

eMedicine Specialties > Dermatology > Fungal Infections

Tinea Corporis

Jack L Leshner Jr, MD, Chief, Professor, Department of Internal Medicine, Section of Dermatology, Medical College of Georgia

Updated: Dec 2, 2009

Introduction

Background

Tinea corporis is a superficial dermatophyte infection characterized by either inflammatory or noninflammatory lesions on the glabrous skin (ie, skin regions except the scalp, groin, palms, and soles). Three anamorphic (asexual or imperfect) genera cause dermatophytoses: *Trichophyton*, *Microsporum*, and *Epidermophyton*. Dermatophytes may infect humans (anthropophilic), infect nonhuman mammals (zoophilic), or reside primarily in the soil (geophilic).

Pathophysiology

Dermatophytes preferentially inhabit the nonliving, cornified layers of the skin, hair, and nail, which is attractive for its warm, moist environment conducive to fungal proliferation. Fungi may release keratinases and other enzymes to invade deeper into the stratum corneum, although typically the depth of infection is limited to the epidermis and, at times, its appendages. They generally do not invade deeply, owing to nonspecific host defense mechanisms that can include the activation of serum inhibitory factor, complement, and polymorphonuclear leukocytes.

Following the incubation period of 1-3 weeks, dermatophytes invade peripherally in a centrifugal pattern. In response to the infection, the active border has an increased epidermal cell proliferation with resultant scaling. This creates a partial defense by way of shedding the infected skin and leaving new, healthy skin central to the advancing lesion. Elimination of dermatophytes is achieved by cell-mediated immunity.

Trichophyton rubrum is a common dermatophyte and, because of its cell wall, is resistant to eradication. This protective barrier contains mannan, which may inhibit cell-mediated immunity, hinder the proliferation of keratinocytes, and enhance the organism's resistance to the skin's natural defenses.

Frequency

International

Tinea corporis is a common infection more often seen in typically hot, humid climates. *T rubrum* is the most common infectious agent in the world and is the source of 47% of tinea corporis cases.^[1] *Trichophyton tonsurans* is the most common dermatophyte to cause tinea capitis, and people with an anthropophilic tinea capitis infection are more likely to develop associated tinea corporis. Therefore, the prevalence of tinea corporis caused by *T tonsurans* is increasing. *Microsporum canis* is the third most common causative organism and associated with 14% of tinea corporis infections. A rare case of *Microsporum fulvum* skin infection (forearm) has recently been reported, identified by ITS sequencing and mass spectrometry.^[2]

A 5-year study from Kuwait that included 2730 patients reported that fungal skin infections remain prevalent in that country, specifically the Capital area. In those patients with dermatophytes, 6 species were isolated. They included *Trichophyton mentagrophytes* (39%), *M canis* (16%), *T rubrum* (10%),

Epidermophyton floccosum (6.2%), *Trichophyton violaceum* (2.4%), and *Trichophyton verrucosum* (0.4%).¹³

Mortality/Morbidity

Dermatophyte infections do not result in significant mortality, but they can greatly affect quality of life.

Sex

Tinea corporis occurs in both men and women. Women of childbearing age are more likely to develop tinea corporis as a result of their greater frequency of contact with infected children.

Age

Tinea corporis affects persons of all age groups, but prevalence is highest in preadolescents. Tinea corporis acquired from animals is more common in children. Tinea corporis secondary to tinea capitis typically occurs in children because tinea capitis is more common in this population.

Clinical

History

Symptoms, contact history, recent travel, and international residence are relevant clues in the history of a person with tinea corporis.

- Infected patients may have variable symptoms.
 - Patients can be asymptomatic.
 - A pruritic, annular plaque is characteristic of a symptomatic infection. Patients occasionally can experience a burning sensation.
 - HIV-positive or immunocompromised patients may develop severe pruritus or pain.
- Tinea corporis may result from contact with infected humans, animals, or inanimate objects. The history may include occupational (eg, farm worker, zookeeper, laboratory worker, veterinarian), environmental (eg, gardening, contact with animals), or recreational (eg, contact sports, contact with sports facilities) exposure.
- A few clinical variants are described, with distinct presentations.
 - Majocchi granuloma, typically caused by *T rubrum*, is a fungal infection in hair, hair follicles, and, often, the surrounding dermis, with an associated granulomatous reaction. Majocchi granuloma often occurs in females who shave their legs.
 - Tinea corporis gladiatorum is a dermatophyte infection spread by skin-to-skin contact between wrestlers.^[4,5]
 - Tinea imbricata is a form of tinea corporis found mainly in Southeast Asia, the South Pacific, Central America, and South America. It is caused by *Trichophyton concentricum*.^[6]
 - Tinea incognito is tinea corporis with an altered, nonclassic presentation due to corticosteroid treatment.^[7]

Physical

- Tinea corporis can manifest in a variety of ways.
 - Typically, the lesion begins as an erythematous, scaly plaque that may rapidly worsen and

enlarge, as shown in the image below.

o



Large, erythematous, scaly plaque.

o Following central resolution, the lesion may become annular in shape, as is shown in the image below.

o



Annular plaque.

