

**AGNI DUSHTI AND ITS ROLE IN THE ETIOPATHOGENESIS OF PSORIASIS: AN
INTEGRATIVE REVIEW OF AYURVEDIC AND MODERN PERSPECTIVES*****Rufshana Yasmin*****Corresponding Author: Rufshana Yasmin**DOI: <https://doi.org/10.5281/zenodo.22159655>**How to cite this Article:** Rufshana Yasmin. (2026). Agni Dushti and Its Role In The Etiopathogenesis of Psoriasis: An Integrative Review of Ayurvedic and Modern Perspectives. World Journal of Pharmaceutical and Medical Research, 12(9), 78–81.

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Article Received on 05/08/2026

Article Revised on 24/08/2026

Article Published on 01/09/2026

ABSTRACT

Background: Psoriasis is a chronic, immune-mediated inflammatory dermatosis with complex etiopathogenesis. Ayurveda classifies it under Kushtha (predominantly Kitibha and Ekakushtha), where Agni Dushti — impairment of digestive and metabolic fire — is identified as a central upstream pathological event. **Objective:** To explore conceptual and mechanistic parallels between Agni Dushti and the modern immunopathogenesis of psoriasis, integrating classical Ayurvedic frameworks with contemporary molecular and dermatopathological evidence. **Methods:** An integrative literature review was conducted using classical Ayurvedic texts (Charaka Samhita, Sushruta Samhita, Ashtanga Hridayam) and peer-reviewed biomedical databases (PubMed, Scopus, AYUSH Research Portal) for the period 2000–2024. **Key Findings:** Agni Dushti leads to Ama formation, Dosha Vaishamya, and Dhatu Dushti — paralleling increased intestinal permeability, systemic inflammation (IL-17, IL-23, TNF-alpha), and altered keratinocyte kinetics in psoriasis. **Conclusion:** Agni Dushti provides a holistic etiological framework for psoriasis, and its targeted correction through Deepana-Pachana therapies may complement modern biologic approaches. Further clinical validation is warranted.

KEYWORDS: Agni Dushti, Ekakushtha, Psoriasis, Ama, Immunopathogenesis, Integrative Review, Ayurveda, Gut-Skin Axis

1. INTRODUCTION

Psoriasis is a chronic, relapsing, immune-mediated inflammatory skin disorder affecting approximately 2–3% of the global population, with Indian prevalence estimated between 0.44–2.8%. Beyond dermatological manifestations, it is associated with psoriatic arthritis, metabolic syndrome, and significant psychosocial burden. Despite advances in biologic therapies targeting the IL-17/IL-23 axis, challenges of recurrence, high cost, and long-term adverse effects necessitate exploration of integrative approaches.

Ayurveda classifies psoriasis-like conditions within Kushtha (skin diseases). Charaka Samhita (CS.Ni.5) describes Ekakushtha and Kitibha as Vata-Kapha predominant Kushtha presenting with Matsyashakala (fish-scale appearance), Aswedana (absence of sweating), and extensive involvement — features closely paralleling psoriasis vulgaris.

Central to Ayurvedic etiopathogenesis is Agni — the biological fire governing all metabolic and digestive

processes. Agni Dushti, particularly Mandagni (hypoactive Agni), initiates a cascade of Ama (toxic metabolic byproduct) formation, Dosha Vaishamya, Srotorodha, and Dhatu Dushti affecting Twak (skin). Contemporary research on the gut-skin axis, intestinal permeability, and microbiome dysbiosis in psoriasis offers compelling parallels to these classical constructs. The present review bridges this gap by analyzing Ayurvedic frameworks alongside contemporary immunopathological evidence.

2. Agni in Ayurveda: Classical Concept and Relevance to Skin

Agni represents the totality of biological transformation at every level of the organism. Charaka Samhita (CS.Su.11/35) declares: 'Agni eva sharira sthitha' — Agni alone is the basis of life. The texts enumerate 13 types of Agni: 1 Jatharagni, 5 Bhutagnis, and 7 Dhatvagnis. Three types hold particular relevance for dermatological pathology:

Table 1: Agni Types Relevant to Skin Disease — Site, Function, and Dushti Effects.

