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Superficial Fungal Infections

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Dr Kelly has disclosed no financial relationships relevant to this article. This commentary does contain a discussion of an unapproved/investigative use of a commercial product/device.

Educational Gap

Superficial fungal infections are often mistaken for other common diseases, and the epidemiology of tinea capitis has changed, requiring adaptation in diagnostic techniques.

Objectives After completing this article, readers should be able to:

1. Recognize the many forms of superficial fungal infections and be aware of their varied clinical presentations and complications.
2. Be aware of other common skin conditions that can mimic superficial fungal infections.
3. Understand proper techniques for diagnosing superficial fungal infections.
4. Be aware of recommended regimens for treating superficial fungal infections.

Introduction

Infections caused by pathogenic fungi and limited to the human hair, nails, epidermis, and mucosa are referred to as superficial fungal infections. Despite the fact that these infections rarely are dangerous or life threatening, they are important because of their worldwide distribution, frequency, person-to-person transmission, and morbidity. Furthermore, particularly severe infections or those refractory to treatment may be the first indication of an underlying immunodeficiency.

Dermatophytosis (tinea or ringworm), pityriasis versicolor (formerly tinea versicolor), and candidiasis (moniliasis) are the three most common types of superficial fungal infections. The dermatophytes are a large group of related fungi that can infect human skin, hair, and nails; they are found in soil (geophilic organisms), on animals (zoophilic), and on humans (anthropophilic). These fungi require keratin for growth and, therefore, they are unable to infect mucosal surfaces. These fungi are found all over the world, although the specific species, and subsequent clinical presentation, vary from region to region.

Dermatophytosis is labeled by the involved area of the body (eg, tinea corporis, tinea capitis, tinea pedis, tinea unguium). Pityriasis versicolor is caused by the yeast form of a dimorphic fungus that is considered part of the normal human skin flora. Candidiasis is caused by a yeast-like fungus of the genus *Candida* (most commonly *C albicans*) that is part of the microflora in the human gastrointestinal tract (including the mouth) and the vagina. Symptoms and signs of candidiasis manifest with a change in the normal host immune system.

This article will review a practical approach to the diagnosis and treatment of dermatophytosis, pityriasis versicolor, and other common diseases that often are mistaken for a superficial fungal infection. Candidiasis will not be discussed further in this review. Tinea capitis will be reviewed in detail because of its high incidence in children, need for systemic therapy, and potential for significant morbidity (particularly scarring alopecia).

Diagnosis

Many clinicians diagnose and treat superficial fungal infections based on clinical presentation alone. However, there are many infectious and noninfectious disorders that can mimic fungal infections. Microscopic confirmation of a dermatophyte or yeast infection is quick

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and painless and can be reassuring to the patient and parent. Proof of infection by fungal culture is recommended when prolonged systemic antifungal therapy is considered, especially with cases of tinea capitis and onychomycosis.

The Wood lamp has been used in dermatology since 1925. It emits long wavelength ultraviolet radiation and can be helpful to the pediatrician for diagnosing a wide variety of dermatologic conditions (Table 1), including tinea capitis and pityriasis versicolor. However, in the United States, a change in the epidemiology of tinea capitis has rendered this technique nearly obsolete for diagnosing this disorder. Tinea capitis in North America is caused predominately by *Trichophyton* sp and is an endothrix infection. The spores (arthroconidia) exist within the hair shaft in an endothrix infection and, therefore, do not fluoresce under ultraviolet light.

Ectothrix infection (usually caused by *Microsporum* sp), by contrast, has arthroconidia on the outer surface of the hairs and fluoresces a brilliant green with a Wood lamp examination. *Microsporum* is the most common cause of tinea capitis in many parts of the world (Fig 1). Thus, a hand-held Wood lamp will be useful to the pediatrician practicing in Brazil, central and eastern Europe, Asia, Africa, and Australia.

Favus, a rare chronic form of tinea capitis caused by *T schoenleinii*, will fluoresce a pale green with examination under a Wood lamp. Dermatophyte and *Candida* infections limited to the skin do not display fluorescence with ultraviolet light. However, tinea cruris and *Candida* intertrigo can be confused easily with erythrasma. Erythrasma is a bacterial infection caused by *Corynebacterium minutissimum*, which fluoresces a brilliant coral red when examined with a Wood lamp. This examination also can be helpful in diagnosing pityriasis versicolor. The organism, *Malassezia furfur*, produces a pale yellow to white fluorescence with a Wood lamp.

Microscopic confirmation of a fungal infection is extremely helpful in diagnosing suspected dermatophytosis, pityriasis versicolor, and *Candida* infection. Superficial scraping or clippings from skin, nails, subungual debris, or hair can be prepared on a slide with potassium hydroxide (KOH) for microscopic review. The KOH destroys the squamous cells without affecting the fungal elements. Skin scraping often is performed with a No. 15 scalpel blade in adults; I prefer to use the edge of a glass slide in children. A thin layer of material is placed on a glass slide and a coverslip is applied. A few drops of 10% to 20% KOH are applied until the area under the coverslip is filled. Gentle heating of the slide will accelerate the destruction of the squamous cells.

Gentle pressure applied to the coverslip prior to visualization will help with layering of the fungal elements and remove trapped air. Low-power visualization with a dimmed condenser will reveal branching hyphae with septations when there is a dermatophyte infection. The KOH preparation in a patient with pityriasis versicolor will reveal short hyphae and clusters of spores, often described as a “spaghetti and meatballs” pattern.

Fungal culture is the gold standard technique for diagnosing dermatophytosis. Culture usually is unnecessary except in cases of tinea capitis and onychomycosis. Positive results often take 7 to 14 days to develop, and the test is expensive. The sample is placed in a fungal culture media (eg, Sabouraud dextrose agar or Dermatophyte Test Medium) that contain antimicrobials to prevent the growth of saprophytic fungi and bacteria. Samples in suspected tinea capitis are obtained most easily with a tap water–moistened cotton swab rubbed vigorously over the scalp. This technique has proved to be reliable and is much more tolerable for a young child than the traditional hair plucking technique. (1) In suspected onychomycosis, material should be obtained from nail clippings and scraping under the nail.

