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Dermatophytoses: Taxonomy, Pathogenesis, Diagnosis, Resistance, and Management

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Abstract

Dermatophytes, belonging to the family Arthrodermataceae, are keratinophilic fungi responsible for superficial mycoses. Dermatophytoses affect the skin, hair, and nails globally. Over the last few years, dermatophytic infections have seen a sharp rise in India, with the main causative agent being antifungal-resistant Trichophyton mentagrophytes genotype VIII. This review provides a detailed overview of dermatophytoses, including taxonomy, pathogenesis, epidemiology, diagnosis, and treatment. Dermatophytes belong to the kingdom Fungi, family Arthrodermataceae, with seven different species, including Trichophyton, Nannizzia, and Epidermophyton. Dermatophytes cause infection by the adhesion of arthroconidia to keratinized tissues followed by invasion via the production of keratinases. Dermatophytoses present with different manifestations, with the majority being tinea corporis (32.4%), followed by tinea cruris (19.7%), onychomycosis (17.9%), and tinea capitis (13.3%) in the Indian population. Epidemiologically, the infections were previously caused by Trichophyton rubrum, which is now being replaced by antifungal-resistant Trichophyton mentagrophytes, responsible for 64% cases. Factors contributing to the epidemiological shift include the widespread misuse of topical corticosteroids, the prevalence of diabetes mellitus, and the effects of COVID-19. Potassium hydroxide (KOH) microscopy is the diagnostic tool with the highest sensitivity, ranging from 70 to 90%, followed by culture and internal transcribed spacer polymerase chain reaction with 95 to 99% sensitivity. However, the main drawback is the high resistance to terbinafine, with mutations in the squalene epoxidase (SQLE) gene, including Leu393Phe and Ala448Thr mutations, seen in 50 to 65% cases in the Indian population. Terbinafine is the drug of choice, with oral terbinafine being the first-line treatment with cure rates ranging from 70 to 90%, although the widespread resistance to terbinafine is the biggest drawback in the treatment of dermatophytoses. Combination therapy is required to overcome the resistance. Prevention is simple, with emphasis on personal hygiene and avoidance of fomites.

Keywords: Dermatophytes, *Trichophyton mentagrophytes*, antifungal resistance, dermatophytosis, terbinafine

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INTRODUCTION

Dermatophytes represent a phylogenetically cohesive clade within Ascomycota's Onygenales, specialized for keratin degradation in keratinized tissues – skin stratum corneum, hair cortex, nail plates [1]. Afflicting over 1 billion annually, these infections impose substantial morbidity, with economic losses exceeding $4 billion in therapeutics alone [5]. Ancient texts, like the Ebers Papyrus (1500 BCE), describe "tegna impetiginosa" scalp alopecia, while modern epidemics underscore adaptive evolution [3].

Structurally dimorphic, dermatophytes produce mycelial colonies in vitro (cottony/pleomorphic)

and arthroconidia (2–5 μm) in vivo for fomite transmission, persisting 4.5 years environmentally [1]. Genomes (22–26 Mb, ~8000 genes) feature expanded protease superfamilies: 20+ subtilisins (Sui1-6), metalloproteases (Mep1-7), sedolisins, upregulated 100-fold on keratin induction (pH 5.5–7.5 optima) [25, 43]. Ecological niches stratify virulence: anthropophiles (*T. rubrum*, *T. tonsurans*) exhibit gene decay (fewer adhesins, reduced MAT loci), yielding chronic mild infections; zoophiles (*M. canis*, *T. verrucosum*) retain inflammatory mannans/phenolics; geophiles (*N. gypsea*) sporadically infect via soil keratin [1, 6, 8, 9,11, 12, 15, 16, 19].

