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INFLAMMATORY EFFECTS OF FINE AND ULTRAFINE URBAN AIR PARTICULATE MATTER EXPOSURE IN ADULT SUBJECTS WITH DIFFERENT HEALTH CONDITIONS

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Introduction and aim: Particulate matter (PM) air pollution increases the risk of cardiovascular and respiratory diseases, but the mechanisms behind this effect remain unclear. The PM-CARE Study, Particulate Matter CArdio-Respiratory Effects, was designed to investigated these mechanisms in susceptible subjects (i.e. people suffering from cardiac or chronic lung diseases) and in healthy subjects. The aim of this abstract is to assess the association between PM exposure and markers of inflammation.

Methods: Three groups of non-smoking adults (n = 81) entered the PM-CARE Study: 34 subjects with chronic ischemic heart disease (Heart group), 20 with chronic asthma or COPD (Lung Group), and 27 without diagnosis of the afore mentioned diseases (Healthy Group). They underwent a 24-h exposure/clinical protocol during their habitual activities in the warm and in the cold season. Individual exposures to PM were given as 24-hour averaged $PM_{2.5-10}$, $PM_{1-2.5}$, $PM_{0.5-1}$, $PM_{<0.5}$ mass concentrations, and particle number concentrations (NC range (µm)), by separately covering the ultrafine (NC > 0.02), the fine (NC 0.3-0.5; NC 0.5-1, and NC 1-2.5), and the coarse (NC 2.5-5; NC 5-10, and NC > 10) fractions, in accordance with particles' aerodynamic diameter (µm). Blood samples were collected at the end of a 24-h protocol: white blood cell count, TNF-α, TNF-α sR-I and II, IL-8, IL-10 measured both in plasma and *in vitro* following stimulation with PHA or LPS. Linear mixed effects models for repeated measurement data were applied.

Results: Heart Group: we found positive associations ($p < 0.05$) between: monocytes and $PM_{0.5-1}$ and NC 0.3-1; TNF-α sR-II and $PM_{0.5-2.5}$ and NC 0.3-2.5; *in vitro* IL-8 and NC 0.5-1.

Lung Group: a negative association ($p < 0.05$) was found between: lymphocytes and $PM_{1-2.5}$ and NC 0.3-5.

Healthy Group: we found positive associations ($p < 0.05$) between: lymphocytes and $PM_{0.5-1}$, as well as NC > 0.02 and NC 0.3-1; TNF-α sR-II and $PM_{1-2.5}$ and NC 0.3-5; *in vitro* IL-8 and NC > 0.02 µm, NC 0.3-1

Conclusion: Increased plasma and *in vitro* levels of cytokine and different inflammatory cells suggest that fine and ultrafine PM exposure exert a pro-inflammatory effect both on individuals with chronic ischemic heart disease and on "healthy" subjects, whereas individuals with lung chronic diseases seemed to be less susceptible. Further refinement to control the numerous confounders is expected to increase the consistency of the results.