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## Pathophysiology and treatment of psoriasis

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### Abstract

The pathogenesis and treatment of psoriasis are reviewed.

Psoriasis is characterized by defects in the normal cycle of epidermal development that lead to epidermal hyperproliferation, altered maturation of skin cells, and vascular changes and inflammation. The condition typically manifests as areas of thickened, flaky, silvery white and reddened skin that may hurt, itch, and bleed. Biochemical markers of psoriasis are changes in levels of keratins, keratinocyte transglutaminase, migration inhibitory factor-related protein, skin-derived antileukoproteinase, involucrin, small protein rich protein 2, filaggrin, and cytokines. Types of psoriasis that may be clinically encountered include plaque psoriasis, guttate psoriasis, erythrodermic psoriasis, and pustular psoriasis. Psoriasis is believed to be genetically linked but can also be triggered by mechanical, ultraviolet, and chemical injury; various infections; prescription drug use; psychological stress; smoking; and other factors. Topical treatment of psoriasis is usually the first line of therapy. Topical treatments consist of emollients and keratolytic agents, anthralin, coal tar, corticosteroids, vitamin D3 analogues, topical retinoids, and topical psoralens plus ultraviolet A (UVA) light. In patients who do not respond adequately to topical therapy, oral or injectable therapy, such as oral retinoids, methotrexate, cyclosporine, tacrolimus, and oral psoralens plus UVA light, may be warranted. Patients receiving systemic treatments should be carefully monitored for adverse effects and drug-drug interactions.

Drug therapy is the mainstay of the treatment of psoriasis. The potential adverse effects and interactions necessitate vigilant monitoring.

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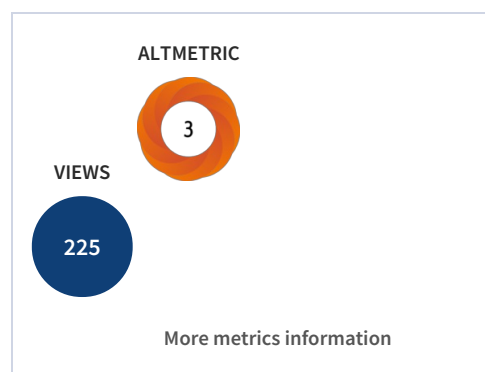
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