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How the skin of patients with psoriasis protects itself from virus infections

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Scientists at Charité – Universitätsmedizin Berlin have discovered why patients with psoriasis are less susceptible to viral infections than patients suffering from atopic dermatitis (atopic eczema). The reason for this is the larger quantity of special proteins present in psoriatic skin, which inhibit viral replication. The interdisciplinary team under the direction of Dr. Robert Sabat from the Department of Dermatology and the Institute of Medical Immunology, in collaboration with the Institute of Virology and the Berlin-Brandenburg Center for Regenerative Therapies (BCRT), has additionally discovered a new function of the immune system. The study is published in the current issue of the journal *Science Translational Medicine*.

Psoriasis and atopic dermatitis are the two most common chronic diseases of the skin, from which over 40 million people suffer in the US and in EU alone. They cause persistent visible changes of the skin that severely impact the quality of life of the patients. In addition, the damaged skin barrier enables pathogens, such as viruses, to penetrate the skin and multiply. Surprisingly, only atopic dermatitis patients show a high incidence of cutaneous viral infections, which exacerbate the course of atopic dermatitis and if not treated promptly, might be life-threatening. The authors of the just-published study show that compared to the skin of patients with psoriasis, the skin of atopic dermatitis patients produces smaller quantities of what are known as antiviral proteins, which inhibit viral replication. In the search for the trigger responsible for these differences in protein production in the two skin diseases, the research team came upon the immune messenger

interleukin-29.

"Of the thirty messengers produced by the immune cells that we examined in psoriatic skin, we only found a correlation with the quantities of antiviral proteins for interleukin-29," explains Dr. Kerstin Wolk from the Institute of Medical Immunology at the Charité, one of the study's two first authors. "In fact, interleukin-29 is present in psoriatic skin, but not in affected skin of atopic dermatitis patients." Removing this immune protein from skin samples taken from patients with psoriasis diminishes the quantity of antiviral proteins in these samples. Using experiments with healthy skin, skin models and isolated cells from the upper layer of the skin, the team additionally showed that interleukin-29 is able to stimulate the production of antiviral proteins, and thereby to protect the skin cells from viral infection.

Moreover, the researchers showed that interleukin-29 is produced by a specific population of immune cells known as Th17 cells. "These cells promote the production of antiviral proteins and thus, anti-viral defences in the skin cells," explains Dr. Katrin Witte from the Institute of Medical Immunology at the Charité, also a first author of the study. Thus, they simultaneously uncovered a new function of the immune system.

"It is conceivable that the therapeutic administration of interleukin-29 or substances that mimic its effects could increase patients' local defences against viruses. This would be applicable not only for atopic dermatitis, but also for other chronic inflammatory diseases that take place in epithelia, such as certain lung conditions in which viral infections represent a cofactor. This treatment could alleviate the course of such illnesses," states Dr. Robert Sabat, Head of the Psoriasis Research and Treatment Center at the Charité.

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