

ASSOCIATION BETWEEN AMBIENT AIR POLLUTION AND BLOOD MARKERS OF INFLAMMATION AND COAGULATION IN INDIVIDUALS WITH POTENTIAL GENETIC PREDISPOSITION ON THE DETOXIFYING PATHWAY

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Background and Aims: The pathophysiological pathways linking particulate air pollution to cardiovascular disease are still not fully understood. We examined the association between ambient air pollutants and blood markers of inflammation and coagulation in three potentially susceptible populations.

Methods: Three panels of non-smoking individuals were examined: 1) with type 2 diabetes mellitus (n=83), 2) with impaired glucose tolerance (n=104), and 3) with a potential genetic predisposition on the detoxifying pathway (n=87) defined by the null polymorphism for *glutathione S-transferase M1* (*GSTM1*) in combination with a certain single nucleotide polymorphism on the *C-reactive protein* (*CRP*) or the *Fibrinogen* gene. Study participants had blood withdrawn up to seven times, scheduled every 4-6 weeks between 3/2007 and 12/2008. In total, 1765 blood samples were investigated for CRP, interleukin 6 (IL-6), soluble CD40 ligand (sCD40L), fibrinogen, myeloperoxidase (MPO), and plasminogen activator inhibitor-1 (PAI-1). Hourly means of PM₁₀, PM_{2.5} (particle diameter <10µm and <2.5µm, respectively), particle number concentrations and nitrogen dioxide were collected at a fixed monitoring site and individual 24 hour averages calculated. Associations between air pollutants and blood markers were analysed using additive mixed models adjusting for long-term time trend and meteorology.

Results: For the panel with potential genetic susceptibility sCD40L and PAI-1 showed negative associations with all air pollutants within 24 hours before blood samples were taken (lag 0). Clear positive associations were seen for CRP and MPO especially with lags of 1 to 3 days (% change of geometric mean and 95% confidence interval for lag 1: 14.5 [5.3;24.6] and 4.9 [0.7;9.3] per interquartile range of PM_{2.5}, respectively). Results for IL-6, fibrinogen and the two other panels were less conclusive.

Conclusions: Patients with potential genetic susceptibility showed a clear association with ambient air pollutants. Our results support the idea that systemic inflammation may aggravate atherosclerotic disease, especially in susceptible populations.