

Dermatomycoses and inflammation: The adaptive balance between growth, damage, and survival.

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Abstract

Dermatomycosis is characterized by both superficial and subcutaneous infections of keratinous tissues and mucous membranes caused by a variety of fungal agents, the two most common classes being dermatophytes and yeasts. Overall, the stepwise process of host infection is similar among the main dermatomycotic species; however, the species-specific ability to elicit a host reaction upon infection is distinct. Yeasts such as *Candida albicans* elicit a relatively low level of host tissue damage and inflammation during pathogenic infection, while dermatophytes may induce a higher level of tissue damage and inflammatory reaction. Both pathogens can, however, manipulate the host's immune response, ensuring survival and prolonging chronic infection. One common element of most dermatomycotic infections is the disease burden caused by inflammation and associated signs and symptoms, such as erythema, burning and pruritus. There is a strong clinical rationale for the addition of a topical corticosteroid agent to an effective antimycotic therapy, especially in patients who present with inflammatory dermatomycoses (e.g., tinea inguinalis). In this review, we aim to compare the pathogenesis of common dermatomycotic species, including *Candida* yeasts (*Candida albicans*), dermatophytes (*Trichophyton*,

Epidermophyton or Microsporum species), and other pathogenic yeasts (Malassezia), with a special focus on unique species-specific aspects of the respective infection processes, the interaction between essential aspects of pathogenic infection, the different roles of the host inflammatory response, and the clinical consequences of the infection-related tissue damage and inflammation. We hope that a broader understanding of the various mechanisms of dermatomycoses may contribute to more effective management of affected patients.

Involved units

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Leibniz-HKI-Authors



Bernhard Hube

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