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**Case 1:** This asymptomatic plaque on the left cheek of a 12-year-old girl was not responding to a cream that her physician had prescribed when the rash began. Is there a simple diagnostic test? Is there an effective topical therapy?

**Case 1: Tinea faciei** is a dermatophyte infection of the face that, in my experience, most often presents in childhood as misdiagnosed eczema that does not respond to topical corticosteroid therapy. As in eczema, the eruption is papulosquamous; however, the morphology and the failure to respond to topical steroids are the key elements in the diagnosis of this presentation. The morphologic features to look for are the active inflammatory border and the tendency to central clearing that results in annular and polycyclic configurations of the plaques. The borders are not only scaly, but often papulovesicular with crusting. Also, if the follicular involvement is prominent, the lesions may look "granulomatous." The lesions appear in small numbers and are unilateral in almost all presentations.

The most useful diagnostic test is the potassium hydroxide (KOH) preparation, which can be performed as an office procedure or in your local laboratory. Take a scraping from the inside edge of the advancing border or--if a blister is present--from the underside of a blister roof. I treat patients based on the KOH results because cultures of dermatophytes may not be available for weeks--a period during which most patients can be cured. The dermatophytes implicated in this infection are commonly zoophilic: direct skin contact with pets is the most common source of infection among affected patients in my practice. I can always imagine the gerbil, kitten, or puppy snuggling up to the cheeks of the infected child. Zoophilic dermatophytes produce the most inflammatory skin reactions but, on occasion, they will resolve spontaneously. The specific dermatophyte is often based on the regional prevalence of the dermatophytes themselves. I attempt to treat these infections topically with an antifungal cream. More often than not, however, there is significant follicular involvement that requires systemic antifungal therapy. If 3 weeks of topical therapy with either ciclopirox or terbinafine does not eradicate the infection, I add systemic therapy with terbinafine tablets for 4 weeks.

I also recommend questioning other members of the patient's family about scaly or itchy skin lesions that may have developed over the preceding weeks. One infected pet can affect an entire family.
Case 2: These 2 adolescents have a skin infection with the same organism. Their presentations are, however, unique. The varied colors explain the name of this condition. How do you decide whether systemic therapy will be indicated? Also, what advice will you give the patients about the chance of recurrent infection?

Case 2: Tinea versicolor, the "many colored fungus," is a superficial skin infection with the yeast Malassezia furfur. The infection most commonly affects persons who live or visit warm, moist climates. In southern climates, up to half the population may be affected. In my northern climate, I often see this infection in patients who have returned from a winter vacation in a warm locale or who use tanning beds. Tanning in these beds requires the use of shared equipment: the heat and sweat produced during light exposure is the perfect environment for the transfer of the organism.

Tinea versicolor presents as slightly pruritic or asymptomatic scaling patches that coalesce to involve large areas of the skin surface of the trunk and extremities—particularly the upper arms and chest. A characteristic feature is the presence of smaller round lesions at the periphery of the larger patches, which you would predict to occur in a superficial skin infection. The term "versicolor" derives from the fact that the patches vary from red to brown to white.

Affected persons tend to have "one-color" infections. The diagnosis is confirmed by KOH examination, which shows multiple short hyphae and round yeast forms ("spaghetti and meatballs"). White patches can be clearly distinguished from vitiligo and post-inflammatory hypopigmentation if fine superficial scales are present when the patches are lightly scratched.

Persons probably carry M furfur as dictated by genetic susceptibility: infection develops when local environmental conditions (heat, humidity, change in skin surface lipids, or altered immunity) favor the development of the hyphal form of the yeast. The organism produces azaelic acid that inhibits normal pigment production: the result is hypopigmentation.

The treatment of tinea versicolor depends on the extent of the infection and patient preferences for topical versus systemic therapy. Topical therapy for extensive infection may be accomplished by the application of selenium sulfide (2.5%) or ketoconazole (2%) shampoos for 1 week. The shampoo is applied for 15 minutes each day and then showered off. Topical imidazole creams applied twice daily for 2 weeks will clear localized areas.

There are also many preparations that remove the stratum corneum that have proved effective. These include propylene glycol and salicylic acid (I refer you to the texts for the exact formulas). Systemic therapy with itraconazole (2.5 mg/kg/d for 7 days) is my favorite option whenever the involvement is extensive or multiply recurrent, when topical agents have failed, and when general medical conditions allow.

It is crucial to counsel patients that the condition is likely to recur, that it may take months for repigmentation to occur, and that applications of medicine beyond the prescribed duration will only
irritate the skin. I advise patients that they can consider re-treatment when they scratch the white patches and this brings up a fine scale that does not develop in the adjacent normal skin.

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