Taxonomic revolution via multilocus sequencing (ITS, TEF3α, RP60S) adopted "One Fungus=One Name," consolidating *Arthroderma* teleomorphs into *Trichophyton* (45 species), Nannizzia (9), *Paraphyton*, *Lophophyton* (1,16,29). *T. mentagrophytes* complex bifurcated: interdigitale (anthro/zoophilic), genotype VIII (*T. indotineae*, India epidemic) (2,28). Table 1 quantifies these divergences – *T. rubrum* (22.5 Mb) versus *N. canis* (24.1 Mb) – correlating size with host range [1].

Pathogenesis initiates with arthroconidia hydrophobins binding corneocyte integrins, resisting complement C3b via melanin (Mf1 gene). Germ tubes secrete KER1-3 keratinases cleaving disulfide bonds, lipases breaching cuticles, and gliotoxin suppressing phagocytes [1]. Invasion confines to non-viable keratin by host IL-17/Th17 barriers; breaches yield Majocchi granuloma (Table 2) [27]. Chronicity reflects IL-10 tolerance in anthropophilic infections [23, 25, 30, 31, 32, 37].

India's "tinea epidemic" exemplifies globalization: T. mentagrophytes VIII surged from 31% (2011–15) to 64% (2016–21), analyzing 56,000 cases amid OTC betamethasone-terbinafine combos fostering resistance (Tables 2, & 3) [2, 3]. Urban tropics amplify via humidity (>70% RH), overcrowding, and diabetes (30% diabetics affected) (3). This review dissects these facets through tables, furnishing actionable insights [41].

PATHOGENESIS AND HOST INTERACTION

Dermatophyte pathogenesis represents a highly specialized, multi-phase process optimized for superficial colonization of keratinized tissues, balancing aggressive invasion with immune modulation to evade clearance while avoiding deep-tissue dissemination [1, 33]. The lifecycle initiates with arthroconidia dissemination via direct contact, fomites, or soil/animals, landing on abraded skin where humidity and temperature (28–32°C) trigger reactivation [33]. These dormant propagules (2–5 × 3–8 μm) possess robust cell walls enriched in β-1,3-glucans and chitin, conferring desiccation resistance up to 4.5 years [1].

Adhesion Phase (0–6 hours): Hydrophobic class I hydrophobins (Hfb1-3) and ALS3-like glycoproteins mediate corneocyte attachment, targeting keratin K1/K10 α-helices via lectin–carbohydrate bonds and electrostatic forces (10–20 nN adhesion strength per atomic force microscopy) [25, 34]. *T. rubrum* upregulates hydrophobins 5–10-fold during early keratin contact, enhancing cell wall hydrophobicity and virulence [34]. Fibrinogen-binding proteins further anchor conidia, resisting desquamation [33].

Germination and Penetration (6–48 hours): Metabolically reactivated, conidia form appressoria-like structures, secreting sulfite via Ssu1 efflux pump to sulfitolyze keratin disulfide bridges (Cys-S-S-Cys → Cys-SH + S-SO3^2-), rendering compact matrices protease-accessible – exclusive to dermatophytes (33,38). This precedes the keratinase cascade: serine-subtilisins (SUB1-6, MFSui1) initiate endoproteolysis of α/γ-keratins into oligopeptides; fungalysins (Mep1-7, M28 family) cleave elastin/hair cuticles; sedolisins (Tri1-3) and aspartyl proteases (Saps1-4) generate amino acids (Ala, Gly, Ser dominant) for assimilation (33,38,43). L-cystine induces *T. mentagrophytes* Cdol (cysteine dioxygenase), upregulated 20-fold on keratin, detoxifying sulfite while fueling cysteine metabolism [38]. Lipases (Lip3-5) hydrolyze sebum triglycerides, phosphatases liberate Pi, and reductases (Thi1) sustain redox balance (25). *T. rubrum* expresses 40+ such genes (vs. 10 in saprobes), with *T. mentagrophytes* VIII showing 2–10x faster kinetics correlating to epidemicity [2, 43]. Penetration rate exceeds stratum turnover (14 days), confining to cornified layers via hyphal septation [1].

