American Journal of Psychiatric ehabilitation

Expert Opinion Article

Viruddha Ahara As An Epigenetic Trigger In Chronic Dermatoses: An Ayurvedic Hypothesis Paper

Dr. Jyoti Chopde[1*], Dr.Sandesh Chokhar[2], Dr. Ashutosh Dubey[3]

MD Kayachikitsa, Associate Professor, Institution: Shri Vivekanand Nursing Homes Ayurved Mahavidyalaya, Rahuri

[2]MD kayachikitsa, Associate Professor, Institution: Shri Vivekanand Nursing Homes Ayurved Mahavidyalaya, Rahuri

[3]PG Scholar, (Department of Kayachikitsa), Institution: Shri Vivekanand Nursing Homes Ayurved Mahavidyalaya, Rahuri

ABSTRACT

Viruddha Āhāra (incompatible foods) is an established etiological concept in classical Āyurveda and has been historically implicated in Kuṣṭha and other skin disorders. Modern epigenetics demonstrates that environmental factors including diet can modify DNA methylation, histone marks and non-coding RNA expression — mechanisms that regulate gene expression relevant to skin barrier, immune response and inflammation. We propose a mechanistic hypothesis: repeated exposure to Viruddha Āhāra produces persistent metabolic and inflammatory perturbations that act as epigenetic triggers, promoting chronicity and relapses in dermatoses (e.g., psoriasis, atopic dermatitis, hidradenitis suppurativa). We review classical definitions, summarize contemporary epigenetic evidence in chronic skin disease, outline plausible molecular pathways linking incompatible diet to epigenomic change, and propose a translational research agenda to test this Ayurvedic–molecular hypothesis.

Keywords: Viruddha Āhāra, epigenetics, psoriasis, atopic dermatitis, Kuṣṭha, methylation, diet, Ayurvedic hypothesis

INTRODUCTION

Viruddha Āhāra (incompatible or ill-combined food) is described in classical texts as food combinations, processing, or timings that disturb normal metabolism and lead to a spectrum of disorders — including skin diseases. Classical authors warn that persistent intake of such foods acts like a toxin that vitiates the Doshas and produces disease.

Concurrently, epigenetic mechanisms (DNA methylation, histone modifications, and non-coding RNAs) are recognized mediators by which environment and diet shape gene expression relevant to immune and barrier functions in skin. Diet and other environmental factors (microbiome, smoking, stress) can produce stable or semi-stable epigenetic changes implicated in chronic dermatoses.

This review synthesizes Ayurvedic concept and modern epigenetic science to advance the working hypothesis that Viruddha Āhāra acts as an epigenetic trigger in chronic inflammatory dermatoses.

AIMS

1. To review classical descriptions and modern interpretations of Viruddha Āhāra with emphasis on skin disease (Kuṣṭha).
2. To summarize contemporary evidence for epigenetic dysregulation in chronic dermatoses (psoriasis, atopic dermatitis).
3. To propose mechanistic pathways linking incompatible diet → systemic perturbation → epigenetic change → chronic dermatoses.
4. To outline study designs and biomarkers to test the hypothesis translationally.

BACKGROUND

Viruddha Āhāra: classical concept and dermatological relevance

Classical Āyurveda catalogs types of Viruddha (food–food incompatibility, processing–related incompatibility, chronobiologic incompatibility etc.) and records that such intake may lead to vitiation of Doshas, formation of Ama (metabolic toxin), and manifestation of Kuṣṭha (skin disorders). Contemporary Ayurvedic reviews link incompatible foods to inflammation and skin pathology.

Epigenetics and skin disease — concise primer

Epigenetic regulation controls gene expression without altering DNA sequence. Key mechanisms include DNA methylation, histone modification, chromatin remodeling, and microRNA-mediated post-transcriptional regulation. These mechanisms respond to environmental cues (diet, microbiota, toxins, stress), and are implicated in barrier dysfunction, immune dysregulation and chronic inflammation in skin diseases such as psoriasis and atopic dermatitis.

