Expert Panel Review of Skin and Hair Dermatophytoses in an Era of Antifungal Resistance

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Abstract

Dermatophytoses are fungal infections of the skin, hair, and nails that affect approximately 25% of the global population. Occlusive clothing, living in a hot humid environment, poor hygiene, proximity to animals, and crowded living conditions are important risk factors. Dermatophyte infections are named for the anatomic area they infect, and include tinea corporis, cruris, capitis, barbae, faciei, pedis, and manuum. Tinea incognito describes steroid-modified tinea. In some patients, especially those who are immunosuppressed or who have a history of corticosteroid use, dermatophyte infections may spread to involve extensive skin areas, and, in rare cases, may extend to the dermis and hair follicle. Over the past decade, dermatophytoses cases not responding to standard of care therapy have been increasingly reported. These cases are especially prevalent in the Indian subcontinent, and *Trichophyton indotineae* has been identified as the causative species, generating concern regarding resistance to available antifungal therapies. Antifungal-resistant dermatophyte infections have been recently recognized in the United States. Antifungal resistance is now a global health concern. When feasible, mycological confirmation before starting treatment

is considered best practice. To curb antifungal-resistant infections, it is necessary for physicians to maintain a high index of suspicion for resistant dermatophyte infections coupled with antifungal stewardship efforts. Furthermore, by forging partnerships with federal agencies, state and local public health agencies, professional societies, and academic institutions, dermatologists can lead efforts to prevent the spread of antifungal-resistant dermatophytes.

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1 Introduction

Dermatophytoses are cutaneous infections caused by dermatophytes, fungal pathogens from the *Trichophyton*, *Epidermophyton*, and *Microsporum* genera [1]. Since they require keratin for growth, dermatophytoses are generally limited to skin, hair, and nails, and thrive in warm (25–28 °C), humid environments [2, 3]. Dermatophytoses are often referred to as 'tinea' infections (Latin for 'worm'), referring to the worm-like borders surrounding ring-shaped patches. The word 'tinea' is combined with the anatomical site to form the complete nomenclature (Table 1) [4].

1.1 Prevalence and Main Causal Organisms

An estimated 20–25% of the global population are affected by dermatophytoses [1]. *Trichophyton rubrum* is the most common causative agent in high-income countries, and *Trichophyton mentagrophytes* species complex, *Microsporum canis,* and *Epidermophyton floccosum* are most prevalent globally. *Trichophyton schoenleinii, Trichophyton soudanese,* and *Trichophyton concentricum* are generally limited to parts of Europe, Asia, and Africa [5, 6]. Epidemiology varies over time due to tourism and migration [2, 5, 7]. Taxonomy changes have divided the genera *Trichophyton* and *Microsporum* and added the new genera *Arthroderma*, *Nannizzia*, *Paraphyton* and *Lophophyton* [8].

1.2 Pathogenesis and Virulence Factors

Dermatophytes infect cytokeratin comprising the stratum corneum in the hair, skin, and nails, and adhere to the host via mannan glycoproteins in the fungal cell walls [9–11]. The mold grows as linear hyphae, enabling spread to deeper layers of keratin-rich structures. To utilize keratin for growth, dermatophytes break down disulfide bridges linking epidermal keratins. Keratin is rich in cysteine. Dermatophytes secrete sulfite, a reducing agent that cleaves cystine bonds via cysteine dioxygenase, contributing to keratin degradation [9–12]. Dermatophytes produce biofilms, which have been reported in both *Trichophyton* and *Microsporum* species. It is hypothesized that biofilms contribute to dermatophyte antifungal treatment failures [13, 14].

1.3 General Risk Factors

Table 2 lists risk factors contributing to dermatophytosis susceptibility.

1.4 Transmission

Dermatophyte transmission is multifactorial [15]. Dermatophytes likely persist on wet surfaces, including in bathrooms and pool facilities [15–17], and may spread via shared clothing, footwear, towels, and bedding, especially with a comprised skin barrier [15, 18,

19]. Pets and stray, farm, laboratory, and wild animals have been implicated in zoonotic spread [20–22].

1.5 Racial Disparities

There are racial disparities in the prevalence of tinea infections, particularly tinea capitis [23, 24]. In a retrospective population-based study of children < 10 years of age, incident rates per 10,000 enrolled were 252.1 claimants for Black children versus 23.1 claimants for White children (p value not reported) [25]. In a prospective, cross-sectional surveillance study of 10,514 kindergarten fifth-grade children, Black versus White and Hispanic children had higher infection rates (12.9%, 1.1%, and 1.6%, respectively; $p < 0.01$) [26]. A commercial database study of tinea capitis in children < 18 years of age estimated the incidence rate to be sevenfold higher in Black versus White children [27]. In a cross-sectional study of 351,629 HIV patients in a tertiary healthcare system, dermatophytoses were more common in HIV- versus non-HIV-infected individuals ($p < 0.0009$) [28].

1.6 Looking Ahead

In a prospective survey-based study of errors made by primary care physicians in managing skin disorders, errors related to diagnosis of dermatophytoses accounted for the largest proportion of errors (32%, 102/319) [29]. In a survey-based study of board-certified dermatologists presented with 13 clinical images of fungal infections, respondents categorized cases with > 75% accuracy in only 31% of cases [30], highlighting that visual inspection alone is unreliable for diagnosis. Dermatologists play a key role not only in accurate diagnosis of superficial fungal infections but also in educating other non-dermatologists about dermatophytoses. The American Academy of Dermatology's guidelines for the treatment of superficial mycotic infections were last published in 1996, necessitating an updated review on diagnosis and management.

2 Tinea Corporis and Tinea Cruris

2.1 Demographics and Geography

Tinea corporis is most caused by *T. rubrum* globally. Other causes include *T. tonsurans*, especially when secondary to tinea capitis infection, and *M. canis*, particularly with exposure to infected house pets [31, 32] (Table 3).

The most common cause of tinea cruris worldwide is *T. rubrum*. *T. mentagrophytes* and *T. interdigitale* are becoming more common infectious etiologies globally, particularly in India [33–35]. Tinea cruris more commonly affects male adolescents and young adult men [36] (Table 3).

2.2 Risk Factors, Clinical Features, and Differential Diagnosis

Risk factors, clinical features, and differential diagnosis of tinea corporis and cruris are listed in Tables 2, 4, and 5, respectively (Figs. 1, 2).

2.3 Diagnostic Methods

The most rapid and cost-effective diagnostic technique for tinea corporis and cruris is direct microscopic examination with 10–15% potassium hydroxide (KOH) preparation using skin scrapings of the affected area(s) showing segmented hyphae [37]. Taking abundant scrapings from the active edge of a plaque or patch increases sensitivity. A fungal culture of skin scrapings on Mycosel agar media is considered the diagnostic gold standard since it can identify the causative organism; however, turnaround time is typically several weeks, with a significant false negative rate.

Polymerase chain reaction (PCR) may rapidly detect the presence of fungal DNA and identify the fungal species [38–43]; however, PCR for dermatophytes is not performed by all laboratories, and widespread adoption has been slow in clinical practice [44, 45].

Reflectance confocal microscopy (RCM) is a non-invasive technique that is being explored for cutaneous fungal infection diagnosis [46–48]. While results are immediate, limitations include difficulty positioning the probe in certain anatomic areas, expensive equipment, and required training.

Dermoscopy is an emerging adjunctive diagnostic technique for dermatophyte infections [49–52]. Useful dermatoscopic features distinguishing tinea corporis from other dermatologic conditions include presence of peripheral dotted vessels, white 'moth-eaten' scale with peripheral distribution and outward peeling direction [50].

2.4 Treatment and Management

Treatment for tinea corporis and cruris is always indicated to prevent infection spread to self and others and prevent secondary bacterial infections. In general, mild or localized infection may be treated once to twice daily with topical monotherapy antifungals, including azoles, allylamines, ciclopirox, butenafine, and tolnaftate [53–56], while more disseminated disease is treated with oral antifungals. The American Academy of Pediatrics (AAP) 2021–2024 Report of the Committee on Infectious Diseases recommended application of topical antifungals directly to lesions and 1–2 cm beyond the lesion border. Although clinical resolution may be apparent after 2 weeks of therapy, treatment is recommended for 4–6 weeks. If there is no clinical improvement after 2 weeks of topical therapy, an alternate diagnosis or systemic therapy may be considered [56].

A Cochrane Database Systematic Review of 129 studies involving 18,086 participants with tinea corporis or cruris supported the use of topical terbinafine and naftifine 1%, and clotrimazole 1%, based on clinical and/or mycological cure rates versus placebo. Azoles and benzylamines had similar mycological cures rates (risk ratio [RR] 1.01, 95% confidence interval [CI] 0.94–1.07) [57, 58]. In a mixedtreatment comparison meta-analysis of 65 articles including 14 topical antifungal treatments for tinea corporis, cruris, and pedis, mycological cure at treatment completion was not significantly different among the topical antifungals studied [59]. However, butenafine, naftifine, and terbinafine, a benzylamine and allylamines, respectively, were the most effective among 13 therapies for sustained cure, defined as maintaining cure from infection for   14 days, compared with azoles. Cost consideration is necessary in most cases since there are limited efficacy data to support one

class over another. Therefore, azoles, which are cheaper on average, may be used as first-line therapy, followed by allylamines for first-line treatment failures [58]. Topical antifungals indicated for tinea corporis and cruris are listed in Table 6.

Position statements released in 2019 by the Canadian Pediatric Society recommend that adjunctive topical corticosteroid therapy be avoided due to the risk of skin atrophy [60]. Topical corticosteroid misuse is hypothesized to contribute to antifungal resistance [61, 62]. Combination topical antifungal-corticosteroid products should be avoided. Antifungal monotherapy is effective and may decrease inflammation and pruritus.

For widespread infection involving multiple areas, disease refractory to topical therapy, or tinea corporis with vellus hair involvement (Fig. 3), oral treatment with either terbinafine or itraconazole is recommended [52]. Griseofulvin and fluconazole are second-line treatments and require longer treatment courses. With partial response, therapy is continued and patient lifestyle factors, including hygiene, which may contribute to incomplete cure are assessed. With no response to treatment, a change to another oral antifungal (i.e. terbinafine to itraconazole) is indicated. Follow-up for at least 4 weeks after clinical cure is recommended to prevent recurrence.

Terbinafine is the recommended first-line therapy in treatment-naïve patients with tinea corporis or cruris [53]. In a randomized controlled trial of 50 patients with tinea corporis treated with 500 mg/day of griseofulvin versus 500 mg/day of terbinafine for 6 weeks, successful treatment rates, defined as either clinical and mycological cure or mycological cure with only slight residual clinical signs of infection, were 73% versus 87%, respectively (p value not reported) [63]. In a study of 64 patients diagnosed with tinea and/or tinea cruris receiving terbinafine 250 mg/day versus griseofulvin 500 mg/day for 2 weeks, mycological cure was 87.1% versus 54.8% at 6 weeks follow up ($p < 0.05$) [64]. In a prospective trial of 60 patients with tinea corporis and/or cruris randomized to terbinafine 250 mg/day versus terbinafine 500 mg/day, complete cures were similar (20% vs. 33.3%; $p = 0.82$) [65].

