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How do I treat atopic dermatitis

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First of all, there is no consensus as to the diagnosis of atopic dermatitis. Many believe that it is a clinical diagnosis for which no testing is necessary. Others believe that it is essential to prove that the patient is truly atopic, by showing atopen specific IgE. If not present, a diagnosis of atopiform dermatitis should be made, and future avoidance of important allergens has no place.

Treatment of atopic dermatitis (AD) depends on its severity. First of all, patients need to be instructed about the necessity to keep the skin in a good condition, avoiding excessive water and soap exposure. Although atopic dermatitis is part of the atopic syndrome, avoidance of allergens does not seem to help. But on the other hand, as many patients have other problems being atopic, such as asthma and rhinitis, avoidance of so called aeroallergens should be advised. Also, it is believed that patients with AD have a dry skin, for which emollients are generally given. There are many modalities available for the active treatment of AD. Their use depends on age and severity. In some countries, guidelines have been developed.

Specific modalities are tar preparations and topical corticosteroids in various strengths. In severe cases, oral prednisone or cyclosporins have a place. Finally, the so called biologics are underway.

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Environmental factors transmitted by the aryl hydrocarbon receptor (AhR) influence the severity of psoriatic inflammation

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Psoriasis is a chronic inflammatory skin disease resulting from the interaction of genetic and environmental factors. Cross-talk between innate, adaptive and epithelial cells underpins the pathological response in this disease. More than 40 disease-associated loci have been identified to contribute to psoriasis. However, environmental risk factors remain less well-defined on a mechanistic basis. The aryl hydrocarbon receptor (AhR), a ligand-activated transcription factor that senses environmental stimuli, has been previously implicated at barrier tissue organ such as the gut as critical regulator of tissue-homeostasis. Here, the author shall present recent data showing how AhR modulates pathology in psoriasis. AhR-activating ligands reduced inflammation in the lesional skin of psoriasis patients, whereas AhR antagonists increased inflammation. Similarly, AhR signalling via the endogenous ligand FICZ reduced the inflammatory response in the Aldara-induced model of psoriasiforme skin inflammation and AhR deficient mice exhibited a substantial exacerbation of the disease, compared to AhR sufficient controls. Non-haematopoietic cells, in particular keratinocytes, were responsible for this hyper-inflammatory response, which involved increased reactivity to pro-inflammatory stimuli and upregulation of AP-1 family members of transcription factors. Thus, these data suggest a critical role for AhR in the regulation of inflammatory responses and open the possibility for novel therapeutic strategies in chronic inflammatory skin disorders.

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