Immune Evasion and Persistence: Dermatophytes orchestrate tolerance. Mf1 melanin quenches H2O2/- OH (ROS); gliotoxin blocks NF-κB, inhibiting IL-1β/IL-6 [25]. Saps degrade IgA1 hinges and C3b opsonins; fumagillin halts phagosome maturation [33]. Trehalose (5–15% dry weight via Tps1/2) osmoprotects against hypertonicity and cathelicidins [25, 45]. Biofilms in onychomycosis – *T. rubrum* EPS matrices (β-glucans 40%, mannoproteins 30%, eDNA) – reduce azole susceptibility 100–1000 fold, with viability >80% post-amikacin challenge.

Host Immunity Bifurcates: Acute (zoophilic/geophilic): Langerhans cells (LCs, CD207+) and dermal DCs (CD1a+) process antigens via Dectin-2/Mincle, activating NLRP3 inflammasome and CARD9 signaling to drive Th17 differentiation (TGF-β, IL-1β, IL-6, IL-23). Th17 cells secrete IL-17A/F (10–50 pg/mL lesional), IL-22 recruiting neutrophils (PMNs) producing NETs/calprotectin and keratinocytes synthesizing HBD-2/3, dermcidin (antifungal peptides killing 90% conidia in vitro) [35, 38]. IL-22 upregulates S100A7/A8/A9, amplifying barrier [35]. Chronic (anthropophilic): Th2 skewing (IL-4/IL-13 20–100 pg/mL) via fungal proteases cleaving PAR-2 receptors promotes Tregs (FoxP3+, TGF-β), suppressing Th17/Th1 (IFN-γ low) for persistence [35, 40]. Guinea pig models confirm Th17 clearance (*T. benhamiae*), Th1 downmodulating inflammation [39]. Defects (STAT3^mut, CARD9^loss) enable deep dermatophytosis [35]. Diabetics show impaired PMN chemotaxis (CXCL8 low), HIV CD4<200 depletes effectors.

Predispositions amplify trauma breaches barriers, occlusion traps moisture, immunosuppression (steroids blunt IL-17). Table 1's ecology predicts: anthropophiles (*T. rubrum*) evade via Th2 tolerance; zoophiles (*N. canis*) provoke Th17 flares [1, 35].

CLINICAL FEATURES AND EPIDEMIOLOGY

Clinical Spectrum (Table 2): Tinea corporis (32.4% India, *T. mentagrophytes* 50%): annular plaques (1–15 cm), polycyclic extension, vesiculation if inflammatory; extensive (>30% BSA) in 20–30% epidemic cases [3, 20]. Tinea cruris (19.7%, *T. rubrum* 40%, *E. floccosum* 25%): symmetric inguinal erythema advancing to buttocks/thighs, maceration, satellite lesions; 60% recurrence untreated [3]. Onychomycosis (17.9%, *T. rubrum* 70%): distal lateral subungual (DLSO 80%), proximal (immunosuppressed), total dystrophic (hyperkeratotic debris); paronychia with Pseudomonas superinfection [10]. Tinea capitis (13.3%, children <12): endothrix (*T. tonsurans* 30%: "black dots," scarring alopecia); ectothrix (*T. violaceum* 25%: fluorescing hairs); favus (*T. schoenleinii*: scutula crusts); inflammatory kerion (zoophilic, *M. canis* 20%, fluctuant boggy swellings resolving scarred) [7]. Tinea pedis (4.1%, *T. rubrum* 60%): interdigital (maceration/erosions), moccasin (hyperkeratosis), vesiculobullous (*T. mentagrophytes*) [13]. Tinea manuum (mirror pedis, unilateral); faciei (non-scaling, photosensitive); barbae (folliculitis); unguium corporis (extensive steroid-abetted); Majocchi (dermal nodules, 1–2%, immunosuppressed) [3, 27]. Inflammatory variants: id reactions (distant vesicles), Majocchi granuloma (perifollicular granulomas on biopsy PAS+) [27].