In a randomized controlled trial of 78 patients with tinea corporis or cruris treated with itraconazole 100 mg/day or griseofulvin 500 mg/day for 15 days, clinical (72% vs. 51%; $p < 0.03$) and mycological cure (87% vs. 57%; $p < 0.03$) were higher for itraconazole versus griseofulvin [66]. A double-blind, randomized controlled trial of 40 patients with clinically and mycologically confirmed tinea corporis or cruris comparing itraconazole 100 mg/day versus griseofulvin 500 mg/day for 15 days showed no difference in complete cure at treatment completion (22.2% vs. 23.8%; $p = 1.000$) nor at 15-day post-treatment follow-up (55.6% vs. 52.4%; $p = 0.672$) [67]. Physicians can consider a minimum dose of itraconazole 200 mg to treat tinea corporis and/or cruris, as underdosing may contribute to resistance development.

Recommended oral therapy dosing for tinea corporis and cruris is listed in Table 7 [64, 68, 69].

3 Tinea Capitis

3.1 Demographics and Geography

Tinea capitis primarily affects children, particularly in the prepubertal years [70, 71]. Black children are most frequently affected in the US (Fig. 4). In a prospective, cross-sectional surveillance study of 10,514 kindergarten fifth-grade children, Black children had the highest rate of *T. tonsurans* tinea capitis infection (12.9%) versus Hispanic (1.6%) and White children (1.1%) [$p < 0.01$] [25]. Although uncommon, tinea capitis may affect adults [72–74].

Common causative organisms of tinea capitis are listed in Table 3. In general, species within *Trichophyton* and *Microsporum* genera cause tinea capitis, including *Microsporum audouinii, Trichopython violaceum, or Trichophyton tonsurans*. In the US, *T. tonsurans* is the most common causative species, followed by *M. canis* (Fig. 5) [23].

3.2 Risk Factors, Clinical Features, and Differential Diagnosis

The hair shaft infection presents in one of three distinct forms: endothrix, ectothrix, or favus (Fig. 6), which drive the clinical appearance. Endothrix and ectothrix infections are the most common presentations [8, 75]. Kerion is an inflammatory presentation of tinea capitis. Risk factors, clinical features, and differential diagnosis are described in Tables 2, 4, and 5, respectively.

3.3 Diagnostic Methods

Physical examination identifies signs of tinea capitis, such as black dots associated with endothrix tinea capitis. Ectothrix infections caused by *Microsporum* species or *T. schoenleinii* may fluoresce a green/yellow color or blue/white color, respectively, while endothrix infections do not exhibit fluorescence Wood's lamp; however, Wood's lamp examination in not reliable for definitive diagnosis [75].

Fungal culture using Mycosel agar culture remains the gold standard of diagnosis for tinea capitis for species identification. Samples are collected using a sterile toothbrush, moistened cotton swab, or by scraping with a no. 15 blade or glass slide over the affected area. Infected hairs often break off and may be sent along with scrapings [76–78]; however, the results may take 2–4 weeks, which may significantly delay therapy initiation and increase transmission risk [8, 75, 76, 79]. Intense inflammation associated with kerion may cause false negative results and, in general scrapings, should be taken prior to initiating antifungal treatment. A KOH preparation may be used to screen for tinea capitis while fungal cultures are pending. KOH is applied to a glass slide and the proximal ends of a hair shaft are examined micro-scopically. The test is positive if fungal spores are found within or outside of the hair shaft (endothrix and ectothrix, respectively).

PCR may be used for tinea capitis diagnosis, although is rarely employed in clinical practice. In a descriptive cross-sectional study of 115 scalp scrapings and hair fragments from children aged 1–16 years diagnosed with tinea capitis, PCR-ELISA had 84% sensitivity and 85% specificity compared with fungal culture used as the gold standard [80].

In a cross-sectional, descriptive study on 129 hair samples from patients clinically diagnosed with tinea capitis, real-time PCR had 89.3% sensitivity and 75.3% specificity compared with fungal culture ($p < 0.05$) [81]. A PCR test for *M. canis* identification achieved 100% sensitivity and specificity in a study of 130 clinical isolates of dermatophytes, 10 yeast/mold isolates, 12 hair and skin samples from animals with/without *M. canis* infection, and 35 patient specimens, 7 of which were positive for *M. canis* [82]. *M. canis* is less susceptible to terbinafine than *Trichophyton* species, requiring prompt therapy to avoid long-term sequelae, including permanent hair loss.

RCM has been used for tinea capitis diagnosis, whereby hyphae appear linear, with high reflectivity and conidia near the hair shaft present as round and hyperreflective [46, 83, 84]. In one case series including four patients with tinea capitis, diagnosis was achieved in approximately 5 min per patient [46].

Trichoscopy, dermoscopy of the hair and scalp, is a useful diagnostic adjunct. Trichoscopic findings include comma hairs, corkscrew hairs, broken hairs, dystrophic hairs, and/or black dots [85–88]. In a cross-sectional study comparing trichoscopic features of 15 children with tinea capitis with 10 patients with alopecia areata, yellow dots, exclamation mark hairs, and vellus hairs differentiated alopecia areata from tinea captitis [85].

3.4 Treatment and Management

Although mycological confirmation before starting treatment is considered best practice, diagnostic methods may not be readily available or may result in significant delays to therapy initiation. The 2014 British Association of Dermatologists tinea capitis guidelines indicate that it is appropriate to begin treatment when tinea capitis is strongly suspected clinically, including in the presence of scaling or kerion [89].

First-line therapy for tinea capitis is oral antifungal therapy, as topical therapy cannot effectively penetrate the hair shaft. Although many controlled trials described herein are exclusive to pediatric patients, the same medications are used in adult patients. Pediatric treatment regimens are described in Table 7, along with commonly accepted adult dosing recommendations [90].

Griseofulvin and terbinafine are US FDA-approved tinea capitis treatments and are the most commonly used therapies [56]. The AAP 2021–2024 Report of the Committee on Infectious Diseases states that higher doses of griseofulvin than are FDA-approved may be used, and high-dose griseofulvin is the standard of care for pediatric patients with *M. canis* tinea capitis (griseofulvin microsize 20–25 mg/kg/day, maximum 1 g/day, or ultramicrosize 10–15 mg/kg/day, maximum 750 mg/day, for 6 weeks and until clinically clear).

For tinea capitis due to *Trichophyton*, both griseofulvin and terbinafine are acceptable first-line therapies, but choice of agent should be tailored to speciation, if known [91]. A 2016 Cochrane review found that 4 weeks of terbinafine versus 8 weeks of griseofulvin treatment had similar efficacy in achieving complete cure across three studies including 328 participants with *Trichophyton* tinea capitis (84.2% vs. 79%; RR 1.06, 95% CI 0.98–1.15) [91]. In two randomized, investigator-blinded, active-controlled trials in children aged 4–12

years with clinically diagnosed and KOH-confirmed tinea capitis, terbinafine hydrochloride oral granules ($n = 1040$, 5–8 mg/kg/day) versus griseofulvin microsize ($n = 509$, 10–20 mg/kg/day) for 6 weeks had higher complete (45.1% vs. 39.2%; $p = 0.024$) and mycological cure rates (61.5% vs. 55.5%; $p = 0.029$) on pooled data at week 10. Terbinafine and griseofulvin had similar efficacy for clinical cure when compared with pooled data at week 10 (63.0% vs. 58.8%; $p = 0.10$). Terbinafine had greater efficacy for mycological, clinical, and complete cure in children with *T. tonsurans* tinea capitis specifically (complete cure 52.1% vs. 35.4%; $p < 0.0001$) [92]. For *Microsporum* tinea capitis, a meta-analysis of two studies including 334 participants found greater complete cure rates with 6–12 weeks of griseofulvin versus 6 weeks of terbinafine (50.9% vs. 34.7%; RR 0.68, 95% CI 0.53–0.86) [91]. If the exact species is unknown, griseofulvin is recommended as first-line therapy in treating tinea capitis [93].

Due to concerns about resistance of *T. tonsurans* to griseofulvin, fluconazole and itraconazole may be considered as non-FDA approved therapeutic alternatives [94] and for patients with contraindications to terbinafine and/or griseofulvin, or in patients who cannot tolerate high griseofulvin doses [95]. Fluconazole is FDA-approved for children <2 years of age, for indications other than tinea capitis. A randomized, multicenter, three-arm trial in children aged 3–12 years comparing fluconazole 6 mg/kg/day for 3 weeks followed by 3 weeks of placebo with fluconazole 6 mg/kg/day for 6 weeks and with griseofulvin 11 mg/kg/day for 6 weeks, showed no differences between mycological (44.5%, 49.6%, and 52.2%, respectively; $p = 0.40$) or clinical cure (87.8%, 89.8%, and 90.9%, respectively; $p = 0.61$) [95]. For griseofulvin, physicians could consider a dose of 20 mg/kg/day. A randomized controlled trial of 34 children and 1 adult comparing itraconazole 100 mg daily for 6 weeks with ultra-micronized griseofulvin 500 mg daily for 6 weeks for mostly *M. canis* tinea capitis showed complete cure 8 weeks after therapy completion (88% vs. 88%; *p*-value not reported) [96]. Another randomized, double-blind controlled trial of weight-based itraconazole and terbinafine each administered for 2 weeks for mostly *T. violaceum* tinea capitis showed complete cure rates of 85.7% and 77.8%, respectively at week 12 ($p > 0.05$) [97]. A multicenter, randomized, single-blinded trial of 200 patients treated with griseofulvin microsize 20 mg/kg/day for 6 weeks, terbinafine 62.5–250 mg (weight dependent) for 2–3 weeks, itraconazole 5 mg/kg/day for 2–3 weeks, or fluconazole 6 mg/kg/day for 2–3 weeks showed similar efficacy (measured as a combination of mycological cure and either clinical cure or only a few residual symptoms) among all arms 12 weeks after the start of therapy (griseofulvin: 92%, 95% CI 88.2–95.8%; terbinafine: 94%, 95% CI 90.6–97.4%; itraconazole: 86%, 95% CI 81.1–90.9%; fluconazole: 84%, 95% CI 78.8–89.2%) [98]. Fluconazole and itraconazole are second-line therapies. Dosing of oral antifungals is described in Table 7.