EVIDENCE THAT DIET & ENVIRONMENT AFFECT SKIN EPIGENOME

1. Multiple studies show DNA methylation alterations in psoriasis and atopic dermatitis (differentially methylated loci in epidermal and immune genes).
2. Reviews identify diet, microbiota, smoking and stress as environmental factors that modulate skin epigenetics and influence disease severity and course.
3. Clinical dietary-intervention studies in inflammatory skin diseases report symptomatic improvement (various diets/nutritional changes), supporting a diet–disease axis amenable to mechanistic exploration.

PROPOSED MECHANISTIC FRAMEWORK (VIRUDDHA → EPIGENOME → DERMATOSIS)

We propose a multi-step mechanistic chain:

1. Exposure Phase (Repeated Viruddha Intake)

- Repeated intake of incompatible foods (e.g., specifically incompatible combos, improper food processing, food with opposing Virya) leads to digestive disturbance (Agni mandya) and formation of Ama in Ayurvedic terms. In modern terms, this produces metabolic stress, low-grade systemic inflammation, altered gut microbiome and oxidative stress.

2. Systemic Signaling Phase

- Metabolic/inflammatory mediators (IL-6, TNFα, oxidative species, LPS from dysbiotic gut) circulate and signal to peripheral tissues including skin. Chronic signaling establishes a pro-inflammatory niche and impacts local resident cells (keratinocytes, fibroblasts, immune cells).

3. Epigenetic Reprogramming Phase

- Persistent inflammatory/metabolic cues recruit epigenetic modifiers (DNMTs, HDACs, HATs) that alter DNA methylation and histone marks at key loci (e.g., skin barrier genes FLG, LOR; cytokine/chemokine genes IL-17, IL-23 related pathways; TLRs). MicroRNAs responsive to dietary/metabolic signals may further regulate immune genes. These changes can be stable (semi-permanent) and predispose to heightened inflammatory responses or defective barrier function.

4. Clinical Manifestation & Chronicity Phase

- Once epigenetic marks predispose skin cells to dysregulated responses, exposures (mechanical stress, microbes, allergens) trigger exaggerated inflammatory flares and impaired resolution — clinically manifesting as chronic or relapsing dermatoses (psoriasis, AD, etc.). Epigenetic memory may explain persistence/recurrence even after removal of immediate triggers.

5. Bidirectional Loop (Perpetuation)

- Chronic skin inflammation further sustains systemic inflammation, perpetuating epigenetic changes and creating a feedback loop that Ayurveda conceptualizes as persistent Dosha vitiation and Ama. Intervening on diet (removing Viruddha Āhāra), restoring Agni, and Rasayana-like interventions may reverse or ameliorate epigenetic states.

SUPPORTING MOLECULAR & TRANSLATIONAL RATIONALE

- DNA methylation differences have been shown in psoriasis and AD patient skin vs control skin — implicating epigenomic dysregulation in pathogenesis.
- Environmental and dietary inputs are known modulators of epigenetic machinery in other tissues; skin is similarly responsive via nutrient-sensing pathways, redox signaling and microbiome-derived metabolites (short-chain fatty acids).
- Observational and interventional nutritional studies report clinical benefit when diets reduce pro-inflammatory components or optimize anti-inflammatory nutrients — compatible with the model that diet-mediated signals alter disease activity (and potentially epigenetic state).

RESEARCH AGENDA — HOW TO TEST THE HYPOTHESIS

Study design (suggested)

1. Cohort/Case–control observational study

- Enroll subjects with chronic dermatoses and matched healthy controls. Conduct detailed dietary assessment focusing on Viruddha-type combinations (validated questionnaire adapted to local dietary practices and Ayurvedic categories). Collect skin biopsies (lesional/non-lesional) and peripheral blood.

