Students of psoriasis have struggled for centuries to distinguish the disease from its ‘mimics.’ With a wide range of clinical presentations, psoriasis may resemble many other dermatoses, inflammatory, infectious, and neoplastic. Characteristics that hint at the presence of psoriasis include family history, aspects of symmetry, distribution, silvery scaling, and nail changes. Some diseases, however, emulate the appearance of psoriasis so closely that a therapeutic trial of topical therapy may be necessary. If uncertainty persists, biopsy, culture, and selected laboratory tests may aid in confirming the presence or absence of psoriasis. Fortunately, with time, the vast majority of cases of psoriasis declare themselves, evolving into the classic form (Table 7).

**Table 7 Psoriasis mimickers with differentiating features.**

<table>
<thead>
<tr>
<th>Mimics</th>
<th>Features distinct from psoriasis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INFLAMMATORY</strong></td>
<td></td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>Flexural distribution, degree of erythema, ‘weeping’ lesions, lack of scale and lesser nail disease, significant pruritus, personal and/or family history of atopy</td>
</tr>
<tr>
<td>Dyshidrotic eczema</td>
<td>Vesicles with ‘tapioca’-like fluid, involvement of sides of fingers, toes and webs, significant pruritus</td>
</tr>
<tr>
<td>Nummular eczema</td>
<td>Lesions do not expand, less silvery scale and erythema, uniform size, distal leg a common site</td>
</tr>
<tr>
<td>Pityriasis rubra pilaris</td>
<td>Orange hue, follicular orientation, patches of normal skin among diseased skin ('islands of sparing'), less broad array of nail disease (no pitting, onycholysis, or ‘oil drop’), hyperkeratotic palms and soles (yellowish)</td>
</tr>
<tr>
<td>Pityriasis rosea</td>
<td>Presence of ‘herald patch,’ ‘Christmas tree’ distribution, collarette of scale, salmon color, self-limiting 2–3 months’ course</td>
</tr>
<tr>
<td><strong>INFECTIOUS</strong></td>
<td></td>
</tr>
<tr>
<td>Tinea capitis</td>
<td>Alopecia, broken hair shafts, possible adenopathy</td>
</tr>
<tr>
<td>Tinea corporis/tinea cruris</td>
<td>Active scaly border, central clearing, asymmetry</td>
</tr>
<tr>
<td>Tinea pedis</td>
<td>Involvement of lateral web space, unilaterality, vesicular, ‘moccasin’-type diffuse scaling</td>
</tr>
<tr>
<td>Tinea unguium</td>
<td>Asymmetry, predominantly toenails, lack of pitting</td>
</tr>
<tr>
<td>Candida</td>
<td>Peripheral pustules (‘satellite’ pustules), moist, whitish appearance, involvement of crural areas and finger webs</td>
</tr>
<tr>
<td>Candida of genitalia (‘balanitis’)</td>
<td>Pustules, diffuse erythema, erosions and fissures</td>
</tr>
<tr>
<td>Secondary syphilis</td>
<td>Early erythematous exanthem, copper-colored lesions (‘pennies’) on palms and soles, mucosal lesions, condyloma lata, history of chancre</td>
</tr>
<tr>
<td><strong>NEOPLASTIC</strong></td>
<td></td>
</tr>
<tr>
<td>Squamous cell carcinoma <em>in-situ</em> (Bowen’s disease)</td>
<td>Sun-exposed distribution, lesions few in number, gradual increase in size, resistant to treatment, potential progression to erosions</td>
</tr>
<tr>
<td>Cutaneous T-cell lymphoma (CTCL)</td>
<td>Often initially diagnosed as psoriasis; asymmetry, more irregular and fewer lesions, wrinkled appearance, ‘bathing-trunk’ distribution common, unresponsive to traditional topical therapies, potential progression to plaques, nodules and tumors</td>
</tr>
</tbody>
</table>
**INFLAMMATORY SKIN DISEASE**

**Eczema**

Eczema appears at the top of the list of dermatoses causing confusion with psoriasis. Although many forms exist, three types – atopic, dyshidrotic, and nummular – particularly resemble subtypes of psoriasis.