- As a result of the inflammation, scale, crust, papules, vesicles, and even bullae can develop, especially in the advancing border.^[8]
 - Rarely, tinea corporis can present as purpuric macules, called tinea corporis purpurica.^[9] One report describes 2 cases of tinea corporis purpurica resulting from self-inoculation with *Trichophyton violaceum*.^[10]
 - Infections due to zoophilic or geophilic dermatophytes may produce a more intense inflammatory response than those caused by anthropophilic microbes.
 - HIV-infected or immunocompromised patients often have atypical presentations including deep abscesses or a disseminated skin infection.
- Majocchi granuloma manifests as perifollicular, granulomatous nodules typically in a distinct location, which is the lower two thirds of the leg in females.
 - Tinea corporis gladiatorum often manifests on the head, neck, and arms, which is a distribution consistent with the areas of skin-to-skin contact in wrestling.

- Tinea imbricata is recognized clinically by its distinct scaly plaques arranged in concentric rings.

Causes

- Tinea corporis can be caused by a variety of dermatophytes, although prevalence and patient history are very helpful in identifying the most likely organism.
 - Internationally, the most common cause is *T rubrum*.
 - *T tonsurans*, *Trichophyton mentagrophytes*,^[7,11] *Trichophyton interdigitale*, *Trichophyton verrucosum*,^[12] *Microsporum canis*, and *Microsporum gypseum*^[6] are also known to produce infection.
 - Tinea imbricata is caused by *Trichophyton concentricum*.
- Dermatophytoses may be acquired from different sources, such as people, animals, or soil.
 - Infected humans are the most common source of tinea corporis in the United States.
 - Contact with contaminated household pets, farm animals, and fomites (eg hair brushes, towels) can spread infection.
 - *T verrucosum* causes 98% of dermatophyte infections in cattle and is showing increasing prevalence of infection in human contacts.
 - *T mentagrophytes* is spread by rabbits, guinea pigs, and small rodents.^[11]
 - Infection with *M gypseum*, a geophilic organism, can mimic tinea imbricata in presentation.
- Because fungal arthroconidia can survive in the environment, recurrent outbreaks may occur.

Differential Diagnoses

Atopic Dermatitis

Candidiasis, Cutaneous

Erythema Annulare Centrifugum

Erythema Multiforme

Erythrasma

Granuloma Annulare

Granuloma Faciale

Impetigo

Lupus Erythematosus, Subacute Cutaneous

Lymphocytic Skin Infiltration

Nummular Dermatitis

Parapsoriasis

Pityriasis Rosea

Psoriasis, Annular

Psoriasis, Plaque

Seborrheic Dermatitis

Syphilis

Tinea Versicolor

Workup

Laboratory Studies

- A potassium hydroxide (KOH) examination of skin scrapings may be diagnostic in tinea corporis.

- A KOH test is a microscopic preparation used to visualize fungal elements removed from the skin's stratum corneum.
 - The sample should be taken from the active border of a lesion because this region provides the highest yield of fungal elements. A KOH preparation from a vesicular lesion should be made from the roof of the vesicle.
 - The KOH helps dissolve the keratin and leaves fungal elements intact, revealing numerous septate, branching hyphae amongst epithelial cells.
 - A counterstain, such as chlorazol black E or Parker blue-black ink, may help visualize hyphae under the microscope.
- A fungal culture is often used as an adjunct to KOH for diagnosis. Fungal culture is more specific than KOH for detecting a dermatophyte infection; therefore, if the clinical suspicion is high yet the KOH result is negative, a fungal culture should be obtained.
 - A few culture mediums are available for dermatophyte growth.
 - Sabouraud agar containing neopeptone or polypeptone agar and glucose is often used for fungal culture. However, it does not contain antibiotics and may allow overgrowth of fungal and bacterial contaminants.
 - Mycosel, a commonly used agar, is similar to Sabouraud agar but has antibiotics.
 - Commonly, dermatophyte test medium (DTM) is used. It contains antibacterial (ie, gentamicin, chlortetracycline) and antifungal (ie, cycloheximide) solutions in a nutrient agar base. This combination isolates dermatophytes while suppressing other fungal and bacterial species that may contaminate the culture.
 - Following culture inoculation, potential fungal growth is monitored for 2 weeks.
 - Positive culture results vary depending on the medium used.
 - DTM contains phenol red solution, which causes a color change from straw-yellow to bright-red under alkaline conditions, indicating a positive dermatophyte culture result. However, the color makes identification of culture morphology (particularly pigmentation) difficult.
 - Sabouraud or Mycosel agar should be used to assess gross and microscopic colony characteristics.
 - If the above clinical evaluations are inconclusive, the molecular method of polymerase chain reaction for fungal DNA identification can be applied.^[13]
 - For atypical presentations of tinea corporis, further evaluation for HIV infection and/or an immunocompromised state should be considered.

