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Review Article

VIRUDDHA AHARA IN THE ETIOPATHOGENESIS OF *KUSHTA*: AN INTEGRATIVE REVIEW THROUGH AYURVEDIC AND BIOMEDICAL PERSPECTIVES

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ABSTRACT

Viruddha Ahara (incompatible diet), a unique concept referring to in-compactible dietary and behavioural practices is, considered as an important etiological factor in pathogenesis of *Kushta* (skin disorders). Classical text describes 18 kinds of *Viruddha Ahara* that adversely affects the metabolism that ultimately leads to diseases. Epidemiological evidence suggests a strong association between *Viruddha Ahara* and *Kushta* with *Samyoga* (incompatible combination) and *Veerya Viruddha* (incompatible potency) as most prevalent. Ayurveda conceptualizes *Kushta* as a systemic disorder originating from *Jataragni Mandya* (reduced digestive fire) that leads to *Tridosha Dushti* (aggravation of bodily humors) affecting the *Twak* (skin), *Rakta* (blood), *Mamsa* (muscle tissue) and *Lasika* (lymph). Even though the gut- skin related studies of contemporary medicine are still developing; the emerging biomedical researches supports these concepts through the mechanisms of Gut–Immune Pathway mediated by Lipopolysaccharide (LPS) initiating the Arachidonic Acid (AA) Pathway. This conceptual review analyses the *Viruddha ahara* in relation to *Kushta* using modern biomedical perspectives, highlighting the gut–immune–skin axis. Understanding these interdisciplinary correlations underscore the importance of dietary regulations for preventing and managing the skin disorders.

INTRODUCTION

Viruddha ahara (incompatible diet) is a unique concept of Ayurveda, referring to dietary incompatibility that leads to disease manifestation. In today's fast moving food culture, discussing the concept of *Viruddha Ahara* as a common risk to skin disorders are essential. Classical Ayurvedic texts describe eighteen types of *Viruddha Ahara*, namely *Desha Viruddha* (geographical incompatibility), *Kala Viruddha* (time incompatibility), *Agni Viruddha* (digestive incompatibility), *Matra Viruddha* (quantity incompatibility), *Satmya Viruddha* (suitability incompatibility), *Dosa Viruddha* (humoral incompatibility), *Samskara Viruddha* (processing incompatibility), *Veerya Viruddha* (potency incompatibility), *Koshta Viruddha* (gut incompatibility), *Avastha Viruddha* (condition incompatibility), *Krama Viruddha* (condition incompatibility), *Parihara Viruddha* (avoidance of dietary rules), *Upachara Viruddha (*therapeutic management incompatibility), *Paaka Viruddha* (cooking incompatibility), *Samyoga Viruddha* (combination incompatibility), *Hridya Viruddha* (palatability incompatibility), *Sampad Viruddha* (quality incompatibility) and *Vidhi Viruddha* (avoidance of rules) [1]. *Kushta* is described in ayurveda as a disease arising from the vitiation of all three *Doshas* predominantly involving the *Twak* (skin), *Rakta* (blood), *Mamsa* (muscle tissue) and *Lasika* (Lymph). The causative factors are improper diet, lifestyle, and suppression of natural urges. An epidemiological study conducted by the Central Council for Research in Ayurvedic Sciences (CCRAS)[2] among patients suffering from *Kushta* revealed a significant association between *Viruddha Ahara* and the disease. The study reported *Samyoga Viruddha* and

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Virya Viruddha as the most prevalent forms of dietary incompatibility, observed in 73% of patients. This was followed by *Karma Viruddha* in 62%, *Matra Viruddha* in 44.33%, and *Samskara Viruddha* in 43.14% of patients. *Avastha Viruddha* was observed in 14.11% of cases Although this study was conducted in 2015, post–COVID-19 observations indicate a further increase in the prevalence and chronicity of skin disorders[3]. Ayurvedic understanding of disease pathogenesis as originating primarily from the *Amashaya*, with the skin serving as one of the sites of its clinical manifestation. So, *Kushta*, in Ayurveda even before thousands of years back is understood as not merely a dermatological disorder but a systemic disease rooted in impaired digestion, metabolic dysfunction. While modern medical science acknowledges the role of diet in triggering or exacerbating skin disease flare-ups, the underlying biochemical mechanisms remain incompletely understood. Moreover, the multidimensional framework of *Viruddha Ahara*- digestion, metabolism, tissue interaction, and disease manifestation has not been adequately explored through contemporary scientific lenses. This article aims to bridge these conceptual gaps by critical analysing *Viruddha Ahara* relation to *Kushta* with emerging modern biomedical perspectives.

AIM

To critically analyse *Viruddha Ahara* in *Kushta* using modern biomedical perspectives

MATERIALS AND METHOD.

The classical texts along with peer-reviewed biomedical literature accessed through PubMed, PMC, and related scientific databases.

Nidanas* (cause) of *Kushta

On analysing the various *Nidana*, we can group them as *Adhyashana* (over eating before digestion) of high fat, sugar and protein rich *Ahara* with less fibre, *Viruddha Ahara*.

