Introduction: Atopic dermatitis (AD) is a chronic and multifactorial inflammatory skin disease, characterized by an altered epidermal barrier and abnormal immune responses. AD immune response involves various dendritic cells such as epidermal Langerhans cells (LC) and inflammatory dendritic epidermal cells (IDEC). Most of clinical studies had been performed on isolated cells, thus it is essential to characterize the cellular events on human epidermal tissue.

Objective: We thus propose to study the dynamic behavior of IDEC and LC, in situ, keeping the human epidermis whole and directly following the atopy patch test (APT) removing

Materials and Methods: The study included fifteen AD subjects who exhibited a positive atopy patch test reaction (dust mite allergen Dermatophagoides pteronyssinus, Derp) according to the criteria of the International Contact Dermatitis Research Group (ICDRG). We analyzed the dynamic behavior of inflammatory cells by using APT as a model for early AD reaction. The suction blister method was used in order to obtain whole epidermis samples. Employing immunostaining and 3D-multiphoton microscopy, high-resolution images of epidermal sheets were analyzed to precisely determine the early dynamic responses of inflammatory cells after allergen exposure.

Results: Derp-APT application provoked rapid and strong infiltration of IDECs (CD206+ cells p=0.0065), and proliferation and activation of LC in the AD subjects' epidermis. Moreover, emollient pre-application (twice a day for 8 days) strengthened the defective skin barrier and had positive effects on inflammatory cells' behavior, characterized by the complete inhibition of IDEC influx and the presence of immature LC.

Conclusions: This study highlights two points: first, the early key role of human dendritic cells, LC and IDEC, in post-APT inflammatory reaction, and second, it demonstrates the
preventive effects of emollient application in this cellular context. This 3D multiphoton epidermal cell visualization contributes to a better understanding of AD pathophysiology.