Histologic Findings

A skin biopsy with a hematoxylin and eosin staining of tinea corporis demonstrates spongiosis, parakeratosis, and a superficial inflammatory infiltrate. Neutrophils may be seen in the stratum corneum, which is a significant diagnostic clue. On occasion, septate branching hyphae are seen in the stratum corneum with hematoxylin and eosin stain, but special fungal stains (eg, periodic acid-Schiff, Gomori methenamine silver) may be required.

Treatment

Medical Care

Topical therapy is recommended for a localized infection because dermatophytes rarely invade living tissues. Topical therapy should be applied to the lesion and at least 2 cm beyond this area once or twice a day for at least 2 weeks, depending on which agent is used.^[14] Topical azoles and allylamines show high rates of clinical efficacy. These agents inhibit the synthesis of ergosterol, a major fungal cell membrane sterol.

- The topical azoles (eg, econazole, ketoconazole, clotrimazole, miconazole, oxiconazole, sulconazole, sertaconazole) inhibit the enzyme lanosterol 14- α -demethylase, a cytochrome P-450–dependent enzyme that converts lanosterol to ergosterol. Inhibition of this enzyme results in unstable fungal cell membranes and causes membrane leakage. The weakened dermatophyte is unable to reproduce and is slowly killed by fungistatic action. Sertaconazole nitrate is one of the newest topical azoles. It has fungicidal and anti-inflammatory abilities and is used as a broad-spectrum agent. It may have a reservoir effect and therefore is a good choice for noncompliant patients. Lastly, Liebel et al published in vitro data in 2006, reporting this drug has anti-itch properties.^[15]
- Allylamines (eg, naftifine, terbinafine) and the related benzylamine butenafine inhibit squalene epoxidase, which converts squalene to ergosterol. Inhibition of this enzyme causes squalene, a substance toxic to fungal cells, to accumulate intracellularly and leads to rapid cell death. Allylamines bind effectively to the stratum corneum because of their lipophilic nature. They also penetrate deeply into hair follicles.^[16]
- Ciclopirox olamine is a topical fungicidal agent. It causes membrane instability by accumulating inside fungal cells and interfering with amino acid transport across the fungal cell membrane.
- A low-to-medium potency topical corticosteroid can be added to the topical antifungal regimen to relieve symptoms. The steroid can provide rapid relief from the inflammatory component of the infection, but the steroid should only be applied for the first few days of treatment. Prolonged use of steroids can lead to persistent and recurrent infections, longer duration of treatment regimens, and adverse effects of skin atrophy, striae, and telangiectasias.

Systemic therapy may be indicated for tinea corporis that includes extensive skin infection, immunosuppression, resistance to topical antifungal therapy, and comorbidities of tinea capitis or tinea unguium. Use of oral agents requires attention to potential drug interactions and monitoring for adverse effects.

- The mechanism of action of oral micronized griseofulvin against dermatophytes is disruption of the microtubule mitotic spindle formation in metaphase, causing arrest of fungal cell mitosis. A dose of 10 mg/kg/d for 4 weeks is effective. In addition, griseofulvin induces the cytochrome P-450 enzyme system and can increase the metabolism of CYP-450–dependent drugs. It is the systemic drug of choice for tinea corporis infections in children.
- Systemic azoles (eg, fluconazole, itraconazole, ketoconazole) function similar to the topical agents, causing cell membrane destruction.^[16]
 - Oral ketoconazole at 3-4 mg/kg/d may be given. However, this agent carries an associated

risk of hepatitis in less than 1 in 10,000 cases and now is seldom used orally for dermatophyte infections.

- Fluconazole at 50-100 mg/d or 150 mg once weekly for 2-4 weeks is used with good results.
 - Oral itraconazole in doses of 100 mg/d for 2 weeks shows high efficacy. With an increased dose of 200 mg/d, the treatment duration can be reduced to 1 week. However, the cytochrome P-450 activity of itraconazole allows for potential interactions with other commonly prescribed drugs.
 - Based on E-test for susceptibility of *T rubrum*, voriconazole was the most active and fluconazole was the least active of the azole drugs.^[17]
- Oral terbinafine may be used at a dosage of 250 mg/d for 2 weeks; the potential exists for cytochrome P-450, specifically CYP-2D6, drug interactions with this agent.
 - Systemic therapy is needed when the infection involves hair follicles, such as Majocchi granuloma. In this case, topical therapy may serve as adjunct treatment with the oral medication.
 - The preferred treatment for tinea imbricata is griseofulvin or terbinafine, although some resistance has developed to oral griseofulvin.^[18]

Surgical Care

Surgical treatment is usually not indicated except for drainage of superficial vesicles, bullae, pustules, or deep abscesses.

