

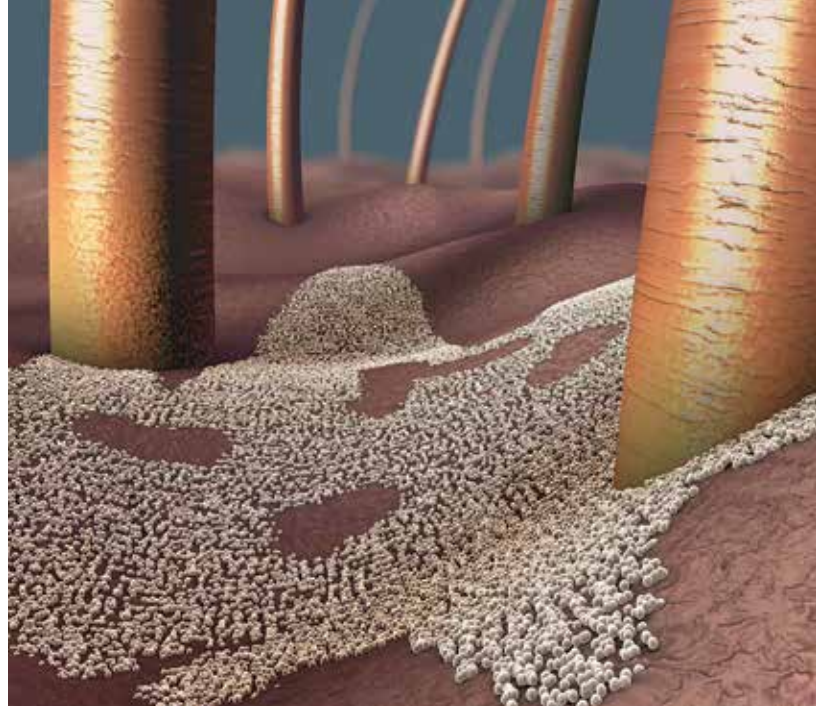
# Overview of cutaneous fungal infections

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Establishing a diagnosis before starting treatment is essential in the management of fungal skin infections. This can be achieved by having a high index of suspicion regarding the many clinical appearances of fungal infections and by using simple diagnostic tests. The three most common fungal conditions include tinea, pityriasis versicolor and candidiasis; however, this article will mention some rarer conditions including tinea nigra and piedra.

## KEY POINTS

- Cutaneous fungal infections present in a large variety of ways, many of which mimic noninfectious conditions. Establishing a diagnosis is essential before starting treatment, especially if oral antifungal therapy is considered.
- If the diagnosis is uncertain, avoid using corticosteroid treatment, which can obscure the fungal infection resulting in tinea incognito.
- Topical antifungal treatment is adequate in most fungal infections; however, for infections of the palms, soles, nails, hairy body areas, tinea incognito, granulomatous lesions and in widespread infection, oral treatment is usually required.
- Referral to a dermatologist is recommended in cases of treatment resistance.
- Investigate for underlying immune deficiency if the infection is widespread or recurrent.



Cutaneous fungal infections are common and are frequently managed by GPs. Fungal infections occur in children and adults of all ages and manifest in a large variety of ways. Nevertheless, the management principles for most superficial mycoses are similar. It is essential to establish the diagnosis microbiologically before starting therapy to avoid masking the underlying true condition with incorrect treatments such as topical corticosteroids.

Most cutaneous fungal infections are caused by either dermatophytes or yeasts. Dermatophytes are organisms that require keratin as a substrate and therefore have no potential to become systemic. They infect hair, nail and skin. There are three genera: *Trichophyton*, *Microsporum* and *Epidermophyton*. Within the genera, there are many species and these tend to vary depending on geographic location. They are classified as zoophilic when the natural host is a nonhuman animal; geophilic when found in the soil; and anthropophilic when the natural host is human. The last type are adapted to humans and therefore tend to cause low-grade but chronic disease. Other dermatophytes can cause highly inflammatory disease.

Another phenomenon found with acute inflammatory fungal infections is the 'id reaction', an immunological response to the fungus causing an eruption of sterile vesicles distant to the infection site. This is most commonly observed on the palms and fingers (Figure 1a).

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**Figures 1a and b.** Tinea pedis. a (left). An 'id' reaction in a patient with acute tinea pedis. b (right). Chronic tinea pedis, or athlete's foot, caused by *Trichophyton*.

*Candida*, a yeast, is a human commensal that can become an opportunistic pathogen. This tends to occur in immunosuppression, diabetes and moist environments, particularly in obese patients and when antibiotic therapy has altered the microbiome. *Candida* species are considerably more limited than the dermatophytes and, when immunosuppression exists, may cause systemic disease. *Candida albicans* is by far the commonest species; however, about 5% of infections are associated with atypical species such as *Candida glabrata*. Vulvovaginal candidiasis is, however, commonly seen in otherwise healthy women.

This article will summarise common cutaneous fungal infections; risk factors for fungal infections; complications of fungal infections; investigations required to diagnose fungal infections; and management of fungal infections.

### Types of cutaneous fungal infection

Cutaneous fungal infections are classified into three main groups:

- dermatophytosis – tinea
- yeast infections – candidiasis and pityriasis versicolor
- other fungal infections with minimal inflammatory response – tinea nigra and piedra. This group is a rare relative to dermatophytes and yeasts.

