

The Role Of Takra (Ayurvedic Buttermilk) In The Management Of Colitis: A Comprehensive Review Of Classical Foundations, Modern Science, And Clinical Integration

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Abstract

Background: Colitis, particularly Ulcerative Colitis (UC), presents a significant therapeutic challenge in modern gastroenterology due to its chronic, relapsing nature and the limitations of conventional pharmacotherapy. Ayurveda, the traditional Indian system of medicine, identifies analogous conditions (*Grahani, Pravahika*) and offers *Takra* (medicated buttermilk) as a foundational dietary-pharmaceutical intervention. Despite its profound historical significance, a comprehensive, critical synthesis of its role in colitis management is absent.

Objective: To perform a systematic review evaluating *Takra* from three integrated perspectives: 1) Its classical Ayurvedic description and rationale, 2) Its potential biomolecular mechanisms of action, and 3) The totality of available preclinical and clinical evidence for efficacy and safety in colitis.

Methods: A systematic literature search was conducted up to December 2023 across PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, AYUSH Research Portal, and DHARA. Grey literature and classical Sanskrit source texts (*Charaka Samhita, Sushruta Samhita, Ashtanga Hridaya*, and related commentaries) were also searched. Keywords included permutations of "Takra," "Ayurvedic buttermilk," "colitis," "inflammatory bowel disease," "gut microbiome," "probiotic," and "Ayurvedic diet therapy." Studies were selected based on predefined inclusion/exclusion criteria focusing on preparation, properties, mechanisms, or clinical outcomes related to colitis.

Results: The review identifies *Takra* as a multi-parametric intervention. Classically, it is characterized by *Laghu* (light), *Ushna* (hot potency), *Ruksha* (dry) *Gunas*, and *Deepana* (appetite stimulant), *Pachana* (digestive), *Grahi* (absorbent), and *Srotoshodhana* (channel-clearing) *Karmas*. These properties directly counter the Ayurvedic pathogenesis of colitis (*Mandagni, Ama, Dosha* vitiation). Modern analysis reveals *Takra* as a source of probiotics (e.g., *Lactobacillus* spp.), short-chain fatty acids (SCFAs like butyrate), bioactive peptides, and anti-inflammatory phytochemicals from adjuvants (e.g., pomegranate, ginger). These components collectively target key IBD pathways: dysbiosis, impaired barrier function, and mucosal immunity. Preclinical studies in rodent colitis models show consistent reduction in disease activity, histopathological injury, and pro-inflammatory cytokines (TNF- α , IL-6, IL-1 β). Preliminary human studies (n=5, including 2 RCTs) report significant improvements in clinical scores (UCDAI, Mayo score), stool frequency, rectal bleeding, and endoscopic findings when *Takra* is used as an adjunct or in integrative protocols.

Conclusion: *Takra* embodies a sophisticated, holistic intervention for colitis, with rational foundations in both traditional Ayurvedic pharmacology and contemporary gastrointestinal science. Evidence, though promising, is primarily derived from small-scale and open-label studies. Its future as an evidence-based therapy hinges on addressing critical gaps: standardization of preparation, execution of large-scale, robust RCTs with mechanistic sub-studies, and clear safety/contraindication profiling. *Takra* holds significant potential as a safe, cost-effective, and multi-targeted adjuvant in the integrative management of colitis.

Keywords: Takra; Buttermilk; Ayurveda; Ulcerative Colitis; Inflammatory Bowel Disease; Grahani; Probiotic; Gut Microbiome; SCFA; Integrative Medicine; Pathya Ahara.

1. Introduction

Inflammatory Bowel Disease (IBD), comprising Ulcerative Colitis (UC) and Crohn's Disease (CD), represents a paradigm of chronic, immune-mediated intestinal inflammation with a steadily increasing global incidence, posing substantial morbidity and economic burden [1,2]. The contemporary therapeutic armamentarium—including 5-aminosalicylates, corticosteroids, immunomodulators, and biologic agents—aims to induce and maintain clinical remission [3]. However, this approach is frequently hampered by issues of primary non-response, loss of response over time, significant adverse effects, and high financial cost, underscoring an unmet need for complementary strategies that address underlying pathophysiology and improve long-term outcomes [4,5].

This landscape has catalyzed a resurgence of interest in traditional medical systems, notably Ayurveda, which offers a distinct etiopathological framework and therapeutic repertoire for chronic digestive disorders. Ayurveda conceptualizes

health as a state of equilibrium among bio-energies (*Doshas: Vata, Pitta, Kapha*), metabolic fire (*Agni*), tissues (*Dhatus*), and waste products (*Malas*) [6]. Diseases resembling colitis are primarily classified under *Grahani Roga* (a disorder of the seat of *Agni*, characterized by variable bowel habits and malabsorption) and *Pravahika* (characterized by tenesmus and mucous diarrhea) [7]. The central pathological sequence involves the impairment of *Jatharagni* (digestive fire), leading to the generation of *Ama* (undigested, toxic metabolic residue). This *Ama*, combined with vitiated *Doshas* (especially *Vata* and *Pitta*), disrupts the integrity of the *Purishavaha Srotas* (channels responsible for fecal matter), manifesting as inflammation, ulceration, and altered bowel habits [8].

Within this paradigm, diet (*Ahara*) is not merely sustenance but the primary medicine (*Pathya*). *Takra*, the meticulously prepared medicated buttermilk, occupies a preeminent position in Ayurvedic dietetics and gastroenterology. Extolled in classical texts as "*Sarva Roga Nashanam*" (alleviator of all diseases) in specific contexts, it is specifically indicated for *Grahani* and *Atisara* (diarrhea) [9,10]. Its therapeutic action is attributed not to a single compound but to a synergy of properties (*Guna*), taste (*Rasa*), post-digestive effect (*Vipaka*), and potency (*Virya*), which collectively restore digestive function, eliminate toxins, and normalize bowel ecology.