Antifungal shampoos may be used as adjunctive therapy by patients and their household members for tinea capitis in the first 1–2 weeks following diagnosis, to reduce transmission and possible re-infection [36]. In a randomized, double-blind controlled trial of 40 children aged 1–11 years diagnosed with tinea capitis and treated with an 8-week course of ultramicronized griseofulvin, selenium sulfide shampoo 1% and ciclopirox shampoo 1% showed equal efficacy in mycological cure 4 weeks after treatment completion when used as adjunctive treatment twice per week (week 12: 91.7% vs. 95.2%; $p = 1$) [99]. A study

of ketoconazole 2% shampoo used as monotherapy for tinea capitis in 16 Black children aged 3–6 years, over an 8-week treatment period, showed 50% reduced viable arthroconidia numbers after 2 weeks of treatment, clinical cure in 93% of children at week 8, and complete cure in 33% of children 12 months post-treatment [100]. It is recommended that children and their families are instructed on disinfecting hair tools following a diagnosis of tinea capitis, to prevent re-infection or transmission to other household members [101].

It is recommended that patients are reassessed after 1 month of therapy to determine clinical response. If there is no response to therapy despite adherence to prescribed medications, an alternative antifungal is recommended [56].

4 Tinea Barbae/Tinea Faciei

4.1 Demographics and Geography

Table 3 lists the most common causative species of tinea barbae and faciei, by region.

4.2 Risk Factors, Clinical Features, and Differential Diagnosis

Tinea barbae may present as an inflammatory or non-inflammatory morphology, and usually involves an ectothrix infection pattern. Tinea faciei often presents similarly to tinea corporis. Risk factors, clinical features, and differential diagnosis of tinea barbae and are listed in Tables 2, 4, and 5, respectively (Figs. 7, 8).

4.3 Diagnostic Methods

Diagnostic methods related to tinea capitis, described in Sect. 3.3, apply to tinea barbae, whereas diagnostic methods related to tinea corporis, described in Sect. 2.3, apply to tinea faciei.

4.4 Treatment and Management

Treatment principles for tinea capitis, described in Sect. 3.4, apply to tinea barbae. Oral antifungal therapy is first-line as topical therapy cannot penetrate the hair shaft adequately. Treatment principles for tinea corporis, described in Sect. 2.4 apply to tinea faciei. Topical antifungal therapy is first-line therapy, while oral therapy is reserved for refractory or widespread involvement.

5 Tinea Incognito

5.1 Risk Factors and Clinical Features

Due to dampening of the local immune response, classic clinical features of dermatophytoses may not be present in patients with tinea incognito. Steroid-modified dermatophytosis has been described as 'tinea pseudoimbricata' due to its similarity in appearance to tinea imbricata caused by *Trichophyton concentricum*, which is endemic in parts of Southeast Asia [102], presenting with concentric erythematous rings and associated with moderate to severe pruritus (Fig. 9) [103]. Risk factors and clinical features for tinea incognito are listed in Tables 2 and 4, respectively.

5.2   Diagnostic Methods

Because tinea incognito is a steroid-modified infection and does not present with classic features typically present with dermatophyte infections, diagnosis may be delayed. A high suspicion for tinea incognito is necessary if a patient describes a rash coupled with corticosteroid administration, resulting in a temporary improvement of the lesions, followed by worsening and spread [104].

Diagnostic methods of tinea incognito mirror those for tinea corporis and cruris, described in Sect. 2.3. It is recommended that diagnostic methods, including KOH preparation and fungal culture, are performed after steroid discontinuation. Dermoscopy may show black dots surrounded by a white-yellow halo, a sign of vellus hair involvement in dermatophytosis [105].

5.3   Treatment and Management

Once tinea incognito has been diagnosed, topical corticosteroids should be stopped and antifungal therapy initiated. If oral corticosteroids are identified as the culprit in cases of tinea incognito, an alternative treatment should be considered. The treatment thereafter follows that of tinea corporis, cruris, and/or faciei, described in Sect. 2.4.

6   Tinea Pedis and Tinea Manuum

6.1   Demographics and Geography

Tinea pedis typically affects post-pubescent adolescents and adults [70] and is most often caused by *T. rubrum, T. interdigitale, T. mentagrophytes, and E. floccosum* [106]. Common causative species are listed in Table 3. In children, *T. tonsurans* is the common causative organism [69]. Tinea pedis occurs more often in older adults and may be more common in males than in females [107–111]. Tinea pedis is a common problem among developed and developing countries, with a global prevalence estimate of 3–15% [109–113]. In a study of 6932 participants with tinea pedis in the All of Us database 2017–2021, higher adjusted odds of tinea pedis were observed in Black and Hispanic participants (odds ratio [OR] 1.29, 95% CI 1.20–1.38, and OR 1.38, 95% CI 1.28–1.48, respectively), individuals   75 years of age (OR 1.45, 95% CI 1.33–1.57), individuals with income <$35,000 (OR 1.09, 95% CI 1.02–1.16), and individuals with a physical disability (OR 1.56, 95% CI 1.08–1.24) [110]. Epidemiology of tinea manuum infections is far less studied than that of tinea pedis.

6.2   Risk Factors, Clinical Features, and Differential Diagnosis

There are three major clinical presentations of tinea pedis, namely interdigital (Fig. 10), hyperkeratotic ('moccasin-type'), and vesiculobullous (inflammatory) tinea pedis, and a rare ulcerative form. *T. rubrum* typically causes chronic tinea pedis, whereas *T. mentagrophytes* is associated with inflammatory lesions [114]. Risk factors, clinical features, and differential diagnosis for tinea pedis (Fig. 11) and manuum (Fig. 12) are listed in Tables 2, 4, and 5, respectively.

6.3 Diagnostic Methods

Diagnosis of tinea pedis and tinea manuum is primarily based on clinical features, coupled with a positive fungal culture, KOH preparation demonstrating hyphae, or PCR.

6.4 Treatment

Patient education regarding hygiene and proper foot care is an important aspect of management for tinea pedis, and consequently tinea manuum. Patients are instructed to keep feet clean and dry, including use of sweat-wicking socks to avoid sweat pooling [115, 116]. Patients are advised to wear shoes in communal areas, including in public gyms, showers, and pools. Although onychomycosis is beyond the scope of this review, proper nail care, including keeping nails short and clean, is also important for managing tinea pedis, as coexisting onychomycosis may preclude cure [38, 116, 117].

Topical antifungal therapy is the mainstay of treatment for tinea pedis. Per the AAP 2021–2024 Report of the Committee on Infectious Diseases, 2 weeks of therapy is sufficient for mild tinea pedis in children. Topical treatment in adults is recommended for at least 4 weeks. A Cochrane Library Systematic Review of 67 randomized controlled trials from 1970 to 2005 included participants using topical therapy who had mycologically diagnosed foot and toenail fungal infections. Of these, 11 trials ($n = 1144$) included in a meta-analysis comparing allylamines and azoles showed an RR of treatment failure of 0.63 (95% CI 0.42–0.94), favoring allylamines [118]. A 2012 systematic review on the efficacy and safety of topical antifungals for the treatment of dermatophytosis included a meta-analysis of 17 randomized controlled trials, including 1781 participants, comparing allylamines, including naftifine and terbinafine (1% or 2%) used for 1–6 weeks, with azoles or imidazoles, including econazole and miconazole (1% or 2%) used for 2–6 weeks, and found no statistically significant difference in mycological cure at the end of treatment [59]. A 2022 systematic review of randomized controlled trials examining 1042 participants with tinea pedis across seven studies found that topical terbinafine and topical butenafine, a benzylamine, were similarly efficacious (RR 1.3, 95% CI 0.4–4.4) [119]. However, limitations included lack of reporting on baseline characteristics in each treatment arm [119]. Taken together, topical treatment with azoles, allylamines, and benzylamines are all effective for tinea pedis treatment. Therefore, choice of therapy may be dictated by cost, availability of a specific class of medication, or physician preference. It has been suggested a cost-effective strategy is to utilize topical azoles as first-line therapy, followed by allylamines in cases of therapy failures [59]; however, more up-to-date studies are needed to validate this recommendation. It is important to ensure that patients complete the treatment regimen to prevent relapses [116].

Newer topical antifungals include sertaconazole 2% cream and luliconazole 1% cream. In a prospective, openlabel, randomized controlled trial of 313 total patients (232 patients with tinea pedis) using sertraconazole solution or cream twice daily for 28 days, efficacy (defined as a negative culture test and reduction of clinical severity) was achieved in 90.6% and 88.9% of patients, respectively [120]. A double-blind, randomized, vehicle-controlled, phase III study of 321 patients with tinea pedis treated with luliconazole cream 1% showed complete clearance at day 42 in 26.4% (28/106) of patients randomized to luliconazole

cream 1%, versus 1.9% (2/103) vehicle ($p < 0.001$) [121]. In two randomized, double-blind, vehicle-controlled studies of 495 subjects with tinea pedis, patients were treated with econazole nitrate 1% foam for 4 weeks, and complete cure (negative KOH, negative fungal culture, complete clinical resolution) was achieved by 24.3% of patients at day 43 for the active drug versus 3.6% for vehicle ($p < 0.001$) [122].

Tinea pedis refractory to topical therapy may be treated with oral antifungals. First-line therapy for refractory tinea pedis is oral terbinafine. A 2012 Cochrane Library systematic review examining systemic treatments for fungal infections of the skin of the foot included 15 trials and 1438 participants. Of these, two trials of 71 participants compared terbinafine and griseofulvin, with a pooled RR of 2.26 (95% CI 1.49–3.44) favoring terbinafine. There was no significant difference in mycological cure between terbinafine and itraconazole (RR 1.07, 95% CI 0.92–1.25), fluconazole and itraconazole (RR 1.06, 95% CI 0.87–1.30), or fluconazole and ketoconazole (RR 1.04, 95% CI 0.92–1.17) [123]. In addition, clinical subtype of tinea pedis did not influence response to oral therapy [123, 124]. The incidence of liver injury with terbinafine is extremely low, but the drug should be avoided in patients with pre-existing liver disease, and baseline liver function testing is recommended [124–128]. Oral itraconazole may be considered as second-line due to hepatoxicity and cardiotoxicity and frequent drug–drug interactions. Oral ketoconazole should be avoided due to more severe risks of hepatotoxicity and due to more favorable safety profiles of terbinafine and itraconazole [116].

Patient preference is important when prescribing therapy for tinea pedis. In a cross-sectional study evaluating antifungal products for tinea pedis on Amazon.com in 2018, functionality was the most cited positive (14%) and negative (35%) feature for over-the-counter antifungal therapy based on consumer rating and reviews, with specific comments related to itching/ burning relief and number of applications required for relief predominating [129]. Non-pharmacologic measures may be used as adjunct therapy, in addition to topical and/or oral antifungal therapy for tinea pedis and manuum. In a cross-sectional survey study of 152 patients with tinea pedis, 55.9% reported use of at least one non-pharmacological therapy for tinea pedis, including cologne, saltwater, vinegar soaks, and henna [130]. Vinegar soaks may be applied using old cotton socks, rather than tub soaks, to increase convenience and compliance [131]. Salicylic acid peels and tea tree oil may also be used to improve clinical symptoms, but are not effective for achieving mycological cure [132, 133].

The treatment algorithm for tinea manuum closely resembles that of tinea pedis, with topical antifungal agents as the preferred choice of therapy. Indications for systemic antifungals for tinea manuum include co-occurring fingernail infection and two feet-one hand syndrome [134, 135].