#### Dermatophytosis – tinea

In most cases, tinea refers to a cutaneous infection caused by a dermatophyte, also

known by the lay term ringworm. At other times, the term tinea is used to describe yeast infections such as tinea (or pityriasis) versicolor and mould infections such as tinea nigra.

Dermatophytosis can occur on any skin surface of the body. In a patient with tinea it is important to carefully examine other areas of the skin including the nails, as having any form of tinea predisposes the patient to infection elsewhere.

Specific nomenclature of tinea caused by dermatophyte infection is based on anatomical involvement. For each of these anatomical sites, specific dermatophyte species are present with some variance depending on geographic location.

#### Tinea pedis

Tinea pedis, also known as athlete's foot, refers to a dermatophyte infection of the foot (Figure 1b) and is commonly caused by *Trichophyton rubrum*, *Trichophyton interdigitale* and *Epidermophyton floccosum*. Of dermatophyte infections, tinea pedis is the most common and is often associated with tinea manuum (hand), tinea cruris (groin) and tinea unguium (nail), with the last acting as a reservoir for recurrent tinea pedis. Tinea pedis is highly contagious with transmission of infection occurring through direct contact with spores shed by infected people. Most often, the infection is acquired by walking barefoot in shared facilities such as swimming pools or locker rooms. Other risk factors predisposing patients to tinea pedis include excessive sweating of the foot and

prolonged use of occlusive footwear.

Based on the clinical presentation, tinea can be broadly classified as either acute or chronic.

**Acute tinea pedis.** Acute tinea pedis, also known as vesiculobullous (inflammatory) tinea pedis, is most commonly associated with *T. interdigitale*. Infections are often recurrent, self-limiting and typically precipitated by activities that cause the feet to sweat. Less frequently, acute ulcerative tinea pedis, characterised by ulcerations and erosions, can also occur and is often associated with secondary bacterial infections.

Patients with acute tinea pedis usually present with unilateral or bilateral blistering and/or scaling of the skin, particularly on the soles, which can be intensely itchy and sometimes painful with a burning sensation.

**Chronic tinea pedis.** Chronic tinea pedis is predominantly caused by *T. rubrum*, an anthropophilic dermatophyte that can be very chronic and recurrent. This condition is characterised by slow, progressive formation of itchy, red erosions and scales in the lateral toe webbing (interdigital tinea pedis). The skin becomes increasingly macerated as the infection develops and interdigital fissures may form. If left untreated, chronic tinea pedis can extend to the sole, heel and lateral sides of the foot (moccasin tinea pedis), appearing as diffuse white scaly plaques. At this stage, nail involvement occurs frequently.

Differential diagnoses for tinea pedis include:

- psoriasis – often salmon-red in colour, well-demarcated, with heavy silvery scale
- candidiasis – often in moist toe webs with satellite pustules
- bacterial infections – often in moist toe webs
- irritant contact dermatitis – associated with excessive sweating
- contact allergic dermatitis – may have contact history to footwear



Figure 2. Tinea unguium, also known as onychomycosis.

- material such as rubber or dyes
- scabies – often involves other areas of the body and finger webs.

**Cellulitis secondary to tinea pedis.** Tinea pedis is a common cause of lower limb cellulitis and its presence should be examined for in all cases of cellulitis, particularly in recurrent cases. If tinea is found in a patient with cellulitis, aggressive treatment of the fungal infection is mandatory to prevent recurrences of the bacterial infection.

#### Tinea manuum

Tinea manuum refers to dermatophyte infection of the hand and is often caused by the same dermatophytes as tinea pedis. Direct contact with infected animals or soil can also predispose to tinea of the hand. The distribution is classically of the palms but there is often asymmetry and the existence of a defined edge to the eruption. Tinea manuum is characterised by fine granular scale in the creases with erythema, and may be associated with itch or burning.

Tinea infections commonly present as ‘two feet, one hand syndrome’ and it is therefore important to examine the feet as well as the hands. The dermatophyte species involved are similar to those that cause tinea pedis.

Differential diagnoses for tinea manuum include:

- psoriasis – often salmon-red in colour, well-demarcated, with heavy silvery scale
- candidiasis – found most often

- between the fingers with satellite pustules or causing paronychia
- irritant contact dermatitis – scaly red, ill-defined rash involving the dorsum of the hands as well as the palms, with patients often atopic and having a strong occupational element with exposure to moist conditions
- allergic contact dermatitis – usually acute, inflammatory and vesicular involving the dorsum of the hands as well as the palms.

#### Tinea unguium

Tinea unguium, also known as onychomycosis, refers to a fungal infection of the fingernail or toenail and is most commonly caused by *T. rubrum* and *T. interdigitale*. Toenails are much more likely to be infected compared with fingernails. Multiple nails are usually infected, although involvement of all nails is uncommon (Figure 2). Tinea unguium initially presents as white, yellow, green and/or black discolouration of the nail, with the distal nail most likely to be infected first. If left untreated or in severe cases, the infected nail can become gradually thickened and disfigured.

Onychomycosis may involve the whole thickness of the nail plate or may be superficial, in which case it presents as thin white plaques on the nail surface. The latter presentation can be caused by dermatophytes but other fungi such as the moulds, for example *Fusarium* and *Aspergillus*, can be found.

Tinea unguium can be difficult to distinguish from noninfective nail dystrophies such as psoriasis. However, it rarely involves every nail as opposed to endogenous diseases. To support the diagnosis, careful examination of the nail itself and other cutaneous signs elsewhere is essential. The diagnosis usually remains unclear and nail clippings should always be sent for culture.