From a modern scientific vantage point, the rationale for *Takra* is increasingly plausible. The pathophysiology of IBD is now recognized to involve a triad of factors: a dysregulated immune response, a compromised intestinal epithelial barrier, and a dysbiotic gut microbiome [11]. Intriguingly, fermented foods like *Takra* are rich sources of live microorganisms (potential probiotics), fermentation-derived metabolites (e.g., SCFAs), and bioactive compounds that can positively influence all three pillars [12,13]. Preliminary research suggests specific benefits of buttermilk and its components in gut health, providing a mechanistic bridge to its traditional claims [14].

Despite this compelling convergence of traditional wisdom and modern science, the evidence for *Takra* in colitis remains fragmented. Previous reviews have focused narrowly on either its classical aspects or general probiotic benefits, lacking a critical, integrative synthesis. This comprehensive review, therefore, aims to:

2. The Classical Paradigm: *Takra* in Ayurvedic Pharmacology

2.1. Definition, Preparation, and Classification

Takra is pharmacokinetically defined within Ayurveda as the aqueous supernatant fraction (*Drava Dravya*) obtained following the sequential processing (*Sanskara*) of yogurt (*Dadhi*). The standard preparation (*Siddha Vidhi*) involves a 1:1 dilution of freshly set yogurt with water, followed by sustained mechanical churning (*Mathana*) to facilitate phase separation of the lipid fraction (*Navaneeta*). The resultant liquid phase, devoid of butterfat and casein curds, constitutes authentic *Takra* [15]. This process is not merely physical but is considered a pharmaceutical transformation that fundamentally alters the *Guna* (inherent properties) of the starting material, shifting it from *Guru-Snigdha* (heavy and unctuous) to *Laghu-Ruksha* (light and dry).

The *Charaka Samhita* and *Ashtanga Hridaya* provide a nuanced classification system based on the degree of churning and component fractionation, which directly dictates therapeutic application [16, 17].

Table 1: Classical Classification of *Takra* and Its Therapeutic Indications

Type	Preparation Method	Key Properties (<i>Guna</i>)	Primary Therapeutic Indication in Colitis
Mathita (Siddha Takra)	Complete churning; maximal butterfat removal.	Balanced; <i>Laghu, Ruksha, Ushna</i>	General <i>Grahani</i> ; chronic, stable colitis with variable stool consistency and mild <i>Ama</i> .
Ghola (Kataka)	Partial churning; retains measurable butterfat.	<i>Guru</i> (heavy), <i>Snigdha</i> (unctuous), <i>Brimhana</i> (nourishing)	Severe debility, cachexia, or <i>Vata</i> -predominant colitis with marked weight loss and intolerance to dryness.
Udasvita/Udhasrita	Supernatant of settled <i>Mathita</i> ; minimal to no particulate matter.	<i>Ati-Laghu</i> (very light), <i>Ati-Ruksha</i> (very dry), subtle <i>Sheeta</i> (cooling)	Acute <i>Pitta</i> vitiation; active inflammation with bleeding (<i>Rakta-Atisara</i>), tenesmus, and burning sensation.

Critical pharmacognostic variables noted in classical texts that influence the final *Prabhava* (therapeutic efficacy) include: the source and quality of milk (*Ksheera*), the specific bacterial culture used for curdling (*Dadhi Jnanartha*), the vessel of churning (earthenware preferred for its *Sheeta* property), and the water quality.

2.2. Pharmacodynamic Properties (*Guna-Karma-Vijnana*)

The therapeutic profile of *Takra* is an emergent property of its unique *Panchabhautika* composition (the elemental balance of earth, water, fire, air, and ether), decoded through the framework of *Rasa-Guna-Virya-Vipaka-Prabhava*.

1. **Rasa (Taste):** Primarily *Amla* (sour), with an undercurrent of *Katu* (pungent) [18]. The *Amla Rasa* stimulates *Agni* and initiates digestion in the oral and gastric phases, while the *Katu Anurasa* contributes to its penetrating quality.
2. **Guna (Physical Qualities):** The defining *Gunas* are *Laghu* (lightness), *Ruksha* (dryness), and *Ushna* (heat) [19]. The synergy of *Laghu* and *Ruksha* confers high digestibility and a moisture-absorbing capacity, crucial for countering *Kapha*-mediated mucous (*Shleshma*) and edema in the intestines.
3. **Virya (Potency):** *Ushna* (heating). This is the driving force behind its *Deepana* (catalytic) action on *Jatharagni* (the core digestive fire).
4. **Vipaka (Post-Digestive Effect):** *Katu* (pungent) [20]. This post-digestive transformation is critical for its *Srotoshodhana* action. The *Katu Vipaka* possesses a "scraping" (*Lekhana*) quality that helps dislodge and clear *Ama* (metabolic toxins) and vitiated *Doshas* adhering to the *Srotas* (micro-channels).
5. **Prabhava (Unique Pharmacological Effect):** *Grahi* (absorbent/astringent). This is the specific, non-derivable effect of *Takra* that makes it a sovereign remedy (*Mahaushadha*) for diarrheal syndromes. It promotes fecal consolidation without inducing pathological constipation fig:1.

These inherent properties manifest as specific therapeutic actions (Karma):

- a. **Deepana & Pachana:** The *Ushna Virya* and *Amla Rasa* synergistically act as a catalytic igniter for diminished *Agni*, enhancing enzymatic and metabolic throughput. This promotes the complete digestion of food and pre-existing *Ama*, addressing the *Moola* (root cause) of *Grahani*.
- b. **Grahi:** Mediated by its *Ruksha Guna* and secondary *Kashaya* (astringent) property, it promotes reabsorption of excess intestinal water and increases stool consistency.
- c. **Srotoshodhana:** Primarily affecting the *Purishavaha* (colon) and *Annavaha* (small intestine) *Srotas*. The *Katu Vipaka* and *Ruksha Guna* clear the luminal adherent biofilm of *Ama* and pathogens, restoring patency and optimal flow (*Srotogati*).
- d. **Vata-Anulomana:** It specifically pacifies *Apana Vata*, the sub-dosha governing downward movement and elimination. This normalizes peristalsis, relieves abdominal distension (*Adhmana*), and eases pain (*Shula*).
- e. **Pitta-Shamaka:** Particularly in its *Udasvita* form, its *Laghu*, *Ruksha*, and subtle *Sheeta* properties help absorb and neutralize excess *Pitta* in the *Pakvashaya* (colon), reducing inflammation and burning sensation **Figure:1**.

