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neutrophils, eosinophils and peripheral blood Ils (PBMCs) in bullous pemphigoid (BP) PKI Luca Borradori¹, Shida Yousefi¹, Hans-Uwe Simon¹ and Dagmar Simon¹

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ng disease iuto-antibodies to type XVII blister formation ny infiltrate. npletely

Methods: In an ex vivo skin model (Sitaru et al. 2002) cryosections are incubated with purified granulocytes and/or PBMCs with or without (wo) prior stimulation, in the presence absence of BP antibodies (BPS). DES is assessed by light

mechanisms.

ler which conditions æ dermal-epidermal

DES

Conclusion: These preliminary results show that neutron are able to induce DES either in the presence of BP antibose upon activation by cytokines and complement factors. Eosinophils are not able to induce DES solely in the presence Eosimophils are not able to induce DES solely in the presence of BP antibodies. PBMCs, and more specific monocytes, are able induce DES upon BPS. Synergistic induction of DES by monocytes and neutrophils is observed. These observations

suggest an active role of these immune cells in skin blister

formation. Further investigations are needed to elucidate the

IV. PBMCs, mainly monocytes, induc

TI

th BPS de

















urine neutrophils downstr d in response to danger s

W. Wei-Lynn Wong³, Mads-Gyrd Hansen⁴, Daniel Bac

the intersection of inflammation and cell death in gene -7 and, to lesser extents, caspase-9 XIA phenotype mediated by the receptor interact minute signaling such as inhibition of the interaction interactivity.

































