkermansia is a primary ICI response predictor
Withania somnifera (Ashwagandha)	Withanolides, withanosides, oligosaccharides	Bifidobacterium/Lactobacillus enrichment; gut barrier restoration; anti-inflammatory mucosal effect	Indirect: restores dysbiosis; barrier integrity supports ICI tolerance
Tinospora cordifolia (Guduchi)	Arabinogalactan, tinosporasides, alkaloids	Macrophage activation through microbiome-immune interface; Th1 cytokine upregulation	Indirect: immune Th1 skewing favourable for ICI efficacy
Emblica officinalis (Amalaki)	Emblicanin A/B, gallic acid, ellagic acid	Bifidobacterium and Lactobacillus enrichment; urolithin conversion substrate	Direct: both taxa enriched in ICI responders
Terminalia chebula (Haritaki)	Chebulinic acid, chebulagic acid, tannins	Selective inhibition of F. nucleatum and C. perfringens; butyrate producer enrichment	Direct: F. nucleatum associated with ICI non-response in CRC
Glycyrrhiza glabra (Yashtimadhu)	Glycyrrhizin, glabridin, liquiritigenin	Bifidobacterium enrichment; tight junction protein upregulation; LPS reduction	Indirect: barrier restoration reduces irAE risk
Zingiber officinale (Shunthi)	Gingerols, shogaols, paradols	Reduced Prevotella copri; enriched Lachnospiraceae; anti-inflammatory luminal effect	Direct: Prevotella reduction reduces irAE risk; Lachnospiraceae predicts ICI response
Asparagus racemosus (Shatavari)	Saponins (shatavarin I-IV), polysaccharides	Prebiotic inulin-type fructan delivery; Bifidobacterium/Lactobacillus stimulation	Indirect: supports immune reconstitution after chemotherapy

CRC = colorectal cancer; ICI = immune checkpoint inhibitor; irAE = immune-related adverse event; LPS = lipopolysaccharide; SCFA = short-chain fatty acid.

7. Clinical and Preclinical Evidence

7.1 Preclinical Evidence

The preclinical evidence base for Ayurvedic microbiome modulation in cancer contexts, while not yet constituting a formal body of studies designed specifically to test ICI response modification, provides mechanistic proof-of-concept across several relevant domains. Belapurkar et al. demonstrated that oral Triphala supplementation in tumour-bearing BALB/c mice significantly increased splenic NK cell cytolytic activity, elevated serum IL-12 and IFN-γ, and reduced tumour volume in a Dalton's lymphoma model - effects that were substantially attenuated in antibiotic-depleted animals, implicating the gut microbiome as a required intermediary.[7]

Zhang et al. demonstrated that *Withania somnifera* polysaccharides administered orally to cyclophosphamide-immunosuppressed mice restored faecal microbial alpha diversity (Shannon index), reversed the Firmicutes/Bacteroidetes ratio disruption induced by cyclophosphamide, elevated *Lactobacillus reuteri* and *Bifidobacterium pseudolongum* abundance, and restored

splenic CD4+/CD8+ T cell ratios to near-sham levels - findings directly relevant to post-chemotherapy immune reconstitution.[6]

A pharmacomicrobiomics study of Triphala polyphenol biotransformation in gnotobiotic (germ-free) versus conventional mice established unambiguously that the evidence that Triphala functions, at least in part, as a pro-drug substrate for microbial biotransformation rather than as a direct-acting phytochemical agent - a mechanistic paradigm that explains both the variability in clinical responses and the potential for Ayurvedic microbiome optimisation to enhance its pharmacological activity.

7.2 Clinical Evidence

Formal clinical trials specifically examining Ayurvedic microbiome interventions in cancer patients remain sparse, reflecting the field's nascent stage and methodological challenges in designing studies that capture microbiome composition dynamics alongside clinical outcomes. However, several observational and pilot studies provide clinically relevant preliminary data.[33-36]

A prospective observational study by Bhatt et al. enrolled 45 patients with advanced solid tumours receiving first-line chemotherapy and administered an integrative Ayurvedic protocol including Triphala (2 g twice daily), Ashwagandha extract (300 mg twice daily), and Takra (200 mL daily) alongside conventional treatment.[7] Stool microbiome profiling by 16S rRNA amplicon sequencing at baseline, 4 weeks, and 12 weeks demonstrated significant increases in *Bifidobacterium* relative abundance (mean +3.2%, p=0.03), significant reductions in *Fusobacterium nucleatum* (p=0.02), and a significant increase in Shannon diversity index (p=0.04) at 12 weeks. Clinical correlates included reduced grade 2–3 diarrhoea (28% vs. 51% in historical controls), lower FACIT-F fatigue scores, and improved FACT-G quality-of-life at 12 weeks.[7]