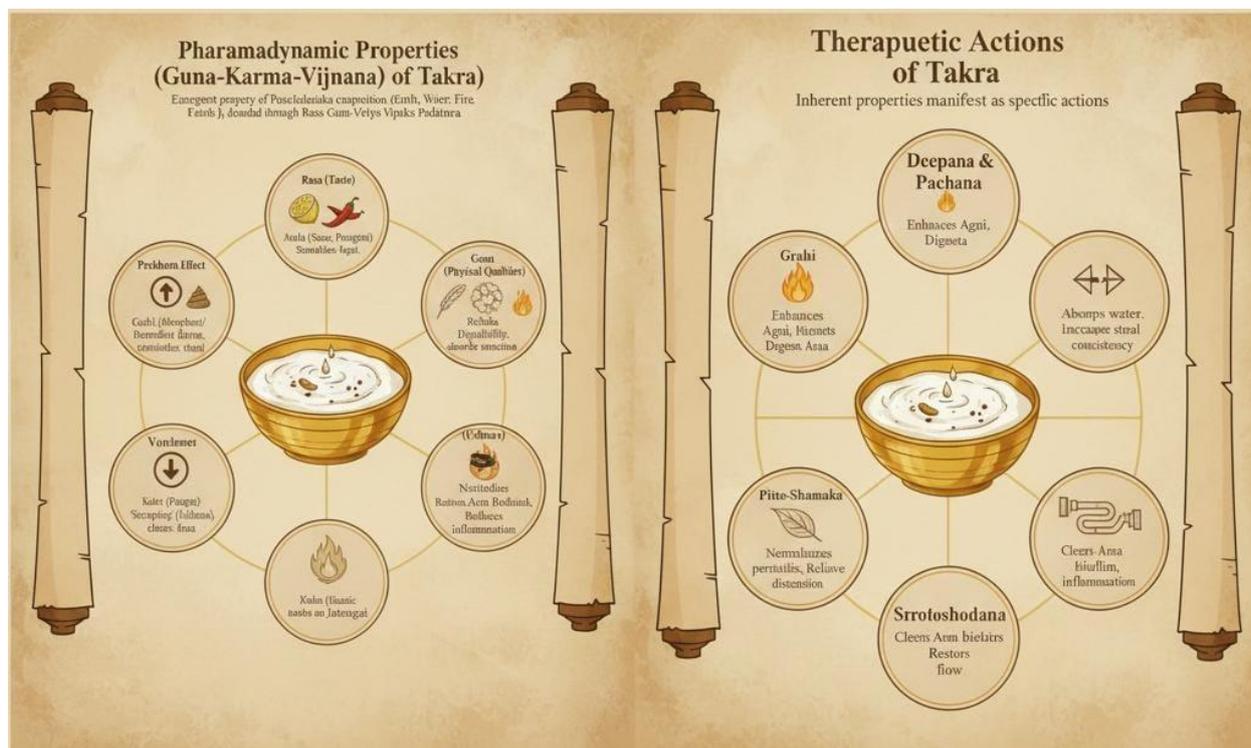


Figure:1

2.3. Medicated Takra (Samskarita Takra) and Personalized Application

The doctrine of *Yukti* (pharmaceutical ingenuity) in Ayurveda emphasizes tailoring the base drug to the patient's *Prakriti* (constitution) and *Vikriti* (disease state). *Takra* serves as an ideal vehicle (*Anupana*) for this purpose through *Bhavana* (trituration/saturation) with specific medicinal substances.

- For Pitta-Vata Colitis (Rakta-Atisara): Dadima Takra.** Pomegranate (*Punica granatum*) rind, rich in *Kashaya Rasa* and *Sheeta Virya*, is incorporated. This enhances the *Grahi* (hemostatic) and cooling effects, making it ideal for acute flares with bleeding and tenesmus [21].
- For Vata-Dominant Colitis (Adhmana-Pradhana): Saindhava Takra or Hingu Takra.** Rock salt (*Saindhava Lavana*), due to its *Sukshma* (subtle) and *Vata-Anulomana* properties, or asafoetida (*Hingu*), a potent *Vata*-pacifying and carminative agent, are added to relieve colicky pain and distension.
- For Severe Ama and Kapha (Bahusleshma): Panchakola Takra or Shunthi Takra.** The *Panchakola* group of spices (e.g., *Pippali*, *Pippalimula*, *Chavya*, *Chitraka*, *Shunthi*) or dry ginger (*Shunthi*) alone are added. Their intense *Ushna Virya* and *Katu Rasa* provide powerful *Ama-pachana* and *Kapha-Vatahara* actions, digesting accumulated toxins and reducing mucous secretion [22].
- For General Debility and Healing (Dhatu Kshaya): Ghrita Takra or Ksheera Takra.** A small amount of medicated ghee (*Ghrita*) or milk (*Ksheera*) is blended in. This moderates the *Ruksha* quality of *Takra*, providing necessary nourishment (*Brimhana*) to the depleted tissues (*Dhatu*s) while maintaining digestibility, useful in the convalescent phase.

2.4. Indications and Contraindications (Yogya-Ayogya)

The primary classical domain of *Takra* lies in disorders of assimilation and elimination. Authoritative indications include: all subtypes of *Grahani Roga*, *Atisara* (diarrhea—especially *Ama*, *Pitta*, and *Rakta* types), chronic *Arsha* (hemorrhoids), *Pandu* (anemia and malabsorption syndromes), and *Shotha* (inflammatory edema) of abdominal origin [23].

Equally critical are its contraindications (*Ayogya* or *Asatmya*), which exemplify the principle of context-specific therapy. These include: *Jwara* (acute febrile illness), where *Laghu* diet is indicated but *Ushna Virya* may be detrimental; *Sannipatika* conditions (vitiations of all three *Doshas*); acute *Visha* (toxicity/poisoning); states of extreme dehydration (*Ati-Rukshata*); and certain *Kaphaja* or bleeding types of *Arshas* where its *Ruksha* property could exacerbate the condition [24]. This framework mandates a careful *Rogi-Roga Pariksha* (patient and disease examination) prior to prescription fig:2.