Table 1. Conditions That Fluoresce With Wood Lamp Examination

Disorder	Fluorescent Organism or Site	Fluorescent Color
Tinea capitis	<i>Microsporum audouinii/canis</i>	Brilliant green
Favus	<i>Trichophyton schoenleinii</i>	Pale green
Pityriasis versicolor	<i>Malassezia furfur</i>	Pale yellow to white
Erythrasma	<i>Corynebacterium minutissimum</i>	Brilliant coral red
Tuberous sclerosis (ash leaf spot)	Hypopigmented skin	Pale white
Vitiligo	Depigmented skin	Bright white
Incontinentia pigmenti	Atrophic streaks on skin	Pale white
Congenital erythropoietic porphyria	Teeth/urine	Red–orange
Erythropoietic protoporphyria	Erythrocytes	Red
Porphyria cutanea tarda	Urine	Red

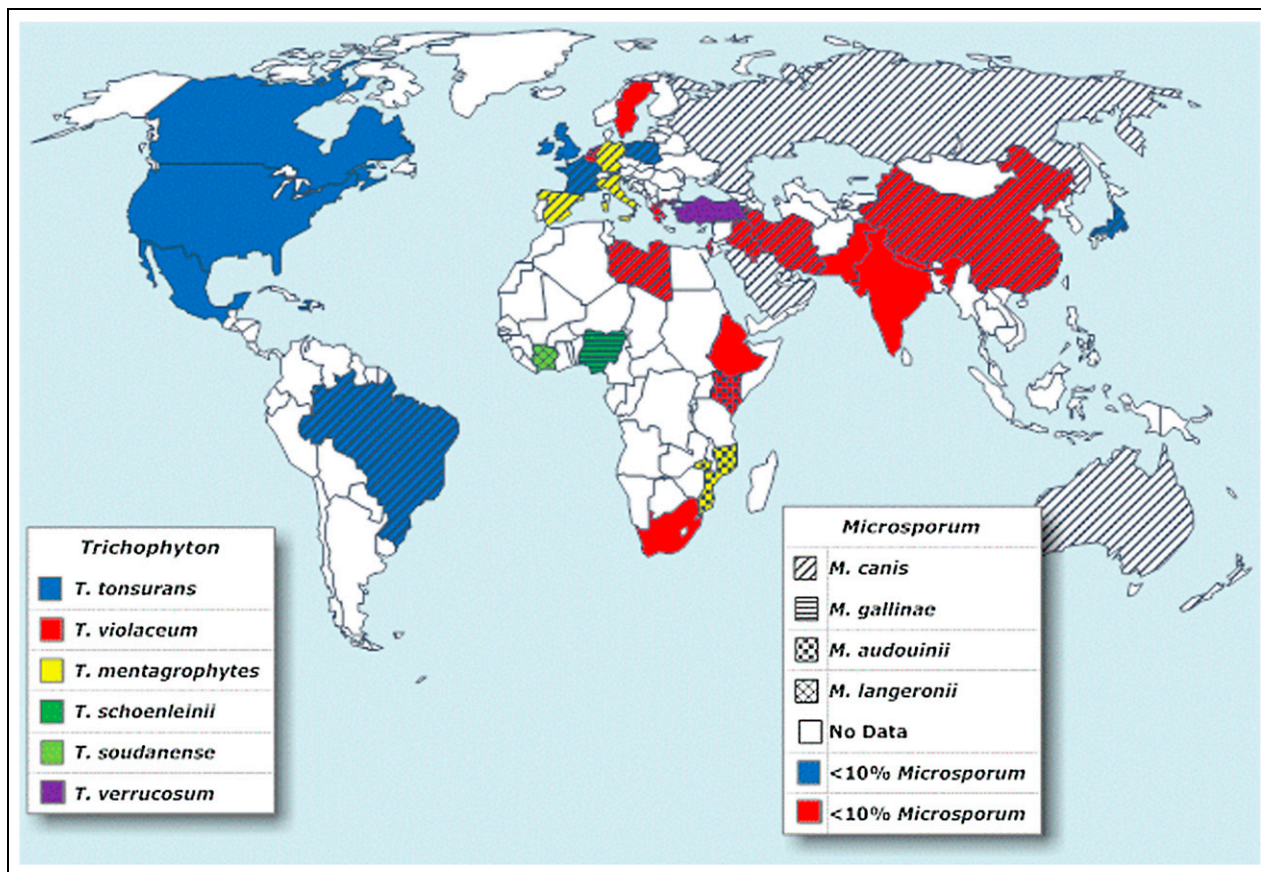


Figure 1. Worldwide epidemiology of tinea capitis. Solid color means that more than 90% of cases in that country are caused by the indicated *Trichophyton* sp. Hatch marks without color indicate that more than 90% of cases in that country are caused by the indicated *Microsporum* sp. Combination of color and hatch marks means that both *Trichophyton* and *Microsporum* spp are significant causes of tinea capitis in that country. Data collected by a review of published articles (MEDLINE) since 1995.

Tinea Capitis

Epidemiology

Tinea capitis is the most common superficial fungal infection in North America. In the United States, disease prevalence among inner-city school-age children is 3% to 8%. (2) This infection mainly affects young children (ages 3 to 9 years), although adults are known to be frequent asymptomatic carriers of the dermatophyte. Tinea capitis is more common with crowded living conditions and low socioeconomic and urban settings and in African American children. The prevalence of this disease varies considerably around the world (eg, <1% in Madrid and Palestine to greater than 50% in Ethiopia, where *Trichophyton violaceum* is endemic). (3)

In North America, the epidemiology of this disease has changed dramatically in the past 50 years. Ectothrix infections in the United States with *M canis* (zoophilic)

and *M audouinii* (anthropophilic) have essentially been eradicated and replaced with the endothrix, anthropophilic dermatophyte *T tonsurans*. Recent studies in the United States and Canada show that tinea capitis is caused by *Trichophyton* sp in more than 95% of cases, with *T tonsurans* the most common species by far. A similar pattern has been noted in Ireland and the United Kingdom.

The worldwide incidence of this disease has increased in the past 30 years. Zoophilic organisms, particularly *M canis*, continue to be a common cause of tinea capitis in South America, central and eastern Europe, the Middle East, Russia, China, and Australia. Only nine of the more than 40 known dermatophyte species are responsible for tinea capitis infections. Within a particular country or region, this disease is caused by only one, two, or three dermatophyte species. Furthermore, species of *Epidermophyton* (a common cause of tinea corporis) do not appear to infect the hair shaft.

Local knowledge of the epidemiology of tinea capitis will help pediatricians who are practicing in international health locales (Fig 1). This knowledge is helpful because the choice of systemic antifungal therapy may be different between *Trichophyton*-predominant and *Microsporum*-predominant regions.

Transmission

Tinea capitis is transmitted commonly by indirect contact with fallen hair and epithelial cells. Direct head-to-head transmission among children is thought to be considerably less common. Transmission of dermatophytes from hair brushes, combs, and shared hats also is common. *Trichophyton* and *Microsporum* have been isolated from barbershop instruments, combs, hair brushes, bedding, clothing, furniture, theater seats, and towels. Co-sleeping has been associated with the spread of tinea capitis. *M canis* has been found to be transmitted to humans by both dogs and cats. Rodent pets, especially guinea pigs, may occasionally transmit *M canis* to humans.

Stray cats are thought to be the most important reservoir for *M canis* infection in urban environments outside of North America. For example, a 2004 Italian study found that 10% of dogs and 28% of cats with a skin condition were infected with a dermatophyte (*M canis* was the most common isolate in both animal groups). (4) Farmers have a unique exposure to *T verrucosum* (zoophilic, from cattle) and *M gypseum* (geophilic), both of which can cause tinea capitis.

The presence of asymptomatic carriage among family members is common and of unclear significance. A study done in South London that screened household contacts

of patients with tinea capitis caused by *T tonsurans* found that 44% had asymptomatic carriage of the dermatophyte. (5) Current guidelines do not recommend the routine screening of family members for the presence of dermatophyte carriage.

Clinical Presentation

Tinea capitis can present with a wide variety of clinical patterns (Table 2; Figs 2–6). An individual may have one, a few, or many of the following features: alopecia, scale, erythema, pustules, “black dots,” tenderness, pruritus, and lymphadenopathy (suboccipital or cervical). Because of the variability of these features, the differential diagnosis of tinea capitis is broad and case specific.