Differential diagnoses for tinea unguium include:

- psoriasis – usually there is other evidence of psoriasis
- traumatic onycholysis



Figure 3. Tinea cruris.

- onychogryphosis – age-related changes often associated with poor peripheral circulation
- lichen planus
- changes secondary to paronychia which usually involve the edge of the nail.

#### Tinea cruris

Tinea cruris, also known as jock itch, refers to a dermatophyte infection of the groin (Figure 3) and is often caused by the same dermatophytes as tinea pedis. This condition occurs far more frequently in men than in women and is associated with sweating.

Patients with tinea cruris present with a red patch high in the inner aspect of the thighs, typically sparing the scrotum. The infection has a well-demarcated border with or without central clearing. Severe cases may involve the perineum, perianal, gluteal cleft and buttock areas.

Differential diagnoses for tinea cruris include:

- candidiasis – usually satellite pustules are seen
- psoriasis – usually no central clearing and deep mahogany in colour but less scale than in psoriasis elsewhere on the body
- seborrhoeic dermatitis – usually no central clearing
- erythrasma – a bacterial infection that fluoresces coral pink under Wood’s light
- contact dermatitis
- intertrigo, particularly in obese patients.

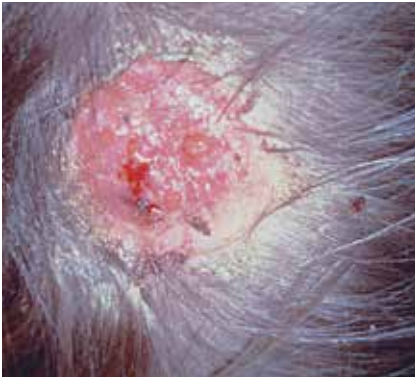


Figure 4. Kerion (inflammatory tinea capitis).

### Tinea capitis

Tinea capitis refers to a dermatophyte infection of the scalp and is caused by *Trichophyton* and *Microsporum* genera. Infection can be contracted from infected individuals and animals. Tinea capitis is almost exclusively seen in children. The commonest appearance is of single or multiple areas of patchy alopecia with scale and broken-off hairs. Inflammation is variable but usually not severe. In Australia, *Trichophyton mentagrophytes* is the commonest organism isolated.

**Endothrix infection.** In endothrix infection, fungal hyphae and spores infect the inner part of the hair shaft. It is caused by the *Trichophyton tonsurans*, *Trichophyton violaceum* and *Trichophyton soudanense*. *T. tonsurans* is the commonest in Australia and is more prevalent in overcrowded housing and more common in Aboriginal and Torres Strait Islander populations. Infected patients often present with scaly, noninflamed patches that may be associated with a round area of hair loss. Because of the destruction of the hair shaft, breakage occurs at the level of the scalp causing ‘black dot tinea’. There may also be diffuse alopecia. There is no fluorescence on Wood’s lamp examination.

**Ectothrix infection.** In ectothrix infection, fungal hyphae and spores infect the external hair shaft. Some dermatophytes associated with this type of infection

include *Trichophyton verrucosum*, *Microsporum canis* and *Microsporum audouinii*. Patchy hair loss is typical. Wood’s lamp examination shows green-yellow fluorescence of the infected hair shaft. These fungi are uncommon in Australia.

**Favus.** Favus is an uncommon, severe form of tinea capitis caused by *Trichophyton schoenleinii*, which spreads along the entire hair shaft. Eventually, this leads to the destruction of the hair shaft leaving behind a yellow cup-shaped crust (scutula) and matted hair.

**Kerion.** A kerion is a dramatic, inflammatory mass usually occurring in hair-bearing skin. Although the patient is well and afebrile, the appearance can be alarming, with tender pustules and boggy swelling. There is rapid, dramatic hair loss (Figure 4), posterior cervical or auricular lymphadenopathy and, rarely, in severe cases, permanent scarring alopecia. In some carrier states, patients may be asymptomatic with only mild scaling to the scalp. In Australia, the zoophilic *T. mentagrophytes* is the commonest cause but recently there has been an increasing prevalence of African species of dermatophytes such as *M. audouinii*, *T. violaceum* and *T. soudanense*. These dermatophytes can cause endothrix or ectothrix infections. As tinea capitis is endemic in Africa, asymptomatic carriers may be seen more frequently and therefore extending treatment to family members is required to prevent a reservoir of infection.

Differential diagnoses for tinea capitis include:

- alopecia areata – hair loss areas are usually smooth
- trichotillomania – there is no inflammation and hair is broken off
- scarring alopecias such as lichen planus or lupus erythematosus
- trauma, particularly from hair-styling techniques where hair is pulled into a tight ponytail.

### Tinea corporis

Tinea corporis refers to dermatophyte



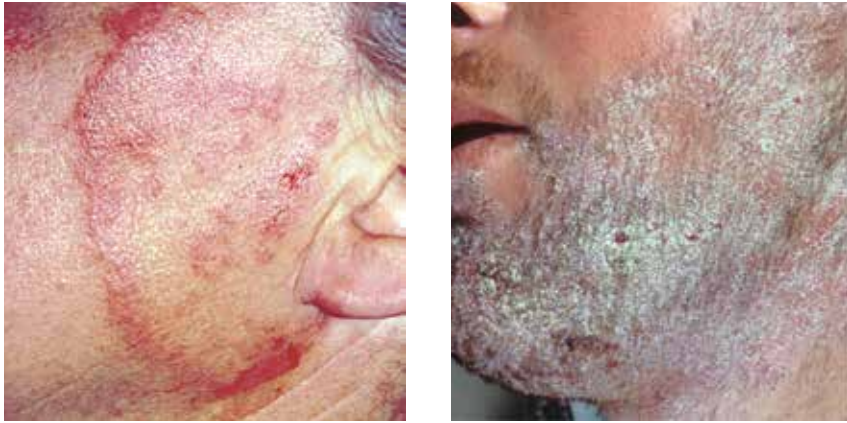
Figure 5. Submammary tinea corporis.