***Nidana* and its possible modern biochemical correlates**

Improper dietary habits play an important role in the pathogenesis of skin disorders. All *Nidana* of *Kushta* are pointing toward different biochemical changes in body. One is Gut–Immune Pathway mediated by LPS.

The regular intake of *Ahara* such as *Phanitha* (jaggery-based preparations)/ meat/ pulses/ oil are characterized by high fat and high protein content with low in dietary fibre. Continues intake of such ahara over a prolonged time leads to significant alterations in gut health.

Effect on intestinal mucosa

Dietary fibres are necessary for nourishment and growth of gut bacteria such as lactobacillus and bifidobacterium. This bacteria's help in maintaining the cell integrity. However, diet with low fibres and result in the reduction of these beneficial bacteria. Simultaneously, there is a significant increase in harmful gram-negative bacteria, such as *Escherichia coli*. These thrive in high-fat dietary, bile salt (sour food intake can increases the secretion of bile) predominant conditions and do not require fibre for growth[4].

Biochemical and Cellular Changes

Gram-negative bacteria possess lipopoly-saccharides (LPS) in their outer cell wall. Extensive proliferation of these bacteria will increase the production and release of LPS into the gut. LPS are endotoxins and they are glycolipids LPS are found in healthy human blood in non-lethal doses does not cause inflammation. In people with obesity and individuals who consumes *Krama* and *Avastha Viruddha Ahara* (eating heavy meals after sexual activity, heat exposure, or strenuous exercise) are susceptible to increased intestinal permeability and in such humans (with gut dysbiosis) the production of LPS and its uptake is directly proportional to the amount of fat intake. Dietary fat increases LPS absorption through chylomicron-mediated transport mechanisms, exacerbating its systemic exposure. LPS may translocate across the intestinal epithelium via transcellular mechanisms, ultimately entering to the bloodstream[5].

LPS-induced postprandial endotoxemia

Once the LPS mixes with blood more than the lethal doses, it induces the postprandial endotoxemia, a condition of low-grade inflammation that may persists for up to eight hours following a meal. The circulating LPS will be recognized by Toll-like receptor 4 (TLR-4), a key pattern-recognition receptor of the innate immune system. The TLR-4 activation with then signal macrophage mediated cytokine production, activating nuclear factor-κB (NF-κB), and assembly of the NOD-like receptor protein 3 (NLRP3) inflammasome.

NLRP3 Inflammasome Signalling and Th17-Mediated inflammatory response

Nuclear factor-κB (NF-κB) will produces pro-interleukin-1β (pro-IL-1β) and pro-interleukin-18 (pro-IL-18) and NLRP3 convert it into biologically active forms, IL-1β and IL-18. These cytokines play an important role in promoting T helper 17 (Th17) cell differentiation and activation. Th17 will bind to their specific respective cellular receptors. It induces the hyper proliferation thereby contributing to chronic inflammatory and immune-mediated pathologies.

Arachidonic Acid (AA) Pathway as a link

In addition to the inflammasome driven cytokine signalling, the AA acts as a link between metabolic stress and cutaneous manifestations. Persistent exposure to LPS will activate phospholipids A2, an enzyme responsible for the release of AA. AA is metabolized through cyclooxygenase (COX) and lipoxygenase (LOX) pathways to generate pro-inflammatory eicosanoids. These mediators activate the dendritic cell and enhance Th17 cell differentiation and signalling. The resulting amplification of the IL-23–IL-17 axis promotes keratinocyte hyper-proliferation and sustained cutaneous inflammation

Milk-Based *Viruddha Ahara:* Digestive and Immune Implications

Certain *Nidanas* in *Kushta* that including simultaneous conception of milk and milk-based products with other protein and fats has not been extensively investigated in contemporary biomedical researches, yet possible inferences can be drawn based on the existing nutritional evidences.

Milk proteins, casein and whey, are known for its delay in gastric emptying and prolonged digestive process. Casein in particular, forms a coagulum in stomach resulting in slower digestion compared to many other dietary proteins. When food contain different proteins are taken together, the overall digestive efficacy is compromised. Meat proteins like myofibrillar protein get absorbed quickly than casein. Such combinations require the coordinated action of multiple digestive enzymes working at different pH levels, potentially impairing the digestive process. As prolonged gastric time, increase satiety while promoting abdominal distension, delayed digestion, and incomplete nutrient absorption[6]. This can contribute to gastrointestinal discomfort, malabsorption, and the accumulation of toxins and intestinal dysbiosis.

Heat–Cold Behavioural *Nidanas (Krama/Avastha Viruddha)*

The practice of alternating cold exposure including cold water immersion following exercise induce hyperthermia, is widely accepted as gold standard recovery intervention. Despite this the ayurvedic literature on such procedure is considered as *Viruddha*. The response to such protocol remains limited, and systematic research is scarce. Some of the potential adverse effects have been documented till now. They are cardio vascular risks Abrupt immersion in cold water following exercise-induced hyperthermia induces peripheral vasoconstriction and elevated blood pressure, imposing acute stress on the cardiovascular system[7]. The neuro muscular impairment is another effect. Cold exposure reduces muscle temperature, slowing nerve conduction velocity and impairing muscle contractility. Next is Immune Modulation and Stress Hormones. Cold exposure acts as an acute physiological stressor, triggering elevated catecholamine and cortisol levels. Following strenuous exercise, immersion in water below 15°C can induce a "cold shock" response characterized by heightened sympathetic activity and altered immune cytokine expression of IL-6, IL-10 and TNF-α. This acute stress over time may influence the process of chronic inflammation in the body [8].