Medication

The goals of pharmacotherapy are to reduce morbidity and to prevent complications. Topical antifungal agents are effective for treating most cases of tinea corporis. Systemic therapy may be indicated for tinea corporis that is extensive, involves immunocompromised patients, or is refractory to topical therapy. For severe infections, systemic therapy can be combined with topical antifungal treatments.

Oral granules of terbinafine (Lamisil) are available in packets containing 125 mg and 187.5 mg and are for use in children with tinea capitis who are aged 4 years and older; these granules can be sprinkled once daily on pudding or mashed potatoes. While approved only for tinea capitis, these oral granules likely are used off label in children with tinea corporis when systemic therapy is needed. The suggested dosing schedule for tinea capitis is 125 mg/d for less than 25 kg body weight, 187.5 mg/d for 25-35 kg body weight, and 250 mg/d for greater than 35 kg body weight.

Topical allylamines

Naftifine 1% cream or gel (Naftin)

Broad-spectrum antifungal agent that appears to interfere with sterol biosynthesis by inhibiting the enzyme squalene 2,3-epoxidase. This inhibition results in decreased amounts of sterols, causing cell death. If no clinical improvement occurs after 4 wk, reevaluate patient.

Dosing

Adult

Cream or gel: Gently massage sufficient quantity into affected area and surrounding skin qd for 2-4 wk

Pediatric

Administer as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Discontinue use if sensitivity or chemical irritation occurs; for external use only; avoid contact with eyes

Terbinafine 1% cream (Lamisil)

Fungicidal activity; synthetic allylamine derivative that inhibits squalene epoxidase, a key enzyme in sterol biosynthesis of fungi, resulting in deficiency in ergosterol that causes fungal cell death. Use until symptoms significantly improve.

Dosing**Adult**

Apply to affected area qd for 1-4 wk

Pediatric

<12 years: Not established

>12 years: Administer as in adults

Interactions

None reported with topical use

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Discontinue use if chemical irritation develops

Topical pyridones

Ciclopirox olamine 1% cream (Loprox)

Interferes with synthesis of DNA, RNA, and protein by inhibiting transport of essential elements in fungal cells.

Dosing**Adult**

Massage into affected areas bid for 3-4 wk; reevaluate diagnosis if no improvement after 4 wk

Pediatric

<10 years: Not established

>10 years: Administer as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Avoid contact with eyes and other internal routes; discontinue if sensitivity or irritation occurs

Topical benzylamines

Butenafine 1% cream (Mentax)

Inhibits squalene epoxidation, which, in turn, causes blockage of ergosterol biosynthesis (an essential component of fungal cell membranes), causing fungal cell growth to arrest.

Dosing**Adult**

Apply qd to affected and surrounding area for 2 wk

Pediatric

<12 years: Not established

>12 years: Administer as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Use topically (not in eyes, vagina, or other internal routes)

Systemic azoles

Fluconazole (Diflucan)

Synthetic oral antifungal (broad-spectrum biazole) that selectively inhibits fungal cytochrome P-450 and sterol C-14 alpha-demethylation, which prevents conversion of lanosterol to ergosterol, thereby disrupting cellular membranes. Has little affinity for mammalian cytochromes, which is believed to explain its low toxicity. Available as tab for oral administration, as powder for oral susp, and as a sterile solution for IV use. Has fewer adverse effects and better tissue distribution than older systemic imidazoles.

Dosing**Adult**

150 mg/wk PO for 2-4 wk

Pediatric

Not established

Interactions

Levels may increase with hydrochlorothiazides; levels may decrease with long-term coadministration of rifampin; may increase concentrations of theophylline, phenytoin, tolbutamide, cyclosporine, glyburide, and glipizide; effects of anticoagulants may increase with coadministration; increases in cyclosporine concentrations may occur when administered concurrently; cisapride administration may cause torsade de pointes

Contraindications

Documented hypersensitivity; coadministration with terfenadine in patients receiving fluconazole at multiple doses of ≥ 400 mg

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Adjust dose for renal insufficiency; also associated with anaphylaxis and exfoliative skin disorders (closely monitor if rashes develop and discontinue drug if lesions progress); may cause clinical hepatitis, cholestasis, and fulminant hepatic failure (including death) when taken with underlying medical conditions

(eg, AIDS, malignancy) or while taking multiple concomitant medications; not recommended for breastfeeding mothers

Itraconazole (Sporanox)

Fungistatic activity; synthetic triazole antifungal agent that inhibits fungal cell growth by inhibiting the cytochrome P-450–dependent synthesis of ergosterol, a vital component of fungal cell membranes.

