classically referred to as *impetigo contagiosa* and *impetigo bullosa*; they differ from each other simply by the size of the pustules. Over the past decade a remarkable shift in etiology has been observed. Whereas in the past impetigo contagiosa was almost exclusively caused by group A β-hemolytic streptococci and impetigo bullosa by *Staphylococcus aureus*, both are now usually caused by *Staphylococcus aureus*.

Pathogenesis. Bacterial species in the epidermis evoke an innate immune response that causes epidermal injury, leading to local serous exudate and formation of a scale crust (scab). The pathogenesis of blister formation in impetigo is related to bacterial production of a toxin that specifically cleaves desmoglein 1, the protein responsible for cell-to-cell adhesion within the uppermost epidermal layers. Recall that in pemphigus foliaceus, which has a similar plane of blister formation, desmoglein 1 is compromised not by a toxin but by an autoantibody (Fig. 25-28). Because there is virtually no involvement of the dermis, once the bacteria are eliminated the lesions heal without scarring.

MORPHOLOGY

Impetigo presents as an erythematous macule, but multiple small pustules rapidly supervene. As pustules break, shallow erosions form, covered with drying serum, giving the characteristic appearance of **honey-colored crust**. If the crust is not removed, new lesions form about the periphery and extensive epidermal damage may ensue. A bullous form of impetigo mainly occurs in children.

The characteristic microscopic feature of impetigo is **accumulation of neutrophils beneath the stratum corneum,** often producing a subcorneal pustule containing serum proteins and inflammatory cells. Special stains reveal the presence of bacteria in these foci. Nonspecific, reactive epidermal alterations and superficial dermal inflammation accompany these findings. Rupture of pustules releases serum, neutrophils, and cellular debris, which layer out and dry to form the characteristic crust.

Superficial Fungal Infections

As opposed to deep fungal infections of the skin, where the dermis or subcutis is primarily involved, superficial fungal infections of the skin are confined to the stratum corneum, and are caused primarily by dermatophytes. These organisms grow in the soil and on animals and produce a number of diverse lesions with characteristic distributions, as follows:

- *Tinea capitis* usually occurs in children and is only rarely seen in infants and adults. It is a dermatophytosis of the scalp characterized by asymptomatic, often hairless patches of skin associated with mild erythema, crust formation, and scaling.
- *Tinea barbae* is a dermatophyte infection of the beard area that affects adult men; it is a relatively uncommon disorder.
- *Tinea corporis*, on the other hand, is a common superficial fungal infection of the body surface that affects persons of all ages, but particularly children. Predisposing factors include excessive heat and humidity, exposure to infected animals, and chronic dermatophytosis of the feet or nails. The most common type of tinea corporis is an expanding, round, slightly erythematous plaque with an elevated scaling border (Fig. 25-40*A*).
- *Tinea cruris* occurs most frequently in the inguinal areas of obese men during warm weather. Heat, friction, and maceration all predispose to its development. The infection usually first appears on the upper inner thighs as moist, red patches with raised scaly borders.
- *Tinea pedis (athlete's foot)* affects 30% to 40% of the population at some time in their lives. There is diffuse erythema and scaling, often initially localized to the web spaces. Most of the inflammatory reaction, however, appears to be the result of bacterial superinfection and is not directly related to the primary dermatophytosis. Spread to (or primary infection of the nails) is referred to as *onychomycosis*. This produces discoloration, thickening, and deformity of the nail plate.
- *Tinea versicolor* usually occurs on the upper trunk and is highly distinctive in appearance. Caused by *Malassezia furfur* (a yeast, not a dermatophyte), the lesions consist

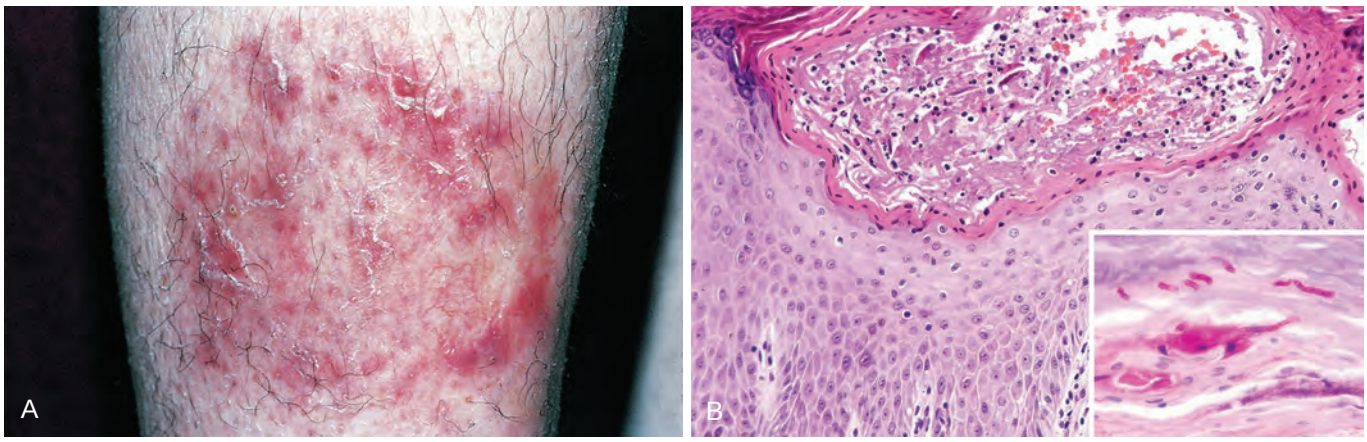

Figure 25-40 Tinea. **A,** Characteristic plaque of tinea corporis. **B,** Routine histology shows a mild eczematous (spongiotic) dermatitis and focal neutrophilic abscesses. A periodic acid–Schiff stain *(inset)* reveals deep red hyphae within the stratum corneum.