infection of the trunk or limbs and is most commonly caused by the anthropophilic *T. rubrum*. Infection contracted from cats or dogs is often due to *M. canis*. However, in Australia, *T. mentagrophytes* infection is more commonly contracted from guinea pigs and pet mice. It can present independently or with other forms of fungal infection such as tinea pedis and tinea unguium (Figure 5).

Patients with tinea corporis present with a scaly, itchy annular (ring-shaped) or arcuate (bowed or curved) rash with clearly defined raised edges and central clearing as the lesion resolves. Pustules may occur in some cases. Infection from animal transmission is much more acute and inflammatory in nature.

Differential diagnoses for tinea corporis are wide and include many rashes that are annular:

- erythema annulare centrifugum – usually with trailing peripheral scale
- nummular/discoid eczema – usually with double-edged peripheral scale
- granuloma annulare – nonscaly annular lesions
- psoriasis – no central clearing, silvery scale and may have other clinical symptoms
- seborrhoeic dermatitis – ill-defined edges
- lichen simplex – history of chronic scratching or rubbing although skin scraping may be required to rule out tinea infection.



**Figures 6a and b.** Tinea faciei and tinea barbae. a (left). Tinea faciei affects the glabrous skin and is more common in children and women than in men. b (right). Tinea barbae is a more severe infection than tinea faciei and affects the beards of men.

**Tinea faciei and tinea barbae**

Tinea faciei refers to a dermatophyte infection of the face and is most often seen in children and women. It is caused by a variety of dermatophytes and infection is acquired from tinea infection elsewhere on the body or from direct contact with infected individuals or animals. It presents as red scaly round lesions that often affect glabrous skin such as the chin and upper lip (Figure 6a).

Tinea barbae refers to a dermatophyte infection of the beard and is often a more severe form of infection compared with tinea faciei resulting in kerion or abscess formation (Figure 6b). It generally affects men, especially farmers as the causative dermatophyte is often from infected animals including cattle (*T. verrucosum*) and horses (*Trichophyton mentagrophytes var equinum*).

Differential diagnoses for tinea faciei and tinea barbae include:

- discoid lupus erythematosus, which can look very similar
- bacterial, viral or candidal folliculitis – skin scraping, or swab may be required to differentiate
- contact dermatitis – contact history
- rosacea – no scales, pustules on an erythematous background
- psoriasis – silvery scale and may have other clinical symptoms

- seborrhoeic dermatitis – ill-defined edges.

**Majocchi's granuloma**

Dermatophyte infections are usually limited to the epidermis. Majocchi's granuloma refers to extension of dermatophyte infections into deeper dermis and subcutaneous tissue. The most common causative dermatophyte for this form of infection is *T. rubrum*. The condition is typically characterised by nodules or abscess formation. Invasion of the dermatophyte into the dermis is thought to be secondary to trauma to the skin or hair follicles, which can occur during shaving. In some cases, in particular immunocompromised patients, inappropriate use of topical corticosteroids to treat dermatophyte infections can also promote the development of Majocchi's granuloma.

**Tinea incognita (tinea incognito)**

Tinea incognita describes the clinical appearance of tinea infection that has been altered by inappropriate use of immunosuppressant medications such as topical or systemic corticosteroids, or calcineurin inhibitors. Due to the reduced inflammation of the tinea infection, typical clinical features of the infection may not be present. For example, tinea incognito lesions are generally less erythematous

and may lack a scaly border. Additionally, the lesions can be more pustular and larger than typical tinea infections. With ongoing use of topical corticosteroid, they may become quite extensive with nodules.

**Yeast infections – pityriasis versicolor and candidiasis**

**Pityriasis versicolor**

Pityriasis versicolor, also known as tinea versicolor, is a yeast infection from the genus *Malassezia* (Figures 7a to c). It can occur at any age but usually affects adolescents and young adults. The most commonly involved areas include the upper chest and upper back, then the face, scalp and groin, and less frequently the antecubital fossae (Figure 7a). In prepubertal children it usually involves the forehead where it is easily confused with pityriasis alba. Pityriasis versicolor is characterised by well-demarcated hyper- or hypopigmented lesions, often coalescing and covered with fine, branny scale. The morphology is of multiple coalescing macules with a characteristic appearance. Hypopigmentation occurs due to damage to melanocytes from azelaic acid compounds in the yeast (Figure 7b). Colouration of pityriasis versicolor varies and depends on a combination of factors including natural skin pigmentation, exposure of the area to sunlight and the severity of the disease.

Although pityriasis versicolor is usually asymptomatic or only associated with mild itch, the appearance often motivates treatment for many individuals. The lesions fluoresce a pale greenish colour under Wood's light.

A less common clinical presentation of *Malassezia* infection is folliculitis. The folliculitis occurs predominantly on the trunk and, in children, on the forehead, with follicular papules and pustules.