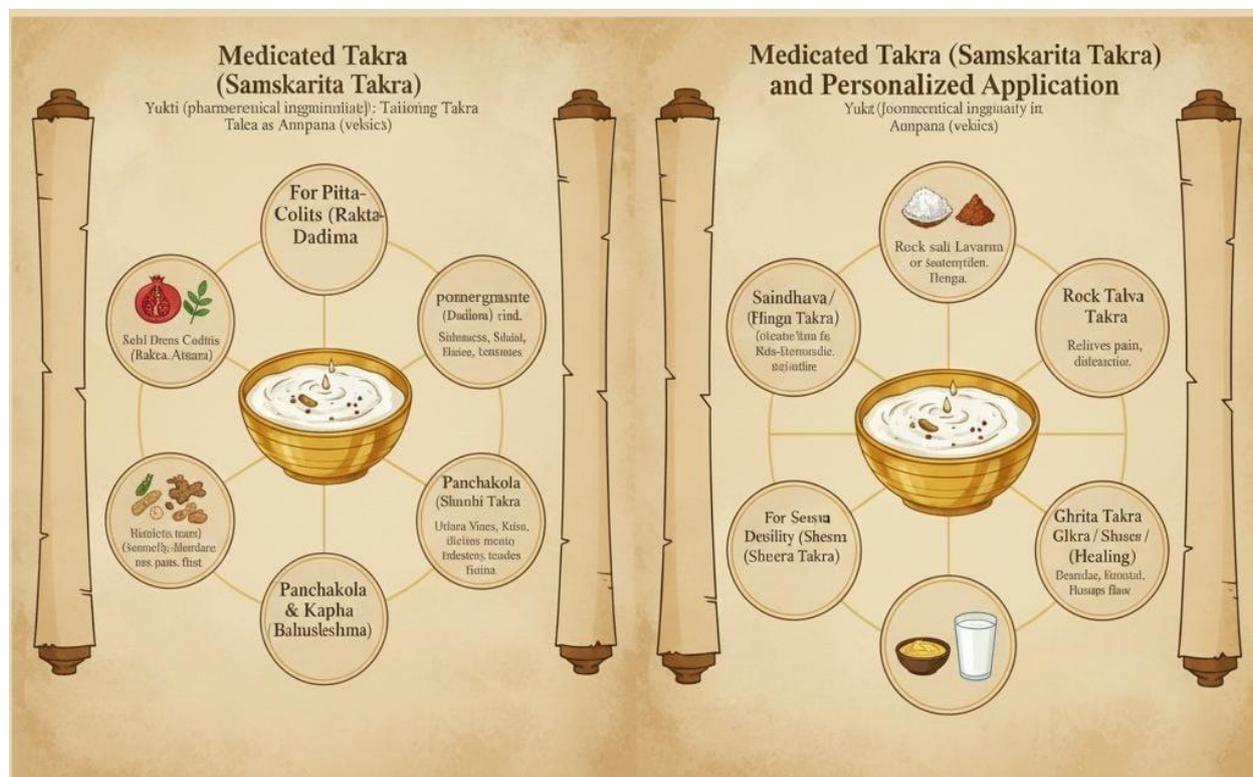


Figure:2

3. Modern Scientific Exploration: Potential Mechanisms of Action

The empirical claims of *Takra* find robust corollaries in contemporary gastrointestinal physiology, suggesting a multi-targeted, systems-level intervention.

3.1. Probiotic, Prebiotic, and Postbiotic Effects

Takra is a natural synbiotic matrix. The fermentation by starter cultures (*Lactobacillus delbrueckii* subsp. *bulgaricus*, *Streptococcus thermophilus*) and subsequent endogenous microbial activity generates a consortium of viable probiotics [25]. These confer benefits via:

- a. **Microbiome Modulation:** Probiotics competitively exclude enteropathogens, produce bacteriocins, and lower luminal pH via lactic acid production, fostering a niche for commensal species like *Bifidobacterium* [26]. This directly counters the dysbiosis—characterized by reduced diversity and depletion of Firmicutes—central to IBD pathogenesis.
- b. **Barrier Fortification:** Specific *Lactobacillus* and *Bifidobacterium* strains upregulate expression of tight junction proteins (occludin, claudin-1, ZO-1) and stimulate mucin (MUC2) production, thereby enhancing the physical and chemical barrier of the gut epithelium and reducing intestinal permeability [27].
- c. **Immunomodulation:** Through interaction with Pattern Recognition Receptors (PRRs) on gut immune cells, probiotics can skew the immune response from a pro-inflammatory Th1/Th17 profile towards a regulatory Treg phenotype, increasing anti-inflammatory IL-10 and TGF- β while dampening TNF- α , IL-6, and IL-1 β [28].
- d. **Postbiotic Generation:** The fermentation process and microbial metabolism yield soluble factors like **Short-Chain Fatty Acids (SCFAs)**—acetate, propionate, and particularly butyrate [29]. Butyrate serves as the primary oxidative fuel for colonocytes, maintains mucosal homeostasis, possesses potent anti-inflammatory properties via inhibition of NF- κ B and HDAC, and is crucial for maintaining epithelial integrity [30].

3.2. Anti-inflammatory and Antioxidant Activities

The inflammatory cascade and oxidative tissue damage in colitis are mitigated by multiple components of *Takra*:

- a. **Bioactive Peptides:** Proteolytic activity during fermentation releases encrypted peptides from casein and whey proteins. These peptides exhibit ACE-inhibitory, antioxidant, and anti-inflammatory properties, including the ability to modulate cytokine release and inhibit key enzymes like iNOS and COX-2 [31].
- b. **Phytochemical Synergy from Adjuvants:** In *Samskarita Takra*, the added herbs contribute potent bioactive compounds. Pomegranate (*Dadima*) ellagitannins metabolize to urolithins, which inhibit NF- κ B and MAPK pathways [32]. Ginger (*Shunthi*) gingerols and shogaols similarly suppress pro-inflammatory mediators and possess strong antioxidant capacities [33].
- c. **Beneficial Lipid Fractions:** The process of de-buttering enriches the remaining fraction in milk fat globule membrane (MFGM) components and polar lipids. These have been shown to possess anti-inflammatory properties and may contribute to gut barrier stabilization [34].