Two US studies have examined the frequency of clinical symptoms and signs in symptomatic children with culture-proved tinea capitis (Table 3). The table shows the prevalence of symptoms or signs in culture-positive groups. Both studies were convenience samples of outpatient children with suspected tinea capitis.

Hubbard (6) found that the presence of occipital or posterior auricular lymphadenopathy was predictive of culture-proved *T tonsurans* infection (positive likelihood ratio = 7.5). In this study, all children with adenopathy and alopecia (100%) and nearly all children with adenopathy and scaling (97%) had cultures confirming *T tonsurans* infection. Conversely, no child without adenopathy and scaling (0%) and only one child without adenopathy and alopecia (6%) had cultures positive for dermatophytosis. Hubbard concluded that posterior auricular adenopathy in combination with pruritus, scaling, or alopecia was highly predictive of culture-proved tinea capitis.

Table 2. Clinical Patterns Associated With Tinea Capitis

Clinical Pattern	Description	Differential Diagnosis
“Black dot”	Well-demarcated alopecia with “black dots” (hairs broken off close to scalp with <i>T tonsurans</i> infection)	Alopecia areata, trichotillomania Note: “exclamation point” hairs and nail pitting suggest alopecia areata.
“Gray patch”	Patchy and scaly alopecia with or without erythema	Seborrheic dermatitis, atopic dermatitis, contact dermatitis, psoriasis, traction alopecia, systemic lupus erythematosus
Diffuse scale	Widespread scaling of scalp with or without erythema	Seborrheic dermatitis, atopic dermatitis, contact dermatitis, psoriasis, pityriasis amiantacea
Diffuse pustular	Scattered pustules with scale, alopecia, and lymphadenopathy	Bacterial folliculitis, sterile folliculitis (associated with hair oils)
Kerion	Dramatic boggy, erythematous, tender plaque with pustules and lymphadenopathy	Pyoderma, neoplasia, pyoderma gangrenosum (uncommon in children)
Favus	Dramatic yellow cup-shaped crusts around the hair	Psoriasis, pityriasis amiantacea

Table adapted from Higgins et al. (9).



Figure 2. Tinea capitis characterized by gray, patchy scaling with alopecia. Lesion was noted after a haircut. (Reprinted from Shy R. Tinea corporis and capitis. *Pediatr Rev.* 2007;28:169. © American Academy of Pediatrics.)



Figure 4. Tinea capitis, showing black dots from broken hairs in area of hair loss. (Reprinted from Shy R. Tinea corporis and capitis. *Pediatr Rev.* 2007;28:169. © American Academy of Pediatrics.)

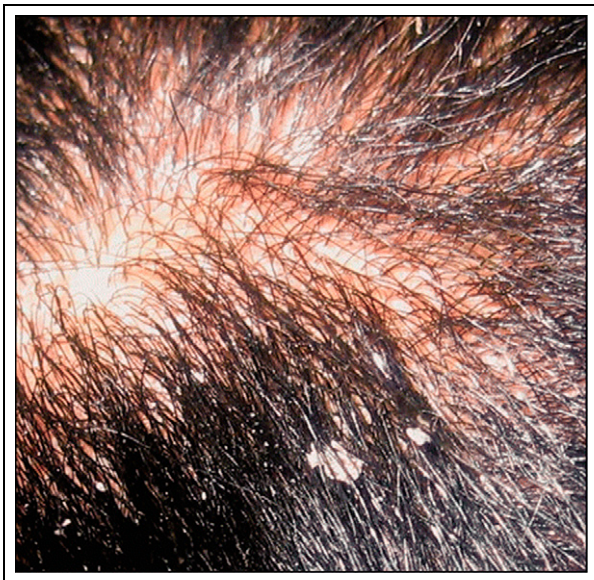


Figure 3. Tinea capitis, showing diffuse flaking and scale that resembles seborrhea. (Reprinted from Shy R. Tinea corporis and capitis. *Pediatr Rev.* 2007;28:169. © American Academy of Pediatrics.)



Figure 5. Tinea capitis that was resistant to high-dose griseofulvin after 6 weeks of treatment. Some of the smaller lesions are new. Culture grew *T tonsurans*. (Reprinted from Shy R. Tinea corporis and capitis. *Pediatr Rev.* 2007;28:170. © American Academy of Pediatrics.)

Table 3. Frequency of Symptoms and Signs in Patients With Culture-Proved Tinea Capitis

Symptom/Sign	Culture-positive*	Culture-positive†
Scaling	97%	91%
Alopecia	90%	75%
Pruritus	81%	71%
Occipital/posterior auricular lymphadenopathy	90%	0%
Occipital lymphadenopathy	0%	27%
Kerion	0%	18%

* Hubbard 1999 (6)
† Lorch Dauk et al 2010 (2)

Lorch Dauk et al (2) also found that of the symptoms and signs they studied, occipital adenopathy was the most predictive of culture-proved tinea capitis (positive predictive value = 84%). This study included children with kerion, who are known to have a high false-negative rate for culture-proved disease. Both Hubbard (6) and Lorch Dauk et al (2) concluded that prescribing empiric therapy for tinea capitis was a reasonable approach in an urban setting when presented with a child demonstrating scaling, alopecia, or pruritus associated with adenopathy (occipital or posterior auricular).

In a 2005 study of 300 nonselected and largely asymptomatic children, Williams et al (7) found that both lymphadenopathy and scalp scaling are common (53% and 22%, respectively) in the pediatric population. Of the 38 children with both scalp scaling and head or neck lymphadenopathy, only four (11%) had culture-proved dermatophyte infection. Seborrheic dermatitis and atopic dermatitis were the most frequent diagnoses associated with scalp scaling and head/neck lymphadenopathy in this study. The authors conclude that scaling of the scalp and head/neck lymphadenopathy are not specific for tinea capitis in the general (and mostly asymptomatic) pediatric population. However, how this information should be applied to the child who presents to the physician with scalp complaints associated with scaling and significant posterior lymphadenopathy is unclear.

Many authors argue that in all cases of suspected tinea capitis, the presence of a dermatophyte should be confirmed by microscopy or culture before starting a prolonged course of systemic therapy. (8)(9)(10) A sample for a KOH preparation can be obtained from a cooperative child using a blunt scalpel or edge of a glass slide. Wiping the area with an alcohol swab may help the material adhere to the scalpel or slide. Alternatively, a toothbrush can be used to collect scale and involved hair.

In the United States, one would expect to find spores (arthroconidia) within the hair shaft typical of *T tonsurans*

infection. Microscopy is the quickest and most inexpensive way to confirm the presence of a dermatophyte. However, KOH preps have a high false-negative rate when performed by inexperienced examiners. As mentioned, culturing the scalp can be done with a moist cotton swab. Culture is more sensitive than a KOH prep and less time intensive for the pediatrician, and it does not require the use of an office microscope. However, false-negative cultures do occur, particularly in the setting of a kerion. The culture may take several weeks to become positive and can be costly (\$60–\$100). (2)

There appears to be no clear standard of care in the United States regarding the diagnosis of tinea capitis. A young child presenting with pruritus, scale, or alopecia of the scalp associated with occipital or posterior auricular adenopathy can be treated reasonably with empiric therapy for tinea capitis. With no adenopathy, a dermatophyte infection is less likely and a confirmatory test is prudent before starting therapy. Similarly, when faced with an empiric treatment failure, a fungal culture should be obtained to identify the presence of a dermatophyte and the specific species.

