

POSTER PRESENTATION

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Modulation of IL-17A and TL1A largely abrogates house dust mite-induced lung inflammation in murine model of allergic airway disease

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Recent studies suggest a role for Th17 responses in the increased airway neutrophilia associated with severe asthma. House dust mite (HDM) is a natural allergen to which asthmatics are often sensitized. Mice repeatedly challenged with HDM extract developed robust airway neutrophilia rapidly evolving into asthma-like disease with increased numbers of eosinophils and lymphocytes in bronchoalveolar lavages (BAL) as well as inflammatory infiltrates, vascular/muscular hypertrophy, interstitial fibrosis, epithelial hyperplasia and mucus accumulation in lung tissues. RNA and protein screening revealed a robust Th17 component post-HDM exposure. We thus evaluated whether IL-17A deficiency could modulate HDM-induced allergic airway disease. Airway neutrophilia was indeed abrogated in IL-17A deficient mice weekly challenged with HDM (acute model), however total BAL cellularity and lung mechanics remained comparable to those of HDM-challenged WT mice. In contrast, IL-17A deficient mice daily exposed to HDM (chronic model) had decreased BAL cellularity associated with reduced numbers of BAL macrophages, neutrophils, eosinophils and lymphocytes. Interestingly antibody neutralization of TL1A, a member of the TNF superfamily known to promote Th2 and Th17 responses, reduced BAL cellularity to baseline levels in HDM-challenged WT mice. Our results thus indicate that targeting Th17 responses can alleviate HDM-induced airway neutrophilia, and can also broadly modulate allergic airway disease.

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