

Evaluation and Treatment of *Malassezia*-Related Skin Disorders

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Malassezia are commensal yeasts found on the sebaceous areas of human skin. Although they are part of the normal skin flora, they play a pathogenic role in several skin conditions, most notably tinea versicolor, *Pityrosporum* folliculitis, and seborrheic dermatitis. *Malassezia* also have been associated with subsets of psoriasis and atopic dermatitis, especially those affecting the scalp. Patients are often distressed by the appearance of *Malassezia*-related diseases, particularly the dyspigmentation of tinea versicolor and the scaling and erythema of seborrheic dermatitis and scalp psoriasis. Treatment of *Malassezia*-related dermatoses generally requires the use of topical or oral antifungal medications, often in combination with antifungal washes and shampoos. In some cases, low-potency corticosteroids are a useful adjunct. Patients with *Malassezia* dermatoses need to be educated on the tendency of these eruptions to recur unless maintenance treatment is continued indefinitely. The appearance of skin affected by *Malassezia* may take months to normalize, even after successful treatment.

Malassezia are commensal yeasts found on the sebaceous areas of human skin. Although they are a part of normal skin flora, *Malassezia* also play a pathogenic role in several dermatological conditions with substantial cosmetic consequences. *Malassezia* yeasts cause the dyspigmented patches of tinea versicolor, the inflamed red papules and pustules of *Pityrosporum* folliculitis, and the erythematous, scaling facial and scalp skin seen in seborrheic dermatitis.¹ There is evidence that

Malassezia may even play a role in atopic dermatitis and psoriasis, especially in cases involving the scalp.

HISTORY AND TAXONOMY

Malassezia yeasts were first described in the mid-19th century on the skin of patients with seborrheic dermatitis. They are named after Louis Charles Malassez, a French scientist who in 1874 described budding yeasts isolated from the skin. Another French physician and microbiologist, Raymond JA Sabouraud, proposed the genus name *Pityrosporum* in 1904 for fungal spores seen on human skin. The *Pityrosporum* genus was later differentiated into *Pityrosporum orbiculare* for types with round spores and *Pityrosporum ovale* for variants with oval shape.²

Until quite recently, the name *Malassezia* was used to denote fungi with hyphal forms seen on the skin of patients with tinea versicolor, whereas the term *Pityrosporum* was used to denote the yeast forms seen in

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Pityrosporum folliculitis. However, a recent taxonomic study has shown that the hyphal and yeast forms are interconvertible and represent the same organism.³ In the new taxonomy, the genus *Malassezia* has incorporated the *Pityrosporum* species and now comprises 12 species: *Malassezia dermatitis*, *Malassezia equi*, *Malassezia furfur*, *Malassezia globosa*, *Malassezia japonica*, *Malassezia nana*, *Malassezia obtusa*, *Malassezia pachydermatis*, *Malassezia restricta*, *Malassezia slooffiae*, *Malassezia sympodialis*, and *Malassezia yamatoensis*.⁴⁻⁶ *Malassezia* species are classified based on morphology, enzymatic properties, and colony characteristics, in addition to use of molecular techniques such as polymerase chain reaction.⁷ In clinical practice, it is not usually necessary to speciate *Malassezia*, as treatment of most of the different species is the same.

MICROBIOLOGY

Malassezia is a dimorphic organism, at times assuming yeast forms and at times assuming hyphal (mycelial) forms. All species of *Malassezia* except *M pachydermatis* require lipid-rich environments, such as human skin or lipid-enriched culture media, as they are unable to synthesize medium-length saturated fatty acids. The lipid-dependent *Malassezia* require specialized media, such as Leeming and Notman agar, Dixon agar, or Littman Oxgall agar with olive oil, for culture. This lipid requirement is clinically important, because it determines where on the body these organisms are typically found (scalp, face, upper trunk). It also means that the organism will be missed in a routine fungal culture, because most fungal media do not contain the fatty acids essential for *Malassezia* to grow.

