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Evidence for a restoration of TLR2 response in epidermal dendritic cells in atopic dermatitis by topical anti-inflammatory therapy

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To the Editor,

Patients suffering from atopic dermatitis (AD) show an attenuated immune response and imbalanced microbiome environment that favors *Staphylococcus aureus* (*S. aureus*).[1] Bacterial products are recognized via Toll-like receptors (TLR) of the immune system leading to maturation of dendritic cells (DC).[2] Recently, we showed that Langerhans cells (LC), DC in the epidermis, from ex vivo AD skin had an inhibited maturation response toward TLR2 stimulation when compared to healthy controls (HC).[3]

Topical anti-inflammatory treatment of AD reduces inflammation and the colonization with *S. aureus*.[4] To test its impact on the TLR2 expression and function on epidermal DC, we analyzed DC freshly isolated from lesional skin of treated (topical steroids or tacrolimus for 2 days [median]) and untreated AD patients as well as HC (for methods see Appendix S1).

LC of HC showed elevated TLR2 and similar CD83 and MHCII expression compared to AD, confirming our previous results (Figure 1). CD83 expression was lower in treated AD. CD80 expression was significantly higher in AD, as described,[5] but independent of the treatment.

アトピー性皮膚炎の皮膚において、樹状細胞のTLR2応答と治療の関連について、Flow cytometryを用いて解析を行いました。