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Skin barrier function genes differentially correlated to resident Staphylococci in lesional and non-lesional skin in atopic eczema

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Host-microbe interaction plays a critical role in the pathogenesis of atopic eczema (AE). It is unclear if changes in the microbiota affect the host skin barrier function, or vice versa, and how they together influence AE. We studied differences in the microbiome of lesional and neighboring non-lesional skin in AE patients and correlated them to changes in epidermal barrier gene expression in RNA sequencing transcriptome. The microbiome in skin swab samples from AE patients (N=14, lesional and adjacent non-lesional site) and 7 healthy controls was sequenced using amplicon based 16S analyses (V1 to V3). The transcriptome was assessed by RNA sequencing from punch biopsies taken at the same sites as the microbiome swabs. The frequency of several taxonomic units of *Staphylococcus aureus* is significantly higher in AE lesional samples, whereas in the AE non-lesional and healthy skin *Staphylococcus epidermidis* is the dominating species. In the transcriptome analysis the majority of tight junction genes show a significant downregulation in AE lesional skin. Linking the microbiome and transcriptome we found a significant negative correlation between the abundance of *S. aureus* with the expression of a number of tight junction genes. On the other hand, a positive correlation between the abundance of *S. epidermidis* and the expression of the same genes was observed. Our results show that different Staphylococci have opposite relationships to tight junction gene expression. Bioinformatic analysis allowed us to identify several groups of barrier function-related genes that differently correlated to the microbiome, potentially indicating influences on lesion development in AE.