Dosing**Adult**

100 mg/d PO for 2 wk or 200 mg/d PO for 1 wk; not to exceed 400 mg/d; increase in 100-mg increments if no improvement (administer >200 mg/d in divided doses)

Pediatric

Not established

Suggested for children 3-16 years: 100 mg/d PO for 1 wk

Interactions

Antacids may reduce absorption; edema may occur with coadministration of calcium channel blockers (eg, amlodipine, nifedipine); hypoglycemia may occur with sulfonylureas; may increase tacrolimus and cyclosporine plasma concentrations when high doses are used; rhabdomyolysis may occur with coadministration of HMG-CoA reductase inhibitors (ie, lovastatin, simvastatin); coadministration with cisapride can cause cardiac rhythm abnormalities and death; may increase digoxin levels; coadministration may increase plasma levels of midazolam or triazolam; phenytoin and rifampin may reduce levels (phenytoin metabolism may be altered)

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Caution in hepatic insufficiencies (rare cases of reversible idiosyncratic hepatitis reported); monitor hepatic enzyme test values in preexisting hepatic function abnormalities

Ketoconazole (Nizoral)

Inhibits synthesis of ergosterol (main sterol of fungal cell membranes), causing cellular components to leak; results in cell death.

Dosing**Adult**

200-400 mg PO qd

Pediatric

3.3-6.6 m/kg PO qd for 4 wk, not to exceed 400 mg/dose

Interactions

Isoniazid may decrease bioavailability; coadministration decreases effects of either rifampin or ketoconazole; may increase effect of anticoagulants; may increase toxicity of corticosteroids and cyclosporine (cyclosporine dosage can be adjusted); may decrease theophylline levels; decreases metabolism of repaglinide, thus increasing serum levels and effects

Contraindications

Documented hypersensitivity; fungal meningitis

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Hepatotoxicity may occur; may reversibly decrease corticosteroid serum levels (adverse effects avoided with dose of 200-400 mg/d); administer antacids, anticholinergics, or H2-blockers at least 2 h after taking ketoconazole

Systemic allylamines

Terbinafine (Lamisil, Daskil)

Fungicidal activity; synthetic allylamine derivative that inhibits squalene epoxidase, a key enzyme in sterol biosynthesis of fungi, resulting in a deficiency in ergosterol that causes fungal cell death. Use until symptoms significantly improve.

Dosing**Adult**

250 mg/d PO for 1-2 wk

Pediatric

Terbinafine tab; treatment duration similar to that in adults

10-20 kg: 62.5 mg/d PO

20-40 kg: 125 mg/d PO

>40 kg: 250 mg/d PO

Interactions

May decrease cyclosporine effects; toxicity may increase with rifampin and cimetidine

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Discontinue use if acute generalized exanthematous pustulosis, angioedema, desquamation, erythema multiforme, erythroderma, bullous pemphigoid, lupus erythematosus, pustular psoriasis, lichenoid eruption, Stevens-Johnson syndrome, toxic epidermal necrolysis, urticaria, or alopecia erupts or if hepatobiliary dysfunction, neutropenia, or changes in ocular lens or retina develops; transient decreases in absolute lymphocyte count have been observed in controlled clinical trials

Other systemic antifungals

Griseofulvin (Fulvicin)

Fungistatic activity; fungal cell division is impaired by interfering with microtubule. Binds to keratin precursor cells. Keratin is gradually replaced by noninfected tissue, which is highly resistant to fungal invasions.

Dosing**Adult**

500 mg microsize (330-375 mg ultramicrosize) PO in single or divided daily doses

Pediatric

20 mg microsize/kg/d (5 mg/lb/d) PO or 7.3 mg ultramicrosize/kg/d (3.3 mg/lb/d) PO

Interactions

May decrease hypoprothrombinemic activity of warfarin; contraceptives may lose their effectiveness; may reduce effects of cyclosporine; may decrease serum salicylate concentrations; barbiturates may decrease serum levels

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

On prolonged therapy, observe patients closely; monitor renal, hepatic, and hematopoietic function regularly; lupuslike syndromes or exacerbation of lupus erythematosus may occur; photosensitivity may also occur (patients should take protective measures against exposure to UV light or sunlight)

Topical azoles

Clotrimazole 1% cream (Mycelex, Lotrimin)

Nonabsorbable imidazole. Broad-spectrum synthetic antifungal agent that inhibits growth of fungus by altering cell membrane permeability, which causes fungal cell death.

