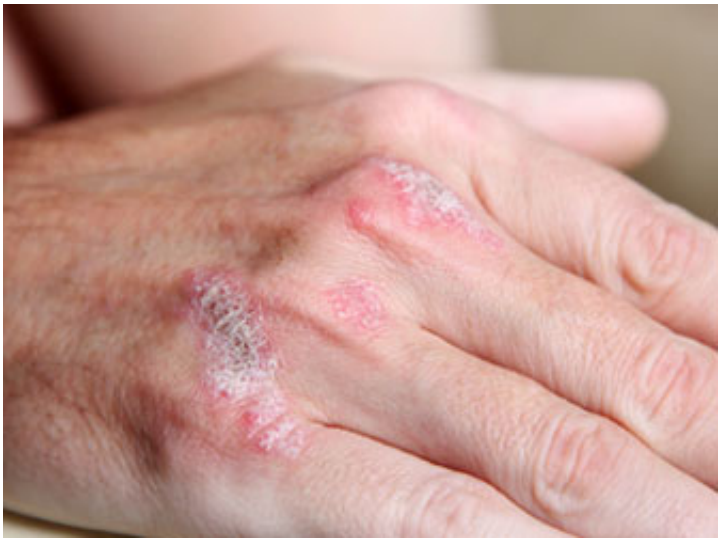
Medical Xpress

Dermatitis could be suppressed as it develops

November 9 2012



Excessive immune reactions against the body's own skin cells can lead to painful and even chronic dermatitis. An international team of researchers at the MedUni Vienna, the MedUni Graz and the Salk Institute in California, led by Herbert Strobl, has now unencrypted the mechanism that contributes towards this unwanted autoimmune reaction being suppressed. This may in future lead to common conditions such as chronic allergic dermatitis or psoriasis being halted as they develop and treated.

The human immune system generally has two tasks: it needs to be able to respond effectively to viruses and bacteria, and also prevent reactions

against the body's own cells. With every inflammation, but also during the natural regeneration process (renewal) of tissue, the body's own cells die. These dead cells represent a constant source of inflammation and are therefore eliminated by 'sentry cells' (macrophages and dendritic cells) which act as the body's own waste disposal system.

These sentry cells are also charged with detecting viruses and bacteria and, with the help of T-cells in the lymph nodes, with triggering a specific immune response in the form of inflammation against them. Says Strobl: "The sentry cells therefore carry out a constant balancing act between preventing inflammation caused by the body's own cells and triggering a desired inflammatory response to pathogens from outside the body."

In the study, which has now been published in the highly respected Journal of Experimental Medicine, the researchers from Vienna and Graz, in collaboration with colleagues from California, have been able to demonstrate that the sentry cells are programmed even during their development to detect the body's own dying cells, eliminate them and suppress any immune reaction to them.

They have also demonstrated that this mechanism is particularly important for the immune regulation of the skin, as well as the receptor responsible for this. The transmitter substance TGF-beta1 is synthesised in the epidermis and causes the presence of the Axl receptor on the cell surface of Langerhans cells (epidermal sentry cells) as well as on other skin cells. Says Strobl: "If this mechanism is literally switched off, dermatitis and excessive allergic reactions occur."

Cortisone is currently the most important drug for the treatment of dermatitis – and although it is used effectively for many skin conditions, there is a demand for alternatives. The new findings regarding the mechanisms behind the development of dermatitis could, says Strobl, in

future lead to the development of alternative medications that boost the receptor. This work has been financed by the Research Fund for Scientific Research in Austria (FWF).

More information: Bauer, T. et al., Identification of Axl as a downstream effector of TGF-β1 during Langerhans cell differentiation and epidermal homeostasis. *JEM* vol. 209 no. 11 2033-2047. doi: 10.1084/jem.20120493.

Provided by Medical University of Vienna