7 Chronic and Recurrent Dermatophytosis

Chronic and recurrent dermatophytosis is not a strictly defined clinical entity. It has been previously described as a dermatophyte infection persisting for > 6 months, with or without recurrence, even with adequate therapy [136–138]. It may be secondary to host, pathogen, environmental, or therapeutic factors [136].

7.1 Risk Factors for Chronic or Recurrent Infection

Risk factors for chronic or recurrent dermatophytosis are listed in Table 2.

7.1.1 Immunosuppressed Patients—In immunocompetent hosts, dermatophytosis is typically limited to the epidermis, with epidermal keratinization, body temperature, lipid composition of the stratum corneum, and innate and adaptive immunity preventing more widespread infections and infections extending into the dermis and sub-cutaneous fat [139, 140]. Host immunosuppression also increases the risk of recalcitrant dermatophytosis. In a cross-sectional study of dermatologic conditions in 4679 patients living with HIV in 2013–2018, HIV-infected patients, even if adequately treated, had increased risk of developing tinea corporis, cruris, capitis, and non-specified dermatophytosis (all $p < 0.0009$), suggesting persistence of immune system susceptibilities despite HIV treatment [28].

7.2 Complications and Impact

7.2.1 Extensive Dermatophytosis—Dermatophytosis may present as either extensive infection or deep and invasive infection. In extensive dermatophytosis, infection is confined to the stratum corneum, but covers a large body surface area or there are numerous discrete lesions [141]. In addition to patients living with HIV/AIDS [142, 143], extensive dermatophytosis has been described in solid organ and bone marrow transplant recipients (Figs. 13, 14), patients with disorders of keratinization, and patients with caspase-recruitment domain-containing protein 9 (CARD9) deficiency [144–148].

A case series described 13 male patients with severe oral, anal, and genital dermatophyte infections often requiring more than 1 month of antifungal therapy, mainly among men who have sex with men ($n = 12$) due to *Trichophyton mentagrophytes* genotype VII. Eight patients had multiple lesions and 7 had co-infection with HIV [149, 150].

7.2.2 Invasive Dermatophytosis—Invasive dermatophytosis presents as either deep dermatophytosis or Majocchi's granuloma (MG), which are recognized as two separate clinical entities [139, 141]. In deep dermatophytosis, the infection extends from the epidermis to dermis. In a case series of 12 patients with severe dermatophytosis following solid organ transplant, deep dermatophytosis was characterized by discrete nodules ($n = 9$), an indolent course (median time between transplantation and severe dermatophytosis, on average, of 16 months [range 2–94 months]), and proximity to a superficial dermatophyte infection ($n = 12$). In a 2016 literature review, severe dermatophytosis was described as "ill-defined infiltrated plaques, nodules, and papules sometimes associated with itching, pain, and discharge" [141].

Deep and invasive dermatophytosis has been reported in solid organ transplant recipients and patients with CARD9 deficiency or HIV/AIDS [151–155], and in patients taking oral or topical corticosteroids and other immunosuppressive agents, or those with atopy, diabetes mellitus, Cushing's disease, hematologic malignancies and disorders, autoimmune disorders, liver disease, and trauma [156–175]. In extremely rare cases, the infection may spread systemically via lymphatic or vascular systems, or via contiguous spread to bone [151, 176].

Although uncommon, deep dermatophytosis has been reported in immunocompetent hosts without a history of steroid use [177].

MG, also known as nodular granulomatous perifolliculitis, is another type of invasive dermatophytosis whereby dermatophytes invade the dermal and subcutaneous tissue [140]. MG presents as nodules, plaques, and papules in hair-bearing skin, most commonly the forearms, hands, legs, or ankles, including areas where trauma to the skin from shaving may occur [22]. Less frequently, MG may involve the face and/or scalp. It presents as one of two forms—a small, perifollicular papular form presenting with localized dermal infection in mostly healthy people, or a second form presenting with deep, subcutaneous, sometimes fluctuant, plaques and/or nodular lesions in immunocompromised individuals [155, 178–182].

7.2.3 Psychological Burden—Chronic dermatophytosis may profoundly impact quality of life (QoL) [183], likely due to physical symptoms, including pruritus, coupled with psychosocial problems from having rashes involving exposed skin [138], which is amplified with chronic dermatophytoses. Several studies have been conducted in communities across India to evaluate the psychosocial impact of chronic dermatophytoses and to quantitate burden of chronic fungal infection on QoL [138, 184–186]. In a cross-sectional study of the impact of chronic and recurrent dermatophytosis on QoL and psychologic morbidity using the Dermatology Life Quality Index (DLQI) and the Perceived Stress Scale (PSS), including 123 patients age 12 years, with 65.9% of participants with dermatophytosis for 6 months, those diagnosed with dermatophytosis for 6–9 months had lower PSS scores versus those with disease for 9–12 months and > 12 months ($p < 0.001$), with over 55% of patients reporting an 'extremely large effect' per the DLQI and involvement of more than one anatomic site a risk factor for higher DLQI score [138]. In a study of 184 children aged 6–12 years with tinea capitis in a semi-urban area of Nigeria, 58.2% had mild-to-severe psychosocial impact, and young females versus males were more often affected (61.5% vs. 41.7%; $p = 0.02$) [187]. While dermatophytoses have a high prevalence, cultural and socioeconomic differences globally may limit generalizing study findings to US communities. Nevertheless, it is well accepted that chronic and recurrent dermatophytoses have a negative impact on psychosocial wellbeing and QoL. Thus, a multidisciplinary approach to address the psychological impact of disease is needed.

7.3 Recalcitrant Dermatophytosis Treatment Guidelines in India

In response to an evolving landscape of recurrent, chronic, and recalcitrant dermatophytosis affecting the Indian subcontinent in recent years, the Indian Association of Dermatologists, Venereologists and Leprologists created the Task Force Against Recalcitrant Tinea (ITART) in 2017 to develop a consensus on the management of tinea affecting the glabrous skin (tinea corporis, tinea cruris, and tinea faciei), which was updated in 2020 [53]. The task force emphasized the importance of maintaining personal hygiene as a key component of managing tinea infections, including thoroughly drying skin after bathing, thoroughly drying clothes after washing, and avoiding sharing soaps, clothes, and other linens.

The 2020 ITART guidelines recommend that patients follow-up 3 weeks after beginning oral therapy to assess clinical response, and state that with inadequate response to terbinafine 250 mg daily after 3 weeks, the dose may be increased to twice daily. However, the FDA-approved dosage is 250 mg daily. The guidelines also recommend topical and oral antifungal therapy combinations for most patients at a longer duration than would otherwise be recommended to dermatophytosis-naïve patients. The Task Force recommended prescribing dermatophytosis-naïve patients itraconazole 100 mg for 3 weeks, and patients with chronic, steroid-modified, or recalcitrant infections, 100 mg twice daily for 4 weeks [53]. Expert consensus is lacking on the most appropriate dosing regimen of itraconazole for dermatophytoses; however, because of concerns that underdosing could lead to resistance, physicians could consider dosing itraconazole 100 mg twice daily at a minimum. ITART also recommends that topical therapy be applied twice daily and for 2 weeks after clinical resolution.

In 2018, an Expert Consensus on the Management of Dermatophytosis in India, including tinea pedis, was developed [188]. Experts recommended tinea pedis of any duration and severity with combination topical and systemic antifungal therapy. Azoles were recommended as first-line topical therapy. Terbinafine 250 mg once daily was recommended for naïve tinea pedis, and itraconazole 200–400 mg/day was preferred for severe or refractory infection. Recommended treatment for naïve tinea pedis is 2–4 weeks, and > 4 weeks for chronic, severe, or recalcitrant infection [188].

8   Emerging Resistance

Chronic and recurrent dermatophytoses may develop due to resistance to antifungal therapies. In the early 1980s–1990s, antifungal resistance among dermatophytes was hypothesized to be due to non-compliance with prescribed treatment, inadequate drug absorption or blood concentration, and drug–drug interactions; resistance was only demonstrated for griseofulvin and typically related to *T. rubrum* tinea corporis [189, 190]. Over the past decade, concerning outbreaks have emerged worldwide, with reports of both clinical and laboratory-confirmed resistance to oral terbinafine and other antifungals, most prominently in the Indian subcontinent [191, 192].

8.1   Pathogenesis

Antifungal resistance may develop for a variety of reasons, including host, pathogen, environmental, and pharmacologic factors [136]. Certain dermatophyte strains, including some *Trichophyton* species, may acquire resistance to antifungal drugs such as terbinafine; these antifungal-resistant strains can cause infection and person-to-person transmission [193]. It is hypothesized that the increased prevalence of antifungal-resistant dermatophyte infections in India stems from overzealous use of antifungal therapies, whether through empiric treatment of suspected but unconfirmed dermatophytosis or through prescription sharing [194–197]. Steroid cream sales in India are estimated at US$329 million, with a large proportion containing an antifungal combination [192]. Corticosteroid-antifungal-antibacterial combination therapies are available over-the-counter in India and other South and Southeast Asian countries at relatively low cost. Additionally, they are overprescribed

for the treatment of a variety of dermatoses, even non-fungal dermatoses. These combination topical therapies often contain high-potency steroids, such as clobetasol, and use of these therapies is likely a major driver of resistance [191, 192]. While these medications may temporarily improve clinical symptoms of dermatophyte infections, including pruritus, they are ultimately problematic in altering the classic morphology of cutaneous fungal infections, leading to delayed diagnosis [102, 191, 192, 198, 199]. These combination topical therapies reduce local cellular immunity at sites of dermatophyte infections, allowing for severe dermatophytosis to develop [191, 192].

8.2 Trichophyton indotineae

The pathogen now widely known as *Trichophyton indotineae* was first identified in 2017 and was referred to as *Trichophyton mentagrophytes* genotype VIII. Although a member of the *T. mentagrophytes* species complex, *T. indotineae* represents a frequently terbinafine-resistant species caused by mutations in the gene encoding squalene epoxidase (SQLE), a key enzyme in the ergosterol biosynthetic pathway whose inhibition results in accumulation of squalene, depletion of ergosterol, and arrest of fungal growth [200]. Mutations in the SQLE gene lead to ineffective enzyme function and cause elevated minimum inhibitory concentrations (MICs) of terbinafine. *T. indotineae* is distinguished from *T. mentagrophytes* in part by sequence differences across the genome including the ITS region [200–203]. Therefore, *T. indotineae* was recognized as a separate species in 2020. This newly identified species is primarily responsible for the epidemic-level prevalence of dermatophyte infections in India [204].

While *T. indotineae* is believed to have been present in India, Australia, Iran, and Oman since 2004 [196], the first clinical cases of what is now known as *T. indotineae* dermatophytosis were described in India after 2014, where chronic and recalcitrant dermatophytosis concerned dermatologists for the past decade [53]. *T. indotineae* dermatophytosis was thereafter recognized in other Asian countries and across Europe [205–215]. The first cases of *T. indotineae* dermatophytoses in the US were reported in February 2023 in New York City, when two patients with severe tinea infections failed to clear despite treatment with oral terbinafine [197]. As such, *T. indotineae* dermatophytosis is now recognized as a worldwide problem [216].