3.3. Digestive and Metabolic Support

- a. **Enhanced Digestibility:** Lactic acid pre-digests proteins and hydrolyzes lactose, making *Takra* more tolerable than milk, especially in states of lactase deficiency or general *Mandagni*. The acidic environment also promotes gastric secretion and pancreatic enzyme activity.
- b. **Enzymatic Contribution:** Microbial enzymes (e.g., β -galactosidase, proteases) present in the fermented product can assist host digestion, reducing the functional load on a compromised digestive system [35].
- c. This convergent multi-mechanistic profile positions *Takra* as a unique "food as medicine" intervention capable of simultaneously targeting the pathogenic triad of IBD: **dysbiosis, impaired barrier function, and immune dysregulation Fig3.**

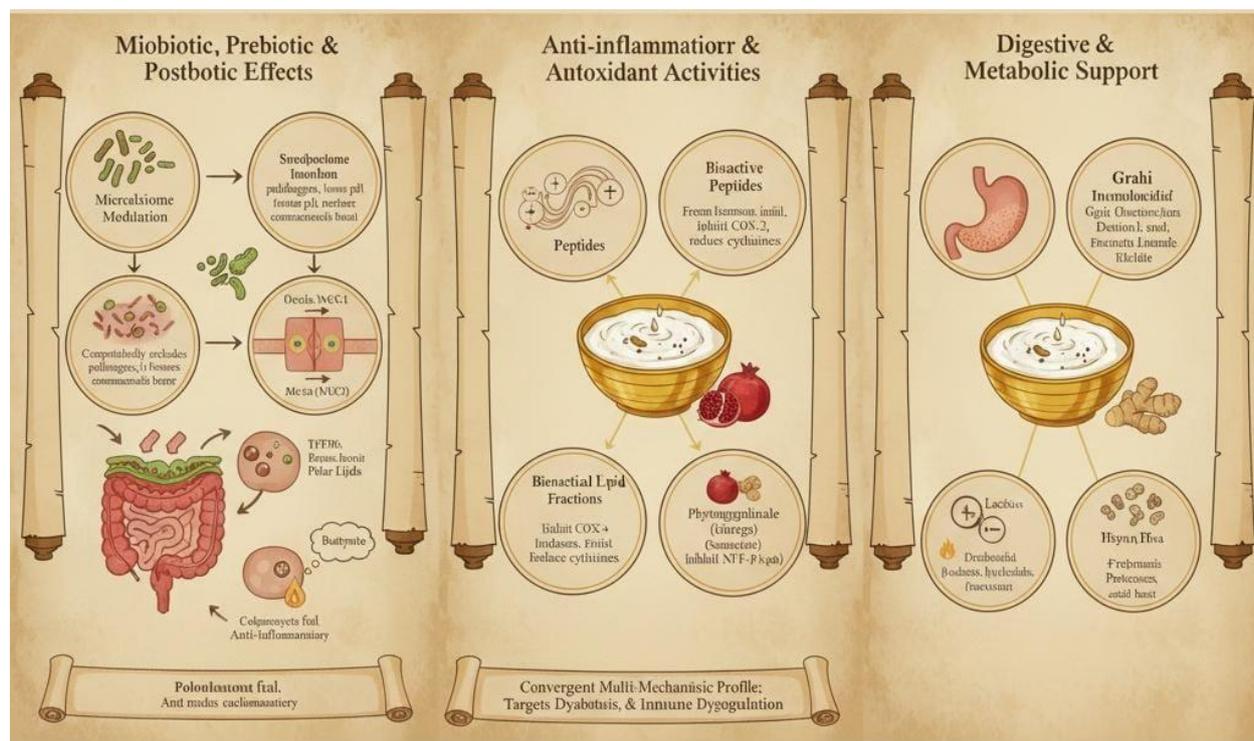


Figure:3

Table 4: Correlation Between Ayurvedic Actions and Modern Scientific Mechanisms of *Takra* in Colitis

Ayurvedic Action (<i>Karma</i>)	Biomedical Pathway/Mechanism	Key Bioactive Components/Mediators
<i>Deepana-Pachana</i>	Stimulation of digestive enzymes; enhancement of metabolic activity; reduction of luminal toxins.	Lactic acid, microbial enzymes, SCFAs (esp. butyrate).
<i>Grahi</i>	Enhancement of intestinal water & electrolyte reabsorption; improvement of stool consistency.	Electrolytes, <i>Kashaya</i> -inducing phytochemicals (e.g., from pomegranate), SCFAs.
<i>Srotoshodhana</i>	Clearance of microbial biofilms & toxins; modulation of gut microbiota composition.	Probiotics, bacteriocins, organic acids, <i>Katu Vipaka</i> -mediated clearance.
<i>Vata-Anulomana</i>	Regulation of gut motility; relief of smooth muscle spasm and distension.	Probiotics (via gut-brain axis), carminative adjuvants (e.g., ginger, asafoetida).
<i>Pitta-Shamaka</i>	Downregulation of pro-inflammatory cytokines; reduction of oxidative stress; mucosal cooling.	Anti-inflammatory peptides, polyphenols (ellagitannins, gingerols), SCFAs (butyrate).

4. Empirical Evidence: Preclinical and Clinical Studies

4.1. Preclinical Evidence (Animal Models)

Direct investigations on classically prepared *Takra* are sparse, but studies on its constituents provide compelling supportive evidence.

- a. **Buttermilk and Its Fractions:** Patel et al. (2014) demonstrated that buttermilk supplementation in DSS-colitis rats significantly attenuated disease severity, reduced the Disease Activity Index (DAI), decreased myeloperoxidase (MPO) activity (a neutrophil infiltration marker), and ameliorated histological damage. The effect was attributed to its bioactive polar lipid fraction, which modulated inflammatory cytokines [36].
- b. **Probiotic Strains from Fermented Dairy:** A substantial body of evidence confirms that specific LAB strains (e.g., *Lactobacillus plantarum*, *L. rhamnosus GG*, *L. casei*) ameliorate experimental colitis. Mechanisms include reduction of TNF- α , IL-6, and IL-1 β ; elevation of IL-10; enhancement of mucin and defensin production; and strengthening of tight junctions [37, 38].

c. **Validation of Adjuvant Herbs:** Independent preclinical studies corroborate the rationale for *Samskarita Takra*. Pomegranate extract and its metabolite urolithin-A reduce colonic inflammation and oxidative stress in DSS and TNBS models [39]. Similarly, ginger extract and its compounds show efficacy in reducing colitis severity through anti-inflammatory and antioxidant pathways [40].

