

## Crosstalk of keratinocytes and T cells during eczematous skin reactions [Abstract]

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P001

### **Crosstalk of keratinocytes and T cells during eczematous skin reactions**

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Background: Allergic contact dermatitis (ACD) and atopic eczema (AE) are eczematous skin diseases in which T cells are directly involved in the induction of keratinocytes' death. However, almost nothing is known how keratinocytes influence T cell activity in the course of allergic diseases.

Objective: The aim of this study was to investigate the outcome of keratinocyte-T cell interactions with the focus on T cell functions such as proliferation and cytokine release in an antigen-specific in vitro model of ACD to Nickel and acute AE to Phleum pratense.

Methods: T cell clones were generated of Phleum pratense sensitised ( $n = 2$ ) or non-atopic Nickel-sensitised ( $n = 5$ ) patients. Autologous monocyte-derived dendritic cells (DC) or EBV-transfected B cells (EBV-B) served as antigen-presenting cells (APC). Autologous primary human keratinocytes were generated by the method of suction blister. Crosstalk of keratinocytes and T cells was analysed by cocubation of keratinocytes, T cells and APC with the endpoints T cell proliferation and cytokine production.

Results: Cocubation of keratinocytes, T cells and APC revealed that keratinocytes variably influence the proliferation of T cells with 77% being blocked and 23% being induced in antigen-specific proliferation capacity. Notably, production of the cytokines IL-4 and IL-10 was regulated independently from the proliferation showing always a reduction in IL-4 and IL-10 release. IFN $\gamma$  was not constantly regulated. Cocubation experiments with Phleum pratense-specific T cell clones show similar results with T cells being regulated in proliferation and cytokine release by keratinocytes. Differentiation and preincubation of keratinocytes with IFN $\gamma$  did not alter the effect on T cells. However, fixation with paraformaldehyde (PFA) abrogates this immune-modulating effect.

Conclusion: Keratinocytes influence actively the effector phase of eczematous reactions. The immune-modulating effect on T cell cytokine production seems to be modified by soluble factors. Suppression of IL-4 and IL-10 in T cells could lead to a Th1 microclimate in the skin favouring the maintenance and chronification of eczematous skin reactions. Thus, keratinocytes seem to be not only the defenceless victim of T cells in eczematous reactions but a modulator of T cell functions.