

AMBIENT AIR POLLUTION AND CARDIOVASCULAR MORBIDITY AMONG POTENTIALLY SUSCEPTIBLE GROUPS

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Background and Aims: Evidence suggests that risks associated with exposure to ambient air pollutants are not uniform across individuals; however, there is considerable uncertainty in estimates for susceptible populations.

Methods: We conducted a five-year study (2003-07) examining the association of fine particulate matter (PM_{2.5}) and gases with cardiovascular hospital admissions in Denver, Colorado. We investigated the relationships between daily counts of cardiovascular disease admissions and same-day concentrations of ambient air pollutants (PM_{2.5} mass, elemental carbon [EC], organic carbon [OC], sulfate, nitrate, source-apportioned PM, gases) among potentially susceptible subgroups defined by comorbid illness (using secondary diagnoses, e.g. hypertension, diabetes), age, race, sex, and payment type.

Results: The mean daily concentration of PM_{2.5} mass was 8.0•g/m³ (standard deviation 5.1•g/m³). We observed increases in cardiovascular disease hospital admissions, particularly for ischemic heart disease, in relation to increases in PM_{2.5} mass, EC, and OC concentrations as well as several of the PM source factors. For the PM species, the associations were strongest for EC (relative risk [RR per interquartile range] = 1.019; 95% confidence interval [CI] 1.010-1.028 per 0.33•g/m³ increase) and OC (RR=1.017; 95%CI 1.006-1.028 per 1.7•g/m³ increase). We did not observe consistent evidence of heterogeneity of these associations by subgroups. Some indication of stronger effects among patients with comorbid ischemic heart disease (IHD), including coronary atherosclerosis, compared to patients without comorbid IHD and among non-white patients compared to white patients was observed, although confidence intervals were wide and overlapping.

Conclusions: Although associations were detected among the total population at relatively low PM mass and component concentrations, we did not observe strong evidence of effect modification by potentially susceptible subgroups. The use of secondary diagnoses may have limited our ability to detect comorbid illness.

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