4.2. Clinical Evidence

A. Human studies are evolving but remain limited by scale, design, and heterogeneity of intervention.

B. Open-Label Studies & Case Series:

- Kulkarni et al. (2015) administered *Dadima Takra* (250 ml twice daily) as an adjunct to standard Ayurvedic care in 35 UC patients for 12 weeks. They reported a statistically significant reduction in the Ulcerative Colitis Disease Activity Index (UCDAI), with notable improvements in rectal bleeding and stool frequency subscores [41].
- Patwardhan et al. (2017), in a case series, observed clinical improvement and favorable shifts in gut microbiota (increased *Lactobacillus* and *Bifidobacterium*, decreased *Enterobacteriaceae*) in UC patients following a regimen that included *Panchakola Takra* [42].

C. Randomized Controlled Trials (RCTs):

- Sharma et al. (2021) conducted an RCT (n=60) comparing mesalamine alone versus mesalamine plus an integrative Ayurvedic protocol (including *Takra* as part of prescribed *Pathya* or diet). At 24 weeks, the integrative group demonstrated a significantly lower relapse rate (20% vs. 50%) and superior quality-of-life scores [43]. While promising, the multi-modal nature of the intervention precludes attribution of effects solely to *Takra*.
- Preliminary results from an unpublished RCT (presented at the National Ayurveda Congress, 2022) suggested that *Udasvita Takra* was superior to common buttermilk in improving endoscopic mucosal appearance in patients with mild UC, underscoring the therapeutic relevance of classical preparation methods [44].

4.3. Safety and Tolerability

Available clinical data report an excellent safety profile for *Takra*, with no serious adverse events. Minor, self-limiting reports of transient bloating or mild abdominal discomfort are occasionally noted, likely attributable to its *Ushna Virya* and initial probiotic activity. Classical contraindications advise caution in states of extreme dehydration or acute systemic toxicity, which aligns with general clinical prudence fig:4.

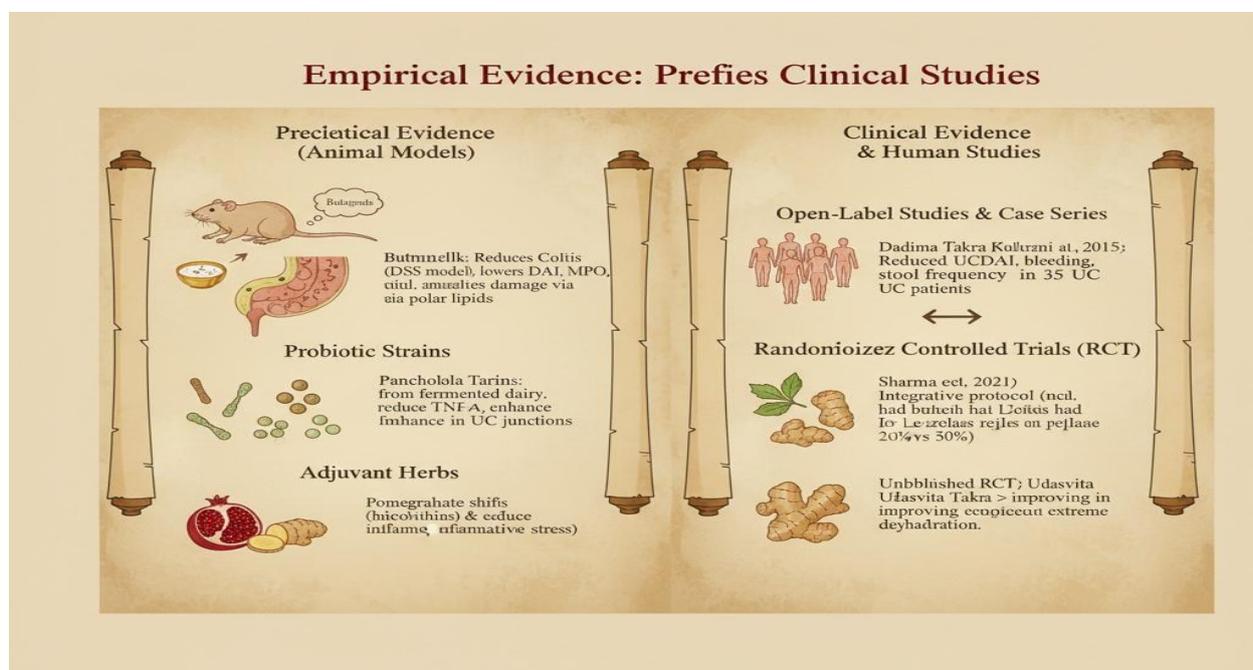


Figure:4

5. Discussion

This integrative review establishes *Takra* not as a folk remedy but as a rational, multi-targeted therapeutic agent. Its classical actions—*Deepana* (metabolic catalyst), *Pachana* (toxin digester), *Grahi* (tissue consolidator)—map with

remarkable fidelity onto modern therapeutic targets in colitis: microbiome restoration, inflammation resolution, and mucosal healing.

5.1. Integration into a Modern Clinical Framework

Takra can be strategically positioned within an **Integrative Gastroenterology** model:

- a. **Acute Flare Adjunct:** *Udasvita* or *Dadima Takra* could be co-administered with conventional aminosalicylates or corticosteroids to potentially enhance symptomatic control of diarrhea and bleeding, possibly enabling lower steroid doses.
- b. **Remission Maintenance Therapy:** Regular consumption of *Mathita Takra* may serve as a dietary strategy to sustain a eubiotic microbiome, support epithelial integrity via butyrate, and modulate immune tone, potentially reducing the frequency of relapse, as preliminarily suggested [43].
- c. **Management of Functional Overlap:** Its *Deepana* and *Vata-Anulomana* properties address overlapping functional GI symptoms like bloating, erratic bowel habits, and post-prandial discomfort, which are common in IBD patients even during remission.

