

OXIDIZED-LDL AND CARBON LOAD OF LUNG MACROPHAGES

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Background and Aims: Epidemiologic studies indirectly suggest that air pollution accelerates atherosclerosis. We hypothesized that individual exposure to particulate matter (PM) derived from fossil fuel would correlate with plasma concentrations of oxidized low-density lipoprotein (LDL), taken as a marker of atherosclerosis. We tested this hypothesis in a susceptible population of patients with diabetes.

Methods: In a cross-sectional study of non-smoking adult outpatients with diabetes we assessed individual chronic exposure to PM by measuring the area occupied by carbon in airway macrophages, collected by sputum induction and by determining the distance from the patient's residence to a major road, through geocoding. These exposure indices were regressed against plasma concentrations of oxidized LDL, von Willebrand factor and plasminogen activator inhibitor 1 (PAI-1)..

Results: We could assess the carbon load of airway macrophages in 79 subjects (58 percent). Each doubling in the distance of residence from major roads was associated with a $0.027 \mu\text{m}^2$ decrease (95 % confidence interval (CI): -0.048 to -0.0051) in the carbon load of airway macrophages. Independently from other covariates, we found that each increase of $0.25 \mu\text{m}^2$ [interquartile range (IQR)] in carbon load was associated with an increase of 7.1 U/L (95% CI: 1.5 to 12.7) in plasma oxidized LDL. Each doubling in distance of residence from major roads was associated with a decrease of -3.1 U/L (95% CI: -5.3 to -0.93) in oxidized LDL.

Conclusions: The observed positive association, in a susceptible group of the general population, between plasma oxidized LDL levels and either the carbon load of airway macrophages or the proximity of the subject's residence to busy roads is compatible with a proatherogenic effect of traffic air pollution.