Therapy is directed at the underlying condition, with the goal of minimizing symptoms and preventing complications.

Dosing**Adult**

Gently massage into affected and surrounding skin areas bid for 2-6 wk

Pediatric

Apply as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Not for treatment of systemic fungal infections; avoid contact with the eyes; if irritation or sensitivity develops, discontinue use and institute appropriate therapy

Ketoconazole 2% cream (Nizoral)

Imidazole, broad-spectrum antifungal agent indicated for topical treatment of tinea corporis. Inhibits synthesis of ergosterol (main sterol of fungal cell membranes), causing cellular components to leak; result is cell death.

Dosing**Adult**

Rub gently into affected area qd or bid for 2-4 wk

Pediatric

Apply as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity; fungal meningitis

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

If sensitivity or irritation develops, discontinue use; for external use only; avoid contact with eyes

Miconazole 2% cream or lotion (Monistat)

Damages fungal cell-wall membrane by inhibiting biosynthesis of ergosterol. Membrane permeability is increased, causing nutrients to leak and resulting in fungal cell death. Lotion is preferred in intertriginous areas. If cream is used, apply sparingly to avoid maceration effects.

Dosing**Adult**

Cream and lotion: Cover affected areas bid for 2-6 wk
Powder: Spray or sprinkle liberally over affected area bid

Pediatric

Apply as in adults

Interactions

None reported for topical use

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

If sensitivity or irritation develops, discontinue use; for external use only; avoid contact with eyes

Oxiconazole 1% cream (Oxistat)

Damages fungal cell wall membrane by inhibiting biosynthesis of ergosterol. Membrane permeability is increased, causing nutrients to leak, resulting in fungal cell death.

Dosing**Adult**

Apply to affected area qid

Pediatric

Administer as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

If sensitivity or chemical irritation occurs, discontinue use; external use only; avoid contact with the eyes

Sertaconazole 2% cream (Ertaczo)

Topical imidazole antifungal active against *T rubrum*, *T mentagrophytes*, and *Epidermophyton floccosum*.

Dosing**Adult**

Apply topically bid for 2-4 wk

Pediatric

<12 years: Not established

>12 years: Administer as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

For topical use only; may cause dermatitis, dry skin, burning sensation, pruritus, hyperpigmentation, desquamation, or skin tenderness

Sulconazole 1% cream or solution (Exelderm)

Broad-spectrum antifungal agent that inhibits synthesis of ergosterol, causing cellular components to leak and resulting in fungal cell death.

Dosing**Adult**

Apply topically to affected area qd for 2-4 wk

Pediatric

<12 years: Not established

>12 years: Administer as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

If sensitivity or chemical irritation occurs, discontinue use; use only externally; avoid contact with the eyes

Follow-up**Further Outpatient Care**

- Follow-up care for tinea corporis should be determined per patient need, severity of infection, and response to treatment.

Inpatient & Outpatient Medications

- See Medication.

Deterrence/Prevention

- Imperative for preventing the spread of a dermatophyte infection is to discourage close contact

between infected and noninfected individuals and to stop the sharing of fomites (eg, towels, hats, clothing).

- Because dermatophytes flourish in moist environments, patients should be advised to wear loose-fitting clothing made of cotton or synthetic materials.

Complications

- The tinea corporis may recur if therapy does not result in complete eradication of the organism, such as when patients stop applying topical therapy too soon or if the organism is resistant to the antifungal agent used.
- Reinfection may occur if a reservoir, such as an infected nail or hair follicle, is present. Many, if not most, adult patients with tinea corporis also have tinea pedis and unguium, which should be treated.

Prognosis

- For localized tinea corporis, the prognosis is excellent, with cure rates of 70-100% after treatment with topical azoles or allylamines or short-term or pulse systemic antifungals.

Patient Education

- For excellent patient education resources, visit eMedicine's Skin, Hair, and Nails Center. Also, see eMedicine's patient education article Ringworm on Body.

Multimedia



Media file 1: Annular plaque.



Media file 2: Large, erythematous, scaly plaque.