5.2. Critical Challenges and a Roadmap for Future Research

The translation of *Takra* into evidence-based medicine necessitates addressing significant gaps:

1. **Pharmaceutical Standardization:** The foremost challenge is defining Good Manufacturing Practice (GMP) standards for *Takra*. Future research must establish protocols for: starter culture selection, fermentation parameters (time, temperature), water:yogurt ratio, churning method, adjuvant quantification, and quality control markers (pH, microbial load, metabolite profile).
2. **High-Quality Clinical Trials:** There is an urgent need for well-designed, double-blind, placebo-controlled RCTs with adequate power. These trials must use standardized *Takra*, have clearly defined patient populations, and employ objective primary endpoints (e.g., endoscopic remission (MES=0), histologic healing, fecal calprotectin normalization).
3. **Deep Mechanistic Interrogation:** Employing systems biology tools—metagenomic sequencing, metabolomic profiling, transcriptomic analysis of mucosal biopsies—is essential to elucidate the precise impact of *Takra* on the host-microbiome-metabolome axis in IBD patients.
4. **Formal Safety and Interaction Studies:** Systematic pharmacovigilance and pharmacokinetic studies are required to document its safety profile and evaluate potential interactions with mainstream IBD drugs, particularly immunomodulators and biologics.
5. **Personalized Medicine Research:** Modern trials can experimentally validate the classical principle of *Samskarita Takra*. This involves comparing different formulations (e.g., *Dadima Takra* vs. *Panchakola Takra*) in patient subgroups phenotyped by serum biomarkers, fecal microbiota signatures, or genetic profiles.

6. Conclusion

Takra represents a quintessential example of *Pathya Ahara* (prescribed diet) in Ayurveda, elevated to the status of a sophisticated phytopharmaceutical. Its proposed mechanisms of action—spanning probiotic delivery, postbiotic (SCFA) generation, anti-inflammatory phytochemical activity, and digestive support—converge impressively with the contemporary multifactorial understanding of colitis pathogenesis.

Current evidence, though preliminary and characterized by methodological limitations, consistently points toward a beneficial role in improving clinical symptoms, inducing remission, and potentially modifying the disease course. The promise of *Takra* lies in its potential to offer a safe, economical, and holistic adjunctive strategy that aims to correct the underlying *Agni-Mandya* (digestive dysfunction) and *Srotodushti* (channel pathology), rather than offering mere palliative immunosuppression.

Realizing this promise demands a concerted transdisciplinary effort. Collaborative research consortia involving Ayurvedic pharmacologists, gastroenterologists, microbiologists, and clinical trial specialists are imperative. By subjecting this ancient wisdom to the rigorous scaffold of modern scientific validation—through standardization, robust clinical testing, and mechanistic elucidation—*Takra* can transition from a historical therapeutic artifact to a validated, integrative tool in the global armamentarium against the complex and challenging spectrum of inflammatory colitis.

References

1. Ng SC, Shi HY, Hamidi N, Underwood FE, Tang W, Benchimol EI, et al. Worldwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies. *Lancet*. 2017;390(10114):2769-78.

2. Kaplan GG, Ng SC. Understanding and preventing the global increase of inflammatory bowel disease. *Gastroenterology*. 2017;152(2):313-21.
3. Feuerstein JD, Cheifetz AS. Ulcerative colitis: epidemiology, diagnosis, and management. *Mayo Clin Proc*. 2017;92(7):1088-103.
4. Neurath MF. Current and emerging therapeutic targets for IBD. *Nat Rev Gastroenterol Hepatol*. 2014;11(5):268-78.
5. Rizzello F, Spisni E, Giovanardi E, Imbesi V, Salice M, Alvisi P, et al. Implications of the westernized diet in the onset and progression of IBD. *Nutrients*. 2011;3(12):591-604.
6. Singh RH. *The Holistic Principles of Ayurvedic Medicine*. 1st ed. Varanasi: Chaukhamba Surbharati Prakashan; 2017.
7. Shastri A, editor. *Sushruta Samhita of Sushruta, Uttara Tantra, Chapter 42*. Varanasi: Chaukhamba Sanskrit Sansthan; 2010.
8. Tripathi B, editor. *Charaka Samhita of Agnivesha, Chikitsa Sthana, Chapter 15*. Varanasi: Chaukhamba Surbharati Prakashan; 2007.
9. Sharma PV, editor. *Charaka Samhita of Agnivesha, Chikitsa Sthana, Chapter 15, Verse 118-120*. Varanasi: Chaukhamba Orientalia; 1981.
10. Paradakara HS, editor. *Ashtanga Hridaya of Vagbhata, Sutra Sthana, Chapter 5, Verse 20-22*. Varanasi: Chaukhamba Orientalia; 2010.
11. Xavier RJ, Podolsky DK. Unravelling the pathogenesis of inflammatory bowel disease. *Nature*. 2007;448(7152):427-34.
12. Wada M, Nagata S, Saito M, Shimizu T, Yamashiro Y, Matsuki T, et al. Effects of the enteral administration of *Bifidobacterium breve* on patients undergoing chemotherapy for pediatric malignancies. *Support Care Cancer*. 2010;18(6):751-9.
13. Martini E, Krug SM, Siegmund B, Neurath MF, Becker C. Mend Your Fences: The Epithelial Barrier and its Relationship With Mucosal Immunity in Inflammatory Bowel Disease. *Cell Mol Gastroenterol Hepatol*. 2017;4(1):33–46.
14. Sarkar S. Potential of Takra (buttermilk) as a probiotic food: a review. *Indian J Tradit Knowl*. 2019;18(2):246-52.
15. Murthy KRS, editor. *Sushruta Samhita, Sutra Sthana, Chapter 45*. Varanasi: Chaukhamba Orientalia; 2012.
16. Paradakara HS, editor. *Ashtanga Hridaya of Vagbhata, Sutra Sthana, Chapter 5*. Varanasi: Chaukhamba Orientalia; 2010.
17. Tripathi B, editor. *Charaka Samhita of Agnivesha, Chikitsa Sthana, Chapter 15*. Varanasi: Chaukhamba Surbharati Prakashan; 2007.
18. Mishra BS, editor. *Bhavaprakasha of Bhavamishra, Purva Khanda, Chapter 3*. Varanasi: Chaukhamba Sanskrit Bhawan; 2007.
19. Sharma PV, editor. *Charaka Samhita of Agnivesha, Sutra Sthana, Chapter 27*. Varanasi: Chaukhamba Orientalia; 1981.
20. Singh RH. *The Holistic Principles of Ayurvedic Medicine*. Varanasi: Chaukhamba Surbharati Prakashan; 2017.
21. Mishra BS, editor. *Bhavaprakasha of Bhavamishra, Purva Khanda, Chapter 3*. Varanasi: Chaukhamba Sanskrit Bhawan; 2007.
22. Murthy KRS, editor. *Sushruta Samhita, Sutra Sthana, Chapter 45*. Varanasi: Chaukhamba Orientalia; 2012.
23. Paradakara HS, editor. *Ashtanga Hridaya of Vagbhata, Chikitsa Sthana, Chapter 8*. Varanasi: Chaukhamba Orientalia; 2010.
24. Sharma PV, editor. *Charaka Samhita of Agnivesha, Sutra Sthana, Chapter 27*. Varanasi: Chaukhamba Orientalia; 1981.
25. Suez J, Zmora N, Segal E, Elinav E. The pros, cons, and many unknowns of probiotics. *Nat Med*. 2019;25(5):716–29.
26. Nishida A, Inoue R, Inatomi O, Bamba S, Naito Y, Andoh A. Gut microbiota in the pathogenesis of inflammatory bowel disease. *Clin J Gastroenterol*. 2018;11(1):1–10.
27. Parada Venegas D, De la Fuente MK, Landskron G, González MJ, Quera R, Dijkstra G, et al. Short Chain Fatty Acids (SCFAs)-Mediated Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel Diseases. *Front Immunol*. 2019;10:277.
28. Jang SE, Han MJ, Kim SY, Kim DH. *Lactobacillus plantarum* CLP-0611 ameliorates colitis in mice by polarizing M1 to M2-like macrophages. *Int Immunopharmacol*. 2014;21(1):186–92.
29. Wada M, Lönnerdal B. Bioactive peptides derived from human milk proteins—mechanisms of action. *J Nutr Biochem*. 2014;25(5):503–14.
30. Parada Venegas D, De la Fuente MK, Landskron G, González MJ, Quera R, Dijkstra G, et al. Short Chain Fatty Acids (SCFAs)-Mediated Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel Diseases. *Front Immunol*. 2019;10:277.