Epidemiology: Global 20–25% prevalence (1–2B cases/year), highest tropics (India 6–61.5% community surveys) [20]. Table 3 (161k isolates): 1939–60 *T. rubrum* (60%, rural zoonotic *M. canis* 20%); 1971–80 *T. violaceum* capitis (57%, urban migration); 2001–10 *T. rubrum* 45%; 2011–15 transitional *T. mentagrophytes* 31%; 2016–21 VIII 64%; 2022–25 proj. 70–75% amid diabetes (140M Indians), TB (2.6M), HIV (2.4M), post-COVID. Males 1.9–3:1 (occlusion); bimodal peaks (children capitis, adults corporis/cruris); urban 70% (crowding, AC use) (20). Zoonoses 15–25% (*M. canis* 70% pet cats/dogs, *T. verrucosum* cattle) [3]. 2024–26 data: North India 68% VIII, recurrence 40–60% (steroids, non-compliance); South 55% [36]. Economic: $500M+ India/year; global $12B [5]. Drivers: OTC combos (clobetasol–terbinafine, 40% misuse), climate (monsoons), migration [2, 20]. Table 3 signals One Health integration [22].

These elucidate pathogenesis-clinical links: protease sulfitolysis enables superficiality; Th17/Th2 dictates inflammation [35, 40]; epidemiology resistance interplay [2, 3].

DIAGNOSIS

Accurate diagnosis of dermatophytoses remains paramount amid rising antifungal resistance and atypical presentations, particularly in endemic regions, like India, where T. mentagrophytes genotype VIII mimics eczema or psoriasis [3, 17]. A tiered approach integrates clinical acumen, direct microscopy, culture, and molecular tools, with Table 4 benchmarking their metrics: KOH (70–90% sensitivity, minutes), culture (gold standard, weeks), PCR (95–99%, hours) [4, 17, 42].

- *Clinical Diagnosis:* Characteristic morphology guides suspicion – annular scaly plaques with advancing borders (corporis), inguinal erythema sparing scrotum (cruris), dystrophic nails (onychomycosis), or alopecia with black dots/kerion (capitis) [3, 42]. However, steroid misuse yields "tinea incognito" (disseminated, pustular, lacking scale), confounding 30–40% Indian cases. Differentials include psoriasis (silver scale, nail pitting), nummular eczema (confluent scale), granuloma annulare (smooth), pityriasis rosea (herald patch), candidiasis (satellite pustules), erythrasma (Wood's coral–red), and subacute cutaneous lupus (sun-exposed) (44). Tinea capitis mimics alopecia areata (no scale), seborrheic dermatitis (greasy), or trichotillomania (broken hairs) [44]. Confirmation essential pre-therapy, averting unnecessary antifungals [42]. Direct Microscopy (KOH Mount): First-line, bedside test scraping active lesion borders (not centers) with scalpel/No.15 blade or swab (safer) [42, 38, 48]. Process: 10–20% KOH (nails/hair: 40%), optional DMSO/urea accelerates; warm slide 10–20s; examine ×10/×40 (400x total) with focus racking for "sandwich-bag" nuclei. Findings: uniform septate hyphae (2–8 μm, branching 45°), arthroconidia (rectangular chains), favoring mosaic patterns; capitis shows endothrix spores (intrafollicular) or ectothrix sheaths. Sensitivity 70–90% (higher active borders), specificity near-100% for hyphae; false-negatives from powdery scale or prior topicals. Enhancers: Calcofluor white (CFW, 85–95%, fluorescence microscopy), chlorazol black E (stains chitin), Parker ink (metachromatic blue hyphae) [4, 43]. Nails: pare distal plate (not debris), sensitivity 50–70%; subungual hyperkeratosis yields debris hyphae.
- *Culture (Gold Standard):* Sabouraud dextrose agar (SDA) + cycloheximide/chloramphenicol (inhibits contaminants/saprobes/bacteria); dermatophyte test medium (DTM) shifts red via alkaline metabolites (pH>8). Inoculate scrapings/hair/nail shards, 25–30°C aerobic, 7–21 days (mold: reverse pigment, urease+); microscopic: macroconidia (club-shaped E. floccosum), microconidia (grape-like T. rubrum), spirals (T. mentagrophytes), spindle (M. canis) [39]. Hair perforation test (Trichophyton+): digest hair in fungal broth. Sensitivity 60–80% (sampling-dependent), specificity 100%, but 2–4 weeks delay therapy; contamination 10–20% [17]. PCR reflex on slow growers.
- *Molecular Diagnostics:* Pan-dermatophyte PCR targets ITS1/2 rDNA (TBF1/2 primers), 95–99% sensitivity/specificity, 4–24h turnaround; multiplex distinguishes T. indotineae (genotype VIII) via SQLE sequencing. qPCR quantifies load (infection vs. colonization); LAMP (loop-mediated) isothermal for POC [45]. MALDI-TOF MS: protein spectra match databases (90–97%, 1–2h), gaps for rare taxa [17]. TineaProbe®/DermaGenius® commercial kits (CE-marked) yield 90%+ accuracy [36].
- *Histopathology:* Refractory/extensive cases; punch biopsy active edge, PAS/GMS stains hyphae (80% sensitive); Majocchi shows granulomas with PAS+ organisms [27, 42]. Differentiates deep invasion.
- *Adjuncts:* Wood's lamp (365 nm UV): green fluorescence (*M. canis*/audouinii), dull copper (T. schoenleinii), absent most Trichophyton (4). Dermoscopy: corkscrew hairs (endothrix), comma/ yellow dots (ectothrix), perifollicular scaling, Morse code (pigment invasion). Reflectance confocal microscopy (RCM): non-invasive hyphae (bright filaments), 90% correlation with histo [43].
- *Pitfalls and Algorithms (Table 4):* Empiric therapy risks resistance (50–65% terbinafine failure, Table 5) [2]. Algorithm: suspect clinically → KOH (rules out 80% non-fungal); atypical/ incognito/recalcitrant → culture/PCR; nails/capitis → mandatory confirmation (griseofulvin/ terbinafine expense) (44). India: genotype VIII mandates ITS/SQLE sequencing. Sensitivity hierarchy: PCR > CFW-KOH > culture > plain KOH. Cost: KOH <$1, culture $5–10, PCR $20–50 [17].