Treatment of Tinea Capitis

Tinea capitis requires systemic antifungal therapy for effective treatment because the dermatophyte is found at the hair follicle root and is not accessible by topical treatments. However, adjunctive topical therapy can decrease transmissibility and improve the mycologic cure rate; see table 4 for dosing of systemic and adjunctive topical therapies. For more than 50 years, griseofulvin has been the treatment of choice for tinea capitis worldwide. This drug has proved to be safe, effective, and relatively well tolerated by children. The availability of griseofulvin has been a major factor in the dramatic reduction of zoophilic dermatophytosis (*Microsporum* sp) found in North America. However, its effectiveness against anthropophilic organisms, in particular *T tonsurans*, has waned in recent years.

This resistance and the need for a prolonged course of therapy have prompted significant interest in alternative, shorter-course treatment options for tinea capitis. Currently, two systemic antifungal agents have been approved by the Food and Drug Administration (FDA) for the treatment of tinea capitis in children: griseofulvin and terbinafine. In addition, there is considerable evidence that itraconazole and fluconazole are effective and safe therapeutic options for children with tinea capitis.

Griseofulvin

Griseofulvin was first isolated from the mold *Penicillium griseofulvum* in 1939. The first successful treatment of a superficial fungal infection by griseofulvin in humans was reported in 1958. The drug is active against all common dermatophytes, including *Trichophyton*, *Microsporum*, and *Epidermophyton* spp. Griseofulvin has no effect on bacteria, *Candida*, *M. furfur*, *Aspergillus*, and other fungi.

Griseofulvin is fungicidal in vitro, but its clinical effect in children and adults with dermatophytosis is fungistatic. This finding is explained by the fact that griseofulvin cannot diffuse into the stratum corneum or hair follicle. Rather, it is absorbed into the keratin precursor cells and prevents the growth of dermatophytes as these cells migrate toward the surface of the skin. Thus, the infection cannot be eradicated until the dermatophyte-infected keratin layers have grown out.

Griseofulvin is a mitotic inhibitor and interferes with nucleic acid, protein, and cell wall synthesis of replicating dermatophyte cells. There is evidence also that griseofulvin has an anti-inflammatory effect, (11) which is unique among the systemic antifungal agents. Dermatophyte resistance to griseofulvin has been demonstrated, but its frequency and clinical significance are unclear.

Recent treatment failures and concerns about increasing resistance (especially in *T tonsurans* infection) have lead most experts to recommend higher doses of griseofulvin (20 to 25 mg/kg/d vs 11 mg/kg/d) and longer treatment courses (6 to 12 weeks vs 4 to 6 weeks) than in the past. In general, *Microsporum* infections require a longer treatment course for clinical and mycologic cure than do *Trichophyton* infections. This difference in course applies to the use of all systemic antifungal agents. Many authors recommend that treatment be continued until mycologic cure is proved. This approach is an expensive and inefficient approach. A more practical approach is to continue griseofulvin therapy for 2 weeks after the symptoms and signs of tinea capitis have resolved. (12) This strategy is endorsed by the American Academy of Pediatrics (AAP).

Griseofulvin is not soluble in water and is poorly and variably absorbed from the gastrointestinal tract. Taking

the medication with a high-fat meal (eg, whole milk, peanut butter) can significantly improve absorption and double peak serum levels. (11) Absorption is also enhanced by microsizing the formulation, creating microcrystalline griseofulvin, which is available in a suspension for accurate dosing in children. An ultracrystalline formulation increases absorption further and therefore requires a smaller total dose (10 to 15 mg/kg/d). However, this formulation is not available as a suspension, limiting its use in children. All formulations of griseofulvin are given as a once-daily dose. When prescribed at the recommended higher doses and longer courses, griseofulvin continues to be effective for tinea capitis. A recent extensive review quoted mycologic cure rates of 80% to 95% and effective therapy rates of 88% to 100% for griseofulvin. (12) The effective cure rate for griseofulvin, defined as a "negative culture \pm negative KOH preparation and few remaining visual signs of infection," was found to be 73.4% in a recent meta-analysis of seven studies. (12) Importantly, in this study, griseofulvin had a higher effective cure rate when used to treat *Microsporum* (88.1%) versus *Trichophyton* (67.9%) infections.

Griseofulvin is a safe medication even when prescribed for extended periods of time. Adverse effects generally are mild but not uncommon and include nausea, headache (up to 15%), urticaria, and other rashes. Typically, adverse effects are not severe enough to require discontinuation of the therapy. Life-threatening effects are extremely rare but include Stevens-Johnson syndrome, toxic epidermal necrolysis, and fatal hepatotoxicity. Griseofulvin can induce photosensitivity and photodermatitis.

Griseofulvin is contraindicated in pregnancy and in patients who have hepatocellular failure or porphyria. Potential for cross-sensitivity exists in patients who have demonstrated hypersensitivity to penicillin. Griseofulvin may decrease the effectiveness of oral contraceptives. In addition, the manufacturer recommends that women wait at least 1 month after griseofulvin therapy before becoming pregnant. Men should wait 6 months after completing therapy before fathering a child.

Baseline laboratory tests are unnecessary in children or adults who do not have a history of or evidence of liver disease. The manufacturer recommends "periodic hematologic, hepatic, and renal function tests ... in patients on long-term therapy." If griseofulvin treatment is required for longer than 8 weeks, laboratory testing with a complete blood count and levels of liver transaminases, bilirubin, blood urea nitrogen, and creatinine is recommended. Therapy should be discontinued if evidence of drug toxicity is found. Griseofulvin is FDA-approved for the treatment of dermatophytoses in children (2 years and older) and adults.

Terbinafine

In 2007, terbinafine became the second FDA-approved drug to treat tinea capitis in children. The drug is an allylamine whose antifungal effect is due to inhibition of squalene epoxidase. This effect leads to decreased synthesis of ergosterol in fungal cell membranes (fungistatic effect) and accumulation of squalene (fungicidal effect). Terbinafine is fungicidal against both *Trichophyton* and *Microsporum* spp in vitro. It is highly lipophilic and accumulates in both fat and skin. Terbinafine remains in the stratum corneum above inhibitory levels for 2 months after discontinuation of the drug. The drug is metabolized in the liver and excreted largely in the urine. It can be given once daily.

There is good evidence that terbinafine is at least as effective as griseofulvin for tinea capitis caused by *T tonsurans*. A recent large multicenter international trial showed better clinical (63% vs 55%) and mycologic (62% vs 47%) cure rates with terbinafine vs griseofulvin in children in the United States. (13) However, the dose of griseofulvin used in these studies was less than standard dose used today in the United States. By contrast, effective cure rates are higher for griseofulvin vs terbinafine in children with tinea capitis caused by *Microsporum* spp. Thus, griseofulvin is considered the first-line systemic therapy for tinea capitis caused by *Microsporum* spp.