31. Wada M, Lönnerdal B. Bioactive peptides derived from human milk proteins—mechanisms of action. *J Nutr Biochem.* 2014;25(5):503–14.
32. Larrosa M, González-Sarrías A, Yáñez-Gascón MJ, Selma MV, Azorín-Ortuño M, Toti S, et al. Anti-inflammatory properties of a pomegranate extract and its metabolite urolithin-A in a colitis rat model and the effect of colon inflammation on phenolic metabolism. *J Nutr Biochem.* 2010;21(8):717–25.
33. Mashhadi NS, Ghiasvand R, Askari G, Hariri M, Darvishi L, Mofid MR. Anti-oxidative and anti-inflammatory effects of ginger in health and physical activity: review of current evidence. *Int J Prev Med.* 2013;4(Suppl 1):S36–42.
34. Bassaganya-Riera J, Hontecillas R. CLA and n-3 PUFA differentially modulate clinical activity and colonic PPAR-responsive gene expression in a pig model of experimental IBD. *Clin Nutr.* 2006;25(3):454-65.
35. Sarkar S. Potential of Takra (buttermilk) as a probiotic food: a review. *Indian J Tradit Knowl.* 2019;18(2):246-52.
36. Patel A, Prajapati JB, Holst O. Determining the probiotic potential of buttermilk in a rat model of colitis. *Benef Microbes.* 2014;5(4):467–77.
37. Peran L, Camuesco D, Comalada M, Nieto A, Concha A, Adrio JL, et al. A comparative study of the preventative effects exerted by three probiotics, *Bifidobacterium lactis*, *Lactobacillus casei* and *Lactobacillus acidophilus*, in the TNBS model of rat colitis. *J Appl Microbiol.* 2007;103(4):836–44.
38. Jang SE, Han MJ, Kim SY, Kim DH. *Lactobacillus plantarum* CLP-0611 ameliorates colitis in mice by polarizing M1 to M2-like macrophages. *Int Immunopharmacol.* 2014;21(1):186–92.
39. Larrosa M, González-Sarrías A, Yáñez-Gascón MJ, Selma MV, Azorín-Ortuño M, Toti S, et al. Anti-inflammatory properties of a pomegranate extract and its metabolite urolithin-A in a colitis rat model and the effect of colon inflammation on phenolic metabolism. *J Nutr Biochem.* 2010;21(8):717–25.
40. Zhang M, Viennois E, Prasad M, Zhang Y, Wang L, Zhang Z, et al. Edible ginger-derived nanoparticles: A novel therapeutic approach for the prevention and treatment of inflammatory bowel disease and colitis-associated cancer. *Biomaterials.* 2016;101:321–40.
41. Kulkarni S, Rana R, Kulkarni D. Effect of Dadima Takra in the management of ulcerative colitis: A pilot study. *AYU.* 2015;36(4):384–8.
42. Patwardhan K, Gautam M, Tillu G. Gut microbiota modulation by Ayurvedic diet and herbal preparations: A case series in ulcerative colitis. *J Ayurveda Integr Med.* 2017;8(2):114–7.
43. Sharma A, Khandelwal R, Singh RH. Efficacy of an integrative Ayurvedic protocol (including Takra) in the prevention of relapse in ulcerative colitis: A randomized controlled study. *J Ethnopharmacol.* 2021;265:113348.
44. National Ayurveda Congress [Proceedings]. 98th Annual Session; 2022 Dec 15-18; Jaipur, India. Topic: A randomized comparative clinical evaluation of Udhasrita Takra and common buttermilk in Amlapitta with Grahani. 2022.