- *Special Sites: Nails:* clippings + debris, clip to 1mm shards; false+ trauma dystrophies [42]. Capitis: epilate fluorescent hairs; kerion treat presumptively (corticosteroids + griseofulvin) pending culture. Household screening: capitis contacts via shampoo cultures avoided; empiric sporicidal (selenium sulfide) [44].
- *Discussion:* The comprehensive analysis of dermatophyte infections through the six meticulously constructed tables provides an integrated framework that bridges taxonomy, clinical practice, epidemiology, diagnostics, pharmacology, and therapeutic strategies, offering healthcare professionals a robust toolkit for evidence-based management [1, 17]. Each table not only consolidates quantitative data but also reveals dynamic interrelationships driving the global dermatophytosis burden, particularly the unprecedented Indian epidemic [2, 3].

TABLES

Table 1: Dermatophyte Taxonomy and Ecology establish the phylogenetic foundation, cataloging eight representative genera-species pairs with precise genomic metrics and virulence determinants [1]. *Trichophyton rubrum*'s compact 22.5 Mb genome encoding SUB3 keratinase and ALS adhesins underpins its 45–50% global prevalence in chronic adult infections, like tinea pedis, reflecting anthropophilic adaptation via reduced inflammatory genes [5]. Conversely, *T. mentagrophytes /interdigitale* genotype VIII's expanded 23.8 Mb repertoire, including SQLE mutations, correlates with its explosive 64% post-2016 dominance in India, transitioning from zoophilic reservoirs to human-to-human transmission [2, 16]. Zoophilic *Nannizzia canis* (24.1 Mb) expresses mannoproteins triggering robust immunity, explaining its 8–10% prevalence in fluorescent Wood's-positive pediatric ringworm from feline sources [1, 4]. Geophilic *N. gypsea* and rare *Lophophyton gallinae* highlight sporadic soil/poultry exposures (<3% cases), emphasizing One Health surveillance [16, 29]. This table's ecological stratification predicts clinical severity: anthropophiles favor chronicity, zoophiles acute inflammation [1].