Clinically, *T. indotineae* dermatophytoses evolve differently than classic tinea infections. *T. indotineae* dermatophytoses present as extensive, severe, and/or refractory tinea corporis, cruris, and/or faciei infections [33], and are characterized by intense pruritus, widespread anatomic involvement, and are highly contagious. These infections are highly inflammatory and lesions may be large and numerous [102, 199].

An added complication of identifying *T. indotineae* dermatophytoses is that traditional culture-based techniques utilized for dermatophytosis diagnosis fail to correctly identify *T. indotineae*, instead resulting in diagnoses of *T. mentagrophytes* or *T. interdigitale* dermatophytoses [53, 217, 218]. Therefore, *T. indotineae* dermatophytosis must be confirmed using molecular-based methods, such as PCR, to avoid misdiagnosis and delay of appropriate treatment. In the US, dermatologists are encouraged to contact their state public

health department, who can assist in identifying appropriate testing protocol and laboratories that can perform genomic sequencing [197].

Optimal treatment guidelines for *T. indotineae* dermatophytosis are lacking and current treatment recommendations are based on individual cases or case series. Due to wide-spread resistance to otherwise first-line oral agent terbinafine, oral itraconazole is the drug of choice for treating *T. indotineae* dermatophytosis. In a 2020 study of 200 patients with microscopy-confirmed tinea infection in India, treatment with 8 weeks of itraconazole was superior to treatment with fluconazole, griseofulvin, and terbinafine ($p < 0.001$) for both clinical and mycological cure [219]. Although the patients included in this study were not confirmed *T. indotineae* cases, patients meeting the inclusion criteria had chronic or chronic-relapsing tinea corporis, cruris, or faciei (duration of tinea 3 months, or tinea present for 3 months that improved but relapsed within 1 month), suggesting possible *T. indotineae* infections. A double-blind, randomized trial of 149 adult patients with tinea corporis/cruris involving > 5% body surface area receiving different itraconazole dose regimens (100, 200, 400 mg/day) showed similar complete cure rates for the 100 mg and 200 mg doses (HR 1.44, 95% CI 0.91–2.30; $p = 0.12$) and treatment duration (mean ± standard deviation [SD] 7.7 ± 4.73 vs. 7.2 ± 3.81 weeks; $p = 0.902$), with 400 mg superior for both outcomes (100 vs. 400 mg groups; HR 2.87, 95% CI 1.78–4.62, $p < 0.01$; 200 vs. 400 mg groups (HR 1.99, 95% CI 1.28–3.09; $p = 0.02$). Although the 200 and 400 mg groups had 63% and 120% higher associated costs versus the 100 mg group in achieving cure [68], use of higher itraconazole doses to achieve complete cure may be more cost-effective overall. A dose of itraconazole 200 mg daily may be used to treat infections unresponsive to first-line oral therapies, with the caveat that treatment may be needed for > 3 months [188, 197]. Topical luliconazole cream may also be used although oral therapy is the mainstay of treatment [220].

Combination therapy with terbinafine and itraconazole has been studied for recalcitrant dermatophytoses, although larger studies are needed to validate its use. In a study of 45 patients with recalcitrant dermatophytosis treated for 4 weeks with terbinafine 250 mg and itraconazole 200 mg once daily versus monotherapy with terbinafine 250 mg twice daily or itraconazole 200 mg twice daily, at 12 weeks, 100% of the combination therapy group achieved complete cure versus 80% and 86.7% in the terbinafine-only and itraconazole-only groups, respectively ($p = 0.207$) [221]. In a study of 60 patients with clinical and KOH-positive tinea corporis, cruris, or faciei, at 3 weeks, 90% of patients treated with combination therapy with terbinafine 250 mg once daily and itraconazole 200 mg once daily achieved complete cure versus 35% and 50% in the terbinafine and itraconazole monotherapy groups, respectively ($p = 0.002$) [222].

Resistance to oral itraconazole among dermatophyte infections has been observed [205, 223–225]. In such instances, voriconazole and posaconazole, two oral azole antifungals ordinarily reserved for invasive mold infections, may be utilized. When antifungal therapies fail, physicians should investigate the cause of treatment failure, including adherence to therapy, confirmation of the diagnosis, and whether resistance is suspected. Antifungal susceptibility testing (AFST) could be pursued but is not yet well established in relation to dermatophyte infections.

It is recommended that itraconazole capsules are administered with high-fat foods and an acidic beverage, such as juice, to increase absorption and bioavailability. In contrast, the oral solution should be administered on an empty stomach, either 1 h before, or 2–3 h after, a meal [226]. Triazole antifungals are also associated with substantial drug–drug interactions and adverse effects [227]. For example, coadministering itraconazole with a proton pump inhibitor may reduce bioavailability via gastric acid suppression. Therapeutic drug monitoring may be required for triazoles to ensure adequate dosing, although target serum drug levels have not yet been established for the treatment of resistant dermatophytoses, and the correlation between serum levels and accumulation in tissue has not been established. Given the relatively infrequent use of these antifungals, dermatologists are encouraged to contact infectious disease specialists and pharmacists before prescribing. Issues with insurance coverage may arise when prescribing these less commonly used antifungal therapies, creating a barrier for patients.

8.3 Terbinafine−Resistant *T. rubrum*

T. rubrum terbinafine-resistant tinea corporis cases have been reported in the US [223]. In a retrospective study of 271 dermatophyte isolates from North American institutions from 2021 to 2022, 18.6% were resistant to terbinafine, including 21 isolates of *T. rubrum* [216].

8.4 Implications for Clinical Practice

Empiric treatment of suspected dermatophyte infections is discouraged. At a minimum, dermatologists can confirm mycological diagnosis using KOH with microscopy (Fig. 15), culture, or PCR before prescribing antifungal therapy [228]. If an antifungal-resistant dermatophytosis is suspected and resources are available, advanced molecular techniques (e.g., genomic sequencing of the internal transcribed spacer region) can help identify the causative organism and guide treatment [197, 229]. US dermatologists can contact their state and local health department and Centers for Disease Control and Prevention (CDC) to receive assistance with testing and report unusual cases. If advanced diagnostics are not available, empiric treatment is reasonable.

The emergence of antifungal-resistant tinea infections has several implications for clinical practice. First, prescribing combination topical antifungal-steroid medications for dermatophyte infections is strongly discouraged [53, 188, 218]. Dermatologists may educate non-dermatologists and patients on avoiding such therapies. Furthermore, dermatologists can promote the importance of not sharing prescriptions among household members, of proper adherence to recommended therapies, and of seeking examination by a dermatologist if their condition is not improving with recommended therapy [194].

9 Future Directions

As infections with *T. indotineae* and terbinafine-resistant *T. rubrum* continue to rise in the US, surveillance efforts will be critical to characterize disease epidemiology, monitor spread, describe treatment practices and outcomes, inform clinical practice, and guide public health interventions [197, 230]. Detection of antifungal-resistant species depends on dermatologists maintaining a high index of suspicion and having adequate access to laboratory testing.

In partnership with federal agencies, state and local public health agencies, professional societies, and academic institutions, dermatologists can lead efforts to prevent the spread of antifungalresistant dermatophytes and reduce barriers to testing and appropriate treatment. Staying abreast of updated information regarding new resistant species and changes in treatment guidelines can ensure that dermatologists are providing relevant and informed care to patients.

AFST for dermatophytes was rarely performed before the emergence of *T. indotineae* [231] but is becoming an increasingly important component of managing recalcitrant tinea infections [232]. The European Committee on Antimicrobial Susceptibility Testing (EUCAST) provides guidelines for conducting *in vitro* AFST. A multicenter validation of six EUCAST methods was performed, whereby 10 laboratories with minimal experience with dermatophyte testing performed susceptibility testing of terbinafine, itraconazole, voriconazole and amorolfine against a blinded panel of 38 terbinafine wild-type and target gene mutant isolates, showing a low error rate and favoring a chloramphenicol and cycloheximide supplemented growth media and spectrophotometric endpoint reading using 50% inhibition [233], a potential standardized method for AFST. Although adoption of this method worldwide may generate reproducible susceptibility data, it requires prolonged incubation periods and specialized laboratory equipment with staff who are trained in at least basic laboratory-based work, and many laboratories throughout the world follow the guidelines outlined by the Clinical and Laboratory Standards Institute rather than EUCAST [233, 234]. The terbinafine containing agar method (TCAM) may be used to identify susceptibility to terbinafine. In one study of 20 molecularly identified isolates, including isolates of *T. indotineae*, eight clinical microbiology laboratories received blinded samples. All laboratories correctly identified the terbinafine-resistant isolates ($n = 5$) and the terbinafine-susceptible isolates ($n = 15$) [231], highlighting the reliability and ease of performing the TCAM. However, TCAM is limited to testing terbinafine susceptibility, precluding use in suspected itraconazole-resistant and other suspected antifungal-resistant dermatophytoses [231]. Molecular methods may be used to detect resistant dermatophytes and may identify molecular mechanisms and target gene mutations that confer antifungal resistance, but these methods are not widely available [235]. In one study of a commercially available PCR assay used to identify *Trichophyton* isolates with common SQLE mutations, including Phe397Leu and Leu393Phe, the assay identified all 40 isolates of *T. indotineae* harboring a mutant SQLE gene out of 90 total *T. indotineae* isolates [234]. While this method may potentially provide rapid information about antifungal resistance, it is currently considered an adjunct to culture-based susceptibility testing techniques, due to the potential for a range of mutations in resistant dermatophytes that are not yet available in molecular assays [235]. Additional large, prospective studies are needed to confirm the reliability of molecular methods to detect antifungal-resistant dermatophytes.

10   Conclusion

Antifungal stewardship is a cornerstone of preventing the spread of antifungal-resistant dermatophytoses [229, 236, 237]. Confirmation of dermatophyte infections with appropriate mycological testing is necessary, followed by adherence to recommended treatment guidelines and consensus statements issued by trusted medical organizations and societies.

High clinical suspicion for resistant isolates of dermatophytes is necessary if patients do not improve with the recommended course of therapy. Furthermore, dermatologist-led engagements with regulatory bodies and public health agencies could help address barriers to providing testing and appropriate treatment and to address the public health threat posed by the spread of these infections [197]. Antifungal stewardship extends beyond specialist care by a dermatologist, to both patients and non-dermatologists, who must be educated on the risks of inappropriate prescribing practices related to antifungal therapy.

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Key Points

Dermatophytoses are fungal infections of the skin, hair, and nails.

Over the past decade, some dermatophytoses have become resistant to antifungal therapies, resulting in chronic or recurrent infections.

Antifungal stewardship is a key aspect of managing and preventing treatment-resistant dermatophytoses.