Malassezia species produce several compounds that cause altered skin pigmentation, leading to the pigmentary changes seen in tinea versicolor including azelaic acid decreases melanin production by inhibiting the melanocyte enzyme tyrosinase, which catalyzes the rate-limiting step in melanin production; malassezin induces apoptosis in melanocytes, reducing their numbers⁸; pityriacitrin is a yellow compound that has been shown to increase UV resistance in vivo and in vitro, preventing tanning of affected skin⁹; melanin-like pigments, which stain with Fontana-Masson silver stains, may cause hyperpigmentation.¹⁰

IMMUNOLOGY

The success of *Malassezia* as commensal organisms is in part due to their ability to evade the human immune system by causing localized immunosuppression. *Malassezia* induce keratinocytes to down-regulate the proinflammatory cytokines IL-1, IL-6, and tumor necrosis factor- α and to up-regulate IL-10. When cultured with

Malassezia, peripheral blood mononuclear cells respond with a similar pattern of immunosuppression. *Malassezia* also produce indole alkaloids called pityriarubins that inhibit the neutrophil respiratory burst and 5-lipoxygenase activity, leading to local immunosuppression.¹¹

SKIN DISTRIBUTION

The density of skin colonization with *Malassezia* depends on age, body site, and comorbid skin conditions, as well as the geographic area. Being lipophilic, *Malassezia* are found in the highest density in sebaceous areas: the scalp, face, and upper trunk. *Malassezia* are found in lower densities in children and older adults, who tend to have relatively low sebum production, and in higher densities in young adults, who tend to have relatively oily skin. There are geographical variations in the densities of different *Malassezia* species, which may be related to the overall heat and humidity of the climate.

Studies from Spain show that up to 70% of normal individuals carry *Malassezia* species on their trunk, whereas studies from Japan, the United Kingdom, and Canada found carriage rates of 40% to 80% of *Malassezia* on the scalp.¹² Thus, *Malassezia* must be considered part of the normal skin flora. They only cause skin disease when certain conditions such as overgrowth, descent into hair follicles, or inflammation are present.

CLINICAL DISEASES

Tinea Versicolor

Malassezia in their hyphal forms cause tinea versicolor, a superficial infection most commonly found on the upper trunk, neck, and upper arms. Tinea versicolor presents as hypo- or hyperpigmented, finely scaled, round or oval patches with distinct borders that may coalesce to form large patches (Figures 1 and 2). At times, the patches may even be erythematous (Figure 3) to salmon-colored. Some patients may experience pruritus, but most are asymptomatic. The presenting complaint is usually related to the distressing appearance of the hypo- or hyperpigmented patches.

Sweating and increased sebum production play a role in tinea versicolor. Consequently, tinea versicolor is more prevalent in warmer, tropical climates where it may affect over 30% of the population.¹³ Affected individuals in temperate climates often experience annual recurrences during the summer months. *M furfur*, *M globosa*, and *M sympodialis* are the most common *Malassezia* species isolated from cases of tinea versicolor.¹²

Tinea versicolor is diagnosed clinically, with seldom a need for biopsy or culture. Under a Wood lamp, tinea versicolor may fluoresce yellow. Gently scraping or stretching a hypo- or hyperpigmented patch of tinea

versicolor will demonstrate the “evoked scale sign” with the production of fine scale. Scraping may be done with a 15 blade or a glass slide held perpendicular to the skin.¹⁴ Historically, the appearance of *Malassezia* on potassium hydroxide preparation of a skin scraping has been described as “spaghetti and meatballs.” However, these authors find the short hyphal forms and tiny spores seen in tinea versicolor more akin to penne pasta with peas (Figure 4). Though rarely biopsied, tinea versicolor shows spores and short hyphae in the stratum corneum that stain positive with periodic acid–Schiff stain or methenamine silver stains, in the setting of an otherwise normal epidermis without inflammatory infiltrate.

The differential diagnosis of tinea versicolor includes pityriasis rosea, vitiligo, hypopigmented mycosis fungoides, erythrasma, pityriasis alba, and seborrheic dermatitis.

Tinea versicolor is treated with topical antifungal preparations, such as creams or washes, and occasionally, oral antifungals (Table 1). In general, topical antifungal agents should be used 1 or 2 times a day for 2 weeks. These modalities also may be used as maintenance regimens, because the condition is frequently recurrent. When oral ketoconazole is used, it is recommended to have the patient exercise to the point of sweating one hour after taking the drug in order to facilitate delivery of the drug to the skin surface. Oral terbinafine is not recommended for tinea versicolor as many *Malassezia* isolates are not susceptible (only *M pachydermatis* and *M sympodialis* have shown sensitivity in vitro), and terbinafine is not delivered efficiently to the skin surface.¹⁵

It is important to counsel patients that the hypo- or hyperpigmentation seen with tinea versicolor may persist for weeks to months, despite adequate treatment with antifungal agents, as the skin takes time to repigment even after the yeasts have been killed. Patients with hypopigmented tinea versicolor should be counseled to practice good sun protection with sunscreens and clothing, because tanning of their unaffected skin will tend to make their hypopigmented lesional skin more apparent by contrast.

