Comparison of Ethylene- and Ozone-Induced Nasal Toxicity: A Similar Mode of Action?

Jack R. Harkema[1], Christina Brandenberger[1], Ong Chee Bing[1], and Jon A. Hotchkiss[2]
[1]Michigan State University, East Lansing, MI and [2]The Dow Chemical Co., Midland, MI

Abstract

Ethylene is a gaseous hydrocarbon (C_2H_4) widely used by the chemical industry. Ozone (O_3) is a major gaseous component of photochemical smog. Interestingly, episodic inhalation exposures to high concentrations of either gas causes nasal pathology characterized by eosinophilic inflammation (rhinitis) and increases in mucus-secreting epithelial cells (mucous cell metaplasia/hyperplasia) in laboratory rodents. Since these toxicant-induced nasal lesions are also associated with overexpression of Th2 cytokine genes, we hypothesized that both ozone- and ethylene-induced nasal toxicity are dependent on the activation of Th2 cytokine-secreting lymphoid cells. This presentation will include our initial findings in lymphoid-sufficient and -deficient mice episodically exposed to ozone. In addition, we will present studies designed to test the hypothesis that both ethylene- and ozone-induced nasal toxicity is lymphoid dependent. Research funded in part by the ACC Olefins Panel and by the USEPA RD83479701.