Fig. 1.
Trichophyton tonsurans tinea corporis on the ventral forearm (Dr. Avrom Caplan)

Fig. 2.
Anterior groin of a male patient with *Epidermophyton floccosum* tinea cruris (CDC/Dr. Lucille K. Georg)

Fig. 3.
Tinea corporis on the lower leg (species unknown) with co-occurring fungal folliculitis (Dr. Avrom Caplan)
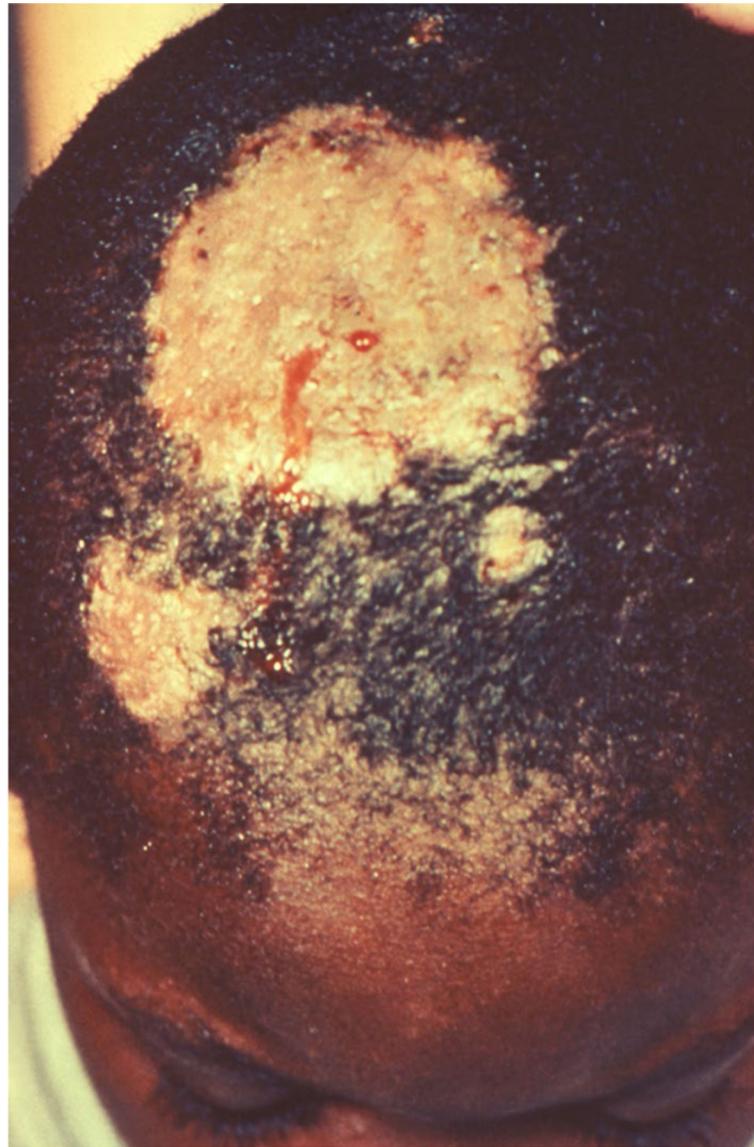
Fig. 4.

Trichophyton mentagrophytes tinea capitis in the scalp of a Black child (CDC)

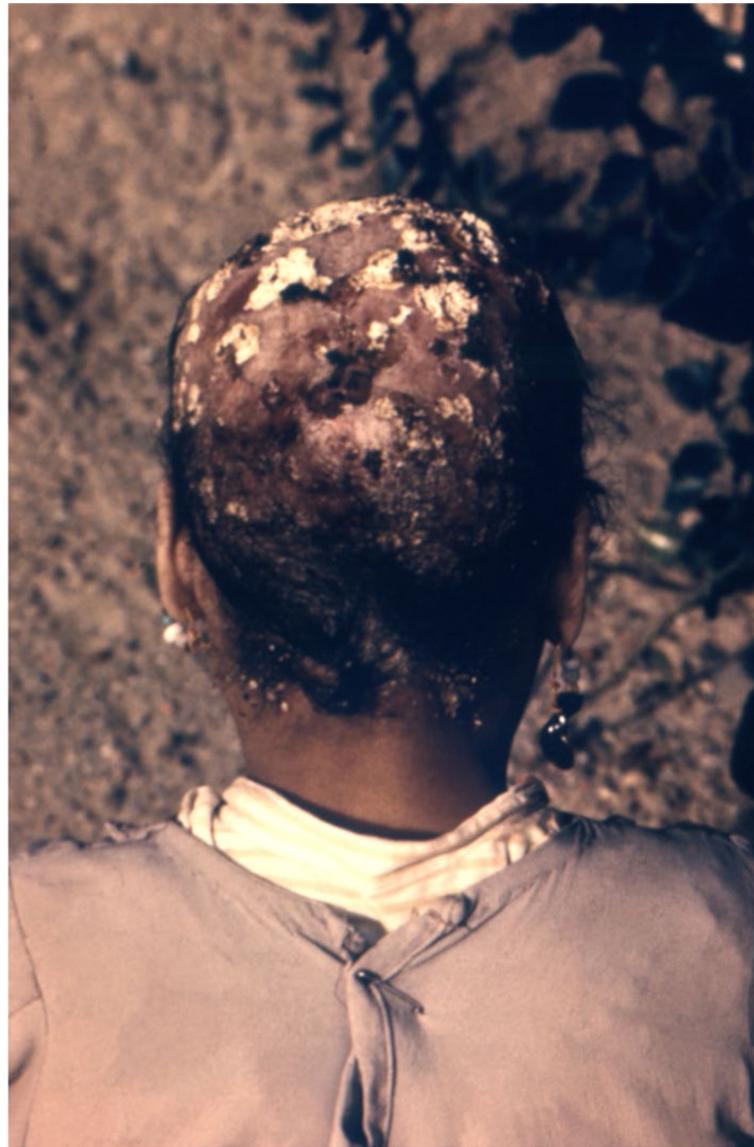**Fig. 5.**
Microsporum tinea capitis (CDC)

Fig. 6.
Trichophyton schoenleinii favus accompanied by alopecia (CDC/Dr. Libero Ajello)

Fig. 7.
Trichophyton schoenleinii mentagrophytes tinea barbae (CDC/Dr. Lucille K. Georg)

Fig. 8.
Tinea faciei of unknown species involving the ear (CDC)

Fig. 9.
Trichophyton concentricum tinea imbricata on the back of a child in Papua New Guinea
(CDC/K. Mae Lennon, Tulane Medical School; Clement Benjamin)

Fig. 10.
Interdigital tinea pedis of unknown species (CDC/Dr. Lucille K. Georg)

Fig. 11.
Tinea pedis (Dr. Shari Lipner)

Fig. 12.
Tinea manuum (Dr. Avrom Caplan)

Fig. 13.
Tinea corporis of the back in a transplant recipient (Dr. Avrom Caplan)

Fig. 14.
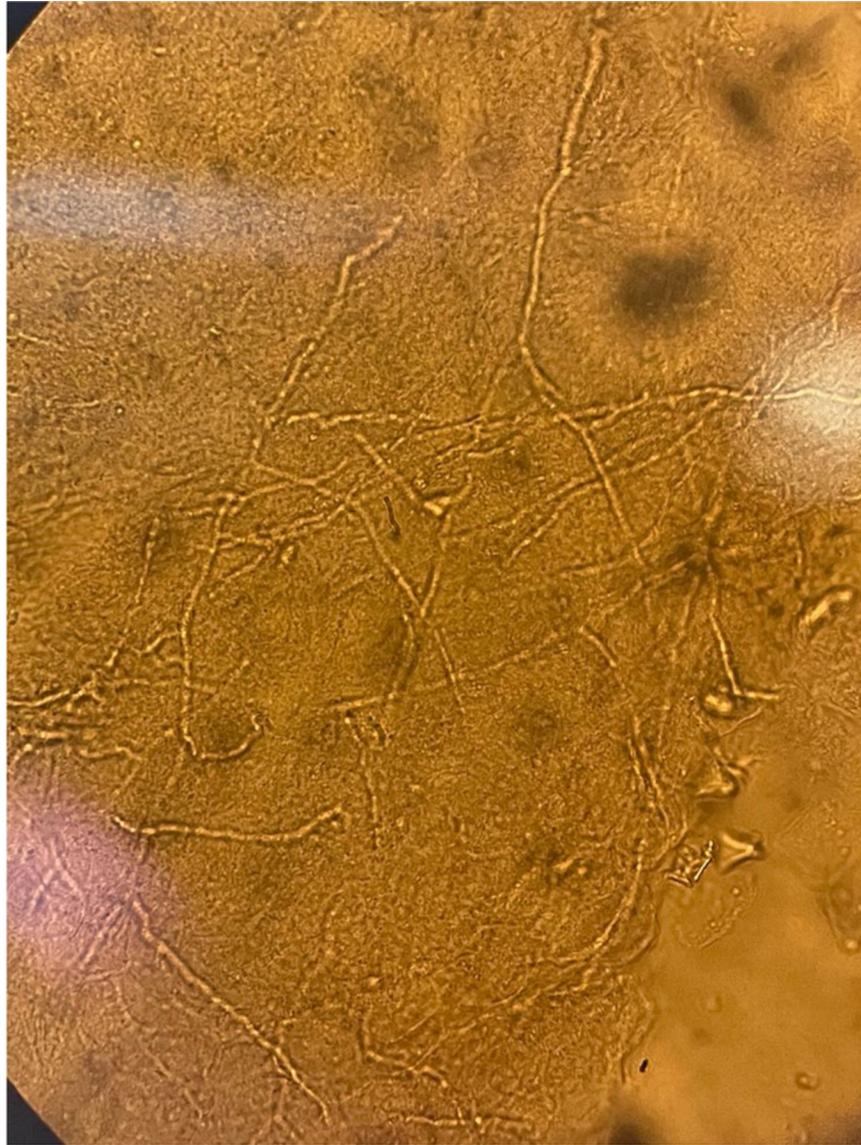Tinea corporis of the distal leg in a transplant recipient (Dr. Avrom Caplan)

Fig. 15.
Trichophyton indotineae KOH preparation (Dr. Avrom Caplan). *KOH* potassium hydroxide

Table 1

Tinea definitions

Type of tinea infection	Definition
Tinea corporis	A superficial fungal infection caused by dermatophytes, with anatomic boundaries defined by the neck, trunk, and extremities
Tinea cruris	A superficial fungal infection involving the crural fold. Commonly referred to as 'jock itch'
Tinea capitis	A dermatophyte infection of the scalp and hair
Tinea barbae	A type of dermatophytic folliculitis; a superficial fungal infection of the hair and hair follicles, involving beards and mustaches in adolescents and adults
Tinea faciei	A dermatophyte infection of the skin of the face in areas without terminal hairs
Tinea incognito	Refers to a tinea corporis, cruris, or faciei infection modified by an anti-inflammatory cream, ointment, or oral therapy that may dampen the local immune response
Tinea pedis	Dermatophyte infection involving the foot/feet. Commonly known as 'athlete's foot'
Tinea manuum	Dermatophyte infection involving the hand(s)
Onychomycosis[a]	Fungal infection of the nails

