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TITLE: INHALATION OF ULTRAFINE PARTICLES ALTERS BLOOD LEUKOCYTE EXPRESSION OF ADHESION MOLECULES IN HUMANS

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ABSTRACT: Ultrafine particles (UFPs; aerodynamic diameter < 100 nm) may contribute to the respiratory and cardiovascular morbidity and mortality associated with particulate air pollution. We tested the hypothesis that inhalation of carbon UFPs has vascular effects in healthy and asthmatic subjects, detectable as alterations in blood leukocyte expression of adhesion molecules. Healthy subjects inhaled filtered air and freshly generated elemental carbon particles (count median diameter - 25 nm, geometric standard deviation similar to 1.6), for 2 hr, in three separate protocols: 10  $\mu\text{g}/\text{m}^3$  at rest, 10 and 25  $\mu\text{g}/\text{m}^3$  with exercise, and 50  $\mu\text{g}/\text{m}^3$  with exercise. In a fourth protocol, subjects with asthma inhaled air and 10  $\mu\text{g}/\text{m}^3$  UFPs with exercise. Peripheral venous blood was obtained before and at intervals after exposure, and leukocyte expression of surface markers was quantitated using multiparameter flow cytometry. In healthy subjects, particle exposure with exercise reduced expression of adhesion molecules CD54 and CD18 on monocytes and CD18 and CD49d on granulocytes. There were also concentration-related reductions in blood monocytes, basophils, and eosinophils and increased lymphocyte expression of the activation marker CD25. In subjects with asthma, exposure with exercise to 10  $\mu\text{g}/\text{m}^3$  UFPs reduced expression of CD11b on monocytes and eosinophils and CD54 on granulocytes. Particle exposure also reduced the percentage of CD4(+) T cells, basophils, and eosinophils. Inhalation of elemental carbon UFPs alters peripheral blood leukocyte distribution and expression of adhesion molecules, in a pattern consistent with increased retention of leukocytes in the pulmonary vascular bed.