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M. Jargosch, Madhumita Bhattacharyya, Claudia Traidl-Hoffmann, Matthias Reiger, F. Lauffer, N. Garzorz-Stark

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Staphylococcus hominis is beneficial for skin architecture, barrier and inflammation in an atopic dermatitis model with reconstructed human epidermis

M Jargosch[1], M Bhattacharyya[2], C Traidl-Hoffmann[2], M Reiger[2], F Lauffer[1] and N Garzorz-Stark[1] *1 Department of Dermatology and Allergy, Technical University of Munich, Munich, Germany and 2 Institute of Environmental Medicine, UNIKA-T, Technical University of Munich and Helmholtz Zentrum Munich, Augsburg, Germany*

Commensal skin microbes are essential components of the epidermal barrier. However, an imbalance of skin microbes is associated with inflammatory skin diseases such as atopic dermatitis (AD). In AD, *Staphylococcus (S.) aureus* is highly abundant and knowingly contributes to disease pathology. In contrast, *S. hominis* is more abundant in non-lesional and healthy skin than in AD, suggesting a beneficial role. To investigate the effects of *S. hominis* on skin barrier, reconstructed human epidermis (RHE) was inoculated with *S. hominis* under type 2 inflammatory conditions, using T cell supernatant (TCS) isolated from T cells derived from AD skin biopsies. AD-TCS led to thickening of the epidermal layer, induction of spongiosis, and upregulation of inflammatory genes (e.g. *CCL2*, *S100A7*, *SERPINB3*). These effects were reversed by *S. hominis* inoculation in a dose-dependent manner. Furthermore, expression of genes related to physiological epidermal differentiation (e.g. *KRT10*, *COL1A1*, *DSC1*, *MMP2*) increased after *S. hominis* inoculation, indicating positive effects on barrier formation. These findings were validated in AD patients, where the trans-epidermal water loss − a marker for epidermal barrier damage − correlated negatively with *S. hominis* abundance. In conclusion, *S. hominis* normalized type 2 related changes of the epidermal architecture, barrier and inflammation in AD. Therefore, skin treatment with *S. hominis* may represent a new potential therapeutic strategy. Moreover, established AD-RHE models allow the analysis of interactions between microbes, keratinocytes, and T cells and can contribute to study the effect of microbes on disease pathology.