**Atopic dermatitis**

A multifactorial condition beginning in childhood, atopic dermatitis (AD) manifests in a variety of ways depending on age and severity. In children under 2 years, characteristic plaques with vibrant erythema and minor scale appear on the face and extensor surfaces of the limbs. In older patients, distribution becomes flexural and plaques thickened with exaggerated skin markings (lichenification), the result of chronic excoriation. Patients report a long personal and/or family history of asthma and allergic rhinitis, known as the atopic diathesis. Paramount among the subjective aspects of the disease, pruritus may be triggered by changes in temperature or humidity. Contact with irritants, such as water, or allergens may also exacerbate symptoms. Indeed, pruritus with resultant excoriation often incites characteristic lesions, leading to the lay description of atopic dermatitis as 'the itch that rashes.' Beyond flexural surfaces, other sites of involvement include periorbital and perioral areas, as well as the hands. Nails may be affected, although typically to a mild degree. Despite similarities with psoriasis, AD remains distinct. Key differentiating features of AD include characteristic history, severe pruritus, distribution of lesions, marked erythema, as well as paucity of scale and nail changes. Often lesions are ‘weepy’ or secondarily infected (impetiginized). Topical steroids...
and emollients are mainstays of treatment with topical immunomodulating agents and systemic antipruritics important additions. In severe cases, systemic corticosteroids, cyclosporin or, on occasion, even azathioprine and mycophenolate mofetil are utilized (184–191).

**Dyshidrotic eczema**
Dyshidrotic eczema, also known as pompholyx, causes symmetric, bilateral vesiculation of the hands and feet. One of the few lesions resembling palmoplantar pustular psoriasis, dyshidrotic vesicles evolve into punctate, scaly papules and even pustules. The ‘tapioca-like’ vesicular fluid, intense pruritus, and involvement of the finger webs and dorsal hands differentiate dyshidrosis from its psoriatic counterpart. Morbidity is great and treatment difficult (192–196).

**Nummular eczema**
A puzzling relative of atopic dermatitis, nummular eczema presents with disk-shaped, scaly plaques frequently on the extremities. Unlike psoriatic plaques, lesions of nummular eczema typically do not expand. Scale is also less exuberant and erythema less uniform. Like AD, nummular eczema may cause extreme pruritus, especially in the elderly (197–200).

**Eczema versus psoriasis**
184–187 Atopic dermatitis. Compared with psoriasis, lesions tend to be less vivid, well-defined, and scaly.
188–191 Psoriasis.
Pityriasis rosea
A self-limited, inflammatory disease possibly related to infection with human herpes virus 6 and 7, pityriasis rosea (PR) begins with a 2–10-cm, salmon-red plaque usually on the trunk, known as the ‘herald patch.’ The plaque precedes the eruption of smaller (1–2-cm) patches, papules, and thin plaques on the trunk and proximal extremities, developing 1–2 weeks later. Classically, lesions develop along skin cleavage lines and display a thin rim of scale. This ‘Christmas-tree’ distribution and ‘collarette’ of scale help to distinguish PR from guttate psoriasis. The eruption is usually self-limited, lasting 2–3 months (201, 202).

Eczema versus psoriasis
192, 193 Dyshidrotic eczema. Note the characteristic clear vesicles.
194–196 Palmoplantar pustular psoriasis with yellowish pustules.

Eczema versus psoriasis
197 Nummular eczema. Lesions are thin, moist, and lack silvery scale.
198–200 Small-plaque psoriasis.
Pityriasis rosea versus psoriasis

201 Pityriasis rosea. The peripheral rim of scale is a distinguishing feature.

202 Guttate psoriasis. Gentle scraping of the surface will elicit silvery scale.
Pityriasis rubra pilaris

203–209 Pityriasis rubra pilaris. Note the more yellow-orange hue and follicular appearance.

210–213 Psoriasis.

Pityriasis rubra pilaris

The scaly plaques of pityriasis rubra pilaris (PRP) strongly evoke psoriasis. Indeed, PRP was originally described as a psoriasis subtype. Decades later, dermatologists began to appreciate the distinct orange hue and follicular distribution of PRP. Waxy plaques affect the palms, soles, trunk, limbs, and scalp. Despite often extensive disease, patches of normal skin intermingle, known as ‘islands of sparing.’ Nails may demonstrate subungual debris, as with psoriasis, but lack pitting, oil drops, and onycholysis. In sum, distinguishing features of PRP include the follicular orientation and color of lesions, ‘islands of sparing,’ more acral distribution, and narrow range of nail changes. Additionally, PRP may be even more refractory to therapy than psoriasis (203–213).
INFECTIOUS DISORDERS

Dermatophyte infection

A diverse group of fungi, the dermatophytes, also termed 'tinea,' infect keratinized tissues of the skin, hair, and nails. While the clinical manifestations of dermatophytic infection range widely, many features mimic psoriasis. Infection of the skin and hair can lead to erythematosus, scaly plaques with an active border, typical of psoriasis. Nail involvement may present with subungual hyperkeratosis identical to psoriatic nail disease. The astute clinician, however, will note asymmetry of dermatophytic lesions, as well as subtle differences in the quality of the active border and central clearing.