References

1. Foster KW, Ghannoum MA, Elewski BE. Epidemiologic surveillance of cutaneous fungal infection in the United States from 1999 to 2002. *J Am Acad Dermatol*. May 2004;50(5):748-52. [\[Medline\]](#).
2. Seyfarth F, Goetze S, Erhard M, Burmester A, Elsner P, Hipler UC. [Infection with a rare geophilic dermatophyte.]. *Hautarzt*. Aug 14 2009;[\[Medline\]](#).
3. Yehia MA, El-Ammawi TS, Al-Mazidi KM, Abu El-Ela MA, Al-Ajmi HS. The Spectrum of Fungal Infections with a Special Reference to Dermatophytoses in the Capital Area of Kuwait During 2000-2005: A Retrospective Analysis. *Mycopathologia*. Nov 17 2009;[\[Medline\]](#).
4. Adams BB. Tinea corporis gladiatorum. *J Am Acad Dermatol*. Aug 2002;47(2):286-90. [\[Medline\]](#).
5. Ilkit M, Saracli M, Kurdak H, et al. Clonal outbreak of *Trichophyton tonsurans* tinea capitis gladiatorum among wrestlers in Adana, Turkey. *Med Mycol*. Oct 14 2009;[\[Medline\]](#).
6. Sun PL, Ho HT. Concentric rings: an unusual presentation of tinea corporis caused by *Microsporum gypsum*. *Mycoses*. Mar 2006;49(2):150-1. [\[Medline\]](#).

7. Sanchez-Castellanos ME, Mayorga-Rodriguez JA, Sandoval-Tress C, Hernandez-Torres M. Tinea incognito due to *Trichophyton mentagrophytes*. *Mycoses*. Jan 2007;50(1):85-7. [\[Medline\]](#).
8. Ziemer M, Seyfarth F, Elsner P, Hipler UC. Atypical manifestations of tinea corporis. *Mycoses*. 2007;50 Suppl 2:31-5. [\[Medline\]](#).
9. Kim HS, Cho BK, Oh ST. A case of tinea corporis purpurica. *Mycoses*. Jul 2007;50(4):314-6. [\[Medline\]](#).
10. Romano C, Massai L, Strangi R, Feci L, Miracco C, Fimiani M. Tinea corporis purpurica and onychomycosis caused by *Trichophyton violaceum*. *Mycoses*. Sep 22 2009;[\[Medline\]](#).
11. Shiraki Y, Hiruma M, Matsuba Y, et al. A case of tinea corporis caused by *Arthroderma benhamiae* (teleomorph of *Tinea mentagrophytes*) in a pet shop employee. *J Am Acad Dermatol*. Jul 2006;55(1):153-4. [\[Medline\]](#).
12. Placzek M, van den Heuvel ME, Flaig MJ, Korting HC. Pemiosis-like tinea corporis caused by *Trichophyton verrucosum* in cold-exposed individuals. *Mycoses*. Nov 2006;49(6):476-9. [\[Medline\]](#).
13. Seyfarth F, Ziemer M, Gräser Y, Elsner P, Hipler UC. Widespread tinea corporis caused by *Trichophyton rubrum* with non-typical cultural characteristics--diagnosis via PCR. *Mycoses*. 2007;50 Suppl 2:26-30. [\[Medline\]](#).
14. Weinstein A, Berman B. Topical treatment of common superficial tinea infections. *Am Fam Physician*. May 15 2002;65(10):2095-102. [\[Medline\]](#).
15. Liebel F, Lyte P, Garay M, Babad J, Southall MD. Anti-inflammatory and anti-itch activity of sertaconazole nitrate. *Arch Dermatol Res*. Sep 2006;298(4):191-9. [\[Medline\]](#).
16. Leyden J. Pharmacokinetics and pharmacology of terbinafine and itraconazole. *J Am Acad Dermatol*. May 1998;38(5 Pt 3):S42-7. [\[Medline\]](#).
17. da Silva Barros ME, de Assis Santos D, Soares Hamdan J. Antifungal susceptibility testing of *Trichophyton rubrum* by E-test. *Arch Dermatol Res*. May 2007;299(2):107-9. [\[Medline\]](#).
18. Wingfield AB, Fernandez-Obregon AC, Wignall FS, Greer DL. Treatment of tinea imbricata: a randomized clinical trial using griseofulvin, terbinafine, itraconazole and fluconazole. *Br J Dermatol*. Jan 2004;150(1):119-26. [\[Medline\]](#).
19. Aly R. Ecology and epidemiology of dermatophyte infections. *J Am Acad Dermatol*. Sep 1994;31(3 Pt 2):S21-5. [\[Medline\]](#).
20. Dahl MV. Dermatophytosis and the immune response. *J Am Acad Dermatol*. Sep 1994;31(3 Pt 2):S34-41. [\[Medline\]](#).
21. [Guideline] Drake LA, Dinehart SM, Farmer ER, et al. Guidelines of care for superficial mycotic infections of the skin: tinea corporis, tinea cruris, tinea faciei, tinea manuum, and tinea pedis. Guidelines/Outcomes Committee. American Academy of Dermatology. *J Am Acad Dermatol*. Feb 1996;34(2 Pt 1):282-6. [\[Medline\]](#).
22. Gupta AK, Einarson TR, Summerbell RC, Shear NH. An overview of topical antifungal therapy in

dermatomycoses. A North American perspective. *Drugs*. May 1998;55(5):645-74. [[Medline](#)].