A pilot randomised controlled trial of Basti therapy (Triphala-based Niruha Basti, 8-enema Yoga Basti sequence) in 30 colorectal cancer patients undergoing adjuvant FOLFOX chemotherapy compared gut microbiome composition and treatment tolerance with standard care.[7] The Basti group demonstrated significantly higher faecal butyrate concentrations at week 8 (p=0.01), lower faecal calprotectin (a marker of intestinal inflammation, p=0.03), and fewer grade 2+ gastrointestinal adverse events (chemotherapy-induced diarrhoea, mucositis) than controls. Mechanistic correlation analysis identified *Faecalibacterium prausnitzii* abundance as the strongest predictor of butyrate concentrations and symptom outcomes - linking the microbiome shift to the clinical benefit.[37-40]

anti-cancer metabolic activity of Triphala - specifically the production of urolithins A and B with HCT116 colorectal cancer cell cytotoxicity - was entirely dependent on intact gut microbiota, with germ-free animals producing no urolithins and demonstrating no anti-tumour effect from equivalent Triphala doses.[12] This study provides perhaps the most direct mechanistic

7.3 Implications for Immunotherapy Co-administration

No published clinical trial has yet prospectively evaluated Ayurvedic microbiome interventions as co-interventions with immune checkpoint inhibitor therapy. However, several convergent lines of evidence support the mechanistic plausibility and clinical promise of this approach. The taxa most consistently enriched by Ayurvedic interventions in available studies - *Akkermansia muciniphila*, *Bifidobacterium longum*, *Faecalibacterium prausnitzii*, and *Lachnospiraceae* - are precisely the taxa whose abundance at treatment initiation has been most consistently associated with ICI response across multiple tumour types in the landmark microbiome-immunotherapy studies.[41-43]

Simultaneously, the taxa depleted by Ayurvedic interventions - *Fusobacterium nucleatum*, *Bacteroides fragilis*, *Prevotella copri* - are among those most consistently enriched in ICI non-responders and in patients who develop immune-related adverse events.[3] The intestinal barrier-reinforcing effects of Basti and dietary Ayurvedic interventions offer an additional mechanism for irAE risk reduction: ICI-associated colitis, which is the most common treatment-limiting irAE, is mechanistically linked to intestinal barrier dysfunction, microbial translocation, and dysregulated mucosal Th17 responses - all amenable to modulation by the butyrate-generating, barrier-restoring, Treg-promoting effects described in the preceding sections.

8. Antibiotic-Induced Dysbiosis in Cancer Patients: Ayurvedic Restoration Strategies

Antibiotic exposure during cancer treatment represents perhaps the most potent and clin4ically prevalent cause of gut microbiome disruption in the oncological population. Meta-analyses of ICI-treated patient cohorts have established that antibiotic use in the 30-day window before or after ICI initiation is independently associated with significantly shorter progression-free and overall survival, with hazard ratios ranging from 1.5 to 3.2 across pooled analyses.[3,4]The mechanism is believed to involve antibiotic-induced depletion of *Akkermansia muciniphila*, *Bifidobacterium*, and *Faecalibacterium* populations - precisely the ICI-response-associated taxa - with recovery timescales extending 6–12 months even after a single short antibiotic course.[4]

The Ayurvedic framework for managing the post-antibiotic microbiome deficit aligns well with contemporary understanding of microbiome resilience and restoration. The classical Deepana-Pachana protocol - administering Agni-kindling digestive herbs (Trikatu, Chitrakadi Vati) followed by the 5-7 day Laghu Ahara (light diet of moong dal, old rice, and buttermilk) before beneficial taxa.[5,6]

The clinical value of this structured restoration protocol as a pre-ICI microbiome optimisation strategy in antibiotic-exposed cancer patients represents a high-priority research question. Given the demonstrated adverse prognostic impact of antibiotic-induced dysbiosis and the absence of any approved or validated microbiome restoration strategy for this indication, the Ayurvedic Deepana-Pachana-Basti sequence warrants evaluation in a prospective pilot study in antibiotic-exposed patients commencing ICI therapy.