Terbinafine, overall, appears to be a very safe drug. A history of allergic reaction to terbinafine is the only contraindication. It is not recommended in patients with chronic or active liver disease. Pretreatment testing of liver transaminases is recommended for all patients. Common adverse effects are nasopharyngitis (10%), headache (7% to 12%), vomiting (5%), and rash (6%). Rare but serious effects include liver failure (1 in 120,000), severe and prolonged disturbance of taste or smell, depression, Stevens-Johnson syndrome, exacerbation of systemic lupus erythematosus, and decreased absolute lymphocyte count. Terbinafine can increase the serum levels of tricyclic antidepressants, selective serotonin reuptake inhibitors, and β -blockers.

Terbinafine is approved for the treatment of tinea capitis in children 4 years and older. It is dispensed in individual packets containing 125 mg or 187.5 mg oral granules. The parent should sprinkle the entire content of the packet on a spoonful of nonacidic soft food such as pudding, mashed potatoes, or ice cream. Applesauce or fruit-based foods should not be used. The child should swallow the spoonful without chewing. Recommended length of treatment is 6 weeks. Longer courses may be required with *Microsporum* infections. A complete blood count with differential should be checked during treatment courses longer than 6 weeks.

Fluconazole

Fluconazole is a broad-spectrum triazole fungistatic agent that inhibits the production of ergosterol in fungi. The drug is not approved by the FDA for use in patients with dermatophytosis. It is, however, approved for use in children with systemic fungal infection (eg, *Candida* sp and *Cryptococcus*).

A number of small studies have demonstrated that fluconazole is effective in the treatment of tinea capitis. Gupta et al found that 2 to 3 weeks of daily fluconazole (6 mg/kg) in children resulted in high clinical and mycologic cure rates at a 12 week follow-up visit (90% and 86% respectively). (8) Similarly, Gupta and his colleagues found that once weekly fluconazole (8mg/kg) administered for 8 to 16 weeks produced complete cure (mycologic and clinical) in 98% of children with tinea capitis. In the subgroup with *T tonsurans* infection, all were cured at 8 weeks. (14)

A study conducted in Iran found that children with tinea capitis caused by *Trichophyton* or *Microsporum* infection had identical cure rates when treated with daily fluconazole (5 mg/kg) for 4 weeks versus griseofulvin (15 mg/kg/d) for 6 weeks. At 8 weeks, cure rates were 76% and 78%, respectively. (15) A large multicenter randomized controlled trial found that treatment of tinea capitis with 6 weeks of fluconazole 6 mg/kg/d or low-dose griseofulvin (11 mg/kg/d) produced comparable, but low, clinical and mycologic cure rates (successful treatment 54% vs 57% at week 10). (16)

Fluconazole, in contrast to other azole antifungals, is very water soluble and, therefore, has excellent bioavailability when taken orally. Fluconazole use is contraindicated in combination with astemizole and terfenadine, both of which have been removed from the US market due to severe adverse effects. The drug is not recommended in patients with liver disease or renal dysfunction or in combination with erythromycin. The FDA considers fluconazole a pregnancy category D medication except for single low-dose therapy for vaginal candidiasis (category C). Adverse effects are less common with fluconazole than with the other systemic antifungals: nausea (4%), vomiting (2%), abdominal pain (2%), diarrhea (2%), headache (2%), and rash (2%). Rare but serious reactions include hepatic failure, anaphylaxis, torsade de pointes, QT prolongation, seizures, exfoliative dermatitis, and agranulocytosis.

The major drawback of fluconazole is its potential for drug interactions with many different medications. Commonly used pediatric medications that may interact with fluconazole are alprazolam, amitriptyline, chloral hydrate, cimetidine, citalopram, clarithromycin, and ergotamine. Fluconazole is available in both tablets and oral solution. Pretreatment testing of complete blood

count, renal function, and liver function is suggested by some authors.

Itraconazole

Itraconazole, like fluconazole, is a triazole and exerts its fungistatic effect by inducing ergosterol deficiency. The drug has an extremely broad spectrum of activity against fungi, including *Aspergillus* and dermatophytes. It is not FDA approved for any indication in children. However, a few studies have shown that itraconazole is as efficacious as terbinafine and griseofulvin in children with tinea capitis. It is contraindicated in patients with congestive heart failure. As with fluconazole, there are many drug-drug interactions with itraconazole. Pretreatment liver function tests should be ordered for all patients. Mild adverse effects are similar to, but more frequent than, adverse effects reported with fluconazole. Rare but serious and life-threatening reactions can occur. Itraconazole is available as capsules or oral solution.

Ketoconazole

Ketoconazole is an imidazole having mainly fungistatic activity against *Trichophyton* sp. The drug is ineffective against *Microsporum*. It has been studied in children with tinea capitis and its effectiveness is comparable to griseofulvin in children with infections caused by *T tonsurans*. However, the risk of hepatotoxicity is unacceptably high with long-term use in children for this indication, given the availability of less toxic and more efficacious alternative medications. Systemic ketoconazole should not be used to treat children with tinea capitis. However, ketoconazole shampoo is a useful and safe adjunct treatment for children with tinea capitis.

Infection Control and Adjunctive Therapy

Topical agents alone are not adequate for the treatment of tinea capitis. However, sporicidal shampoos have been found to reduce the carriage of viable spores and may reduce the risk of transmission in the early stages of systemic therapy. (9) Topical therapy also may shorten and improve the mycologic cure rate in conjunction with oral agents. Selenium sulfide (1% and 2.5%) and ketoconazole (2%) are the best studied shampoos for this indication. There is no proven advantage of the prescription strength (2.5%) selenium sulfide over the 1% formulation, which is available over-the-counter.

A recent study of children in the United States found ciclopirox shampoo 1% to be as effective as selenium sulfide when used to treat tinea capitis along with oral griseofulvin. Other experts have recommended zinc pyrithione (1% and 2%) and povidone-iodine (2.5%) shampoo. Sporicidal shampoos may aid also in the physical removal of adherent

infected scales from the scalp. All shampoos should be applied for 5 minutes 2 or 3 times weekly for 2 to 4 weeks.

Virtually all experts and practice guidelines recommend allowing children with tinea capitis to return to school once they have started appropriate systemic and topical treatment. However, children should not be allowed to share headgear (eg, baseball helmets) or participate in sports with scalp-to-scalp contact (eg, wrestling) until they are cured. Personal items (eg, combs, brushes, scissors, hats) should be disinfected with bleach. Bedding (pillow cases, sheets) should be washed in hot water with or without bleach. In the rare child in the United States who had *M canis* infection, the family pet (cat, dog, guinea pig) is the likely source of the infection and should be evaluated by a veterinarian. Anthropophilic species such as *T tonsurans* are more contagious than zoophilic species. A US study found that over 30% of households that had a child with tinea capitis had at least one asymptomatic carrier of the dermatophyte. Because of this, the AAP recommends that household contacts should be examined for evidence of tinea capitis. (17) The British Association of Dermatologists recommends that close contacts of patients with *T tonsurans* should be screened for evidence of tinea capitis and tinea corporis and “appropriate mycologic samples taken ... even in the absence of clinical signs.”(9) This is a time-intensive and very costly strategy of unproven effectiveness in controlling infection. A reasonable approach endorsed by many experts is to treat all close personal contacts of a child with tinea capitis with a sporicidal shampoo for several weeks.

