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Long-term effects of total and source-specific particulate air pollution on incident cardiovascular disease in Gothenburg, Sweden

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ABSTRACT

Background and aims: Long-term exposure to air pollution increases cardiopulmonary morbidity and mortality, but it is not clear which components of air pollution are the most harmful, nor which time window of exposure is most relevant. Further studies at low exposure levels have also been called for. We analyzed two Swedish cohorts to investigate the effects of total and source-specific particulate matter (PM) on incident cardiovascular disease for different time windows of exposure.

Methods: Two cohorts initially recruited to study predictors of cardiovascular disease (the PPS cohort and the GOT-MONICA cohort) were followed from 1990 to 2011. We collected data on residential addresses and assigned each individual yearly total and source-specific PM and Nitrogen Oxides (NO_x) exposures based on dispersion models. Using multivariable Cox regression models with time-dependent exposure, we studied the association between three different time windows (lag 0, lag 1–5, and exposure at study start) of residential PM and NO_x exposure, and incidence of ischemic heart disease, stroke, heart failure and atrial fibrillation.

Results and discussion: During the study period, there were 2266 new-onset cases of ischemic heart disease, 1391 of stroke, 925 of heart failure and 1712 of atrial fibrillation. The majority of cases were in the PPS cohort, where participants were older. Exposure levels during the study period were moderate (median: 13 μg/m^3 for PM_{10} and 9 μg/m^3 for $PM_{2.5}$), and similar in both cohorts. Road traffic and residential heating were the largest local sources of PM air pollution, and long distance transportation the largest PM source in total. In the PPS cohort, there were positive associations between PM in the last five years and both ischemic heart disease (HR: 1.24 [95% CI: 0.98–1.59] per 10 μg/m^3 of PM_{10}, and HR: 1.38 [95% CI: 1.08–1.77] per 5 μg/m^3 of $PM_{2.5}$) and heart failure. In the GOT-MONICA cohort, there were positive but generally non-significant associations between PM and stroke (HR: 1.48 [95% CI: 0.88–2.49] per 10 μg/m^3 of PM_{10}, and HR: 1.50 [95% CI: 0.90–2.51] per 5 μg/m^3 of $PM_{2.5}$, in the last five years). Effect estimates were stronger for women, non-smokers, and higher socioeconomic classes. Exposure in the last five years seemed to be more strongly associated with outcomes than other exposure time windows. Associations between source-specific PM air pollution and outcomes were mixed and generally weak. High correlations between the main pollutants limited the use of multi-pollutant models.

Conclusions: The main PM air pollutants were associated with ischemic heart disease and stroke (in women) at the relatively low exposure levels in Gothenburg, Sweden. The associations tended to be stronger for women than for men, for non-smokers than for smokers, and for higher socioeconomic classes than for lower. The associations could not be attributed to a specific PM source or type, and differed somewhat between the two cohorts. The results of this study confirm that further efforts to reduce air pollution exposure should be undertaken in Sweden to reduce the negative health effects in the general population.

1. Introduction

Air pollution is the single largest environmental health risk, estimated to cause around 5.5 million annual premature deaths globally (Forouzanfar et al., 2015), half a million in Europe (EEA, 2014; WHO, 2014), and several thousand in Sweden (Forsberg et al., 2005). The epidemiological evidence for this association is solid, consisting of a large number of mainly US and European cohort studies (Dockery et al.,

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1993; Pope et al., 1995; Hoek et al., 2002; Nafstad et al., 2004; Elliott et al., 2007; Raaschou-Nielsen et al., 2012; Cesaroni G et al., 2013; Beelen et al., 2014a). Negative health effects have been most consistently associated with particles, especially fine particles < 2.5 μm ($PM_{2.5}$). It is less known which types and sources of particles cause the adverse health effects, and studies of this have been called for NRC (2001) and Brook et al. (2010).

The evidence for an association between air pollution and specific diseases is not as strong as the association with mortality. In the US, the strongest associations between particles and mortality have been those for cardiovascular diseases, particularly ischemic heart disease (Pope et al., 2004). The European ESCAPE study found no associations between air pollution and cardiovascular mortality, except possibly $PM_{2.5}$ and cerebrovascular disease mortality (Beelen et al., 2014b), but did find associations between air pollution and cardiovascular events (Cesaroni et al., 2014). In a previous study of long-term residential exposure to Nitrogen oxides (NO_x) in Gothenburg, we found an association between NO_x exposure and mortality but not incident myocardial infarction (Stockfelt et al., 2015). Incident disease represents the primary cardiovascular outcome, and only a minority of events are initially fatal. In addition, case fatality also depends on age and other individual factors, and hospital registries generally have a high validity.

Individual susceptibility to the health effects of air pollution may differ, due to either biological differences or behavioral differences affecting exposure. Age, pre-existing cardiovascular disease, obesity, low socioeconomic status, and smoking, as well as both female *and* male sex, have been associated with increased susceptibility, but the evidence is not consistent and requires more investigation (Brook et al., 2010). The temporal risk relationship between air pollution and cardiovascular disease also remains uncertain. Longer-term studies have generally found larger risk estimates than shorter-term studies, but the majority of these risks seem to manifest within one to two years of follow-up (Brook et al., 2010; Pope, 2007). It is important to separate the accumulation of acute events caused by repetitive exposures from the long-term progression of chronic atherosclerosis, since the former is potentially reversible.