Differential diagnoses for pityriasis versicolor include:

- pityriasis alba – white scaly areas seen on face of children with atopic eczema

- pityriasis rosea – usually red lesions in a fir tree pattern with trailing inward facing scale
- seborrhoeic dermatitis – on face, mid-chest and mid-back
- dermatophyte infection – lesions often have peripheral scale with central clearing
- vitiligo – complete depigmented macules and patches without scale.

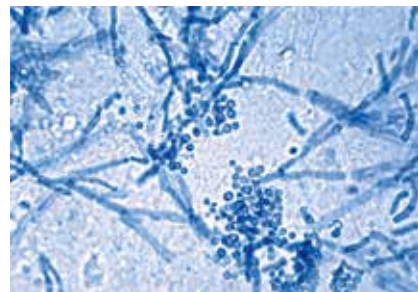
### Candidiasis

Candidiasis is an infection caused by the yeast *Candida* with the most common causative organism being *Candida albicans*. Patients who are immunosuppressed, have diabetes or are using corticosteroids and long-term antibiotics are at higher risk of developing candidiasis. The use of antibiotics can destroy the natural bacterial flora on the skin surface, which can then result in the overgrowth of *Candida* as an opportunistic infection. *Candida* can affect many sites on the body (Figures 8a to e).

**Oral candidiasis.** Oral candidiasis, also known as oral thrush, is common and affects the mouth (Figure 8a). In addition to patients who are immunosuppressed, those using inhaled corticosteroids without properly rinsing their mouths afterwards have increased risk of developing oral candidiasis. The infection presents as discrete white patches on the inner surface of the mouth. Over time, these patches can become confluent forming a pseudomembrane, which can be scraped off leaving an underlying erythematous and sometimes ulcerated area. Although most patients are asymptomatic, oral candidiasis may be associated with a cottony sensation, loss of taste and difficulty eating or swallowing secondary to pain.

Differential diagnoses for oral candidiasis include:

- lichen planus – fine white reticulate scale on mucosa (Wickham's striae)
- herpetic infection – painful mucosal ulcers



**Figures 7a to c. Pityriasis versicolor.** a (above left). Pityriasis versicolor most commonly affects the upper trunk and shoulders. b (above). Depigmented lesions in a patient with pityriasis versicolor. c (left). *Malassezia* as viewed microscopically in 10% KOH, showing the 'spaghetti and meatballs' appearance of the hyphae and spores.

- erythema multiforme – mucosal ulcers with ocular ulcers and acral target lesions
- pernicious anaemia – raw ulceration, particularly on the tongue.

**Vulvovaginal candidiasis.** Vulvovaginal candidiasis, also known as vaginal thrush, is a common candidal infection of the vulva or vagina. Acute infection causes a thick white vaginal discharge with associated itch, burning and an occasional sensation of dysuria. The vaginal wall is often erythematous and oedematous with white plaques adhering to the wall (Figure 8b). A red scaly rash with satellite pustules can sometimes be seen on the groin and thigh, suggesting spread of the infection. Chronic infection is more subtle with nonspecific redness, pain and itch. Vulvovaginal candidiasis is most symptomatic with an elevated oestrogen state that occurs in premenstruation and pregnancy or

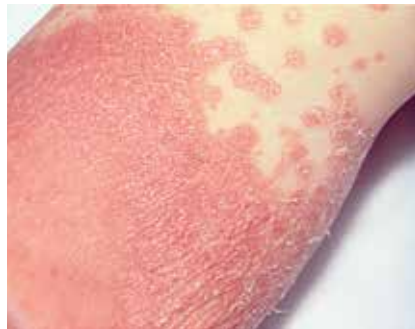
with use of exogenous oestrogen.

Differential diagnoses for vulvovaginal candidiasis include:

- bacterial vaginosis
- skin conditions such as contact dermatitis, psoriasis, lichen planus, lichen sclerosus or atopic eczema.

**Male genital candidiasis.** Male genital candidiasis affects the glans of the penis. It is usually transmitted from an affected sexual partner and is more prevalent in uncircumcised males. Initially, small papules and/or pustules appear on the glans, which may later degrade and discharge leaving behind erythematous erosions with surrounding white scale (Figure 8c). Associated swelling and tenderness can also occur and, in severe cases, inflammation of the urethra or phimosis can result.

Differential diagnoses for candidal balanitis include:



**Figures 8a to e. Candidiasis infection.** a (above far left). Oral candidiasis, also known as thrush. b (above left). Vulvovaginal candidiasis, also referred to as thrush. c (above right). Balanitis caused by candidiasis. d (far left). Candidiasis between the toes, the cause of about 1% of all cases of athletes foot. e (left). Cutaneous candidiasis, seen in intertriginous zones, characterised by satellite lesions.

- bacterial balanitis
- contact allergic dermatitis
- flexural psoriasis
- Reiter's syndrome
- lichen sclerosus
- lichen planus
- plasma cell balanitis
- fixed drug eruption
- penile intraepidermal neoplasia
- scabies.

**Other fungal infections with minimum inflammatory response**

**Piedra**

Piedra is a fungal infection that affects hair follicles and can occur at any age. Piedra is classified into two clinical subtypes based on the clinical appearance and causative organism. Both subtypes are usually asymptomatic.

White piedra is caused by *Trichosporon beigelii* and is prevalent in countries with temperate and semitropical climates. It can affect any hair-bearing areas and

presents with multiple lightly pigmented soft nodules on the hair follicles and shafts.

Over time, these nodules eventually cause the hair to break. Topical treatment is often adequate for white piedra.