Because tinea versicolor tends to recur in susceptible individuals, it also is important to counsel patients to continue maintenance treatment with antifungal washes or creams 1 or 2 times each week, especially during the summer in temperate climates; oral ketoconazole, itraconazole, or fluconazole also may be used prophylactically on a monthly basis.^{16,17}

Pityrosporum (Malassezia) Folliculitis

When *Malassezia* grow within hair follicles and cause inflammation, *Pityrosporum* folliculitis, may result.



Figure 1. Hypopigmented case of tinea versicolor: slightly scaly hypopigmented macules and patches have coalesced on the shoulder and upper trunk.



Figure 2. Hyperpigmented case of tinea versicolor: tan macules and patches are symmetrically located over the upper back.



Figure 3. Erythematous case of tinea versicolor.

TABLE 1

Treatment of Tinea Versicolor

| Shampoos and Washes | Topical Anti-fungal Agents | Oral Anti-fungal Agents | Maintenance Regimens |
|--|---|-------------------------------------|--|
| Ketoconazole 2% shampoo applied daily for 5 min for 3 d | Ketoconazole lotion 2% daily | Ketoconazole 400 mg weekly for 2 wk | Ketoconazole cream 2% once weekly |
| Selenium sulfide 2.5% lotion applied daily for 10 min for 10 d | Econazole nitrate cream 1% daily | Fluconazole 300 mg weekly for 2 wk | Ketoconazole shampoo 2% 2 to 3 times weekly |
| | Clotrimazole cream 1% daily | Itraconazole 200 mg daily for 7 d | Selenium sulfide lotion 2.5% applied as wash to scalp and body once weekly |
| | Miconazole cream 2% once or twice daily | | Itraconazole 400 mg PO once monthly |
| | Ciclopiroxolamine solution 1% daily | | Fluconazole 200 mg PO once monthly |
| | Terbinafine cream 1% twice daily | | |

Abbreviation: PO, by mouth.

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Figure 4. Potassium hydroxide preparation of scale from tinea versicolor stained with chlorazol black E (original magnification $\times 40$) shows short hyphae and spores.

Pityrosporum folliculitis presents as chronic, pruritic monomorphic, follicular-based papules and pustules on the upper trunk, neck, and arms (Figures 5 and 6). Risk factors for *Pityrosporum* folliculitis include

immunosuppression (eg, human immunodeficiency virus, organ transplantation, diabetes mellitus) and the recent use of broad-spectrum antibiotics or prednisone.

The diagnosis of *Pityrosporum* folliculitis is based on clinical presentation, microscopy, and a patient's response to antifungal therapy. On microscopy, a scraping of the pustule will show budding yeast forms and spores, rather than the hyphae seen in tinea versicolor. Biopsy is usually not necessary to diagnose *Pityrosporum* folliculitis, but would show dilated follicular ostia with keratin-plugging, cellular debris, and a mixed inflammatory infiltrate. Within the follicle, budding yeast forms and spores may be seen. The differential diagnosis of *Pityrosporum* folliculitis includes bacterial folliculitis, acne vulgaris, and eosinophilic folliculitis.

In treating *Pityrosporum* folliculitis, it is advisable to use oral antifungal agents, as topical agents do not penetrate well into the hair follicles. Discontinuing oral antibiotics or other triggering medications, such as prednisone, also may be helpful. See Table 2 for recommended treatment options. Patients should be counseled about the high likelihood of recurrence of *Pityrosporum*

TABLE 2

Treatment of *Pityrosporum* (*Malassezia*) Folliculitis

Topical Creams and Lotions (recommended as adjunct therapy with oral antifungal agents)

Oral Antifungal Agents

Maintenance Regimens

| | | |
|----------------------------------|---|--|
| Ketoconazole lotion 2% daily | Ketoconazole 200 mg daily for 2–4 wk | Ketoconazole 400 mg orally once a week |
| Econazole nitrate cream 1% daily | Fluconazole 100–200 mg daily for 2–3 wk | Ketoconazole shampoo 2% 2–3 times weekly |
| Clotrimazole cream 1% daily | Itraconazole 200 mg daily for 1 wk | Selenium sulfide lotion 2.5% used as a shampoo and body wash once weekly |
| | | Itraconazole 400 mg once a month |
| | | Fluconazole 200 mg once a month |

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Figure 5. *Pityrosporum* folliculitis: monomorphic follicular papules and pustules on the chest.



