AIR POLLUTION PARTICULATE MATTER DIFFERENTIALLY ACTIVATE MONOCYTE AND MACROPHAGE PATHWAYS OF INFLAMMATION AND CANCER.

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Air pollution causes an inflammatory response in the airways. This study aimed to examine the proinflammatory and prooncogenic properties of coarse carbon black (CB), urban dust (UD), and nanoparticle carbon black (NPCB) in naive and transduced THP-1 cells and human monocytes and macrophages isolated from peripheral blood, mononuclear cells (PBMCs) using CD14+ magnetic beads. Phorbol-myristate was used as a monocyte-macrophage transition inducer. Cells were quiesced overnight in serum-free media and then were treated with 100 μg·mL−1 coarse CB, UD, or NPCB for 24 hours. Cells were stained with fluorescent antibodies (Cell Signaling) against H2A.X, H2A.X phosphorylated at Ser 139, NFκB, and phosphorylated (Ser 539) NFκB. As a positive control cisplatin, tert-butyl hydroperoxide, and lipopolysaccharide were used. UD and NPCB resulted in a visible narrowing, and prolongation of the binary fluorescence dispersion plots indicating that both variables are positively interrelated. In quiescent monocytes and macrophages, there is a different ratio of phospho- to nonphospo-proteins on binary scatterplots. Both UD and NPCB increased mostly activated H2A.X and NFκB levels. In macrophages, UD activated predominantly H2A.X while NPCB increased active NFκB. These changes may be relevant to the regulation of inflammation and immune response.