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HUMAN CELL MUTAGENICITY OF RESPIRABLE AIRBORNE PARTICLES: SEASONAL, SPATIAL, AND CHEMICAL VARIATIONS

D. Pedersen[1], J. Durant[2], K. Taghizadeh[3], A. Lafleur[3], H. Hemond[1], G. Cass[4]
[1]*Department of Civil & Environmental Engineering, Massachusetts Institute of Technology, Cambridge, USA*
[2]*Department of Civil & Environmental Engineering, Tufts University, Medford, USA*
[3]*Center for Environmental Health Sciences, Massachusetts Institute of Technology, Cambridge, USA*
[4]*Earth and Atmospheric Sciences, Georgia Institute of Technology, Atlanta, USA*

Respirable airborne particles (PM2.5) have been linked with many cardiopulmonary diseases. Genotoxicity of PM2.5 collected at 5 sites in the northeastern U.S. (n=53-60 samples) was tested in a mutation assay based on the h1A1v2 line of human B-lymphoblastoids expressing P450 CYP1A1 cDNA. Samples from each site were separated into bimonthly composites, and annual composites were fractionated by HPLC into 4 non-polar, semipolar, and polar fractions. Our objectives were to test for seasonal, spatial, urban-rural, and chemical variations in the human cell mutagenicity of PM2.5 over a regional scale (300-500 km), and to identify individual compounds which contribute significantly to the mutagenicity of the aerosol. Mutagenicity per microgram of organic carbon (mutagenic potency) was significantly higher during winter and in the semipolar chemical fraction. Wintertime samples and semipolar fractions collected at sites in upstate New York were roughly twice as mutagenic as those collected 400 km away in Massachusetts. Only slight differences in mutagenic potency were observed among sites within each state (up to 100 km distant) despite pronounced urban-rural differences. 150 known or suspected organic mutagens, primarily polycyclic aromatic compounds, were quantified by GCMS in three annual composites and their fractions. Approximately 20% of total sample mutagenicity was attributed to individual compounds, including various PAH and a PAH-ketone. Total attributed mutagenic potency was higher at the urban sites than at the background site, but no interstate differences were evident, implying that linear sums of the mutagenicity of these compounds do not adequately account for the human cell mutagenicity of PM2.5.