Type of Agni	Site of Action	Physiological Function	Effect of Dushti
Jatharagni	Amashaya & Grahani	Primary digestion; Rasa formation	Mandagni → Ama; Vishamagni → irregular digestion
Ranjaka Pitta	Yakrit & Pleeha	Converts Rasa to Rakta Dhatu	Impaired Rakta → skin discoloration, inflammation
Bhrajaka Pitta	Twak (Skin)	Complexion, luster, thermoregulation	Dushti → Twak Vikara, hyperpigmentation, scaling
Dhatwagni	Each of 7 Dhatus	Dhatu conversion and nourishment	Dhatu Kshaya/Dushti → pathological tissue changes

Mandagni is the most clinically significant form in Kushtha. It leads to incomplete digestion, resulting in Ama — a sticky, toxic metabolic byproduct that obstructs Srotas and initiates systemic Dosha vitiation. Vishamagni and Teekshnagni also contribute variably based on the patient's Dosha constitution.

3. Nidana (Etiology): Causative Factors of Agni Dushti in Psoriasis

Charaka Samhita (CS.Ni.5) enumerates specific etiological factors for Kushtha, many of which directly cause Agni Dushti. The following table integrates Ayurvedic Nidana with modern biomedical correlates:

Table 2: Ayurvedic Nidana for Kushtha with Modern Biomedical Correlates.

Ayurvedic Nidana (Hetu)	Modern Correlation / Risk Factor
Viruddha Ahara	Dietary triggers: gluten, alcohol, processed foods, dairy
Adhyashana / Ajeerna Bhojana	Metabolic syndrome, obesity, insulin resistance
Guru, Snigdha, Abhishyandi Ahara	High glycaemic index diet promoting systemic inflammation
Vegadharana	Autonomic dysfunction, gut motility disorders
Manasika Hetu: Chinta, Shoka	HPA axis dysregulation, elevated cortisol, mast cell activation
KrimiJa Hetu (microbial triggers)	Streptococcal infection, Candida overgrowth, gut dysbiosis
Ratri Jagarana	Circadian rhythm disruption, melatonin suppression

4. Samprapti (Pathogenesis): From Agni Dushti to Ekakushtha

Samprapti represents the sequential pathophysiological progression from causative factors to clinical

manifestation. In Ekakushtha, Agni Dushti occupies the pivotal second stage, serving as the gateway through which Nidana Sevana translates into systemic disease:

Table 3: Samprapti of Ekakushtha — Ayurvedic Stages with Modern Pathological Correlates.

Samprapti Stage	Ayurvedic Pathogenesis	Modern Pathological Equivalent
1. Nidana Sevana	Viruddha Ahara, psychological stress, infection	Dietary triggers, HPA stress axis, Group A Streptococcus
2. Agni Dushti (Mandagni)	Impaired Jatharagni → Ama formation	Reduced digestive enzyme activity; intestinal dysbiosis
3. Dosha Vaishamya	Vata-Kapha dominance in Ekakushtha	Epidermal barrier dysfunction (Vata) + immune dysregulation (Kapha-Pitta)
4. Srotorodha	Blockage of Rasavaha, Raktavaha, Swedavaha Srotas	Impaired lymphatic drainage; occluded sweat glands
5. Dhatu Dushti	Rasa → Rakta → Mamsa → Twak (Bahudoshavastha)	Th1/Th17 activation, keratinocyte hyperproliferation
6. Ekakushtha	Matsyashakala, Aswedana, Kandu, Ruk	Psoriatic plaques, PASI elevation, Ki-67 upregulation, pruritus

5. Modern Immunopathogenesis: Parallels with Agni Dushti

Psoriasis pathogenesis is driven by a dysregulated immune cascade initiated by plasmacytoid dendritic cells (pDCs) producing IFN-alpha. This activates myeloid DCs that prime naive T-cells toward Th1 and Th17

lineages. The resultant cytokine milieu — particularly IL-17A, IL-17F, IL-22, IL-23, and TNF-alpha — drives keratinocyte hyperproliferation, reduced differentiation, and neutrophil recruitment. The table below maps classical Agni Dushti constructs to contemporary molecular findings:

Table 4: Integrative Mapping — Agni Dushti Concepts and Modern Immunopathological Equivalents.