Table 1. Dermatophyte taxonomy and ecology.

Genus	Key species	Ecology	Genome size (Mb)	Primary genes	Global prevalence (% human cases)	Key distribution notes	Reference
Trichophyton	*T. rubrum*	Anthropophilic	22.5	SUB3, KER1	45–50%	Worldwide, adults	1
Trichophyton	*T. mentagrophytes*/ interdigitale (VIII)	Zoophilic/ Anthropophilic	23.8	SQLE, Mep3	25–30% (64% India post-2016)	Asia surge	2
Trichophyton	*T. tonsurans*	Anthropophilic	23.2	Endothrix, TUB2	10%	Africa/USA capitis	1
Trichophyton	*T. violaceum*	Anthropophilic	22.9	Follicular	5–8%	Africa/India	7
Nannizzia	*N. canis* (*M. canis*)	Zoophilic	24.1	Mannoproteins	8–10%	Cat/dog transmission	1
Nannizzia	*N. gypsea* (*M. gypseum*)	Geophilic	23.5	Soil keratinases	2–3%	Rural exposure	16
Epidermophyton	*E. floccosum*	Anthropophilic	22.0	Macroconidia	3–5%	Groin specialist	1
Lophophyton	*L. gallinae*	Zoophilic	23.7	Poultry-related	<1%	Avian zoonosis	29

Table 2: Clinical Types and Manifestations correlate etiological agents with lesion morphology, inflammatory profiles, and India-specific prevalence from 161,000+ analyzed cases [3]. Tinea corporis, driven by *T. mentagrophytes* (50%) over *T. rubrum* (30%), presents annular scaly plaques with mild-moderate response, accounting for 32.4% burden – reflecting genotype VIII's accelerated growth [3, 28]. Tinea cruris (19.7%) favors *E. floccosum* (25%), yielding pruritic inguinal plaques responsive to

azoles. Onychomycosis (17.9%), *T. rubrum*-dominant (70%), manifests dystrophic onycholysis persisting for years due to biofilms, necessitating prolonged systemic therapy [10, 15]. Tinea capitis (13.3%) shifts from *T. tonsurans* endothrix "black dots" to *T. violaceum* in children, with kerion risk from zoophiles [7]. Interdigital tinea pedis (4.1%) and rare Majocchi granuloma underscore site tropisms, where follicular invasion evades topical agents [13, 27]. This table guides syndromic diagnosis, linking agent ecology to response patterns.

Table 2. Clinical types and manifestations.

Infection type	Common agents (% isolation)	Key features	Inflammatory response	Duration if untreated	India prevalence (% cases)	Reference
Tinea corporis	*T. mentagrophytes* (50%), *T. rubrum* (30%)	Annular plaques, scaling	Mild-moderate	Months	32.4%	3
Tinea cruris	*E. floccosum* (25%), *T. rubrum* (40%)	Inguinal plaques, pruritus	Moderate	Weeks-months	19.7%	3
Tinea unguium	*T. rubrum* (70%), *T. mentagrophytes* (15%)	Nail dystrophy	Low	Years	17.9%	10
Tinea capitis	*T. tonsurans* (30%), *T. violaceum* (25%)	Alopecia, black dots	Variable (kerion)	Months	13.3%	7
Tinea pedis	*T. rubrum* (60%), *T. mentagrophytes* (20%)	Interdigital maceration	Mild	Chronic	4.1%	13
Tinea faciei/barbae	*M. canis* (20%), *T. verrucosum* (15%)	Facial folliculitis	High (kerion)	Weeks	5–7%	27
Majocchi granuloma	*T. rubrum* (80%)	Follicular nodules	Granulomatous	Persistent	Rare (<1%)	27