Black piedra is caused by *Piedraia hortae* and is more common in tropical climates. It predominantly affects the scalp although it can affect other hair-bearing areas. Nodules formed are firmer, darker and more adherent to the hair follicle/shaft compared with white piedra.

Infrequently, patients present with hair loss only. Oral antifungal therapy is often indicated for black piedra, with shaving of the hair being the optimal treatment.

Differential diagnoses for piedra include:

- pediculosis (lice infestation)– eggs are pale in colour and do not cover the hair follicles/shaft.

**Tinea nigra**

Tinea nigra is caused by a brown mould called *Hortaea werneckii*, which is often found in soil. It is more prevalent in humid climates and associated with hyperhidrosis. It affects the palms and soles and presents as a solitary hyperpigmented macule or patch that progressively grows over time. The infection is very responsive to treatment with clearance of lesions within two to four weeks of topical antifungal therapy.

Differential diagnoses for tinea nigra include:

- benign naevi – pigment network under dermoscopy examination
- extraneous pigment – history of previous skin inflammatory lesion which resolved
- malignant melanoma.

**Risk factors**

Fungal skin infections can affect anyone, with most infections occurring in



**Figures 9a and b.** a (left). Skin infection caused by the granular strain of *Trichophyton rubrum* in an Aboriginal woman. b (right). Tinea unguium in an Aboriginal patient.

otherwise healthy individuals. Immunosuppressed populations, including the elderly, people with diabetes, those with HIV/AIDS and those using immunosuppressants, are at higher risk of developing persistent fungal infections. However, healthy persons with the anthropophilic *T. rubrum* may have very persistent infection. These populations are also more likely to develop treatment-resistant or systemic fungal infections. Aboriginal and Torres Strait Islander populations are also at higher risk of certain dermatophyte strains,

particularly *T. tonsurans*. However, *T. rubrum* (tinea corporis and tinea unguium) and *T. violaceum* (tinea capitis) are found to be more common in these populations (Figures 9a and b). When examining nonspecific skin lesions in at-risk populations, a greater suspicion for cutaneous fungal infection should be maintained. Additionally, there should be a low threshold to investigate for any associated comorbidities such as diabetes or other immunocompromised states when fungal infections are unresponsive to standard therapy.

Environmental factors also play an important role in the proliferation of fungal infections. In general, fungal infections thrive in warm and moist environments. Areas of the body that are warmer naturally and more prone to sweat include intertriginous zones such as between the toes, groin and under the breasts. When combined with external factors such as warm climates, exercise, poor hygiene, close living quarters with other infected individuals and/or other risk factors as described above, fungal infections tend to proliferate freely.

**Complications**

In general, the most significant complication of fungal skin infections is the potential progression towards a secondary bacterial infection. Bacterial infections occur as fungal infection inherently disrupts normal skin integrity. Development of increased erythema extending beyond the site of the fungal infection with associated fever should promptly raise suspicion for cellulitis especially in at-risk populations. Treatment with antibiotics should be commenced in any case involving secondary bacterial infection.

**Investigations**

Establishing the correct diagnosis of fungal infection is imperative. This is because incorrect diagnosis often leads to incorrect management, frequently with topical corticosteroids. Because topical corticosteroids can mask and worsen any underlying fungal infection, always obtain a skin specimen sample to confirm the diagnosis before using them if there is any doubt.

**Specimen collection**

Skin, nail and hair from infected sites can be collected to confirm the presence of fungal infection:

- skin specimen – cleanse the skin with alcohol and allow to dry. Using the edge of a blade, gently scrape the scale from an advancing border onto a glass slide or specimen pot

Treatment	Dosage	Duration
Terbinafine (dermatophytes only)	1% cream or gel, once or twice daily	1 to 2 weeks
Bifonazole	1% cream, once daily	2 weeks
Clotrimazole	1% cream, twice daily	2 weeks
Econazole	1% cream, twice daily	2 weeks
Miconazole	2% cream, twice daily	2 weeks
Ketoconazole	2% cream, once daily	Continue for several days after symptoms resolve
Nystatin	100,000 units/g cream, twice daily	2 weeks
Aluminium acetate wet dressings (chronic tinea pedis)	–	20 minutes, two to three times per day
Tea tree oil-soaked cotton wool (chronic tinea pedis)	–	Overnight