Agni Dushti Concept (Ayurveda)	Modern Molecular / Pathological Equivalent
Ama (undigested metabolic toxins)	Endotoxemia, LPS from leaky gut, Damage-Associated Molecular Patterns (DAMPs)

Rakta Dushti	Activated T-cells (Th1/Th17), elevated CRP, ESR, TNF-alpha
Bhrajaka Pitta Dushti	Keratinocyte hyperproliferation (elevated Ki-67), reduced apoptosis, acanthosis
Srotorodha	Impaired lymphatic drainage, VEGF-driven pathological angiogenesis
Vata-Kapha Dosha dominance	IL-17A/IL-23 axis activation + epidermal barrier dysfunction (filaggrin deficiency)
Aswedana	Eccrine gland occlusion, altered transepidermal water loss (TEWL)
Twak Parushya / Khara Twak	Parakeratosis, stratum corneum retention, scaling (Auspitz sign positive)

A particularly significant parallel exists between Ama and endotoxemia. Ama is characterized as Picchila (sticky), Guru (heavy), and Durgandha (foul-smelling), capable of causing Srotorodha — properties that parallel circulating lipopolysaccharide (LPS) derived from gram-negative intestinal bacteria in conditions of increased gut permeability. LPS activates TLR-4 receptors, initiating NF-κB-mediated systemic inflammation — a mechanistic correlate of Ama-induced Dosha Dushti.

6. Gut-Skin Axis: Agni Dushti as the Central Mechanistic Link

The gut-skin axis has emerged as a critical pathophysiological concept in dermatology. Patients with psoriasis demonstrate distinct gut microbiome alterations including reduced *Faecalibacterium prausnitzii*, *Akkermansia muciniphila*, and *Roseburia intestinalis*, with enrichment of *Candida*, *Escherichia coli*, and select Firmicutes species. These findings resonate with Ayurvedic constructs: Agni Dushti impairs the Annavaha Srotas, leading to Purisha Vaha Srotas Dushti — functionally analogous to gut dysbiosis and altered intestinal permeability.

Key integrative insights include: (1) increased intestinal permeability in psoriasis allows microbial LPS translocation, paralleling Ama formation from impaired Jatharagni; (2) Deepana-Pachana herbs such as Trikatu and Triphala demonstrate prebiotic and microbiome-modulating properties; (3) Takra (buttermilk) contains *Lactobacillus* species mirroring probiotic interventions that reduce PASI scores; and (4) Virechana achieves gut mucosal healing functionally analogous to intestinal permeability restoration. Psychological stress-induced HPA activation further reduces intestinal mucus thickness, directly correlating with the Manasika Hetu → Agni Dushti → Ama pathway.

7. Therapeutic Implications: Correcting Agni Dushti in Psoriasis

Ayurvedic management follows the Shodhana (purification) and Shamana (palliation) paradigm, with Deepana-Pachana as the foundational step before all other interventions:

Table 5: Ayurvedic Therapeutic Interventions Targeting Agni Dushti in Psoriasis.