Figure 6. *Pityrosporum* folliculitis: inflammatory follicular pustules on the shoulder of a patient on corticosteroids (note striae present).

folliculitis and the necessity of continued prophylactic antifungal washes.

Seborrheic Dermatitis

Seborrheic dermatitis is a very common, chronic, unsightly eruption presenting as erythematous, ill-defined scaly, greasy patches on the scalp, face, and upper trunk (Figure 7). On the face, seborrheic dermatitis presents as erythema and scale most often of the eyebrows, glabella, nose, and paranasal folds. Increased incidence of

seborrheic dermatitis is seen in immunocompromised and neurologically impaired patients.

Malassezia are thought to play a role in seborrheic dermatitis by metabolizing triglycerides found in sebum into glycerol and free fatty acids, which can cause inflammation and scaling.¹⁸ Scalp swabs from patients with seborrheic dermatitis show higher levels of *Malassezia* colonization, primarily of *M restricta* and *M globosa*, than swabs taken from healthy controls.¹⁹ Treating seborrheic dermatitis patients with antifungal washes



Figure 7. Patient with oily skin and seborrheic dermatitis. Scale and erythema are evident at the paranasal folds.

and oral agents has shown to improve the dermatitis with improvement correlating with decreased numbers of *Malassezia*.^{20,21}

Seborrheic dermatitis is treated with topical antifungal agents and antifungal washes (Table 3). In addition, low potency topical corticosteroids, calcineurin inhibitors, and sulfur preparations are useful for their anti-inflammatory properties. In rare cases oral antifungal agents are necessary. Because seborrheic dermatitis tends to be a very chronic condition, it is important to emphasize that the patient continue with maintenance or prophylaxis regimens consisting of antifungal shampoos and body washes in combination with topical antifungal creams (Table 3).

Psoriasis

Psoriasis is a T cell-mediated systemic disease manifesting in the skin as erythematous, scaly plaques with a predilection for the extensor extremities, umbilicus, gluteal cleft, and scalp (Figure 8). Scalp psoriasis can be quite itchy and cosmetically distressing for patients, with scaling of the scalp producing copious dandruff.

Although *Malassezia* are not likely to play a pathogenic role in plaque-type psoriasis occurring in nonsebaceous areas, several lines of evidence suggest a role for *Malassezia* in psoriasis of the head and neck: increased levels of *Malassezia* colonization on the scalp, ears,

and face of patients with psoriasis²²; increased levels of inflammatory cytokines and proteins in samples of psoriasis colonized by *Malassezia*²³; increased circulating antibodies to *Malassezia* proteins in psoriatics compared to unaffected controls²⁴; and efficacy of antifungal shampoos such as ketoconazole for scalp psoriasis.

Given this evidence, treatment of recalcitrant scalp psoriasis should include antifungal shampoos such as ketoconazole and may require oral antifungal agents in doses similar to those used for the treatment of *Pityrosporum* folliculitis (Table 2).

Atopic Dermatitis

Atopic dermatitis is a chronic inflammatory skin disorder related to decreased skin barrier functioning and abnormal immune regulation. Although generally presenting in children, who have low rates of *Malassezia* carriage, atopic dermatitis may persist into adulthood and may involve sebaceous areas such as the face and scalp. It is in these latter cases that *Malassezia* are proposed to play a role.

Immunologically, patients with atopic dermatitis appear to have a heightened response to *Malassezia* suggesting the yeast is active in the disease process. Patients with atopic dermatitis, particularly those with head and neck involvement, have higher rates of positive skin prick test for Type I hypersensitivity to *Malassezia* than do healthy controls and higher levels of immunoglobulin E (IgE) specific to *Malassezia* than to other fungi.^{25,26} Some studies have shown that oral ketoconazole and itraconazole or topical ciclopiroxolamine are more effective than placebo in treatment of atopic dermatitis of the head and neck.²⁷⁻²⁹

There is evidence, however, arguing against a role for *Malassezia* in atopic dermatitis, including the observation that children, who have the highest prevalence of atopic dermatitis, have the lowest rates of *Malassezia* carriage.³⁰ Even atopic adults have a lower carriage rate of *Malassezia* (56%) than patients with seborrheic dermatitis (88%) or normal controls (83%).³¹ Since elevated IgE levels are common in patients with atopic dermatitis, the observed increase in *Malassezia*-specific IgE levels may only reflect a generally heightened immune response. Studies of antifungal treatment of atopic dermatitis of the head and neck have shown conflicting results, with several blinded studies showing no improvement.³²