Complications

Kerion celsi (kerion) is a highly inflammatory form of tinea capitis that can lead to scarring and permanent hair loss if not treated aggressively (Fig 6). Kerion often is confused with pyoderma because it is tender, erythematous, and associated with pus. However, it is rare for bacterial infections to cause alopecia. Hairs plucked from a kerion are painless; this is not true with pyoderma. Culturing the kerion for bacteria is unnecessary, as are systemic antibacterial medications.

The treatment of choice for a kerion remains griseofulvin, regardless of the dermatophyte species causing the infection. Griseofulvin has anti-inflammatory properties and this property may help the kerion to heal sooner than when treated with other systemic antifungal agents. Expert opinion on the use of systemic corticosteroids for kerion is mixed. Despite the lack of compelling evidence, many advocate for the use of prednisone (1 to 2 mg/kg/d) for 1 or 2 weeks in addition to griseofulvin and adjuvant topical therapy in patients with kerion.



Figure 6. Kerion with boggy lesion, multiple pustules, and hair loss. (Reprinted from Shy R. Tinea corporis and capitis. *Pediatr Rev.* 2007;28:171. © American Academy of Pediatrics.)

Tinea capitis and, rarely, tinea corporis may be associated with a papular hypersensitivity dermatitis (dermatophytid reaction, or “id” reaction). These papules or vesicles, having varying degrees of inflammation, may be pruritic. The lesions are seen on the face, trunk, and extremities of children with tinea capitis at the time of the diagnosis or, more commonly, after initiation of systemic therapy. The lesions characteristically will be concentrated along the hairline and may be found in an atopic dermatitis–like distribution. This rash is a manifestation of an immune response and is not a drug reaction. Oral antifungal treatment should be continued and topical corticosteroids or systemic antihistamines are used as needed.

Tinea Corporis

Dermatophyte infection of glabrous (nonhairy) skin of the face, trunk, and extremities is known as tinea corporis ringworm). The hyperkeratized skin of hands (tinea manuum) and feet (tinea pedis), despite involving nonhairy areas, have different clinical presentations, epidemiology, and treatment recommendations from tinea corporis infections. *T rubrum* (anthropophilic) is the most common cause of dermatophytosis worldwide and the most common cause of tinea corporis in North America. *T mentagrophytes*, *Epidermophyton floccosum* (both anthropophilic species), and the zoophilic species *M canis* are other causes of tinea corporis in the United States and Mexico. Geophilic species (eg, *M gypseum*) are extremely rare causes of tinea corporis in North America (<1%).

Anthropophilic infections are transmitted person-to-person, and children often become infected by spores shed by a household contact (typically an adult with tinea

pedis or onychomycosis). Skin-to-skin contact among young children and older children participating in sports (tinea corporis gladiatorum) are other common sources of this infection. In the United States, tinea corporis caused by *M canis* is transmitted to children by domestic animals (kittens and puppies).

In its classic form, tinea corporis is diagnosed easily by history and physical examination alone. The classic presentation is the subacute onset of a single (or few) well-demarcated annular erythematous pruritic plaque(s) with a raised border and central clearing (Fig 7). The border can be papular, vesicular, or pustular. Children with this presentation can be managed appropriately by empiric therapy with a topical antifungal medication. However, the presentation in clinical practice can be variable.

A study of the clinical signs and symptoms associated with tinea corporis found that scaling, erythema, pruritus, central clearing, and concentric rings, when measured independently, were neither sensitive nor specific for dermatophytosis. (18) Furthermore, when multiple plaques are present, they coalesce often into unusual, polycyclic patterns. Underlying immunodeficiency can lead to extensive and severe presentations of tinea corporis that are difficult to treat with topical therapy alone.

Finally, prescription and over-the-counter topical therapy prior to presentation to the physician can alter the appearance of tinea infections dramatically (this result is known as “tinea incognita” and is most often found when there has been topical corticosteroid use). Recently, tinea incognita has been described with the use of topical calcineurin inhibitors (pimecrolimus and



Figure 7. Tinea corporis on body and tinea facie. Note the erythematous border and scaling. (Reprinted from Shy R. Tinea corporis and capitis. *Pediatr Rev.* 2007;28:166. © American Academy of Pediatrics.)

tacrolimus). Both classes of medication are used to treat children with atopic dermatitis, and parents may use them inadvertently to treat tinea corporis. For all these reasons, the pediatrician should maintain a low threshold for testing annular scaly skin lesions with a KOH preparation for microscopy. It is interesting to note that, despite the lack of inflammation with tinea incognito, the yield on finding hyphae with microscopy is extremely high.

Many other skin conditions can mimic tinea corporis (Table 5). The “herald patch” of pityriasis rosea can appear identical to the classic description of tinea corporis (Fig 8). The herald patch occurs most commonly on the trunk, proximal extremities, or neck and rarely is itchy. Within 1 to 3 weeks, a generalized eruption occurs with smaller papules and plaques (both surrounded by a “collarlet of scale”) on the trunk and proximal extremities. The ovoid lesions follow the lines of skin cleavage, producing a “Christmas tree” pattern on the back. Pityriasis rosea is common in school-age children, adolescents, and young adults. A skin surface scraping and KOH preparation for microscopy can reliably distinguish the herald patch from tinea corporis.

Granuloma annulare is another relatively common skin disorder that can present very similarly to tinea corporis and occurs in both adults and children (Fig 9). The classic presentation is the subacute onset of an annular, nonscaly, nonpruritic, smooth, flesh-colored plaque on the dorsum of the hands or feet (although it can occur anywhere on the body). Approximately 85% of patients present with a single lesion. Disseminated granuloma annulare (more than 10 lesions) occurs in 15% of patients and usually in patients younger than 10 or older than 40 years. The lesions slowly enlarge peripherally and often develop “central clearing” and a raised, mildly erythematous border similar to tinea corporis.

The key to distinguishing granuloma annulare from tinea corporis is that there are no epidermal changes with a granuloma annulare lesion. In fact, histologically the epidermis is normal in this disorder. The inflammatory process is in the dermis. There is no scale, vesicles, or pustules with granuloma annulare. A skin scraping will reveal no hyphae with a KOH-prepared slide under microscopy. The cause is unknown and treatment generally is unnecessary because granuloma annulare is a self-limited disorder.

Tinea faciei is a variant of tinea corporis that occurs on the nonhairy areas of the face. The clinical appearance can be subtle, and treatment may require a longer course than tinea corporis. Tinea cruris usually occurs in adolescent males and young men. The eruption usually is bilateral, spares the scrotum and penis, and commonly is pruritic. Tinea cruris can be confused with *Candida* intertrigo,

whose characteristics include scrotal involvement and satellite lesions, as well as erythrasma. KOH preparation for microscopy and a Wood lamp examination can help distinguish among these entities. Treatment of tinea cruris is similar to that for tinea corporis.

