Cache Valley PM2.5 Activates Akt and Inflammatory Pathways and Increases Cell Proliferation in Human Lung Cells

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Northern Utah’s Cache Valley frequently has some of the nation’s highest concentrations of PM2.5 particulate pollution (CVPM). In other locales, epidemiological studies have linked PM2.5 exposure to various cardiovascular and cardiopulmonary diseases, including heart attack, stroke, COPD, Alzheimer’s, and lung cancer. The purpose of this study was to determine the gene-level responses in BEAS-2B human lung cells exposed to PM2.5 (1 and 12 g/ml; 24 hr) collected in Logan UT onto stainless steel plates using a calibrated MOUDI impactor. Whole-genome microarray (Affymetrix Human 2.0) revealed significantly impacted genes principally related to the inflammatory and immune pathways, as well as activated serine/threonine Akt (*aka* protein kinase B or PKB)-dependent pathways. Subsequent qPCR analysis showed that CVPM exposure significantly increased expression of cytochrome P450 (CYP) 1A1, the main bioactivator of carcinogenic polycyclic aromatic hydrocarbons, as well as differential expression of various inflammatory markers including IL-6, CD40LG, and PLAG27 (p < 0.05). Immunoblotting confirmed phosphorylation of Akt at Thr308. Flow cytometry showed that CVPM elicited a significant increase in number of actively-dividing cells compared to control. In total, these data are similar to those obtained in earlier studies where CVPM was collected and extracted from Teflon filters. Akt is a proto-oncogene that plays an important role in cell division and carcinogenesis, thus our data affirms the hypothesis CVPM may induce carcinogenic pathways. This research is supported in part by a generous gift from the Marriner S. Eccles Charitable Foundation.