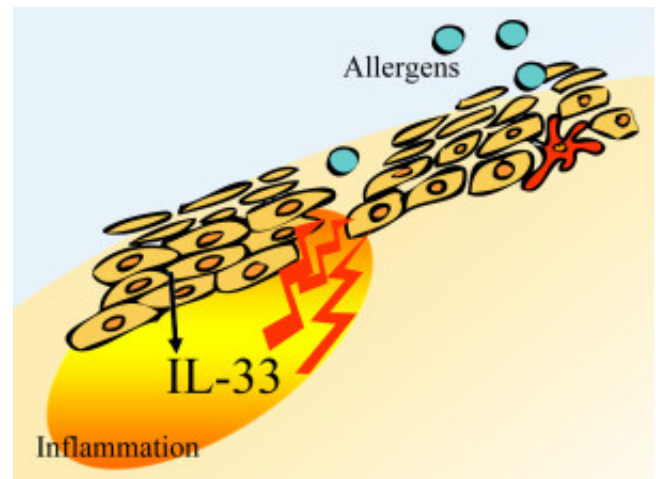


The inflamed skin in atopic eczema contributes to the disrupted skin-barrier

Atopic dermatitis, also known as atopic eczema or neurodermitis, is a chronic allergic skin disease affecting 15 - 30 % of children and 2 - 10 % of adults in industrialized countries. Patients suffer from inflamed, red and dry skin and strong itch. In many patients, the skin-barrier seems to be disturbed and an increased water-loss through the skin can be measured. Genetic mutations that lead to a weakened skin-barrier cannot explain this picture in total, since being detected in some, but not all patients.

In our research project, we were interested in the question, whether the ongoing skin inflammation leads to the weakened skin-barrier. Inflamed skin cells are able to produce a bouquet of small biologically active compounds. A recently discovered one has been termed Interleukin-33 (IL-33).



We found that when skin cells had contact to IL-33 in the petri dish, they intended to produce less of the protein filaggrin, also often referred to as *the skin's glue*. We observed the same when we applied IL-33 to human skin biopsies and looked for filaggrin: a strong reduction in this major skin-barrier protein became visible. To finally mimic the situation in the patient, where IL-33 most probably is present over days and weeks, we bred artificial skin in the petri dish with or without IL-33. While the artificial skin developed nicely under our test conditions, we found massive differences regarding the skin-barrier. First, the protein filaggrin was nearly absent, and second, the skin was much more permeable for a test allergen from peanut. This implies, that allergens may enter the inflamed skin easily, leading to an even stronger inflammation, creating a vicious circle.

Overall, with this study we highlight that the inflammation in atopic dermatitis may sustain itself and that an early intervention is beneficial. IL-33 may therefore represent an interesting target for new therapy strategies.

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Publication

[IL-33 impacts on the skin barrier by downregulating the expression of filaggrin.](#)

Seltmann J, Roesner LM, von Hesler FW, Wittmann M, Werfel T.

J Allergy Clin Immunol. 2015 Jun