

Global patterns of gene expression following inhalation of toluene diisocyanate suggest development of allergic rhinitis (37.4)

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ABSTRACT

Rhinitis is often a comorbid and preceding disease in workers afflicted with toluene diisocyanate (TDI) asthma. We hypothesized that TDI rhinitis is driven by an allergic immune response rather than innate inflammation. Global patterns of gene expression were examined via microarray in a murine model of TDI rhinitis and compared to acute nasal exposure to lipopolysaccharide (LPS). Histological analysis showed extensive inflammation of the nasal mucosa with eosinophils and neutrophils dominating in the TDI and LPS models, respectively. Effector cell recruitment correlated with expression of chemokines directed towards eosinophils in TDI rhinitis and neutrophils in LPS rhinitis. Genes involved in antigen processing/presentation, lymphocyte activation/regulation, cytokine-receptor signaling and humoral immunity were differentially regulated in TDI rhinitis. In contrast, inflammatory, innate immune and defense responses as well as response to pathogens/biotics were over represented in LPS rhinitis. These results indicate that exposure to TDI results in an antigen-driven immune response in the nasal mucosa suggestive of allergic rhinitis. This work was supported in part by an NIEHS IAG (Y1-ES-0001 - Immunotoxicity of Workplace Xenobiotics) The findings and conclusions have not been formally disseminated by NIOSH and should not be construed to represent any agency determination or policy.

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