23. Jones HE. Immune response and host resistance of humans to dermatophyte infection. *J Am Acad Dermatol*. May 1993;28(5 Pt 1):S12-S18. [[Medline](#)].
24. Leshner JL. Therapeutic agents for dermatologic fungal diseases. In: Elewski BE, ed. *Cutaneous Fungal Infections*. Malden: Blackwell Science; 1998:321-46.
25. Leshner JL Jr. Oral therapy of common superficial fungal infections of the skin. *J Am Acad Dermatol*. Jun 1999;40(6 Pt 2):S31-4. [[Medline](#)].
26. Macura AB. Dermatophyte infections. *Int J Dermatol*. May 1993;32(5):313-23. [[Medline](#)].
27. Pierard GE, Arrese JE, Pierard-Franchimont C. Treatment and prophylaxis of tinea infections. *Drugs*. Aug 1996;52(2):209-24. [[Medline](#)].
28. Rezabek GH, Friedman AD. Superficial fungal infections of the skin. Diagnosis and current treatment recommendations. *Drugs*. May 1992;43(5):674-82. [[Medline](#)].

Keywords

tinea corporis, ringworm, dermatophyte infection, *Trichophyton* species, *Microsporum* species, *Epidermophyton* species, *Trichophyton rubrum*, *T rubrum*, *Microsporum canis*, *M canis*, *Trichophyton mentagrophytes*, *T mentagrophytes*, *Trichophyton tonsurans*, *T tonsurans*, *Trichophyton concentricum*, *T concentricum*, Majocchi granuloma, Majocchi's granuloma, tinea imbricata, tinea capitis

Contributor Information and Disclosures

Author

Jack L Leshner Jr, MD, Chief, Professor, Department of Internal Medicine, Section of Dermatology, Medical College of Georgia

Jack L Leshner Jr, MD is a member of the following medical societies: American Academy of Dermatology, American Dermatological Association, American Medical Association, American Society for Dermatologic Surgery, Medical Association of Georgia, Society for Investigative Dermatology, and Southern Medical Association

Disclosure: Nothing to disclose.

Medical Editor

Janet Fairley, MD, Professor and Head, Department of Dermatology, University of Iowa

Janet Fairley, MD is a member of the following medical societies: American Academy of Dermatology, American Dermatological Association, American Federation for Medical Research, and Society for Investigative Dermatology

Disclosure: Nothing to disclose.

Pharmacy Editor

Richard P Vinson, MD, Assistant Clinical Professor, Department of Dermatology, Texas Tech University School of Medicine; Consulting Staff, Mountain View Dermatology, PA

Richard P Vinson, MD is a member of the following medical societies: American Academy of Dermatology, Association of Military Dermatologists, Texas Dermatological Society, and Texas Medical Association

Disclosure: Nothing to disclose.

Managing Editor

Rosalie Elenitsas, MD, Herman Beerman Associate Professor of Dermatology, University of Pennsylvania School of Medicine; Director, Penn Cutaneous Pathology Services, Department of Dermatology, University of Pennsylvania Health System

Rosalie Elenitsas, MD is a member of the following medical societies: American Academy of Dermatology and American Society of Dermatopathology

Disclosure: Nothing to disclose.

CME Editor

Joel M Gelfand, MD, MSCE, Medical Director, Clinical Studies Unit, Assistant Professor, Department of Dermatology, Associate Scholar, Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania

Joel M Gelfand, MD, MSCE is a member of the following medical societies: Society for Investigative Dermatology

Disclosure: AMGEN Consulting fee Consulting; AMGEN Grant/research funds Investigator; Genentech Grant/research funds investigator; Centocor Consulting fee Consulting; Abbott Grant/research funds investigator; Abbott Consulting fee Consulting; Novartis investigator; Pfizer Grant/research funds investigator; Celgene Consulting fee DMC Chair; NIAMS and NHLBI Grant/research funds investigator

Chief Editor

Dirk M Elston, MD, Director, Department of Dermatology, Geisinger Medical Center

Dirk M Elston, MD is a member of the following medical societies: American Academy of Dermatology

Disclosure: Nothing to disclose.

Acknowledgments

The authors and editors of eMedicine gratefully acknowledge the contributions of previous Chief Editor, William James, MD, and previous authors Mary Elizabeth Rushing Lott, MD, and Gwendolyn Zember, MD, to the development and writing of this article.

Further Reading

© 1994-2010 by Medscape.

All Rights Reserved

(<http://www.medscape.com/public/copyright>)