Ayurvedic Intervention	Pharmacological / Therapeutic Action	Level of Evidence
Deepana-Pachana (Trikatu, Chitrakadi Vati)	Digestive enzyme stimulation; anti-inflammatory via Agni correction	Preclinical + Classical authority (CS.Ci.15)
Virechana (Therapeutic Purgation)	Pitta Shodhana; gut detoxification; Ama elimination	Classical authority; case series evidence
Takra Pana / Takradhara	Probiotic effect; anti-inflammatory via gut-skin axis	Emerging clinical evidence; <i>Lactobacillus</i> studies
Khadirarishta + Manjistha	Immunomodulation; blood purification; antioxidant	Ethnopharmacological data; in vitro studies
Panchakarma (Vamana + Virechana)	Complete Dosha Shodhana; metabolic reset	RCT evidence in psoriasis (CTRI-registered studies)
Neem (Nimba) + Turmeric (Haridra)	Anti-inflammatory, antimicrobial, keratinocyte modulation	Clinical trial evidence; curcumin reduces PASI scores

The classical therapeutic sequence begins with: (1) Deepana-Pachana (7–14 days) to correct Agni and digest Ama, followed by (2) Snehana-Swedana to mobilize Doshas, and then (3) Shodhana (Vamana or Virechana as appropriate). This protocol aligns with modern integrative principles of restoring gut health before addressing systemic inflammation.

8. DISCUSSION

This integrative review demonstrates that Agni Dushti provides a coherent upstream framework for understanding psoriasis etiopathogenesis. The construct of Ama — arising from impaired Jatharagni — serves as

a conceptual bridge between Ayurvedic metabolic toxin accumulation and the modern understanding of endotoxemia, gut dysbiosis, and the gut-skin axis. The systematic mapping of Samprapti stages to contemporary immunopathological events reveals that Vata-Kapha Dosha dominance aligns mechanistically with epidermal barrier dysfunction (filaggrin deficiency — Vata quality) combined with IL-17/IL-23-driven immune hyperactivation (Kapha/Pitta excess), suggesting that Ayurvedic Dosha theory may encode phenotypic information about immune-inflammatory subtypes.

However, significant limitations must be acknowledged. Assessment of Agni status relies on subjective parameters (Mandagni Lakshanas: Aruchi, Gaurava, Tandra, Alasya) without validated biomarker correlates. Future research should establish measurable biological surrogates — candidate markers include fasting serum LPS-binding protein, intestinal fatty acid binding protein (I-FABP), stool microbiome profiling, and inflammatory cytokine panels. The mechanistic parallels proposed, while conceptually coherent, require direct experimental validation through RCTs measuring both Ayurvedic endpoints and biomedical outcomes (PASI score, cytokine profiles, microbiome indices).

9. CONCLUSION

Agni Dushti, specifically Mandagni, represents a clinically meaningful and mechanistically coherent upstream pathological event in the etiopathogenesis of psoriasis (Ekakushtha). The formation of Ama from impaired Jatharagni, with subsequent Dosha Vaishamya, Srotorodha, and Dhatu Dushti, parallels the gut dysbiosis, increased intestinal permeability, endotoxemia, and Th17-driven cutaneous inflammation of modern psoriasis pathogenesis. The gut-skin axis emerges as the key terrain where both frameworks converge.

Agni-corrective interventions (Deepana-Pachana, Virechana, Takra) offer rational, evidence-consonant approaches that may function as effective adjuncts to biologic therapy, particularly for patients in resource-limited settings. Translation of Agni Dushti into validated biomarker frameworks represents the most critical research priority for advancing both Ayurvedic scientific credibility and integrative clinical practice.

REFERENCES

1. Charaka Samhita, Nidana Sthana, Chapter 5 (Kushtha Nidanam). In: Sharma PV (ed). Chaukhamba Orientalia; 2005.
2. Sushruta Samhita, Nidana Sthana, Chapter 5. In: Singhal GD (ed). Chaukhamba Sanskrit Pratishthan; 2007.
3. Ashtanga Hridayam, Sutra Sthana, Chapter 10 (Doshadivijnaneeyam). In: Srikanthamurthy KR (trans). Krishnadas Academy; 2001.
4. Griffiths CEM, Armstrong AW, Gudjonsson JE, Barker JNWN. Psoriasis. Lancet. 2021; 397(10281): 1301-1315.
5. Fabbrocini G, Cameli N, Officer MV, et al. The role of the gut-skin axis in atopic and inflammatory skin diseases. G Ital Dermatol Venereol. 2018; 153(5): 651-659