[a] Outside the scope of this review and will not be explored independently

Table 2

Risk factors

Type of tinea infection	Risk factors
All tinea infections	Contact with an individual harboring a dermatophytosis Secondary spread of infection from another affected anatomic region Hyperhidrosis Diabetes mellitus Poor hygiene Obesity Immunosuppression Living in a hot, humid, tropical climate Occlusive clothing/footwear/headwear Contact sports Use of antibiotics or corticosteroids (topical and systemic) Contact with infected animals (pets, stray, farm, laboratory, and wild animals) Being of low socioeconomic status, due to: Crowded living conditions Increased skin-to-skin contact Reduced access to hygienic products, including soaps, shampoos, detergents, etc. Increased proximity to animals, including house pets (i.e. dogs, cats) or livestock (i.e. cattle, horses) [2, 4]
Tinea corporis	Tinea gladiatorum is a subtype of tinea corporis caused mainly by *T. tonsurans* (common among athletes) Skin-to-skin contact during athletics (example, wrestling) Contact with contaminated training equipment
Tinea cruris	Moisture in the intertriginous groin area Obesity due to apposition of skin folds
Tinea capitis	Decreased sebum production, which leads to decreased fatty acid production and raises the pH of the scalp Significant hormonal changes (pregnancy, menopause, puberty) that decrease sebum production Fomites: sharing combs, hairbrushes, hats, pillows Short hairstyles that allow for ease of colonization of the scalp, including immunosuppression causing impaired hair shaft growth
Tinea pedis	Participation in sports, including marathon running [238] Wearing occlusive, closed-toe shoes for long periods of time while working, including miners and soldiers [112, 240, 241] Frequenting public swimming pools, showers, and gyms [5, 111, 242, 243], especially without donning appropriate footwear Cultural practices that involve bare feet, such as entering a place of worship [244]
Tinea manuum	Contact with the skin of a foot infected with tinea pedis Contact with infected clothing, towel, or soil [3] Recurrent trauma to the hands, such as in individuals who perform manual labor [134]
Tinea barbae	Contact with house pets or other animals via occupational exposure to cattle, horses, etc. [243] Contact with improperly cleaned razor, beard brush, or other facial tool [246] Coarse beard hair
Tinea faciei	Skin-to-skin contact, including wrestling [247]
Tinea incognito	Misdiagnosis of an existing dermatophyte infection Application of steroid cream, tacrolimus ointment, or other anti-inflammatory topical treatment to a dermatophyte infection Systemic glucocorticoids Immunocompromised
Chronic and/or recurrent dermatophytosis	Reduced IFN-γ+ cells, Th1 cells, IL-17+ cells, elevated IL-4+ cells, increased serum IgE levels and a diminished delayed type hypersensitivity intradermal skin test response [189, 248]

Type of tinea infection	Risk factors
	History of tinea in a family member [189]
	History of corticosteroid use [189]
	Sharing towels in the home [189]
	Some comorbid conditions, including atopy, diabetes mellitus [137]
	Poor hygiene
	Steroid use
	Low socioeconomic status

IFN interferon, *Th* T-helper, *IL* interleukin, *Ig* immunoglobulin

Table 3

Predominant organisms responsible for tinea infections worldwide

Type of tinea infection	Region	Species
Tinea corporis, tinea cruris, and tinea faciei	Americas	*T. rubrum, T. tonsurans, M. canis, E. floccosum*
	Europe	*T. rubrum, T. tonsurans, M. canis*
	Africa	*T. rubrum, T. mentagrophytes, M. canis, T. soudanense, T. violaceum, M. audouinii, Nannizzia (Microsporum) gypsea, E. floccosum*
	Middle East	*M. canis, T. violaceum, T. rubrum,*
	Far East and Southeast Asia	*T. mentagrophytes, T. interdigitale, T. rubrum, T. concentricum, E. floccosum*
	Indian subcontinent	*T. rubrum, T. verrucosum, T. mentagrophytes, T. indotineae*
Tinea capitis	Americas	*T. tonsurans, M. canis*
	Europe	*M. canis, T. tonsurans, T. soudanense, T. violaceum, M. audouinii*
	Africa	*T. violaceum, T. soudanense, M. audouinii*
	Middle East	*T. violaceum, M. canis, T. tonsurans*
	Far East and Southeast Asia	*T. violaceum, T. schoenleinii, M. canis, T. tonsurans*
	Indian subcontinent	*T. violaceum, M. audouinii, T. schoenleinii, T. tonsurans*
Tinea barbae	Worldwide	*T. verrucosum, T. mentagrophytes*
Tinea pedis	Americas	*T. rubrum, T. mentagrophytes, T. interdigitale, E. floccosum*
	Europe	*T. rubrum, T. interdigitale*
	Africa	*T. rubrum, T. mentagrophytes, T. interdigitale, T. violaceum*
	Middle East	*T. rubrum, T. mentagrophytes, T. interdigitale*
	Far East and Southeast Asia	*T. rubrum, T. mentagrophytes, T. interdigitale*
	Indian subcontinent	*T. rubrum, T. mentagrophytes, T. interdigitale*

Table 4

Clinical features

Type of tinea infection	Clinical features
Tinea corporis	Annular, erythematous, and pruritic patches or plaques with scaly and active raised borders, and central clearing as the infection advances [36, 37] Pustules may appear along the border of the patch or plaque Multiple areas can be affected with coalescing plaques
Tinea cruris	Annular, erythematous, and pruritic patches or plaques with scaly and active raised borders, and central clearing as the infection advances [36, 37] Always involves the crural fold Infection may spread to the thighs, genital, pubic, perineal, and perianal skin Penis and scrotum in men and labia majora in women are commonly spared [249, 250] Often presents with pruritus Pain may be a prominent feature if maceration or a secondary bacterial infection are present [250, 251]
Tinea capitis, endothrix	A type of non-inflammatory tinea capitis Characterized microscopically by arthroconidia within the hair shaft Typically caused by *T. tonsurans, T. violaceum, and/or T. soudanese* infections. Hair breakage occurs at the level of the scalp, resulting in the appearance of 'black dots', which are representative of the broken, distal ends of hairs Does not fluoresce under Wood's lamp [6, 75]
Tinea capitis, ectothrix	A type of non-inflammatory tinea capitis characterized microscopically by arthroconidia outside of the hair shaft, occurring at the mid-follicular level. Hyphae then grow towards the bulb of the hair Typically caused by *T. verrucosum, M. canis, M. audouinii, Nannizzia nana, and N. gypsea* Often present as scaly patch(es) with accompanying inflammation Hair loss occurs, with breakage of the hair above the level of the scalp by at least 2–3 mm Does fluoresce under Wood's lamp [6, 74]
Tinea capitis, favus	A type of inflammatory tinea capitis characterized microscopically by hyphae and air spaces within hair shafts Almost uniquely caused by *T. schoenleinii* Begins as erythema surrounding a hair follicle, that progresses to the classic presentation of a scutulum lesion The scutulum (plural scutula) is a concave yellow crust containing fungal hyphae, neutrophils, and epidermal cells that appears on the scalp and cover areas of severe alopecia. Scutula may coalesce to form plaques, which may lead to secondary bacterial infections [94, 252] If left untreated, inflammation may lead to scarring [252]
Tinea capitis, kerion	An inflammatory-type presentation of tinea capitis, most often caused by zoophilic dermatophytes [253] Usually presents as a solitary, painful, boggy plaque on the occipital scalp Begins as a folliculitis and a scaly lesion containing short hairs. It then progresses to an erythematous, tender, inflamed boggy plaque, that is covered with pustules producing a copious purulent discharge [254] Patients often experience fever and cervical lymphadenopathy Major and minor features: Major features: tenderness to palpation, alopecia within the lesion, numerous pustules and purulent drainage, and scaling Minor features: a dermatophytid reaction, a type of secondary immunologic reaction caused by dermatophytosis resulting in eczematous scaly patches or plaques at a site distinct from the main infection [255], regional lymphadenopathy (namely cervical), short hairs on dermoscopy, boggy plaques, clear demarcation of borders, overlying erythema, and pruritus [253]
Tinea pedis, interdigital	Mainly caused by *T. rubrum* May present with scaling, erythema, maceration, or fissuring, particularly in the fourth interdigital space [112] Generally spares the dorsal foot, but may spread to adjacent plantar surfaces Typically pruritic and may be foul smelling [112] Further subcategorized into dermatophytosis simplex, a dry phenotype with mild peeling scale, and wet tinea pedis, which presents with wet, macerated interdigital spaces [106]
Tinea pedis, hyperkeratotic ('moccasin-type')	Typically caused by *T. rubrum* Named for its distribution Entire plantar surfaces and lateral feet are typically involved bilaterally, while the dorsal feet remain clear [112]

Type of tinea infection	Clinical features
Tinea pedis, vesiculobullous (inflammatory)	Presents on a spectrum, from slight scaling to diffuse hyperkeratosis, with chronic erythema of the plantar surface Occasionally, thick scales and fissures develop along with papules along the line of erythema of the foot [106] More commonly caused by *T. mentagrophytes* Typically involves the medial foot, foot arches, and the sides of the toes Characterized by tense vesicles, pustules, or bullae May be accompanied by burning pain and intense pruritus that may impact ambulation While bullae often contain clear fluid, purulence may ensue with bacterial superinfection, particularly with *Staphylococcus aureus* or *group A Streptococcus* infection [106, 112]
Tinea pedis, rare ulcerative form	Rare Most often caused by *T. interdigitale* Presents with ulcers and vesicular lesions that spread rapidly and are prone to bacterial superinfection [112] There may be maceration involving the interdigital spaces and the plantar surface of the foot [112] Immunocompromised patients are at greatest risk [256]
Tinea manuum	Typically presents as scaling and erythema of the palm, dorsum, and/or interdigital spaces of one or both hands [257] May resemble tinea corporis with an erythematous plaque and an active border [134] 80% of patients with tinea manuum have co-occurring tinea pedis [2, 239] but tinea manuum may also present independently Clinical features often similar to those of moccasin-type tinea pedis [112] Two feet-one hand syndrome describes involvement of unilateral tinea manuum and bilateral tinea pedis, often with co-occurring onychomycosis, a characteristic distribution that likely results from direct hand-to-foot contact while scratching affected feet [239, 258–260]
Tinea barbae, noninflammatory	Affects the hair and hair follicles of beards and mustaches in adult and adolescent males Caused by anthropophilic (humans as primary host) organisms, such as *T. tonsurans and T. rubrum* Flat, scaly plaques with central clearing, vesicles and pustules may be present at the active border [261] May be difficult to distinguish from tinea faciei [261]
Tinea barbae, inflammatory	Affects the hair and hair follicles of beards and mustaches in adult and adolescent males Caused by zoophilic (animals as primary host) organisms, such as *T. mentagrophytes and T. verrucosum* [262] Kerion lesion develops, with erythema, boggy, tender, weeping nodule or plaque. Pustules and draining sinuses may be present [263, 264] Hair easily plucked and appears brittle Hair loss occurs within the infected areas Pain and pruritus usually mild or non-existent Usually unilateral Constitutional symptoms, including fever, may be present Superinfection of kerion with bacterial organisms may occur May result in alopecia and scar formation if left untreated [262]
Tinea faciei	The most frequently misdiagnosed superficial fungal infection [265] Affects the glabrous skin of the face Erythematous, annular plaques with scaly borders [70] Begins with scaly papules that extend outward in a ring, with the central area becoming hypoor hyper-pigmented [70, 265] Pustules may be present [265]
Tinea incognito	Classically refers to tinea corporis, cruris, or faciei modified by a topical steroid cream applied due to misdiagnosis of dermatophyte infection as another dermatosis, commonly eczema or psoriasis [104, 105, 266] Also known as 'steroid-modified tinea' May also be caused by other anti-inflammatory creams, such as tacrolimus ointment, or systemic glucocorticoids [104] Raised margins and scale typically associated with dermatophytosis may be subtle, making diagnosis challenging May present with inflammatory papules and pustules [267] Affected area may become extensive with unusually shaped borders due to suppression of the local immune response and allowing the fungus to grow [267] Secondary changes caused by long-term steroid use may be present, such as skin atrophy, purpura, or telangiectasia