Exposure levels in the Nordic countries are generally low compared with many other countries, often even below current guidelines (EEA, 2012). The dose-response relationship between air pollution and health effects has usually been found to be approximately linear, with no sign of a lower threshold (Beelen et al., 2014b; Cesaroni G et al., 2013). The slope may even be steeper at low exposure levels (Pope et al., 2009). If low levels of air pollution can be established to have health effects, this would motivate both lower limit values and further efforts to reduce exposure in areas with low exposure.

This study used two well-characterized cohorts recruited from the general population of Gothenburg to assess the long-term effects of air pollution on incident cardiovascular disease at the relatively low exposure levels in the Nordic environment. We examined different time windows of exposure, and different types and sources of exposure. The study was part of a national Swedish collaboration using multiple cohorts.

2. Study population and methods

2.1. Study population, covariates, and outcomes

The study population consisted of two cohorts recruited from the general population in Gothenburg to study predictors of cardiovascular disease: the Primary Prevention Study (PPS) cohort and the GOT-MONICA cohort. Recruitment and characteristics of the PPS cohort have been described in detail previously (Wilhelmsen et al., 1972, 1986; Stockfelt et al., 2015). Briefly, a random third of all men in Gothenburg born between 1915 and 1925 were invited (participation rate: 75%) and screened in 1970–73. At baseline, the participants filled out questionnaires on background data and cardiovascular risk factors, and were examined by health care professionals. The GOT-MONICA cohort is part of the MONICA project ("Multinational Monitoring of Trends and Determinants in Cardiovascular Diseases"), an international multi-cohort study of risk factors for cardiovascular diseases (Tunstall-Pedoe, 1988). Recruitment and characteristics of the cohort have been described previously (Wilhelmsen et al., 1997). Briefly, participants were recruited in 1985, 1990, and 1995 via random selection from all residents in Gothenburg aged 25–64 years at the time of inclusion (participation rates: 63%, 69%, and 72%). The participants filled out questionnaires on background data and cardiovascular risk factors, and were examined by health care professionals.

In both cohorts, the medical examination included systolic and diastolic blood pressure, cholesterol levels, and height and weight, which were combined into BMI. The questionnaires included questions on smoking habits, physical activity (occupational and leisure-time), diabetes mellitus diagnosis, hypertensive medication, psychological stress, and marital status. For the PPS cohort, questionnaire data was also used to assign occupational class (in this study used as an indicator of social class) and family history of acute cardiovascular events. For the GOT-MONICA cohort, the questionnaires in 1990 and 1995 also included education level, but the questionnaire in 1985 did not, and so this covariate had to be excluded from the main covariate model. Covariate data was combined into categories (smoking class, marital status, occupational class, education level, family history of acute myocardial infarction, antihypertensive medication, persistent stress, diabetes mellitus, gender, enrollment year) or used as continuous variables (BMI, blood pressure, cholesterol levels) in the statistical analysis.

In 1990, at the start of our study period, 5850 participants in the PPS cohort and 4500 in the GOT-MONICA cohort were alive and residing within the study area. Background data and covariates for these participants are presented in Table 1. The participants in the PPS cohort were older and were all male, while the GOT-MONICA participants were young to middle-aged and included roughly equal numbers of men and women. A greater proportion of the PPS cohort were married. Smoking, hypercholesterolemia, and hypertension were more prevalent in the PPS cohort, largely due to time trends in the population (Wilhelmsen et al., 2008), though some of the difference could also be explained by age and gender differences, and an overestimation of blood pressure in the PPS cohort (Wilhelmsen et al., 2004).

All participants were followed during the study period of 1 January 1990 to 31 December 2011 on the basis of their unique Swedish personal identification number. Individual addresses for each year were retrieved from Statistics Sweden, manually checked and corrected for inconsistencies such as spelling mistakes, and given geographical coordinates so that individual yearly exposures could be assigned. All individual addresses were also assigned an area-level socioeconomic variable using data from Statistics Sweden on Small Areas for Market Statistics (SAMS, Statistics Sweden, 2016).). In the city of Gothenburg there are 836 SAMS-areas (mean and median sizes of 1 and 0.1 km^2 respectively) with an average population of about 650 persons in each. We used mean income in each area in the year 1994 to represent the economic status of each neighbourhood.

We used data on cause-specific mortality from the Swedish national register on cause of death as well as data from the Swedish national hospital discharge register, both according to versions 9 and 10 of the International Classification of Diseases (ICD). We only used the first case of each outcome (new onset) during the observation period, and censored the participant onwards. We also excluded participants with incident disease in the preceding five years, using the registries for 1985–1989. For incident cases of ischemic heart disease (IHD), stroke and heart failure (HF) we only used the main diagnosis for hospital admission/death, but for atrial fibrillation (AF) we also included the underlying diagnoses. Validation studies have shown high validity in the Swedish hospital discharge register for the outcomes used in this study (Ingelsson et al., 2005; Smith et al., 2010; Hammar et al., 2001;

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