2. Epigenomic profiling

- Perform genome-wide DNA methylation profiling (e.g., EPIC array or whole-genome bisulfite sequencing), histone mark ChIP-seq (targeted if resources limited), and miRNA sequencing in skin and peripheral blood. Correlate epigenetic signatures with reported Viruddha exposure and clinical severity.

3. Microbiome and metabolomics

- Analyze gut and skin microbiome (16S/shotgun) and circulating metabolites (SCFAs, bile acids, oxidative markers) to identify mediators linking diet to epigenome.

4. Intervention arm (pilot RCT)

- Randomize a subset of patients to a) dietary correction removing identified Viruddha combinations + Ayurvedic supportive measures (e.g., digestive restoration) vs b) standard care. Primary endpoints: change in clinical severity (PASI/EASI), secondary endpoints: changes in

epigenetic marks and systemic inflammatory biomarkers at 12–24 weeks.

Candidate biomarkers & assays

- Differentially methylated positions (DMPs) near FLG, LOR, S100 genes, cytokine loci (IL17A, IL23A).
- Global 5-mC/5-hmC quantification, DNMT and HDAC expression/activity assays.
- miRNA panels implicated in skin inflammation (e.g., miR-146a, miR-155).
- Cytokine panels (IL-17, IL-23, IL-4, IL-13, TNFα), CRP, and oxidative stress markers.
- Gut microbiome taxa and metabolites (butyrate, propionate) known to affect histone acetylation.

IMPLICATIONS FOR AYURVEDIC PRACTICE & INTEGRATIVE CARE

- If validated, the model offers a molecular basis for classical guidance to avoid Viruddha Āhāra in patients with Kuṣṭha and supports dietary correction as an epigenetically rational therapeutic adjuvant.
- Ayurvedic protocols that restore Agni, clear Ama, and modulate Dosha (pathya-apathya guidance plus Rasayana) could be studied for epigenetic reversal potential.

LIMITATIONS & CAUTION

- Association ≠ causation: observational dietary data are prone to bias; hence rigorous prospective and interventional studies are essential.
- Epigenetic changes can be cell-type specific; careful sampling (lesional vs non-lesional epidermis) and analytical deconvolution are mandatory.
- Viruddha Āhāra is a culturally contextual concept — translating exposures into reproducible variables for molecular studies requires validated instruments and cross-disciplinary collaboration.

CONCLUSION

Classical Āyurvedic teachings about Viruddha Āhāra and modern epigenetic science converge on a plausible model: chronic intake of incompatible foods may create metabolic–inflammatory milieus that reprogram the skin epigenome, lowering the threshold for chronic inflammatory dermatoses and their recurrence. A focused translational program (dietary assessment, epigenomic profiling, microbiome/metabolome characterization, and interventional trials) can test this Ayurvedic hypothesis and potentially open epigenetic targets for prevention and integrative therapy in chronic skin disease.

KEY MESSAGES (Bullet points)

- Viruddha Āhāra is classically linked to Kuṣṭha (skin disease) by Dosha vitiation and Ama formation.
- Diet and environmental factors modify the skin epigenome and are implicated in disease pathogenesis.
- DNA methylation and other epigenetic changes are consistently observed in psoriasis and atopic dermatitis.
- A testable framework links Viruddha intake → systemic inflammation/microbiome changes → epigenetic reprogramming → chronic dermatoses.
- Proposed studies: observational epigenomic correlation and small RCTs of dietary correction + Ayurvedic care with epigenetic endpoints.

SELECTED REFERENCES

1. Viruddha Āhāra: A critical view. (Review discussing incompatible foods in Ayurveda).
2. Role of Viruddha Ahara in Kuṣṭha (skin disease). IJRAP / JAIMS article (discusses Viruddha–skin links).
3. Skin Epigenetics: environmental factors including diet, microbiota, stress. (review).
4. Recent progress on DNA methylation in psoriasis; epigenetic changes implicated in psoriasis pathogenesis.
5. Dietary intervention and inflammatory skin diseases: systematic evidence of benefits. (dietary intervention review).