Sudden cold exposure following heat induces rapid cutaneous vasoconstriction and heat induced vasodilation, leading to transient ischemia and reactive hyperthermia[9]. This changes the stratum corneum lipids, that disrupt the skin barrier. Thermal stress also stimulates keratinocyte and dermal immune cells by elevating IL-1, IL-6, and TNF-α[10]. Repeated heat–cold cycles can enhance oxidative stress and immune activation, potentially aggravating dryness, irritation, or inflammatory skin conditions[11].

Genetic predisposition, Psychological Stress and Immune Axis

Here, *Paapa Karma* may be understood as the cumulative influence of individual's history or the prenatal, intergenerational factors. Supporting this view, several studies have shown that prenatal maternal stress, anxiety, and emotional disturbances are associated with increased risk of psoriasis in offspring[12]. Researchers have proposed multiple explanations for these associations, including the overlap of gene involved in the mood regulation and inflammatory skin disorders.

In addition maternal anxiety can lead to alteration in the DNA methylation pattern in the developing foetus, which may impair normal skin function and promote inflammatory process[13]. Elevated maternal cortisol levels by HPA axis can also enter in to the fetal circulation and interact with the mast cells and program them to be more prone for inflammation and cause early skin disorders[14].

The summary of biochemical changes that can be caused due to *Nidanas* are mentioned in Table.no.1

Table 1: Possible Biochemical changes by exposure to *Nidana*

Nidanas	**Examples**	**Ayurvedic understanding**	**Possible Patho-physiological Changes**
Genetic Predisposition	*Paapa karma*	*Sahajavyadhi* in Ayurveda.	HLA associations (HLA-Cw6, HLA-DR7, HLA-B13, and HLA-Bw57) in psoriasis[15] FLG mutation in atopic dermatitis
Regular intake of *Guru* (heavy), *Snigdha* (moist), *Abhishyandi* (that can create obstruction in channels) *Ahara*, excessive *Snehana* (olation)	High in fat and protein. Less in fibre	*Kleda Vardanam*	Obesity, LPS-Mediated Gut–Immune Pathway
Samyoga Viruddha	Intake of *Hayanaka (*type of rice), *Yavaka (Hordeum vulgare), Ciṇaka (Panicum miliaceum), Udalaka (Paspalum scrobiculatum), Koraduṣa (Paspalum scrobiculatum)* along with milk, curd, buttermilk.	*Ama Visha Utpatti* (production of toxin)	Various Protein–fat–incompatibility→ immune activation[16], enzyme dysfunction
Avastha Viruddha	Immersion in cold water immediately after heat exposure. Eating heavy meals after sexual activity, heat exposure, or strenuous exercise.	The *Ushnatha* (heat) stays inside the body and keep the *Swedavaha* (channel of sweat) *Shrotas* obstructed and *Kleda* (moist) stays inside. By the *Ushnata* trapped inside, *Raktha Dushti* also happens.	Autonomic nervous system imbalance causing incomplete digestion, LPS translocation and inflammation.[17]
Virya Viruddha	Consumption of fish with milk	Mixing of *Sheeta Ushna Veerya* may affect the homologation and *Raktha Dushti* due to *Ama Visha Utpatti.*	Delayed digestion causing dysbiosis, malabsorption and stress.[6]
Krama Viruddha	Consuming *Vidahi ahara* (food that cause burning sensation)/heavy food, *Lakuca (Artocarpus lacucha)*, *Mulaka*) *Raphanus sativus* L), and *Kakamaci* (*Solanum nigrum)* during indigestion.	*Ama Utpatti* due to delayed digestion	Induces more bile secretion that makes and environment to grow gram negative bacteria[4].
Repeated *Viruddha Ahara Sevena*		*Tridosha Dushti*	Persistent immune activation, Th17 pathway dominance, chronic systemic inflammation.
Manaseeka Nidana	*Chinta* (thought), *Bhaya* (fear)	*Vataprakopa.*	Cortisol imbalance and worsens the flares.[18]

CONCLUSION

Ayurvedic pathophysiology describes *Kushta* as a systemic disorder and it is not extensively studied by the modern researchers. This is a humble attempt to correlate the findings for *Acharyas* to available research findings. Critical analysis of *Viruddha Ahara* reveals its potential role as a dietary trigger capable of initiating and these biochemical and immunological disturbances. Thus, *Viruddha Ahara* emerges as a possible etiological factor linking classical Ayurvedic concepts with modern gut–immune–skin axis models. This integrative understanding underscores the importance of dietary regulation in both prevention and management of chronic inflammatory skin diseases and highlights the need for further interdisciplinary research.

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