## AIR POLLUTION REDUCES EPC LEVELS

A large body of epidemiological and experimental data exists to support a connection between exposure to airborne pollutants and poor cardiovascular health. Induction of endothelial dysfunction by the fine particulate matter (PM) not filtered by the respiratory tract (<2.5 μm; $PM_{2.5}$) is one of the proposed mechanisms for the adverse effects of air pollution. Timothy O'Toole and colleagues hypothesized that exposure to PM might cause depletion of endothelial progenitor cells (EPCs), leading to endothelial dysfunction and cardiovascular injury.

The investigators recruited 16 healthy college students from Provo, UT, USA to participate in the study. This city is situated in a valley where temperature inversion leads to a temporary increase in the concentration of $PM_{2.5}$ in the atmosphere. "We were able to measure changes in EPC levels only because we ... studied humans who were transiently exposed to high PM levels," explains Dr O'Toole. Blood EPC levels were measured during two periods of low pollution ($PM_{2.5}$ <10 μg/m$^3$), one period of moderate pollution ($PM_{2.5}$ ~20–40 μg/m$^3$), and one period of high pollution ($PM_{2.5}$ >40 μg/m$^3$). O'Toole *et al*. also conducted an animal study in which 28 mice were exposed either to air with a high concentration of $PM_{2.5}$, or to filtered air, for 6 h per day for 9 consecutive days.

In both humans and mice, exposure to $PM_{2.5}$ was inversely correlated with EPC levels (particularly $CD31^+/CD34^+$ cells). In humans, $PM_{2.5}$ exposure was also associated with increased levels of platelet–monocyte aggregates, HDL cholesterol and, particularly, nonalbumin protein. "We do not fully understand the reasons for these changes," says Dr O'Toole, "but they may be reflective of a systemic inflammatory response." The researchers now plan to investigate the mechanisms by which $PM_{2.5}$ suppresses EPCs and whether exposure also causes functional defects in these cells.

*Alexandra King*

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