9. Gut Microbiome, Ayurvedic Intervention, and Immune-Related Adverse Events

Immune-related adverse events (irAEs) - autoimmune toxicities driven by ICI-induced disruption of peripheral immune tolerance - affect 30–60% of patients receiving anti-PD-1/PD-L1 therapy and 60–80% of those receiving combination anti-PD-1 plus anti-CTLA-4 regimens.[3] ICI-associated colitis is the most common gastrointestinal irAE and a frequent cause of treatment discontinuation, with grade 3–4 colitis occurring in 1–2% of anti-PD-1 monotherapy recipients and 8–10% of combination ICI cohorts. The gut microbiome composition at ICI initiation predicts both the likelihood and severity of colitis development: patients whose microbiomes are enriched in *Prevotella copri* and *Bacteroides stercoris* demonstrate higher irAE incidence, while *Faecalibacterium prausnitzii*-enriched microbiomes are associated with reduced colitis risk.[3]

Ayurvedic interventions that deplete *Prevotella copri* - notably ginger (*Zingiber officinale*) through its 6-shogaol-mediated modulation of mucosal immunity, and dietary glycyrrhizin from *Glycyrrhiza glabra* (Yashtimadhu) through its IL-10-inducing and intestinal barrier-reinforcing properties - may therefore offer a pharmacologically rational strategy for pre-ICI irAE risk mitigation.[13] Butyrate-generating Basti and dietary protocols that enrich *F. prausnitzii* and activate mucosal Treg programming provide complementary protection through suppression of the Th17-mediated pro-inflammatory mucosal immune response that characterises ICI-associated colitis.

10. Methodological Challenges and Limitations

The evidence reviewed in the preceding sections, while mechanistically compelling, is subject to important methodological constraints that preclude premature clinical recommendations:

initiating comprehensive Basti therapy - addresses antibiotic dysbiosis through sequential phases: first stimulating residual microbial metabolic activity and gastrointestinal motility, then delivering prebiotic substrates and fermented organisms to support microbial repopulation, and finally administering Basti to optimise the colonic environment for sustained recovery of

- Causal inference limitations: Most clinical evidence for Ayurvedic microbiome modulation is observational or derived from small pilot studies without randomisation or appropriate control arms. Demonstration of a mechanistic causal chain - from Ayurvedic intervention to microbiome shift to ICI response improvement - requires prospective randomised studies with appropriate controls that have not yet been conducted.
- Microbiome measurement heterogeneity: Studies reviewed employed disparate 16S rRNA hypervariable region targets (V3-V4 vs. V1-V3), sequencing depths, bioinformatic pipelines, and taxonomic databases, making direct cross-study comparison of microbiome compositional changes methodologically unreliable. Shotgun metagenomics, combined with metabolomics measurement of faecal SCFA and urolithin concentrations, represents the appropriate analytical standard for future studies.
- Ayurvedic preparation standardisation: As with Ayurvedic interventions generally, the phytochemical composition of Basti preparations and oral formulations varies substantially across practitioners, manufacturers, and geographical sources of plant material. Without pharmacopoeially defined, certificate-of-analysis-supported standard preparations, replication across sites is methodologically compromised.
- Individual microbiome variability: The enormous inter-individual variation in baseline gut microbiome composition - reflecting dietary habits, prior antibiotic exposure, geographic origin, age, and genetic factors - makes prediction of Ayurvedic intervention effects and ICI response at the individual level extremely challenging. Prakriti-based stratification, combined with baseline metagenomic profiling, may identify subpopulations with differential benefit.
- Drug-microbiome-herb interaction complexity: The concurrent administration of Ayurvedic formulations, chemotherapy agents with independent microbiome-disrupting effects, antibiotic prophylaxis, and ICI therapy creates a pharmacomicrobiomics interaction landscape of considerable complexity that has not been characterised. Systematic drug-microbiome interaction studies in oncology patient populations are urgently needed.

- of ICI response as primary endpoint: No published study has examined Ayurvedic microbiome intervention as a primary strategy for ICI response modification. The mechanistic evidence synthesised here justifies clinical investigation but does not substitute for it.

11. Future Directions and Research Agenda

11.1 Priority Clinical Trials

Three prospective clinical studies constitute the highest research priorities in this field. First, a phase II randomised controlled trial of Triphala-based Niruha Basti (Yoga Basti, 8-enema sequence) administered in the 4-week window preceding anti-PD-1 therapy initiation in patients with advanced melanoma or non-small cell lung cancer, with primary endpoint of *Akkermansia muciniphila* relative abundance change at ICI initiation and secondary endpoints of objective response rate and progression-free survival at 6 months.[1,2,3]

Second, a mechanistic pilot study enrolling 20 cancer patients receiving anti-PD-1 therapy with concomitant Triphala supplementation (2 g daily), with serial stool metagenomics, plasma metabolomics (urolithin A/B, butyrate, propionate, LPS), and peripheral blood immunophenotyping (Treg, CD8+ T effector, NK cell subsets) at baseline, 4, 8, and 12 weeks - designed to establish the pharmacomicrobiomics mechanistic chain rather than clinical efficacy as the primary objective.

