## ACTIVATION OF NFκB AND H2A.X IN MONOCYTE-MACROPGAGE CELLS EXPOSED TO AIR POLLUTANTS

A. Niechoda, K Milewska, J Roslan and A. Holownia

Department of Pharmacology, Medical University of Bialystok, Mickiewicza 2c, Poland, agata.niechoda@gmail.com

Exposure to inhalable particulate matter causes inflammation in the airways which can lead to cancer. Our study aimed to assess ex-vivo responses of human monocytes and monocyte-derived macrophages to carbon black (CB), urban dust (UD), and nanoparticulate carbon black (NPCB), focusing on their pro-inflammatory and DNA-damaging properties. We have used naive and transduced peripheral blood mononuclear cells (PBMCs) isolated from the blood of healthy donors. Cells were grown overnight in serum-free media and then treated with 100 μg·mL−1 coarse CB, UD, or NPCB for 24 hours. Changes in H2A.X and NF-κB expressions were assessed in single and double fluorescence experiments, using specific rabbit fluorescent monoclonal antibodies against H2A.X, H2A.X phosphorylated at Ser 139 (γH2A.X) NFκB, and phosphorylated (Ser 539) NFκB (P-Ser536). In monocytes, only UD increased γH2A.X protein. NF-κB was activated by UD and slightly stronger by NPCB. In MDM cells H2A.X pathway was stimulated by UD only, while NF-κB pathways were activated mostly by NPCB and less by UD. Concerning binary scatterplots, there was a strong polarization of cells near NF-κB ax for NPCB and near H2AX ax for UD. We believe that our results highlight important molecular mechanisms of tumor-promoting inflammation.