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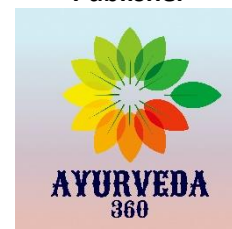
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DOI: [10.63247/3048-7390.vol.1.issue6.11](https://doi.org/10.63247/3048-7390.vol.1.issue6.11)**A Comprehensive Review on Dermatophytosis With Special Reference to Dadru**Gill G.K.¹, Yadav C.R.²

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ABSTRACT**Introduction:**

Dermatophytosis is a globally prevalent superficial fungal infection involving keratinized tissues, primarily caused by dermatophytes such as *Trichophyton*, *Microsporum*, and *Epidermophyton*. Tinea corporis and tinea cruris are among the most common clinical variants, closely resembling the Ayurvedic condition *Dadru Kushtha*, marked by circular, pruritic, erythematous lesions with recurrent tendencies. The increasing incidence, therapeutic resistance, and chronicity observed in clinical settings demand a comprehensive understanding through both biomedical and traditional perspectives. This study is aimed to review the clinicopathological features and therapeutic approaches of dermatophytosis with special reference to its correlation with *Dadru* in Ayurvedic literature.

Methods:

A narrative synthesis was conducted using contemporary dermatological literature and classical Ayurvedic texts, focusing on clinical-pathological parallels.


Discussion:

Dermatophytosis affects approximately 20-25% of the global population, with heightened prevalence in tropical regions like India. The condition typically presents with annular plaques, raised inflammatory margins, central clearing, and pruritus. Pathogenesis involves enzymatic degradation of keratin and host immune activation.


Conclusion:

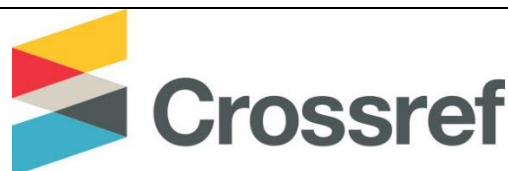
Management of dermatophytosis, especially *Dadru* like phenotypes, necessitates an integrative approach involving targeted antifungal therapy and individualized preventive strategies. Recognizing its clinical diversity and aligning therapeutic goals with both modern and traditional paradigms may enhance treatment outcomes.

Keywords: *Dadru*, Dermatophytes, Dermatophytosis, Keratinophilic Fungi, Tinea.

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Introduction

Fungal infections impose a significant burden in dermatological practice, with dermatophytosis constituting the most prevalent clinical manifestation. Dermatophytosis refers to a group of superficial fungal infections caused by dermatophytes (filamentous fungi) with a unique capacity to invade and utilize keratin as a nutrient source. These infections are primarily restricted to keratinized structures such as the outer epidermis, nails, and hair.^[1] These infections, colloquially termed ringworm or tinea, are often categorized according to the site of involvement (e.g., tinea corporis, tinea cruris).^[2] While typically superficial, certain cases—particularly in immunocompromised hosts, can demonstrate deeper invasion. Dermatophytic infections elicit a spectrum of host responses, ranging from mild desquamation to marked inflammation, depending on the organism's virulence and ecological source (anthropophilic, zoophilic, or geophilic).^[3] In Ayurvedic parlance, the condition correlates with *Dadru Kushtha* distinguished by elevated circular lesions (*Mandala*), itching (*Kandu*), erythema (*Raga*), and papulo-vesicular eruptions (*Pidika*).^[4]

This review aims to synthesize contemporary evidence on the etiopathogenesis, epidemiological

distribution, clinical morphology, diagnostic approaches, and evolving therapeutic strategies for dermatophytosis, with a particular emphasis on correlating its clinical manifestations with the Ayurvedic construct of *Dadru*.

Materials & Methods

This review adopts a narrative approach, extracting and synthesizing content from contemporary dermatological literature, peer-reviewed journals, and classical Ayurvedic treatises. A comprehensive literature search was conducted using key terms such as 'dermatophytosis,' 'dermatophytes,' and Boolean combinations like 'dermatophytosis AND prevalence.

Literature Review

Etiology

Classification of Mycoses: Cutaneous fungal infections are stratified based on depth and anatomical involvement. Superficial mycoses, including dermatophytosis, are limited to the stratum corneum, hair shafts, and nails. Subcutaneous and systemic mycoses involve deeper dermal structures or internal dissemination.