Tinea pedis is most common in adolescents and young adults but can occur in children. There are several clinical variants of tinea pedis. The intertriginous type presents with inflammation and maceration of the lateral toe webs. Vesicular tinea pedis shows inflammation, vesicles, or bullae. Moccasin-type tinea pedis presents with diffuse plantar hyperkeratosis, erythema, scaling, and fissuring. This variant can be confused easily with psoriasis or juvenile plantar dermatosis. Rarely, erythrasma or secondary syphilis can have a similar appearance. KOH preparation for microscopy confirms the diagnosis in all variants. Moccasin-type tinea pedis can be very resistant to topical therapy; systemic therapy (similar to that for tinea capitis) may be required.

Tinea manuum is uncommon in childhood, usually occurs in association with tinea pedis, and is unilateral (“two-foot, one-hand syndrome”). Hyperkeratosis of the palm with scale is typical and treatment is the same as for tinea pedis.

Topical antifungal agents are the treatment of choice for tinea corporis, tinea faciei, tinea manuum, tinea pedis, and tinea cruris. There are many available antifungal medications used to treat dermatophytoses. The cream should be applied to the affected area and to at least 2 cm of surrounding normal skin. Treatment should be continued for at least 1 week after clinical cure. Treatment of tinea pedis and manuum generally requires a longer treatment course than tinea corporis or cruris.

For decades, the mainstay of topical therapy for dermatophytoses has been the imidazoles. This is a group of fungistatic, safe, effective, and (mostly) inexpensive topical drugs that can be curative for most dermatophytoses in 2 to 6 weeks. They have broad antifungal activity against dermatophytes, *Malassezia*, *Candida*, and Gram-positive bacteria. A few agents in this class are formulated for once-daily application (Table 6).

The allylamines and benzylamines are newer, fungicidal antifungal agents that have excellent activity against dermatophytes and molds and moderate activity against *Candida*. Naftifine and terbinafine appear to have additional intrinsic anti-inflammatory activity. These agents and butenafine can be applied topically once daily with higher cure rates and reduced treatment courses, compared to the azoles. It should be noted that nystatin is ineffective against dermatophytes. Topical antifungal agents in combination with corticosteroids are not recommended because these preparations are less effective and have higher risk of side effects.

Table 4. Systemic and Adjunctive Topical Treatment for Tinea Capitis

Medication	Formulation	Dosage	Duration	Monitoring	Other
Griseofulvin	Microsized suspension: 125 mg/5 mL	20–25 mg/kg/d (max 1 g)	6–8 wk (may need up to 16 wk)	No baseline laboratory studies	Take with fatty food; bitter tasting
	Tablets: 250 mg Ultramicrosized tablets: 125, 250 mg	10–15 mg/kg/d (max 750 mg)	Once or twice daily	Complete blood count, levels of liver transaminases, bilirubin, blood urea nitrogen, creatinine every 8 wk	Headache, nausea, rash are common; \$4.50/d (for a 20-kg child)
Terbinafine	Oral granules : 125 mg and 187.5 mg per packet	<25 kg = 125 mg 25–35 kg = 187.5 mg >35 kg = 250 mg	Once daily for 6 wk	Liver transaminase measurement is advised for all patients prior to starting therapy	Entire packet is sprinkled on a spoonful of nonacidic food (eg, pudding or mashed potatoes) and swallowed; \$14/d (for a 20-kg child)
Fluconazole	Tablets: 50, 100, 150, 200 mg Suspension: 10 mg/mL and 40 mg/mL in 35 mL bottle	6 mg/kg/d or 6–8 mg/kg/wk	Once daily for 3 wk or once weekly for 8–12 wk	Baseline complete blood count, liver function test, renal function tests to be considered	Pulse therapy (once weekly) is attractive alternative in children when medical adherence a problem. Not FDA-approved for tinea capitis; many drug interactions
Itraconazole	Capsules: 100 mg Suspension: 10 mg/mL	5 mg/kg/d 3 mg/kg/d	Once daily for 2–4 wk	Measurement of liver transaminase levels is advised for all patients prior to starting therapy	Not FDA-approved for children; not FDA-approved for tinea capitis; many drug interactions
Ketoconazole	2% Shampoo	Topically to scalp for 5 min, then rinse	2 or 3 times weekly for 2–4 wk	None	Decreases viable spores on patients and asymptomatic carriers; all patients and household contacts should be treated
Selenium sulfide	1% Shampoo				
Povidone-iodine	2.5% Shampoo				
Ciclopirox	1% Shampoo				

Table 5. Disorders That Mimic Tinea Corporis

Disorder	Classic Presentation	Distinguishing Characteristics (From Tinea Corporis)
Pityriasis rosea	Adolescent with single annular nonpruritic plaque on neck, trunk, proximal extremities. Paposquamous eruption follows in 1–3 wk on trunk and proximal extremities.	“Herald patch” is rarely itchy. Papules and plaques have a “collarlet of scale.” Exanthem follows the lines of cleavage and produces a “Christmas tree” pattern on back. Potassium hydroxide (KOH)–prep is negative for fungi.
Granuloma annulare	Child with one or more annular flesh-colored, pink or reddish nonpruritic, smooth, nonscaly plaque(s) on dorsum of hand or foot.	No surface changes (ie, no scale, vesicles, or pustules). Mild erythema can be present. KOH–prep is negative for fungi and skin biopsy shows dermal inflammation.
Fixed drug eruption	Localized, well-demarcated, dusky erythematous round or oval plaque with or without bullae. Triggered by sulfa drugs, acetaminophen, ibuprofen, and some antibiotics.	History of prescription or over-the-counter medication use. Recurs in same location with repeat exposure to drug. Starts as single lesion. With recurrence may develop new lesions and increased size. Residual hyperpigmentation is common. KOH–prep is negative for fungi.
Psoriasis	Lesions begin as small, red papules with fine scale that coalesce to form annular, erythematous, well-demarcated plaques with gray or silver scale. Can be solitary or many plaques. Center of plaque may involute and produce central clearing, mimicking tinea corporis.	Psoriasis is suggested by the Auspitz sign (punctuate bleeding with removal of scale), Koebner phenomenon (local flare of psoriasis in response to trauma such as a scrape), geographic tongue, nail pitting. 70% of children who have psoriasis have a family history of psoriasis. Psoriatic arthritis and uveitis are rare in children. KOH–prep is negative for fungi.
Nummular eczema	Discrete, annular, erythematous, pruritic plaque(s) with or without lichenification and hyperpigmentation (Fig 10). Extensor surfaces of extremities are most common sites.	No central clearing. No “active” border. Very itchy. Associated xerosis. KOH–prep is negative for fungi. Do not treat with high-potency corticosteroid until tinea corporis ruled out.
Erythema migrans	Flat, erythematous, rapidly expanding (days), asymptomatic patch in patient who lives in or recently visited a Lyme-endemic area. Classic “bull’s eye” rash. History of tick bite is not typical.	Usually not palpable. No scale. No pruritus. Only with exposure to Lyme-endemic regions (Mid-Atlantic, Northeast, Great Lakes). KOH–prep is negative for fungi. Known <i>Ixodes</i> tick bite greatly increases likelihood of erythema migrans rash. Lyme titers NOT helpful.
Erythema marginatum	Annular, rapidly expanding (hours), evanescent, pink, nonpruritic rash with serpiginous, slightly raised border. Most often on trunk, especially abdomen.	Rapid expansion and evanescent nature of this rash are incompatible with tinea corporis. No scale. Characteristic of acute rheumatic fever in children (<10%). Uncommon in adults. Also associated with hereditary angioedema.
Discoid lupus erythematosus	Well-demarcated, indurated, red-to-purplish plaques with adherent scale and fine telangiectasia. Most common on the head or face of postpubertal girls. Often associated with scarring and requires consultation with a dermatologist.	Serology (antinuclear antibody) may be normal and systemic symptoms of systemic lupus erythematosus may be absent. Purple lesions and telangiectasias not consistent with tinea corporis or tinea faciei. KOH–prep negative for fungi.



