SOURCE-SPECIFIC AIR POLLUTION AND SYSTEMIC INFLAMMATION IN ISCHEMIC HEART DISEASE PATIENTS

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Background and Aims: Associations between particulate air pollution (PM) and cardiovascular health have been observed in numerous studies. Exposure to PM can accelerate systemic inflammation, which has been hypothesized to play an important role in the development of cardiovascular disease. Our aim was to evaluate the association between source-specific PM and systemic inflammation among ischemic heart disease patients.
Methods: Fifty-two ischemic heart disease patients were followed for six months with biweekly clinical visits (minimum of three) including a blood-withdrawal in Kotka, Finland, between November 2005 and May 2006. Blood levels of high-sensitive C-reactive protein (CRP) and fibrinogen were determined to be used as indicators of systemic inflammation. Daily levels of air pollution were measured at a central outdoor measurement site. Source-apportionment for fine particles (< 2.5 µm in diameter) was conducted using positive matrix factorization 3.0.2.2. by the U.S. Environmental Protection Agency. Immediate and delayed associations of source-specific air pollution with levels of fibrinogen and log-transformed CRP were analyzed using mixed models with random patient effects adjusting for time-trend, temperature and relative humidity.
Results: There were 507 successful repeated clinical visits in total. Looking at 5-day average concentration, CRP had stronger association with air pollution than fibrinogen. The %-changes for CRP were 2.9% (95%-confidence interval (CI): 0.3 to 5.5%) for long-range transported PM, 31.1% (95%-CI: -6.4 to 83.8 %) for traffic emissions, 2.3% (95%-CI: 0.2 to 4.5%) for biomass burning and -16.1% (95%-CI: -27.2 to -3.3%) for pulp industry per 1µg m^{-3} increase in pollutant concentration. We found no association for seasalt.
Conclusions: Our results suggest that several sources of ambient air pollution are associated with systemic inflammation, a risk factor for coronary heart disease.