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Immune mediated skin inflammation is the same in atopic dermatitis patients with or without filaggrin mutation

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Much evidence proved that inflammatory cytokines can impair skin barrier. The opposite question whether skin barrier alterations can affect keratinocyte (KC) immune responses, remained less studied. We sought to investigate if the immune mediated skin inflammation, KC function, T and dendritic cell (DC) count differ between severe atopic dermatitis (AD) patients with or without filaggrin (FLG) mutation.

Lesional skin biopsies of severe FLG mutant and wild type (Wt) AD patients and controls were analyzed by immunohistochemistry. FLG and inflammatory T helper (Th)2 polarising cytokines (TSLP, IL-33) and CCL27 chemokine, characteristically expressed by KCs in lesional AD skin, were immunostained. Histological severity markers (Ki67 positivity and epidermal thickness) and CD3+ T cell and CD11c+ DC count were also investigated. Slides were digitalized, which allowed us to compare protein levels. Correlations were calculated.

Compared to controls, in both AD groups, FLG levels were found significantly lower, whilst the same level of FLG expression was measured in the AD groups. Concerning the levels of the investigated parameters no significant differences were found between the two AD groups. The expression of AD specific cytokines and chemokine showed significant correlation with histological severity markers.

Our findings suggest that the immune mediated skin inflammation in FLG mutant and Wt AD patients is mediated by the same amount of immune cells and KC derived cytokines and chemokines. Results of the correlations demonstrated that KC's immune activation is connected rather to the severity of the disease, then to the origin of barrier alterations.