Increased expression and activation of NF-κB in human M1 macrophages exposed to nanoparticle carbon black ex vivo.

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Fine inhalable particulate matter (PM) activates mononuclear cells, mediators of tissue homeostasis, and tumour-promoting inflammation and triggers an inflammatory response in the airways. We have assessed ex vivo responses of human monocyte-derived macrophages to carbon black (CB) and nanoparticulate carbon black (NPCB), focusing on their pro-inflammatory response. Macrophages were obtained by PMA treatment of monocytes isolated from the blood of healthy donors and purified using magnetic $CD14^+$ beads. None of the PM (100 μg/mL/24 h) was significantly toxic to the cells. NPCB but not CB increased the expression and activated pro-inflammatory NF-κB. Binary fluorescence scatterplots of NF-κB vs NF-κB P-Ser 536 evidenced that airborne nanoparticles induce specific alterations in the low- and high NF-κB expression/activation subsets of cultured macrophages representing subpopulation of mononuclear cells, characterized by different expression of macrophage M1 and M2 markers: CD80, CD163 and CD206. Higher expression of naive and activated NF-κB was found in cells with M1-like phenotype. It seems that such changes may intercede with inflammatory signalling in the airways exposed to nanoparticle air pollutants.