Table 3: Epidemiological Trends in India chronicles a half-century evolution across ~161,000 isolates, revealing a paradigm shift from *T. rubrum–M. canis* baseline (1939–1960, rural zoonoses) to *T. violaceum* capitis peaks (1970s urban migration) and post-2011 *T. mentagrophytes* VIII surge (64–70% by 2025) fueled by OTC steroid–antifungals, diabetes, and post-COVID immunosuppression [2, 3, 22]. Analyzing 56,000 recent cases, it projects endemicity amid 1.9:1 male predominance and tropical humidity. This temporal mapping implicates iatrogenic drivers, urging pharmacovigilance [20].

Table 3. Epidemiological trends in India (Decades).

Decade	Dominant agent (% of total isolates)	Total cases analyzed	Key risk factors	Shift notes	Reference
1939–1960	*T. rubrum* (60%), *M. canis* (20%)	~5,000	Rural, zoonotic	Early baseline	3
1971–1980	*T. violaceum* (57%), *T. tonsurans* (15%)	~10,000	Urban migration, children	Capitis peak	3
2001–2010	*T. rubrum* (45%), *T. mentagrophytes* (25%)	~40,000	Humidity, overcrowding	Stable anthropophilic	3
2011–2015	*T. mentagrophytes* (31%), *T. rubrum* (35%)	~50,000	Steroid misuse begins	Transitional	3
2016–2021	*T. mentagrophytes* VIII (64%), *T. rubrum* (20%)	~56,000	OTC combos, resistance	Epidemic surge	2
2022–2025	*T. mentagrophytes* VIII (70%)	~Ongoing	Post-COVID, diabetes	Genotype dominance	22

Table 4: Diagnostic Methods Comparison benchmarks six modalities by metrics critical for resource-stratified settings [4, 17]. KOH microscopy's rapid 70–90% sensitivity detects hyphae but lacks speciation, operator-dependent at 10–20% mounts. Calcofluor white boosts to 85–95% via fluorescence. Culture on SDA/DTM (gold standard, 60–80%) identifies macroconidia/microconidia over 2–4 weeks,

but risks contamination. ITS-PCR achieves 95–99% precision in hours, indispensable for resistance genotyping. MALDI-TOF (90–97%) offers library-dependent speed, while PAS histopathology suits deep invasions. This hierarchy prioritizes molecular escalation for recalcitrant cases.

Table 4. Diagnostic methods comparison.

Method	Sensitivity/ specificity	Time to result	Species identification	Cost (relative)	Limitations	Reference
KOH Microscopy	70–90% / High	Minutes	No (hyphae only)	Low	Operator-dependent	4
Calcofluor White	85–95% / High	Minutes	No	Low	Fluorescence microscope req.	4
Culture (SDA/DTM)	60–80% / Gold std	2–4 weeks	Yes (macro/micro)	Low	Slow, contamination risk	17
ITS-PCR	95–99% / 98%	4–24 hours	Yes	High	Lab infrastructure	17
MALDI-TOF MS	90–97% / 95%	1–2 hours	Yes (library-based)	Medium-High	Database gaps for rare spp.	17
Histopathology	80% / High	24–48 hours	Partial	Medium	Invasive, non-specific	4

Table 5: Antifungal Resistance Profiles exposes the terbinafine crisis in Indian *T. mentagrophytes* VIII, where SQLE mutations (Leu393Phe, Ala448Thr, Phe397Leu) yield 50–65% resistance (MIC 0.5–>16 μg/mL) versus 5–10% globally – 10x wild-type growth amplifying spread [2, 26]. Erg11 alterations drive fluconazole (80–90%) and itraconazole (20–30%) failures; griseofulvin retains 90–95% susceptibility via β-tubulin fidelity [22, 26]. Topical amorolfine's 30–40% SQLE cross-resistance signals combo needs [24]. This pharmacogenomic atlas mandates pre-therapy testing.

Table 5. Antifungal resistance profiles (India focus).

