SHORT-TERM EXPOSURE TO PM2.5, PM1 AND BLOOD COAGULATION IN HUMANS.

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Background

Health effects of short- and long-term exposure to airborne particulate matter (PM) are only partially known. In addition to respiratory effects, effects on the cardiovascular system have been repeatedly described. Epidemiological studies consistently showed an increased cardiovascular mortality and increased rates of cardiovascular hospital admissions following acute episodes of urban pollution. After acute exposure, the following changes in healthy adult subjects have been described: increased plasma viscosity; elevated reactive protein C; heartbeat frequency alterations; increased risk of implanted cardioverter-defibrillator discharges (implying a response by the autonomous nervous system).

Study Hypothesis

Two main explanatory hypotheses have been proposed: 1) the particulate matter deposited in the airways elicits the release of inflammatory mediators capable of affecting the cardiac function and the coagulation process; 2) the smallest particles (aerodynamic diameter <100nm) reach the blood stream from the alveolar space and directly affect the coagulation processes. In addition to particle size, the number of inhaled and deposited particles appears important (although often overlooked in favour of mass measures). It should be noted that, given the same particles mass, the finest particles occupy a more extended surface.

Design

We designed a study in order to evaluate acute effects of exposure to PM on the coagulation process in humans. We planned to examine 50 healthy adult subjects, age 20-55 years, smokers and non-smokers, free from blood coagulation (possibly) related disorders and living in urban polluted areas. The candidates hold jobs in clean industrial environments, virtually free from PM exposure due to production needs (e.g., semiconductors, and pharmaceutical). Their basal blood coagulation function is assessed before the work shift. They later spend six hours in clean rooms, PM "free". Coagulation markers are then assessed at the end of the "non-exposure" period. Subsequently, the outdoor/indoor exposure period (commuting, social and domestic life) begins. For a 14-hour period, PM1 and PM2.5 exposure is evaluated through 1) selection of granulometric fraction and gravimetric analysis (mass definition); 2) elemental analysis (inorganic elements composition); ultra-fine particle counting (from which to derive an exposure estimate). At the end of the exposure period, a further blood coagulation markers assessment is planned.



Sources and images of urban pollution



Two possible study designs. The clean-room workers approach was selected. Feasibility: red, poor; blue, good.

Perspectives

Analyses explore possible changes of coagulation function markers in relation to exposure/non-exposure periods, and to varying levels/types of exposure. This observation may provide a plausible biological explanation for the epidemiologically established link between air pollution and acute myocardial infarction, with obvious bearing on prevention policy and primary health care.



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