Figure 8. Herald patch of pityriasis rosea on neck. (Reprinted from Shy R. *Tinea corporis and capitis. Pediatr Rev.* 2007;28:168. © American Academy of Pediatrics.)



Figure 10. Nummular eczema, showing typical crusting without central clearing. (Reprinted from Shy R. *Tinea corporis and capitis. Pediatr Rev.* 2007;28:167. © American Academy of Pediatrics.)



Figure 9. Granuloma annulare. Note the lack of scaling. (Reprinted from Shy R. *Tinea corporis and capitis. Pediatr Rev.* 2007;28:167. © American Academy of Pediatrics.)

Pityriasis Versicolor

Pityriasis versicolor (formerly *tinea versicolor*) is a common superficial fungal infection caused by the yeast form of *M. furfur*, a dimorphic fungus that is part of the normal cutaneous flora. The condition is characterized by multiple, oval, hypopigmented or hyperpigmented, thin plaques with scale and macules distributed over the trunk, proximal arms, and, less commonly, the neck and face. This rash is most common in adolescents and young adults, but it does occur in prepubertal children, in whom facial involvement is common.

Patients often present in the summer when the lesions fail to tan and the condition becomes more apparent. The

prevalence of this disorder is as high as 40% (in adults) in tropical climates. Temperate regions have a significantly lower prevalence (1% to 4%) in adults. Pityriasis versicolor usually is asymptomatic; however, mild pruritus sometimes is noted.

Pityriasis versicolor can be confused with postinflammatory hypopigmentation, vitiligo, pityriasis alba, tinea corporis, seborrheic dermatitis, or pityriasis rosea. Vitiligo presents with completely depigmented macules or patches without scale. A Wood lamp can help distinguish these two conditions because pityriasis versicolor will fluoresce pale yellow and vitiligo will fluoresce a bright white. Pityriasis alba occurs in atopic patients, presents with hypopigmented macules and patches without scale, and is usually limited to the face. In clinical practice, pityriasis versicolor usually is diagnosed by clinical features alone. However, a KOH preparation for microscopy is confirmatory and reveals short hyphae and grape-like clusters of spores (“spaghetti and meatball” appearance).

There are several effective topical and systemic therapies for pityriasis versicolor. However, the pigmentary changes may take months or years to resolve completely, and recurrences are common (as high as 60% in the first year after treatment). A detailed systematic review of interventions for pityriasis versicolor was published in 2010. (19) The authors concluded that most topical treatments are effective and well tolerated and that, overall, longer duration of treatment and higher concentrations of the topical antifungal agents increased cure rates.

Selenium sulfide 2.5% shampoo or lotion, applied daily for 10 minutes prior to rinsing, for a 1- to 2-week course is effective and inexpensive. However, this agent is more likely than other topical agents to cause drying of the skin

Table 6. Topical Antifungal Therapy for Dermatophytoses and Pityriasis Versicolor

Class	Drug	Over-the-Counter	Approval in Children by FDA	Frequency	Duration	Comments
Allylamines	Terbinafine 1%	Yes	> 11 y	Once or twice daily	2–4 wk	90% cure rate
	Naftifine 1%	No	No	Once daily	2–4 wk	Higher cure rate than azoles; expensive
Benzylamine	Butenafine 1%	Yes	> 11 y	Once or twice daily	2–4 wk	75% cure rate of tinea pedis with 1 week of twice-daily application
Imadazoles	Clotrimazole 1%	Yes	Yes	Twice daily	2–6 wk	No proven difference in efficacy among the azoles
	Econazole 1%	No	No	Twice daily	2–6 wk	
	Ketoconazole 2%	Yes	No	Once daily	2–6 wk	
	Miconazole 2%	Yes	Yes	Twice daily	2–6 wk	
	Oxiconazole 1%	No	Yes	Once daily	2–6 wk	
Other	Sulconazole 1%	No	No	Once daily	2–6 wk	
	Ciclopirox 0.77%	No	> 9 y	Twice daily	2–6 wk	1% shampoo for adjunct therapy for tinea capitis
	Tolnaftate 1%	Yes	Yes	Twice daily	4–6 wk	Less effective than imidazoles; inexpensive
	Selenium sulfide 2.5%, 1%	Yes	Yes	Twice weekly or once daily	2 wk	Adjunct therapy for tinea capitis; daily therapy for pityriasis versicolor
	Zinc pyrithione 1%	Yes	Yes	Twice weekly or once daily	2 wk	Adjunct therapy for tinea capitis; daily therapy for pityriasis versicolor

and it has a strong, unpleasant odor. Zinc pyrithione shampoo 1% applied for 5 min/d for 2 weeks produced a 100% cure rate in one small study. Ketoconazole 2% shampoo is not FDA approved for use in children but is effective when applied daily for 5 days in adults. The shampoo is applied daily to affected areas for 5 to 10 minutes before being washed off. Monthly applications may help prevent recurrences.

Single-dose ketoconazole (400 mg orally) or fluconazole (400 mg orally) has become a popular treatment because of its convenience. However, these agents are not FDA approved for this indication in children, and these high-dose regimens are less effective than longer treatment courses and are associated with a high rate of adverse effects. (19) The authors of the systematic review recommended ketoconazole (200 mg/d for 10 days), itraconazole (200 mg/d for 7 days), or fluconazole (300 mg/wk for 2 to 4 weeks) as the most effective and safe systemic antifungal regimens in adults. Drug interactions are common with these medications, and consultation with a pharmacist is recommended prior to prescribing these medications for off-label use in any patient taking medication chronically.

Summary

- Tinea capitis, tinea corporis, and pityriasis versicolor are common superficial fungal infections in the pediatric population.
- Tinea capitis is the most common dermatophyte infection worldwide. In North America, the cause is almost exclusively *T tonsurans*. Diagnosis of tinea capitis usually can be made by clinical features alone, especially when occipital or postauricular lymphadenopathy is present. Skin scrapings prepared with potassium hydroxide for microscopic examination, or a cotton swab for fungal culture, usually are diagnostic.
- Treatment of tinea capitis requires systemic antifungal therapy. Terbinafine and griseofulvin are both effective against *T tonsurans* and are FDA-approved for this indication in children.
- Adjunctive topical therapy for the patient and household contacts decreases transmission of this infection.
- Topical antifungal therapy usually is effective for tinea corporis and pityriasis versicolor. However, recurrences of pityriasis versicolor are common.

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Brendan P. Kelly
Pediatrics in Review 2012;33:e22
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