Antifungal agent	Target enzyme	Key mutations (T. mentagrophytes VIII)	Resistance rate (India, 2021–25)	MIC range (μg/mL)	Global comparison	Reference
Terbinafine	SQLE (Erg1)	Leu393Phe, Ala448Thr, Phe397Leu	50–65%	0.5–>16	5–10%	2
Itraconazole	Erg11 (CYP51)	K143R, G448S (rare)	20–30%	0.25–4	10–15%	26
Fluconazole	Erg11	Y111F, substitutions	80–90%	4–>64	40–60%	26
Griseofulvin	β-tubulin	Del (Lys55-Ala56)	5–10%	1–8	<5%	22
Amorolfine	Ergosterol	SQLE variants	30–40%	0.03–2	Emerging	24

Table 6. Treatment algorithms by severity and site.

Severity/site	First-line therapy (duration)	Alternatives (if resistant)	Topical adjunct	Monitoring	Cure rate (reported)	Reference
Localized	Topical terbinafine/ azole (2–4 weeks)	None	Clotrimazole BID	Clinical at 4 weeks	85–95%	21
Extensive corporis	Oral terbinafine 250 mg/day (2–4 weeks)	Itraconazole 200 mg/day (2 weeks)	+ Terbinafine cream	Culture at 8 weeks	70–90%	18
Onychomycosis	Terbinafine 250 mg/day (12 weeks)	Itraconazole pulse (3 months)	Amorolfine lacquer	Mycology at 6/12 months	60–80%	10
Tinea capitis	Griseofulvin 20 mg/kg/day (8 weeks)	Terbinafine (if >20 kg)	Ketoconazole shampoo	Culture + Wood's	80–90%	21
Resistant cases	Terbinafine + itraconazole combo (4–6 weeks)	Fosravuconazole (investig.)	UV fomite control	Susceptibility testing	50–70%	22

Table 6: Treatment algorithms stratify by severity/site, endorsing topical allylamines (85–95% localized cure) escalating to oral terbinafine (250 mg/day, 70–90% extensive corporis) or griseofulvin (capitis, 80–90%) [18, 21]. Onychomycosis demands 12-week pulses (60–80%), resistant strains combos with monitoring (50–70% success) [10, 22]. Adjuncts, like ketoconazole shampoo and UV fomites, enhance outcomes.

These Tables Interweave: Taxonomy (1) predicts resistance (2,5); epidemiology (3) fuels clinical patterns (2); diagnostics (4,17) inform therapy (6) amid resistance (5). India's genotype VIII exemplifies globalization risks, demanding integrated networks (19,22). Limitations: regional focus, static snapshots – future updates via real-time genomics are essential (37).

CONCLUSION

Dermatophytoses embody a paradigm of fungal opportunism, where ecological mastery of keratin degradation intersects human susceptibility amplified by modern confounders – steroid abuse, climate shifts, migration. From *T. rubrum*'s stealthy persistence to *T. indotineae*'s aggressive resistance, these pathogens demand vigilant surveillance. Advances in genomics unmask virulence circuits, heralding targeted inhibitors beyond ergosterol pathways (e.g., protease antagonists). Clinically, syndromic management yields to mycological precision: PCR-guided therapy curtails overtreatment, curbing resistance. Public health imperatives encompass antifungal stewardship, fomite decontamination (UV, hypochlorite), and One Health integration tracing zoonoses. In high-burden locales, like India, where genotype VIII threatens endemicity, national mycology networks and susceptibility registries are pivotal. Future horizons gleam with CRISPR-edited models dissecting host–pathogen dyads, mRNA vaccines priming Th17 immunity, and AI-phenotyping accelerating diagnostics. Ultimately, conquering dermatophytes hinges on interdisciplinary synergy – microbiologists decoding evolution, clinicians optimizing regimens, policymakers enforcing pharmacovigilance – ensuring these ancient foes yield to contemporary science. This review arms practitioners with the intellectual arsenal to navigate this dynamic frontier, mitigating morbidity in an era of fungal resurgence.

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