**TABLE 2. ORAL ANTIFUNGAL THERAPIES FOR DERMATOPHYTOSIS AND PITYRIASIS VERSICOLOR**

Treatment	Dosage – Adult	Dosage – Paediatric	Duration*	Other notes
Terbinafine <sup>†</sup>	250 mg, once daily  If CrCl less than 50 mL/min use 125 mg, once daily	<b>Use in 1- to 18-year-olds:</b> <b>10 to 20 kg:</b> 62.5 mg, once daily <b>20 to 40 kg:</b> 125 mg, once daily <b>Above 40 kg:</b> 250 mg, once daily  <b>For tinea capitis, higher dosage can be used:</b> <b>10 to 25 kg:</b> 125 mg, once daily <b>25 to 35 kg:</b> 187.5 mg, once daily <b>Above 35 kg:</b> 250 mg, once daily	<b>Skin:</b> 2 to 4 weeks <b>Scalp:</b> 4 to 6 weeks <b>Fingernails:</b> up to 6 weeks <b>Toenails:</b> up to 12 weeks	<ul style="list-style-type: none"> <li>• Obtain baseline FBE/UEC/LFTs and monitor if treating for more than 6 weeks</li> <li>• Gastrointestinal side effects are common</li> <li>• Metallic taste in mouth, rarely</li> </ul>
Fluconazole	<b>Skin (tinea):</b> 150 mg, once weekly or 50 mg, once daily <b>Nail (tinea):</b> 150 to 300 mg, once weekly <b>Pityriasis versicolor:</b> 400 mg orally as single dose	<b>Use in 1 month- to 18-year-olds:</b> 6 to 12 mg/kg, once daily	<b>Skin:</b> up to 6 weeks  <b>Nail:</b> 3 to 12 months	<ul style="list-style-type: none"> <li>• Obtain baseline FBE/UEC/LFTs and monitor monthly</li> <li>• Gastrointestinal side effects are common</li> </ul>
Itraconazole <sup>†</sup>	200 mg, once or twice daily	<b>Use in 1 month- to 12-year-olds:</b> 5 to 7.5 mg/kg, daily in 1 or 2 doses  <b>Use in 12- to 18-year-olds:</b> 100 to 400 mg, once daily	<b>Skin:</b> daily for 1 to 2 weeks <b>Fingernails:</b> twice daily for one week per month and repeat treatment up to 2 months <b>Toenails:</b> twice daily for one week per month and repeat treatment up to 3 to 4 months <b>Pityriasis versicolor:</b> once daily for 1 to 2 weeks <b>Pityriasis versicolor prophylaxis:</b> 1 day per month for 6 months	<ul style="list-style-type: none"> <li>• Obtain baseline LFTs and serum potassium and monitor further if treating for more than 1 month</li> <li>• Gastrointestinal side effects are common</li> </ul>
Griseofulvin <sup>†</sup>	<b>Skin and scalp tinea:</b> 500 mg, once daily <b>Nails (tinea):</b> 1 g, once daily (note newer drugs work better than griseofulvin in the nails)	<b>Use in 1 month- to 12-year-olds:</b> 10 to 20 mg/kg (maximum 1g), once daily  <b>Use in 12- to 18-year-olds:</b> 500 mg to 1 g, once daily	<b>Skin:</b> up to 12 weeks <b>Scalp:</b> up to 8 weeks <b>Nails:</b> up to 12 months	<ul style="list-style-type: none"> <li>• Monitor complete blood count during prolonged treatment</li> </ul>

Abbreviations: CrCl = creatinine clearance rate; FBE = full blood examination; LFTs = liver function tests; UEC = urea, electrolytes and creatinine.

\* Paediatric duration may differ. Continue until clinical resolution, cultures should be repeated 3 to 4 weeks after cessation of treatment and consider a repeat course if the result is positive.

<sup>†</sup>Terbinafine and griseofulvin are not effective for *Malassezia* yeast.

<sup>†</sup>Dosages for itraconazole are based on Sporanox capsules. Sporanox and Lozanoc capsules and oral liquid formulas are not bioequivalent.

- nail specimen – cleanse the nail with alcohol and allow to dry. Clip the nail as short as possible and, using the edge of a blade, scrape the subungual tissue into a specimen pot
- hair specimen – pluck the affected hair with tweezers and collect the proximal portion involving the bulb.

Using a blade, scrape the skin of the affected scalp onto a glass slide or specimen pot.

**Potassium hydroxide (KOH) test**

The KOH test is a relatively quick and inexpensive test that uses microscopy to confirm the presence of dermatophyte

or yeast such as *Candida* and *Malassezia*. Collected specimens are placed on a glass slide and KOH is added, which dissolves the epidermal keratinocytes, leaving behind the fungal elements. Microscopy is then used to view branch-like structures (septate hyphae) in dermatophytes, ‘spaghetti and meatballs’ appearance

**TABLE 3. TOPICAL THERAPY OPTIONS FOR PITYRIASIS VERSICOLOR**

Treatment	Dosage	Duration
Econazole	1% solution topically to wet skin, left overnight	3 nights
Selenium sulfide	2.5% shampoo topically to wet skin left for at least 10 minutes or diluted 1:4, left overnight	3 times a week for 3 months
Ketoconazole	2% shampoo topically to wet skin left for at least 5 minutes and wash off	5 days
Miconazole	2% shampoo topically, once daily for 10 minutes and wash off	10 days

(hyphae and spores, Figure 7c) in *Malassezia* yeast and pseudohyphae in *Candida*. The test is very high in specificity but low in sensitivity as the results are highly dependent on the quality of the specimen and the operator’s skill.

In deeper infections involving the dermis or subcutaneous tissue such as Majocchi’s granuloma, the KOH test will be negative as it can only demonstrate fungus in the stratum corneum. In this case, a skin biopsy should be performed.

