

**P002** Neuro-immune interaction in allergic asthma: The role of the low-affinity pan-neurotrophin receptor p75NTR  
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Blocking of p75NTR mediated signals in a mouse model of asthma reduced allergic inflammation (AI) and inhibited the development of a sensory nerve hyperreactivity (Kerzel et al., AJRCMB 2003). However, it remains unclear if neurotrophins are directly involved in the development of airway inflammation via p75NTR expressed on immune cells, or indirectly by induction of neuropeptide release from hyperreactive sensory nerves (neurogenic inflammation). In order to identify the underlying mechanism, p75NTR KO were reconstituted with WT bone marrow (BM) or p75NTR KO BM, respectively, and WT mice were reconstituted with p75NTR KO BM or WT BM, respectively. After systemic OVA sensitization followed by OVA aerosol exposure, bronchoalveolar lavage (BAL) was performed. Differential cell count and cytokine levels of the BAL revealed that recipients of WT BM developed an enhanced AI compared to the recipients of BM from p75NTR KO mice. This effect was independent from p75NTR expression in sensory nerves and the sensory nerve hyperreactivity. The development of a sensory nerve hyperreactivity was restricted to recipients of the WT genotype, but independent from p75NTR expression in bone-marrow derived immune cells and the development of an AI. Our results therefore indicate that p75NTR signalling in sensory neurons do not contribute to the development of AI, and neurotrophins may be directly involved in the development of AI via p75NTR expressed on immune cells.