Table 5

Differential diagnosis

Type of dermatophytosis	Differential diagnosis
Tinea corporis	Subacute cutaneous lupus erythematosus Granuloma annulare Erythema annulare centrifugum Nummular eczema Tinea versicolor Cutaneous candidiasis Pityriasis rosea Allergic contact dermatitis Irritant contact dermatitis Atopic dermatitis Seborrheic dermatitis Psoriasis Impetigo Lyme disease
Tinea cruris	Candidal intertrigo Seborrheic dermatitis Inverse psoriasis Erythrasma Allergic contact dermatitis Irritant contact dermatitis Lichen simplex chronicus
Tinea capitis	Alopecia areata Trichotillomania Seborrheic dermatitis Atopic dermatitis Allergic contact dermatitis Irritant contact dermatitis Psoriasis Traction alopecia Systemic lupus erythematous Psoriasis Pityriasis amiantacea Bacterial folliculitis Sterile folliculitis Pyoderma
Tinea pedis	Erythrasma Interdigital *Candida* infection Atopic dermatitis Allergic contact dermatitis Irritant contact dermatitis Palmoplantar eczema Dyshidrotic eczema Palmoplantar psoriasis Pitted keratolysis Juvenile plantar dermatosis Keratolysis exfoliative Palmoplantar keratoderma Palmoplantar pustulosis Scabies Gram negative toe web infection Plantar psoriasis Friction blisters Autoimmune blistering disorders Pityriasis rubra pilaris
Tinea manuum	Psoriasis Allergic contact dermatitis Irritant contact dermatitis Atopic hand dermatitis Occupational dermatitis Dyshidrotic eczema Keratolysis exfoliative
Tinea barbae	Bacterial folliculitis Perioral dermatitis Pseudofolliculitis barbae Contact dermatitis

Type of dermatophytosis	Differential diagnosis
	Herpes simplex
	Sporotrichosis
	Acne vulgaris
	Acne rosacea
Tinea faciei	Atopic dermatitis
	Seborrheic dermatitis
	Allergic contact dermatitis
	Irritant contact dermatitis
	Psoriasis
	Rosacea
	Actinic keratosis
	Perioral dermatitis
	Cutaneous lupus erythematosus
	Polymorphous light eruption
	Nummular eczema
	Sarcoidosis
	Granuloma annulare

Table 6

Topical antifungals

Drugs	Indication	Dose frequency	FDA age approvals
Allylamines			
Naftifine 2% cream	Tinea corporis Tinea cruris Tinea pedis	Once daily for 2 weeks	Tinea corporis: age ≥ 2 years Tinea cruris and tinea pedis: age ≥ 12 years
Terbinafine 1% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/cruris: once daily for 1 week Tinea pedis (interdigital): twice daily for 1 week Tinea pedis (moccasin): twice daily for 2 weeks	Tinea corporis, cruris, and pedis: age ≥ 12 years
Azoles and imidazoles			
Clotrimazole 1% cream or solution	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis and tinea pedis: twice daily for 4 weeks Tinea cruris: twice daily for 2 weeks	Age ≥ 2 years
Econazole 1% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/cruris: once daily for 2 weeks Tinea pedis: once daily for 1 month	Age ≥ 2 years
Ketoconazole 2% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/tinea cruris: once daily for 2 weeks Tinea pedis: once daily for 6 weeks	Age ≥ 2 years
Luliconazole 1% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/cruris: once daily for 1 week Interdigital tinea pedis: once daily for 2 weeks	Tinea corporis: age ≥ 2 years Tinea cruris and tinea pedis: age ≥ 12 years Age ≥ 2 years
Miconazole 2% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis: twice daily for 4 weeks Tinea cruris: twice daily for 2 weeks Tinea pedis: twice daily for 4 weeks	Age ≥ 2 years
Oxiconazole 1% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/cruris, tinea pedis: once to twice daily for 2 weeks	Age ≥ 2 years
Sertaconazole 2% cream	Tinea pedis	Twice daily for 4 weeks	Age ≥ 2 years
Sulconazole 1% cream or solution	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/cruris: once daily for 3 weeks Tinea pedis: twice daily for 4 weeks	The safety and effectiveness in pediatric patients have not been established
Benzylamine			
Butenafine 1% cream	Tinea corporis Tinea cruris Tinea pedis	Tinea corporis/cruris: once daily for 2 weeks Interdigital tinea pedis: twice daily for 1 week or once daily 4 weeks	Age ≥ 12 years
Other			
Ciclopirox 0.77% cream	Tinea corporis Tinea cruris Tinea pedis	Twice daily for 4 weeks	Age ≥ 10 years

Drugs	Indication	Dose frequency	FDA age approvals
Ciclopirox 0.77% gel	Tinea corporis Tinea cruris Tinea pedis	Twice daily for 4 weeks	Age 16 years
Tolnaftate 1% cream or solution	Tinea corporis Tinea cruris Tinea pedis	Twice daily for 4 weeks	Age 2 years

Table 7

Oral antifungals

Drug	Preparation	Indications	Adult dose	Pediatric dose	Duration	Example adverse effects	Laboratory monitoring
Griseofulvin microsize	Tablet, suspension	First-line therapy for *Trichophyton* tinea capitis First-line therapy for *Microsporum* tinea capitis Second-line therapy for refractory tinea corporis/cruris infection or with vellus hair involvement	*Tinea capitis*[a] Optimal treatment regimen unclear *Tinea corporis/cruris* 500–1000 mg/day	*Tinea capitis* 20–25 mg/kg/day *Tinea corporis/cruris* 10–20 mg/kg/day	6–12 weeks 2–4 weeks	Gastrointestinal distress Headache Hepatotoxicity Granulocytopenia Leukopenia	Not necessary before initiation of therapy If therapy extends past 8 weeks or multiple courses given, check liver function panel and complete blood count
Griseofulvin ultramicrosize	Tablet	First-line therapy for *Trichophyton* tinea capitis First-line therapy for *Microsporum* tinea capitis Second-line therapy for refractory tinea corporis/cruris infection or with vellus hair involvement	*Tinea capitis*[a] 10–15 mg/kg/day *Tinea corporis/cruris* 375–500 mg/day	*Tinea capitis* 10–15 mg/kg/day *Tinea corporis/cruris* 5–15 mg/kg/day	6–12 weeks 2–4 weeks	Gastrointestinal distress Headache Hepatotoxicity Granulocytopenia Leukopenia	Not necessary before initiation of therapy If treatment extends past 8 weeks or multiple courses given, check liver function panel and complete blood count
Terbinafine tablets	Tablet	First-line therapy for *Trichophyton* tinea capitis Second-line therapy for *Microsporum* tinea capitis First-line therapy for refractory tinea corporis/cruris infection or with vellus hair involvement First-line therapy for refractory tinea pedis	*Tinea capitis*[a] 250 mg/day *Tinea corporis/cruris* 250 mg/day	*Tinea capitis* 10–20 kg: 62.5 mg daily, 20–40 kg: 125 mg daily, 40 kg: 250 mg daily *Tinea corporis/cruris* 10–20 kg: 62.5 mg daily, 20–40 kg: 125 mg daily, 40 kg: 250 mg daily *Tinea pedis* 10–20 kg: 62.5 mg daily, 20–40 kg: 125 mg daily, 40 kg: 250 mg daily	4–6 weeks (*Trichopyton*); 8–12 weeks (*Microsporum*) 1–2 weeks 2 weeks	Gastrointestinal distress Headache Taste disturbances Hepatotoxicity Pancytopenia	Liver function panel prior to therapy initiation If treatment extends past 6 weeks, check liver function panel and complete blood count
Fluconazole	Tablet, suspension	Second-line therapy for *Trichophyton* tinea capitis Second-line therapy for *Microsporum* tinea capitis Second-line therapy for refractory tinea corporis/cruris infection or with vellus hair involvement Second-line therapy for refractory tinea pedis	*Tinea capitis*[a] 6 mg/kg/day— *Tinea corporis/cruris* 150–200 mg once weekly *Tinea pedis* 150 mg once weekly	*Tinea capitis* 6 mg/kg/day *Tinea corporis/cruris* 6 mg/kg once weekly *Tinea pedis* 6 mg/kg once weekly	3–6 weeks 2–4 weeks 2–6 weeks	Gastrointestinal distress Headache Hepatotoxicity Prolonged QT interval	None

Drug	Preparation	Indications	Adult dose	Pediatric dose	Duration	Example adverse effects	Laboratory monitoring
Itraconazole	Capsule, solution	Second-line therapy for *Trichophyton* tinea capitis Second-line therapy for *Microsporum* tinea capitis Alternative first-line therapy for refractory tinea corporis/cruris infection or with vellus hair involvement with contraindication to terbinafine or terbinafine treatment failure Second-line therapy for refractory tinea pedis	*Tinea capitis*[a] 5 mg/kg/day *Tinea corporis/cruris* 200 mg/day *Tinea pedis* 200 mg twice daily	*Tinea capitis* 3–5 mg/kg/day *Tinea corporis/cruris* 3–5 mg/kg/day *Tinea pedis* 3–5 mg/kg/day	2–6 weeks 1 week (longer courses required in some cases) 1 week	Gastrointestinal distress Headache Dizziness Hepatotoxicity Cardiotoxicity (triad of hypertension, hypokalemia, peripheraledema)	Liver function panel prior to therapy initiation If treatment extends past 4 weeks, check liver function panel

[a] Optimal treatment regimens for tinea capitis in adults are not well known