## AIR POLLUTANTS, β2 INTEGRINS AND MONOCYTE/MACROPHAGE TRANSITION

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Particulate matter (PM) is a mix of heterogeneous components which includes both organic and inorganic particles. Exposure to PM triggers inflammatory response in the airways. The aim of this study was to examine the effect of coarse carbon black (CB), urban dust (UD), nanoparticle carbon black (NPCB) and NPCB + benzo(a)pyrene (NPCB-BaP) onTHP-1 cell adhesion (CD11b/CD18 integrins) and monocyte/macrophage transition. THP-1 cells were grown for 24 hours in cell-dedicated culture medium supplemented with 100 microg/mL of CB, UD, NPCB or NPCB-BaP. Cells were subsequently stained with fluorescent antibodies against CD11b/CD18, CD14 and CD68 epitopes. As a positive control (transition inducer) phorbol-12-myristate-13-acetate(PMA, 200ng/mL; 48h) was used. Marker protein expression was analysed on Beckton flow cytometer using appropriate isotype controls. PMA treatment increased CD11a/CD18 in THP-1 cells by 2 and 3 fold, respectively and decreased CD14 expression. All PM except for NPCB-BaP slightly increased CD11b and CD18 markers, but highly stimulated PMA-induced activation. Macrophages produce different reactive oxygen species that contribute to the pathogenesis of several inflammatory diseases and tissue remodelling. Our data indicate that different PM can stimulate cell adhesion and possibly also diapedesis and, to some extent, also monocyte /macrophage transition, however further study are needed to define their specific phenotype and functional changes. It seems that THP-1 activation model can be further explored, and validated to improve its diagnostic and predictive value in PM-induced chronic inflammation and tissue remodelling.