97th AACR Annual Meeting
April 1-5, 2006
Washington, DC

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Abstract Number: 5207

Presentation Title: Altered gene expression in response to diesel exhaust particulate matter (SRM 1650a) in MCF-7 cells detected with DNA microarrays

Presentation Start/End Time: Wednesday, Apr 05, 2006, 8:00 AM -12:00 PM

Location: Exhibit Hall, Washington Convention Center

Poster Section: 19

Poster Board Number: 8

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Human exposures to polycyclic aromatic hydrocarbons (PAHs) occurs in the form of complex mixtures. Some of these PAHs are carcinogenic. Standard Reference Material (SRM) 1650a-Diesel particulate matter was determined by the National Institute of Standards and Technology to contain a mixture of specific PAHs and nitro-substituted PAHs. In this study the gene expression patterns were investigated in MCF-7 cells (derived from a human mammary carcinoma) exposed (24h) to SRM 1650a alone or in combination with the carcinogenic PAH benzo[*a*]pyrene (BP) or dibenzo[*a,l*]pyrene (DBP). Gene expression was monitored using high density oligonucleotide arrays (Affymetrix U I33A). Exposure of MCF-7 cells to SRM 1650a alone or in combination with BP or DBP resulted in altered gene expression patterns of many RNA species. The data reported here were based on 2-fold up-regulation or down-regulation of genes that were statistically significant ($p \leq 0.05$). Global gene expression analysis revealed 30, 127 and 19 genes that were significantly altered in response to SRM 1650a, SRM 1650a plus BP, or DBP, respectively. Functional classification of differentially expressed genes indicated that several of the genes were either directly or indirectly involved in metabolism of PAHs, cell cycle regulation, proliferation, DNA repair and tumor suppression. In a previous study we have shown that CYP1A1 and CYP1B1 were up-regulated on exposure to BP (*Cancer Research*, 2005, 65:1251-58). In this study, an induction of the same genes was noticed on co-treatment with SRM 1650a plus BP. Unlike SRM 1650a alone or co-treatment with BP, expression of CYP1A1 was not observed when the cells were co-treated with SRM 1650a and DBP, although expression of CYP1B1 was evident. These results not only provide a transcriptional signature to chemical carcinogen exposure, but also assist in the development of specific and sensitive biomarkers for use in studies of exposure and susceptibility, both in mechanistic and epidemiological studies. Supported in part by grant CA28825, NCI, DHHS. Disclaimer: The views expressed in this abstract are those of the authors and do not represent the policies of the U.S. EPA or NIOSH, CDC.

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