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Abstract

Diisocyanate exposure is a leading causes of work-related respiratory allergies including occupational rhinitis and asthma. We have shown that inhalation of toluene diisocyanate (TDI) caused allergic rhinitis in mice with marked upregulation of interleukin (IL)-5 in the nasal mucosa. The present study utilized antibody-mediated neutralization of IL-5 to test the hypothesis that IL-5 plays a key role in allergic and/or eosinophilic inflammation in TDI-induced rhinitis. Mice were exposed to 50 ppb TDI vapor for 4 h/day for 12 weekdays. Gene expression was determined using Illumina whole-genome microarrays and Taqman PCR. Gene Ontology and network analysis confirmed that IL-5 was a key cytokine in many of the upregulated immune networks. In addition, TDI inhalation upregulated Th2 cytokine (IL-4, -5, -13, -10) and chemokine (Ccl11, Ccl24) expression and eosinophil infiltration into the nasal mucosa of mice with TDI rhinitis. The expression of the eosinophil genes eosinophil peroxidase (Epx) and siglec F were upregulated (7.7 fold, P=0.001 and 27.5 fold, P=0.001, respectively) in the nasal mucosa of mice with TDI rhinitis supporting the presence of eosinophilic inflammation. Neutralization of IL-5 did not affect the development of the cytokine/chemokine response driving recruitment of eosinophils. However, treatment with anti-IL-5 reduced infiltration of eosinophils and attenuated the expression of Epx (1.7 fold, P=0.667) and siglec F (4.4 fold, P=0.606) in the nasal mucosa. These results support a role for IL-5 in the regulation of eosinophilic airway inflammation in TDI-induced rhinitis.

Supported in part by an NIEHS-NIOSH Interagency Agreement (Y1-ES-0001).

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