Ozone-Induced Changes in Mouse Intestinal Goblet Cells

Isabel M. Parzecki, Candace R. Longoria, Carol R. Gardner,
Peihong Zhou, Jeffrey D. Laskin, Laurie B. Joseph
Rutgers, The State University of New Jersey

Ozone (O3) is an industrial pollutant known to reduce lung function due to inflammation and cell damage. The purpose of this study was to determine the effects of inhaled O3 on gut epithelium. We hypothesize that inhaled O3 induces changes in intestinal mucin production. Transgenic female mice (CD11b-DTR, JAX #006000) were injected with either 25 mg/kg IP diphtheria toxin (DT) to deplete infiltrating macrophages or PBS (control). One hour following injections, animals were exposed to O3 (0.8 ppm, 3 hr) or air. Twenty-four hours later, animals received a 2nd dose of DT or PBS and were sacrificed 48 hours post-O3 or air exposure. Distal colon was collected, washed with PBS, and prepared for histology and immunohistochemistry. Histological localization of goblet cells was determined using alcian blue/periodic acid-Schiff stain which binds to mucins. O3 induced an increase in the average number of goblet cells per colonic crypt; DT/O3 > O3 > DT/Air > Air. Mucin 2, an oligomeric glycoprotein secreted by goblet cells, coats and protects the epithelial lining of intestines and airways. Mucin 2 expression was upregulated following both DT/O3 and O3 exposure compared to air controls. Taken together, these data suggest that O3 increased production of goblet cells and secretion of Mucin 2, an essential component of gut homeostasis. Future studies will include investigation of the effects of O3 on intestinal integrity, distal ileum, and microbiome of DTR/WT mice and the characterization of Mucin 2 expression and goblet cell number in WT mice. Supported by NIH R25ES020721, ASPET SURF Program, P30ES005022, and U54AR055073.