OTHER MALASSEZIA-RELATED CONDITIONS

Several other less common dermatological conditions may have links to *Malassezia* yeasts. Confluent and reticulated papillomatosis of Gougerot-Carteaud is an

TABLE 3

Treatment of Seborrheic Dermatitis

| Topical Antifungal Agents | Topical Corticosteroids and Calcineurin Inhibitors | Oral Antifungal Agents | Washes and Shampoos |
|---|---|---|---|
| Ketoconazole cream, gel, or foam 2% daily | Hydrocortisone cream 1% daily | Itraconazole 200 mg daily for 7 d, followed by 200 mg daily for the first 2 d of each month | Ketoconazole shampoo 2% used as a shampoo and face wash 2–3 times weekly |
| Econazole nitrate cream 1% daily | Desonide cream 0.05% daily | Ketoconazole 200 mg daily for 7 d, followed by 200 mg once weekly | Selenium sulfide lotion 1% or 2.5% used as a shampoo and face wash 2–3 times weekly |
| Clotrimazole cream 1% daily | Pimecrolimus cream 1% daily | | Zinc pyrithione shampoo 1% used 2–3 times weekly |
| Ciclopirox gel 0.77% twice daily | Hydrocortisone 1%/salicylic acid 2%/sulfur 3% cream | | Ciclopiroxolamine shampoo 1% or 1.5% twice weekly |

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Figure 8. Scalp psoriasis; very thick scaly plaques are concentrated on the postauricular scalp and, in this case, involve the ears and neck. Photograph courtesy of Dr. Megan Bernstein.



Figure 9. Confluent and reticulated papillomatosis; hyperpigmented plaques are confluent over the upper trunk. Photograph courtesy of Dr. Kenneth E. Greer.

idiopathic condition presenting as gray-to-brown papules and plaques that coalesce on the upper trunk, forming a peripheral reticulated pattern (Figure 9). *Malassezia* have been reported in some cases of this disorder, including

one case report of a family in which 3 teenaged siblings had concurrent tinea versicolor and confluent and reticulated papillomatosis.³³ Some patients have shown improvement with selenium sulfide washes.³⁴ However,

most studies have failed to document the presence of *Malassezia* in patients with confluent and reticulated papillomatosis or a notable response to antifungal agents.³⁵ This condition is now thought more likely to have a bacterial etiology,³⁶ as it reliably responds to minocycline, a medication with no efficacy against *Malassezia*.³⁴ Given the clinical similarities between the 2 conditions, it is possible that the cases of confluent and reticulated papillomatosis that responded to antifungal treatment actually represented tinea versicolor.

Malassezia pachydermatis, the one *Malassezia* species that is not lipophilic, is a zoophilic species commonly found on dogs, cats, and small mammals. On human skin, *M pachydermatis* may cause a granulomatous skin infection that presents as verrucous papules and plaques. On biopsy, the involved skin shows hyperkeratosis, acanthosis, a mixed dermal infiltrate, and follicular dilated, neutrophilic microabscesses, and includes multinucleated giant cells. In one case report of *M pachydermatis* infection that a dog owner acquired from her pet, the infection was resolved with itraconazole 200 mg per day for 2 weeks, saturated potassium iodide 10% 30 mL per day for 2 weeks, 200 mg fluconazole per day for 10 weeks, 400 mg oral ciprofloxacin per day for 3 weeks, and 5 courses of cryotherapy.³⁷

CONCLUSION

Although a part of normal skin flora, *Malassezia* have a pathologic role as the causative agents for tinea versicolor and *Pityrosporum* folliculitis and play a role in seborrheic dermatitis. *Malassezia* may exacerbate psoriasis and atopic dermatitis of the head and neck area as well. Treatment of tinea versicolor and *Pityrosporum* folliculitis requires topical and sometimes systemic antifungal agents. In seborrheic dermatitis, antifungal agents are used together with anti-inflammatory medications such as topical corticosteroids. Likewise, in psoriasis and atopic dermatitis of the head and neck, topical and, rarely, oral antifungals targeting *Malassezia* can play a useful role along with corticosteroids.

With the high recurrence rate of *Malassezia*-related diseases, patient education and maintenance regimens are key to minimizing the cosmetic impacts of this ever-present yeast.

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