Tinea capitis

A disease mainly of young children, tinea capitis evolves from infection of the scalp hair shaft by a dermatophyte, most commonly *Trichophyton tonsurans*. Of the various manifestations of tinea capitis, the noninflammatory subtypes are most often confused with psoriasis. Lesions appear circular with abundant scale and relatively sharp demarcation. Alopecia with broken hair shafts also hints at tinea. Regional lymphadenopathy may be present with more inflammatory forms. Wood’s lamp examination and microscopy of affected hairs is usually sufficient to clinch the diagnosis, with fungal cultures occasionally required. Oral, not topical, antifungals are the standard treatment (214–217).
Tinea corporis
Dermatophytoses of the skin subdivide according to anatomic location, with separate designations for the groin, feet, hands, and all other surfaces. Tinea corporis, for instance, refers to dermatophyte infection of any epidermal location other than the scalp, groin, feet, or hands. *Trichophyton rubrum* is the most common culprit. Circular or annular asymmetric plaques produce marked erythema and scale. As with other dermatophytoses, the border appears relatively more ‘active’ than the remainder of the lesion. Microscopic evaluation with the application of potassium hydroxide (KOH) to a collection of scale collected from the active border followed by gentle heating will reveal septate hyphae. Biopsy specimen treated with periodic-acid Schiff (PAS) may be more sensitive for the detection of fungi. Unlike hair and nail dermatophytosis, treatment with topical antifungals is normally adequate (218, 219).

Dermatophyte infection versus psoriasis
214, 215 Tinea capitis. Alopecia and minor scaling are clues to diagnosis
216, 217 Scalp psoriasis.
218 Tinea corporis. Note the active, scaly border.
219 Flexural psoriasis; minimal scale noted in this form.
Tinea cruris
Closely related tinea cruris differs from the corporal subtype in anatomic location (groin), relative paucity of scale except at the border, and propensity for confluence of plaques. As with tinea corporis, *T. rubrum* causes most cases\(^8\). The diagnostic approach and treatment options are similar (220–222).

Tinea pedis
Excessive moisture predisposes to dermatophyte infection of the foot, known as tinea pedis or ‘athlete’s foot.’ Several categories exist, however, the so-called ‘moccasin-type’ most closely resembles nonpustular, plantar psoriasis. Fine, whitish scale and leathery hyperkeratosis encasing the foot characterize the clinical appearance of this type of tinea pedis. Other manifestations of dermatophytosis may coexist, such as interdigital and bullous lesions, which distinguish the infection from psoriasis (223–226).

Tinea unguium
Tinea unguium refers to dermatophyte infection of the nail, also termed ‘onychomycosis.’ In its primary form, tinea unguium affects healthy nails causing discoloration and subungual hyperkeratosis similar to psoriatic nail disease. Alternatively, pre-existing nail damage, such as onycholysis in psoriatics, predisposes to secondary onychomycosis, leading the two conditions to often coexist. Patients with depressed immunity, such as those with diabetes and HIV/AIDS, are prone to infection, usually by *T. rubrum* or *T. mentagrophytes*. While often indistinguishable from nail psoriasis, tinea unguium may be differentiated by asymmetry, sparing of the fingernails, and lack of pitting. A KOH preparation of clippings from the nail bed may secure the diagnosis, but PAS staining of clippings and/or fungal culture of debris are often required (227–230).

Dermatophyte infection versus psoriasis
220 Tinea cruris in a psoriasis patient using topical corticosteroids.
221 Tinea cruris. The relative paucity of scale differentiates this from psoriasis.
222 Psoriasis of the groin.
223, 224 Tinea pedis. Note the asymmetry and web involvement.
225, 226 Psoriasis of the foot.
227, 228 Tinea unguium. Pitting is not observed.
229, 230 Nail psoriasis.
**Candida infection**

Another infectious psoriasis mimic, the yeast species *Candida albicans* affects warm, moist areas of the body, including axillae, inguinal folds, gluteal cleft, perineum, finger webs, angles of the mouth, and breast folds. Erythematous papules coalesce to form eroded plaques with characteristic peripheral pustules. These so-called ‘satellite’ pustules distinguish cutaneous candidiasis from psoriasis. A KOH preparation reveals budding yeast with ‘pseudohyphae,’ confirming the diagnosis. Keeping affected areas dry and use of topical antifungal agents are first-line treatments (231–237).