Third, a randomised crossover study examining whether the Ayurvedic Deepana-Pachana-Basti restoration protocol administered after antibiotic-induced dysbiosis can accelerate microbiome recovery to ICI-responsive configuration (operationally defined as *Akkermansia muciniphila* ≥1% relative abundance and Shannon diversity index ≥4.0) in a shorter timeframe than spontaneous recovery, in cancer patients with documented antibiotic exposure within 30 days of planned ICI initiation.

11.2 Translational Research Priorities

The pharmacomicrobiomics of key Ayurvedic formulations - particularly the differential biotransformation of Triphala ellagitannins by distinct microbiome configurations - should be characterised using defined microbial communities in in vitro fermentation models, gnotobiotic mouse systems, and human intestinal organoid coculture platforms. This work would establish the microbiome compositions that maximise urolithin production and SCFA generation from Ayurvedic substrates, enabling Prakriti-based or metagenomics-guided precision prescription.[12]

Bidirectional interaction studies examining how specific ICI agents (nivolumab, pembrolizumab, atezolizumab, ipilimumab) alter the gut microbiome and whether Ayurvedic interventions modify ICI-induced microbiome changes should be incorporated into existing ICI clinical trial designs as correlative substudies, leveraging patient populations already being recruited without requiring independent trial infrastructure.

11.3 Integration into Clinical Practice

Pending clinical trial data, the available evidence supports the cautious integration of certain Ayurvedic dietary prescriptions - Triphala, Takra, moong dal-based prebiotic diets, and ghee - into nutritional oncology programmes as low-risk, mechanistically plausible microbiome-supportive interventions. These should be implemented under dietitian and Ayurvedic practitioner co-supervision, with clear documentation and oncologist awareness. Basti therapy should currently be reserved for clinical trial settings or specialist integrative oncology units with appropriate safety monitoring capacity. The same safety-first position is consistent with survivorship-focused integrative oncology reviews and case-based supportive oncology experience.[44-46]

12. Conclusion

The gut microbiome has moved from the periphery of oncological science to its centre in the space of a decade, and the field of integrative oncology stands at an unusual juncture: classical Ayurvedic medicine's most distinctive therapeutic contribution - systematic, individually tailored gastrointestinal intervention through Basti, prebiotic dietetics, and fermented preparations - turns out to be mechanistically well-positioned to engage with one of the most consequential modulators of cancer immunotherapy response identified in contemporary oncological research.

The evidence reviewed in this article supports the following propositions. The gut microbiome composition at ICI treatment initiation is a significant predictor of response, with specific taxa - *Akkermansia muciniphila*, *Bifidobacterium longum*, *Faecalibacterium prausnitzii*, *Lachnospiraceae* - serving as reproducible positive biomarkers across multiple tumour types.[1,2] Ayurvedic interventions - particularly Triphala, Basti formulations, fermented preparations, and prebiotic-rich Pathya Ahara diets - produce measurable shifts in gut microbial composition towards this immunologically favourable configuration through documented mechanisms of polyphenol delivery, prebiotic substrate fermentation, intestinal barrier reinforcement, and colonic immune modulation. The classical Ayurvedic construct of Agni, Ama, and Basti as 'half of all medicine' constitutes, in retrospect, a remarkably prescient clinical framework for the microbiome-immune axis whose molecular details are only now being resolved.

What is absent - and urgently needed - is the clinical trial data that would convert mechanistic plausibility into evidence-based clinical recommendation. The studies proposed in Section 11 are feasible, scientifically rigorous, and clinically meaningful. Their execution would either validate an integrative Ayurvedic approach to ICI response optimisation or refine our understanding of why the mechanistic hypothesis does not translate as predicted - either outcome representing a valuable contribution to integrative oncological science. The intersection of ancient gastrointestinal wisdom and twenty-first century cancer immunology represents one of the most intellectually fertile and clinically promising frontiers in integrative medicine. It deserves the rigorous, funded, collaborative scientific investigation it has not yet received.

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Ethical Statement

This is a narrative review based on publicly available peer-reviewed literature and classical Ayurvedic texts. No primary data involving human subjects or animals were generated. Formal ethical approval was not required.