

Effect of atopic skin stressors on NMFs and skin cytokines

K.A. Engebretsen, S. Kezic, I. Jakasa, A. Hedengran, A. Linneberg, L. Skov, J.D. Johansen and J.P. Thyssen

This summary relates to https://doi.org/10.1111/bjd.16487

British Journal of Dermatology, 179, 679-688, September 2018

Summary

Atopic Dermatitis (AD) is a common type of eczema. There is often a greater risk of getting AD by having a deficiency of NMFs (natural moisturising factors) that come from filaggrin in the skin. Filaggrin is a protein in your skin that plays a vital role in the skin barrier function and in releasing acids that help in water retention. While it is now known that AD is caused by the gene mutation of filaggrin, there has been little research done into environmental factors that can cause a deficiency of filaggrin in the body. A Danish study funded by the Lundbeck Foundation has attempted to discover what types of environmental skin stressors can impact on levels of NMF and cytokines. Cytokines are cell molecules that stimulate the movement of cells towards inflammation, infection and trauma. In the study, 40 healthy volunteers (18-49) were exposed to hard, soft and chlorinated water, Sodium Lauryl Sulfate (SLS a chemical found in foaming products such as shampoo), house mite dust, cat allergen, Staphylococcal enterotoxin B (SEB - a protein commonly associated with food poisoning), cooling and histamine (a chemical created by the immune system). The volunteers were tape stripped (this means applying tape to the skin and then removing it and looking at the tape under a microscope) and the biological changes recorded after 24 and 48 hours for NMFs and after 24 hours for cytokines. At 24 hours, a significant decrease of NMFs was recorded for hard and soft waters, house dust mites and SEBs with an increase in cytokines compared with control tests. The study concluded that exposure to skin stressors causes NMF levels to decrease, with an additional increase in various types of cytokines, in healthy people. This data highlights that environmental data might play a role in AD pathophysiology.