AIR POLLUTANT NANOPARTICLES PRIME MACROPHAGES FOR INFLAMMATORY RESPONSE

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Fine inhalable particulate matter (PM) triggers an inflammatory response in the airways. We assessed ex vivo responses of human monocyte-derived macrophages to carbon black (CB), urban dust UD), and nanoparticulate carbon black (NPCB), focusing on their pro-inflammatory properties. The human mononuclear cells were isolated from the blood of healthy donors and were differentiated in culture into monocyte-derived macrophages by phorbol 12-myristate 13-acetate. Cells were incubated in RPMI medium with 100 µg/ml PM at 37°C for 24 hours and IL-1beta, IL-6 and TNFalpha relase were analyzed. Only NPCB increased IL1beta produced by monocytes while IL6 and TNFalpha were unchanged. The transition of cells by PMA highly increased inflammatory response, resulting in a 2-3 times increase in both interleukins and TNFalpha. In macrophages only NPCB significantly increased TNFalpha. LPS induced a strong proinflammatory reaction and elevated all measured parameters about 10 times. Slightly higher, yet not significant values were observed in macrophages pretreated with PM. Both UD and NPCB increased IL-1beta which is important in acute-phase responses to infection and injury, and NPCB additionally elevated multifunctional proinflammatory cytokine - TNFalpha. Our data show that particulate matter molecules can prime subsets of immune cells for inflammatory response.