**TABLE 4. ORAL ANTIFUNGAL THERAPY OPTIONS FOR ORAL AND MUCOSAL CANDIDIASIS**

Treatment	Dosage	Duration
Miconazole gel	<b>Use in adults and children over 2 years of age:</b> 2% gel, 2.5 mL topically then swallowed, four times daily after meals <b>Use in children under 2 years of age:</b> 1.25 mL topically then swallowed, four times daily after meals	1 to 2 weeks, continue for at least 7 days after symptoms resolve
Amphotericin B lozenges	<b>Use in adults and children over 2 years of age:</b> 1 lozenge (10 mg) sucked then swallowed, four times daily after meals	1 to 2 weeks, continue for 2 to 3 days after symptoms resolve
Nystatin drops	<b>Use in adults and children:</b> 100,000 units/mL, 1 mL topically then swallowed, four times daily after meals	1 to 2 weeks, continue for 2 to 3 days after symptoms resolve
Fluconazole	<b>Use in adults:</b> 150 mg, for cutaneous and vulvovaginal candidiasis 50 to 200 mg, daily for oropharyngeal candidiasis <b>Use in children:</b> 3 to 12 mg/kg, once daily	Single oral dose 1 to 2 weeks
Itraconazole*	<b>Use in adults:</b> 100 to 200 mg, once daily <b>Use in children:</b> 5 to 75 mg/kg, once daily in 1 to 2 doses)	2 weeks
Voriconazole	<b>Use in adults and children over 12 years:</b> Above 50 kg, 6 mg/kg, intravenously, every 12 hours for the first 24 hours followed by 4 mg/kg, intravenously, every 12 hours <b>Or</b> Above 40 kg, 400 mg, orally every 12 hours for 2 doses, followed by 200 to 300 mg orally twice daily Under 40 kg, 200 mg, orally every 12 hours for 2 doses, followed by 100 to 150 mg, orally twice daily <b>Use in children 12 to 15 years and less than 50 kg and children 2 to 12 years:</b> 9 mg/kg, intravenously every 12 hours for 2 doses followed by 8 mg/kg, intravenously twice daily or 9 mg/kg, orally twice daily.	2 to 4 weeks
Posaconazole (reserved for serious refractory fungal infections)	<b>Use in adults and children over 13 years:</b> 200 mg, once daily for 1 day then 100 mg, once daily <b>Use in serious refractory fungal infection for adults:</b> 300 mg, intravenously twice daily for 1 day then 300 mg, intravenously, once daily	–

\*Dosages for itraconazole are based on Sporanox capsules. Sporanox and Lozanoc capsules and oral liquid formulas are not bioequivalent.

## Microscopy and culture

Skin, nail and hair specimens can be sent for microscopy, to provide immediate confirmation, and culture in a specimen pot with results returned within three to six weeks. It is important to interpret microscopy results alongside clinical findings because of the high false-negative rates, particularly in nail clippings. With cultures, false-negative results can occur due to insufficient sample, previous treatment with antifungal therapy or non-specific species. When fungal infection is suspected, consider retesting or a trial of therapy to see if there is a clinical response.

## Wood's light

Wood's light is rarely helpful for diagnosing tinea as most species responsible for infections do not fluoresce. It does confirm the differential diagnosis of erythrasma, however, and in pityriasis versicolor, affected areas may fluoresce a pale green colour under Wood's light. It is also useful in some cases of tinea capitis.

## Skin biopsy

Consider a skin biopsy if the above methods are inconclusive. A punch biopsy under local anaesthetic can be performed. The specimen is placed in a formalin-containing pot and is reviewed by a histopathologist, with or without the addition of periodic acid-Schiff (PAS) stain. This is a very reliable test.

## Management options

As a general principle, it is important to advise patients about nonpharmacological measures to reduce the risk of infection including drying the skin after a shower, using drying powder on intertriginous zones and wearing nonocclusive clothing, underwear and footwear.

## Dermatophyte infection

Treatment of dermatophytosis with either topical or oral antifungal agents is dependent on the location and severity of the infection. Topical therapies including

terbinafine, bifonazole, clotrimazole, econazole and miconazole are used to treat local infection of the trunk, limb, face or interdigital areas (Table 1). For localised chronic tinea pedis with maceration, aluminium acetate wet dressings (Burow's solution diluted 1:20 with water) can be applied for 20 minutes, two to three times per day, or tea tree oil-soaked cotton wool can be applied overnight between the affected toes.

Oral therapies including terbinafine, itraconazole, fluconazole and griseofulvin are used for hair-bearing areas, palms, soles and nails (Table 2). Generally, a higher dosage is recommended for treatment of tinea capitis. Oral therapy should also be considered in infections that have failed topical therapy, recurrent or widespread infection, tinea incognita and deep infections, as topical agents are not able to penetrate deeper layers of the skin.

## Pityriasis versicolor

Both topical and oral treatments are appropriate for pityriasis versicolor. Topical options such as econazole, selenium sulfide and ketoconazole are available for treatment of pityriasis versicolor (Table 3). Oral therapy (fluconazole, itraconazole) is indicated when the infection fails to respond to topical treatment and often for folliculitis (Table 2). Repeated topical or oral therapy and even prophylactic treatment with oral itraconazole can be considered in recurrent pityriasis versicolor.

## Candida

Candidiasis can be treated both topically and orally. Topical therapy such as imidazole cream or nystatin is usually commenced initially (Table 1). Low-potency corticosteroids such as 1% hydrocortisone cream can be added to the treatment regimen if there is concurrent inflammation. Oral agents such as fluconazole and itraconazole are indicated if topical agents are contraindicated or if the infection is severe (Table 4).

## When is referral required?

Referral to a dermatologist should be considered in patients with cutaneous fungal infections if:

- the condition is not responsive to treatment despite three to four weeks of therapy
- the infection is worsening and spreading
- there are complications in the treatment.

## Conclusion

Cutaneous fungal infections can affect many areas of the body and present in a variety of ways. Recognition of common clinical patterns and use of simple diagnostic tests can aid in diagnosis and allow for optimal management. Always interpret investigation results with clinical examination. A negative test with a strong clinical suspicion warrants retesting or a trial of antifungal therapy. Consider investigating for underlying comorbidities in unusual, recurrent or severe cases. If there are complications in treatment or non-response, referral to a dermatologist is indicated. MT

## Further reading

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