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Outdoor Particulate Matter, Cardiovascular Health and Incidence of Coronary or Ischemic Stroke Events: A Population-Based Study

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INTRODUCTION

Long-term exposure to outdoor particulate matter of size<2.5 microns (PM2.5) has been associated with increased risk of cardiovascular diseases (CVD) incidence [1], although with substantial between-study heterogeneity. In addition, pathways and factors of individual susceptibility to PM2.5 have been poorly characterized so far. The American Heart Association (AHA) proposed the Life's Simple 7 (LS7) metric of cardiovascular health by combining lifestyle (not smoking, healthy diet, engaging in sufficient physical activity) and health (normal body weight, maintaining low values of blood pressure, total cholesterol and glucose) components [2]. The mediating role of LS7, and whether the risk excess due to PM2.5 is exacerbated in individuals with a "poor" cardiovascular health, remain to be ascertained.

AIMS

To investigate the association between long-term exposure to PM2.5 and the incidence of first coronary heart disease or ischemic stroke event, and the contribution of LS7 on explaining the risk excess, in a population-based cohort in Northern Italy. Furthermore, to assess the role of LS7 as an effect modifier for the association.

METHODS

STUDY POPULATION

The RoCAV study [3] is a population-based cohort of n=3777 50+ years old residents in the city of Varese (Lombardy region, northern Italy) at the time of recruitment (2013-2016; 64% participation rate). For the aims of these analyses, we retained individuals free of coronary heart disease and stroke at baseline (n=3313, 62% men).

CARDIOVASCULAR HEALTH

Participants underwent a comprehensive baseline examination, assessing cigarette smoking (MONICA questionnaire), dietary intake (EPIC food frequency questionnaire), habitual physical activity (Baecke questionnaire), as well as clinical and laboratory parameters (fasting blood lipids and glucose, blood pressure and anthropometric measures). From these, we calculated the LS7 metric on a 0-to-14-points scale [2], further categorized as poor (0-4 points), intermediate (5-9 points) and ideal (10-14 points) [4]. We categorized the two LS7 components of lifestyle (LS7-ls) and health (LS7-h) in a similar way.

PM EXPOSURE

Monthly concentrations for PM2.5 over the period 2000-2019 were retrieved from the EXPANSE project models [5], at a spatial resolution of 25mt. Individuals' concentrations were attributed from spatial linkage at the residential address at baseline, geo-referenced; the exposure metric was the average concentration in the 12 months before the month of baseline visit.

STUDY ENDPOINTS

Individuals were followed-up through record linkage with Electronic Health Records (hospital discharge and mortality) provided by the Local Health Agency. We selected discharge codes suggestive of myocardial infarction and unstable angina (ICD-IX codes: 410-411) or elective coronary revascularization; cerebrovascular infarction (ICD-IX 433 or 434) or endarterectomy with stenosis at cerebral or pre-cerebral arteries (ICD-IX 433.1, 433.3, 434.0). Selected records were then reviewed to identify and retain the first index case. Fatal cases were identified from underlying causes of deaths suggestive of coronary deaths (ICD-X codes: I21-I25) or ischemic stroke (I63). Censorship occurred at the date of death from

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other causes or emigration outside the study Region, as ascertained by contacting the municipality of residency. The study endpoint is the occurrence of first coronary heart disease or ischemic stroke, fatal or non-fatal, before Dec 31st, 2022.

STATISTICAL ANALYSES

Due to the low number of events in women, the analyses were carried out on men and women combined, and on men only. We first estimated the rate ratios for the LS7 categories ("ideal" as reference) from Poisson regression models, adjusting for age and sex. Then, we estimated the Hazard Ratios (HR) and 95%CI for 1 interquartile range (1.93 µg/m3) increase in PM2.5 using nested Cox models with attained age during follow-up on the time scale, and adjusting for: sex and education (Model1), and further for LS7 (Model2). We computed the percent explained by LS7 on the log scale as 100[log(HRM2)-log(HRM1)]/[log(HRM1)]. Finally, we investigated individual susceptibility by adding to Model2 a LS7PM2.5 interaction and by reporting the corresponding p-value from a Wald chi-square test (2df). These analyses were repeated for LS7-ls and LS7-h components.

RESULTS

Mean±SD PM2.5 concentrations were 18.6±1.8 µg/m3. During 7.1 years of median follow-up time, we observed n=196 CVD events for a rate of 8.6 per 1,000 person-years (men: n=150 events, 10.8 per 1,000 py). "Poor" vs. "ideal" LS7 resulted in a 2.34-fold (95%CI: 1.33-4.10) increased event rate (men: 2.13, 1.12-4.06). In Model1, PM2.5 was associated with increased CVD risk, in the overall sample (HR=1.11, 95%CI: 0.95-1.29) and in men (HR=1.19, 95%CI: 1.00-1.43). Further adjustment for LS7 explained 13.4% (men and women) and 7.2% (men) of the risk excess, respectively, mainly due to the LS7-ls component. We found evidence of interaction between PM2.5 and the LS7-ls component (Wald test p-values: 0.03 [men and women] and 0.007 [men]). In men, the HRs for PM2.5 in the "poor" (40% of the sample) and in the "ideal" (15% of the sample) LS7-ls categories were 1.49 (95%CI: 1.11-1.98) and 0.67 (0.45-1.00), respectively. Conversely, no interaction was observed for LS7, nor for the LS7-h component.

CONCLUSIONS

In our cohort, representative of contemporary outdoor PM2.5 levels in a North Italian population, long-term exposure to PM2.5 was associated with an increased risk of first major CVD event, especially in men. AHA LS7 explained only a small proportion of the association. Adults engaging poor lifestyles are more susceptible to the detrimental effect of PM, and can benefit the most from policies reducing pollution levels.

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