Dermatophyte Characteristics:

Dermatophytes are specialized fungi inhabiting keratinized tissues but sparing mucosa. The primary genera include *Trichophyton*, *Microsporum*, and

Epidermophyton, each displaying tissue-specific predilections.^{[5],[6]}

- *Trichophyton* species commonly infect skin, nails, and hair; *T. rubrum* is the most ubiquitous.
- *Microsporum* affects skin and scalp, particularly in pediatric tinea capitis.
- *Epidermophyton floccosum* is restricted to skin and nails, especially in tinea cruris and pedis cases.

Ecological and Transmission

Classifications: Based on transmission ecology, dermatophytes are further categorized as:^[7]

- **Anthropophilic:** Primarily infect humans, typically causing chronic, non-inflammatory lesions.
- **Zoophilic:** Acquired from animals, often inciting intense inflammation.
- **Geophilic:** Originating from soil; moderately inflammatory and usually self-limiting

Epidemiology

Global estimates suggest that dermatophytosis affects approximately 20–25% of the population.^[8] Notably, India exhibits regional heterogeneity, with northern areas reporting prevalence as high as 61.5%.^[9] Factors such as humidity, overcrowding, and corticosteroid misuse contribute to its endemicity.

Pathogenesis

The pathogenesis of dermatophytosis entails a stepwise interaction between the dermatophyte organism and host epidermal defenses, ultimately culminating in the characteristic inflammatory cutaneous lesions. This process can be broadly categorized into three interrelated phases: adhesion and colonization, keratin degradation and penetration, and host immune response.^{[10],[11]}

a) Adhesion and Colonization

The pathogenic cascade initiates when arthroconidia (the infectious fungal spores) come into contact with keratinized epithelial surfaces such as the stratum corneum. This initial adherence is not passive but is mediated by:

- Fungal adhesins, including carbohydrate-specific proteins and peptidases
- Hydrophobic interactions between fungal cell walls and host keratin
- Secretion of dermatophyte-specific proteases, enhancing surface binding

Once adhered, the arthroconidia germinate into hyphae, which extend radially, often in multiple directions, thereby laying the groundwork for annular lesion formation.

b) Keratin Degradation and Epidermal Invasion

Following successful colonization, dermatophytes initiate the degradation of host keratin, their principal nutrient source. This process unfolds via two major biochemical pathways:

- **Sulfitolysis:** Dermatophytes secrete sulfite-reducing enzymes, disrupting cystine disulfide bridges within keratin filaments. This structural cleavage is essential to unravel the proteinaceous barrier.
- **Proteolysis:** A diverse arsenal of proteolytic enzymes such as keratinases, endoproteases, and exoproteases, facilitates the breakdown of partially denatured keratin. These enzymes also liberate low molecular weight peptides and amino acids to sustain fungal metabolism.

As a result, the dermatophyte progressively penetrates the stratum corneum, advancing toward deeper non-viable epidermal layers, without breaching the viable dermis in immunocompetent individuals.

c) Host Immune Response and Inflammatory Manifestations

The progression of fungal elements within the epidermis is typically accompanied by the diffusion of fungal metabolites such as secreted enzymes, metabolic byproducts, and cell wall components into the Malpighian layer.

These act as pathogen-associated molecular patterns (PAMPs) and activate pattern recognition receptors (PRRs) on host keratinocytes and resident immune cells.

Key outcomes of this immunological engagement include:

- **Erythema and edema**, mediated by vasodilation and local cytokine release
- **Pruritus**, triggered by interleukin-mediated nociceptor activation
- **Vesicle or pustule formation**, particularly in zoophilic infections due to intense delayed-type hypersensitivity (Type IV reaction)

Clinical Features

Dermatophytosis typically presents with:

- Annular plaques exhibiting raised, inflamed borders
- Centrifugal progression with central clearing
- Associated signs: scaling, pruritus, papulo-vesicles, and in chronic cases, lichenification or post-inflammatory hyperpigmentation

These features are a direct result of localized fungal proliferation, tissue degradation, and host inflammatory responses.^[12]

Clinical Spectrum

The disease manifestations vary by anatomical site and fungal species:

- **Tinea corporis:** Round, red, scaly lesions on the trunk/limbs
- **Tinea capitis:** Patchy hair loss with inflammation, common in children
- **Tinea barbae:** Inflamed pustules or kerion in the beard region
- **Tinea faciei:** Erythematous facial patches, sometimes steroid-modified
- **Tinea pedis:** Peeling, fissuring, or vesicles between toes and soles
- **Tinea cruris:** Itchy plaques on groin/thighs with central clearing
- **Tinea manuum** and **Tinea unguium:** Unilateral hand scaling or nail dystrophy

Diagnostic Approaches

Different diagnostic approaches used in clinical practice for the identification of dermatophytes are mentioned below:^[13]

- Wood's Lamp Examination:** Useful for *Microsporum* infections exhibiting green-yellow fluorescence.
- KOH Mount Microscopy:** A rapid, low-cost method revealing hyphae in skin scrapings.
- Fungal Culture:** The gold standard; employs Dermatophyte Test Medium (DTM) to confirm species identity, though colony morphology may be altered.

- Histopathology and PCR-based diagnostics** are reserved for atypical or resistant cases.

Therapeutic Modalities

Therapeutic approach for the management of dermatophytosis hinges on lesion location, extent, chronicity, and host factors. It includes the following approaches and measures:^[14]

- **Topical Antifungals:** Azoles (clotrimazole, miconazole), allylamines (terbinafine), and ciclopirox formulations serve as first-line options for localized lesions.
- **Systemic Therapy:** Indicated in extensive, resistant, or nail and scalp involvement. Agents include terbinafine, itraconazole, and griseofulvin.
- **Preventive Measures:** Include maintaining dryness, avoiding occlusive clothing, and treating fomites.

Discussion

The resurgence and chronic persistence of dermatophytosis, particularly in the Indian subcontinent, reflect a multifactorial pathogenesis involving microbial adaptability, host predisposition, and therapeutic lapses, including unsupervised use of topical corticosteroids. Increasing reports of antifungal resistance further underscore

the need for species-specific diagnosis and rationalized treatment strategies.

In the Ayurvedic framework, *Dadru Kushtha* is understood as a manifestation of *Pitta-Kapha Dosha* vitiation, with *Rakta*, *Tvak*, *Mamsa*, and *Lasika* serving as the primary *Dushya*

(affected tissues).^{[15],[16]} The cardinal features such as *Raga* (redness), *Kandu* (itching), *Utsanna Mandala* (raised lesions), and *Pidika* (eruptions), exhibit strong parallels with the clinical presentation of *tinea corporis* and *cruris*.

This correlation is summarized below:

Table 1: Clinical Feature Correlation between Dermatophytosis and *Dadru*

Dermatophytosis Clinical Feature	Ayurvedic Correlate (<i>Dadru</i>)	Explanation
Annular erythematous lesion with raised margin	<i>Raga</i> (redness), <i>Utsanna Mandala</i> (raised plaque)	<i>Pitta-Rakta</i> vitiation causing inflammatory elevation
Central clearing with peripheral spread	<i>Mandala Rupa</i> (circular configuration)	Centrifugal growth pattern linked to <i>Kapha-Pitta Prakopa</i>
Pruritus (itching)	<i>Kandu</i> (itch)	Indicative of <i>Kapha-Vata</i> aggravation in the superficial <i>Srotas</i>
Vesiculation or pustules in inflammatory forms	<i>Pidika / Srava</i> (pustule / exudation)	Result of <i>Rakta-Pitta Dushti</i> leading to <i>Srava</i> (discharge)

From a modern biomedical standpoint, these features are largely explained by the keratin-degrading activity of dermatophytes and the immune-mediated inflammatory cascade they provoke. Notably, the chronicity and tendency for relapse in *Dadru* mirror the clinical behavior of dermatophytosis, particularly in contexts involving biofilm formation and persistent subclinical colonization.

Hence, effective management must transcend symptomatic pharmacological suppression. It demands a comprehensive correction of both internal imbalances and

external risk factors. An integrative therapeutic model, merging antifungal pharmacotherapy with *Ayurveda*-guided lifestyle, dietary, and internal detoxification protocols, may provide a more sustainable and recurrence-resistant approach to management.

Conclusion

Dermatophytosis, when viewed through the dual lenses of biomedical and Ayurvedic frameworks, underscores the necessity of holistic intervention. With the rising prevalence and resistance patterns, tailored therapeutic strategies, early diagnosis, and public awareness assume

critical importance. A clearer understanding of the pathophysiological convergence between *Dadru* and tinea

infections can refine clinical protocols and inform future integrative research trajectories.

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