Candida also affects the nonkeratinized epithelium of the genitals, as does psoriasis. The term ‘balanitis’ refers to candidiasis of the prepuce and glans penis. Discrete or coalescent pustules develop on erythematous, often eroded, skin. Diffuse erythema and pustules suggest balanitis as opposed to psoriasis. Diagnosis is often made clinically (238, 239).

**Secondary syphilis**

With its multitude of expressions involving the skin and other organ systems, syphilis is known as ‘the great imitator.’ Secondary syphilis, developing in untreated patients 2–6 months after initial infection with the spirochete, may very closely resemble guttate psoriasis and pityriasis rosea. Interestingly, patients with early secondary syphilis develop a faint, erythematous exanthema prior to the guttate-like papular eruption. In the latter, copper-colored papules of various sizes up to 1 cm develop gradually over the trunk and limbs, ultimately involving the palms and soles. Lesions characteristically lack signs of inflammation, but scaling may be present. Mucosal lesions, especially on the mouth and genitalia, are frequent. A typical eruption in the setting of a positive screening, followed by treponemal antibody test confirms the diagnosis. Though rarely performed, dark field microscopy of samples from papular lesions will demonstrate spirochetes (240, 241).

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**Candida infection versus psoriasis**

231–233 Psoriasis.
234–237 Candidiasis. Note the discrete pustules at the periphery.
238 Balanitis. Note the moist, non-scaly quality of the lesion.
239 Psoriasis of the genitals.

**Secondary syphilis versus psoriasis**

240 Secondary syphilis. The characteristic copper color of the lesions distinguishes the eruption from psoriasis. Palms and soles are frequently involved.
241 Guttate psoriasis.
NEOPLASMS

Most ominous of the masquerade syndromes, neoplasms affecting the skin may cause scaly, erythematous plaques identical to psoriasis. Lesions may be so similar, in fact, that only resistance to treatment guides the clinician away from a diagnosis of chronic psoriasis and toward consideration of neoplasia. In such cases, histology aids in the diagnosis, although sequential biopsies may be required (see below).

**Squamous cell carcinoma in situ**

Well-circumscribed, isolated erythematous plaques with scale just as adequately describes the lesions of squamous cell carcinoma in situ (SCCIS), or Bowen’s disease, as psoriasis. Lesions favor areas of sun damage, a risk factor for Bowen’s disease, including the ears, scalp, lower lip, upper chest, back, and hands. Males appear more likely to develop SCCIS on the head and neck, whereas women the lower extremities. Other exposures increasing risk include human papilloma virus (HPV), arsenic, heating devices, and iatrogenic radiation. Treatment recalcitrance and/or progression to invasive disease suggest Bowen’s disease rather than psoriasis. Paucity of scale, which is less than silvery, as well as a dull background of erythema aid in distinguishing SCCIS from psoriasis (242–244).

**Cutaneous T-cell lymphoma**

A rare neoplasm of helper T cells, cutaneous T-cell lymphoma (CTCL) encompasses a variety of related conditions, including mycosis fungoides, lymphoma cutis, and Sézary syndrome. Erythematous patches may progress to plaques, which may slowly evolve into nodules or tumors and, in some cases, erythroderma. In this final stage, extensive hematological and even visceral involvement may be seen. The early, ‘patch’ stage of CTCL demonstrates erythematous plaques with mild scale similar to psoriasis or dermatophytoses, hence the term ‘mycosis.’ Indeed, patients with ‘patch’ stage CTCL often carry the diagnosis of psoriasis for many years. Further confusing the picture, these lesions may respond well to both topical corticosteroids and phototherapy initially, as with psoriasis. Biopsy specimens during the patch stage may not consistently demonstrate the characteristic epidermotropism (homing of T cells to the epidermis) and activated CD4+ lymphocytes. Consequently, serial biopsies with special studies, including FISH analysis, may be necessary to confirm the diagnosis.

**Squamous cell carcinoma versus psoriasis**

242, 243 Bowen’s disease. Lesions are isolated, well-circumscribed, eroded, and resistant to anti-psoriatic therapies.

244 Annular psoriasis.
such as T-cell receptor gene rearrangement and flow cytometry, are important for diagnosis.

Thus, the clinician must be wary of the isolated, psoriasiform plaque minimally responsive to topical therapy. If such a lesion raises suspicion for CTCL, initial work-up should include at minimum a full lymph node examination, biopsy of the lesion for routine histology and special studies mentioned above, and complete blood count with peripheral smear. Early lesions respond well to phototherapy, particularly psoralen with ultraviolet A (PUVA). Progression to Sézary syndrome, with generalized erythroderma, warrants consultation with a hematologist/oncologist for appropriate systemic therapy (245–249).