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Associations between short-term exposure to particulate matter and ultrafine particles and myocardial infarction in Augsburg, Germany

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ABSTRACT

Background: Short-term exposure to increased particulate matter (PM) concentration has been reported to trigger myocardial infarction (MI). However, the association with ultrafine particles remains unclear.
Objectives: We aimed to assess the effects of short-term air pollution and especially ultrafine particles on registry-based MI events and coronary deaths in the area of Augsburg, Germany.
Methods: Between 1995 and 2009, the MONICA/KORA myocardial infarction registry recorded 15,417 cases of MI and coronary deaths. Concentrations of PM < 10 μm (PM_{10}), PM < 2.5 μm ($PM_{2.5}$), particle number concentration (PNC) as indicator for ultrafine particles, and meteorological parameters were measured in the study region. Quasi-Poisson regression adjusting for time trend, temperature, season, and weekday was used to estimate immediate, delayed and cumulative effects of air pollutants on the occurrence of MI. The daily numbers of total MI, nonfatal and fatal events as well as incident and recurrent events were analysed.
Results: We observed a 1.3% risk increase (95%-confidence interval: [−0.9%; 3.6%]) for all events and a 4.4% [−0.4%; 9.4%] risk increase for recurrent events per 24.3 μg/m^3 increase in same day PM_{10} concentrations. Nonfatal events indicated a risk increase of 3.1% [−0.1%; 6.5%] with previous day PM_{10}. No association was seen for $PM_{2.5}$ which was only available from 1999 on. PNC showed a risk increase of 6.0% [0.6%; 11.7%] for recurrent events per 5529 particles/cm^3 increase in 5-day average PNC.
Conclusions: Our results suggested an association between short-term PM_{10} concentration and numbers of MI, especially for nonfatal and recurrent events. For ultrafine particles, risk increases were notably high for recurrent events. Thus, persons who already suffered a MI seemed to be more susceptible to air pollution.

Introduction

Ischemic heart disease which includes myocardial infarction (MI), angina pectoris, and heart failure is the leading cause of death globally. It is estimated that 55 million people died due to these causes worldwide in 2011 (http://www.who.int/mediacentre/factsheets/fs310/en/). Chronic risk factors for MI mainly affect the progression of atherosclerosis. Nonmodifiable risk factors are family history, male sex and advanced age. Modifiable factors are mostly lifestyle-dependent and include smoking, hyperlipidemia, psychosocial factors, abdominal obesity, history of diabetes or hypertension, physical inactivity, and chronically increased markers of inflammation (Yusuf et al., 2004; Culic, 2007).

Abbreviations: BMI, body mass index; CPC, condensation particle counter; GCV, generalized cross-validation criteria; IQR, interquartile range; KORA, Cooperative Health Research in the region of Augsburg; MI, myocardial infarction; MONICA, monitoring of trends and determinants in cardiovascular disease; RR, relative risk; PM, particulate matter; PNC, particle number concentration; STEMI, ST segment elevation MI.

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Transient risk factors have the potential to trigger plaque rupture and thrombosis followed by the onset of MI or other acute coronary syndromes. These include heavy physical exertion, emotional stress, sexual intercourse, coffee or alcohol consumption, but also air pollution. A comparative risk assessment of these transient factors showed that although the individual risk increase in association with air pollution is very small, the magnitude of the risk on a population level is considerable since many people are exposed to this trigger (Nawrot et al., 2011). Additionally, the exposure to elevated air pollution concentrations is often inevitable for the population due to its ubiquitous nature.

Results from previous studies on particulate matter with an aerodynamic diameter <10 μm (PM_{10}) and <2.5 μm ($PM_{2.5}$) and MI were somewhat controversial reporting positive (Peters et al., 2001; Zanobetti and Schwartz, 2005; Pope et al., 2006) but also no associations (Sullivan et al., 2005; Lanki et al., 2006; Berglind et al., 2010; Bhaskaran et al., 2011). One reason might be the small effect size which requires large study populations to reduce the standard errors. A recent review and meta-analysis combining 19 studies on PM_{10} and 13 studies on $PM_{2.5}$ found significant positive associations for both pollutants (Mustafic et al., 2012). Due to their small size ultrafine particles (aerodynamic diameter <0.1 μm) are deposited in the alveoli and may even cross the lung-blood barrier and translocate into the circulation. They derive from fresh industrial and vehicle combustion and have only a short lifetime because they agglomerate and coalesce into larger particles. Since ultrafine particles have only little effect on PM mass but contribute most to the number of particles within PM, particle number concentration (PNC) is often used as a proxy. As their surface area-to-mass ratio is much larger compared to PM_{10} and $PM_{2.5}$, their potential of carrying diverse toxic materials on the surface is high. However, studies on ultrafine particles as trigger for MI are sparse (Forastiere et al., 2005; Peters et al., 2005; von Klot et al., 2005; Lanki et al., 2006; Gardner et al., 2014) and often consider data collected over limited periods. Therefore, the objective of this analysis was to assess the effects of short-term exposure to PM_{10}, $PM_{2.5}$ and PNC on registry-based MI cases and coronary deaths in the area of Augsburg, Germany, for the period 1995–2009.

Materials and methods

MONICA/KORA myocardial infarction registry

The population-based Augsburg MI registry was founded in 1984 as part of the WHO MONICA project and since 1996 has been continued by the Cooperative Health Research in the region of Augsburg (KORA). The registry records all cases of MI and coronary deaths among persons aged 25 to 74 with a principal residence in the city of Augsburg, Southern Germany, and the two adjacent rural districts. Altogether, the study population consists of about 400,000 inhabitants aged 25 to 74 years. According to the MONICA protocol (Tunstall-Pedoe et al., 1994), hospital admissions are continuously monitored and MI patients, who survive at least 24 h, are asked for an interview concerning the event, medication and family history. If the patient deceases between the second and 28th day after admission the MI is considered as fatal, otherwise as nonfatal. Coronary deaths are fatal cases outside the hospital or within the 24 h after admission. They are identified by checking all death certificates within the regional health departments together with information of the last treating physician and/or coroner. While the MI diagnosis was clinically redefined in 2000, we used the MI diagnosis established in 1985 over the whole period for consistency (Löwel et al., 2005). The diagnostic criteria included: chest pain lasting more than 20 min that is not relieved by the administration of nitrates; Q waves on electrocardiographic examination that suggest an evolving myocardial infarction; subsequent increases in the level of creatine kinase, aspartate aminotransferase, or lactate dehydrogenase to more than twice the upper limit of normal, or both. The study was approved by the ethics committee of the Bavarian Chamber of Physicians and performed in accordance with the Declaration of Helsinki.

Air pollution and meteorological data

Particulate air pollutant concentrations were measured at several monitoring stations in the city of Augsburg (Online Supplement Fig. 1). From 1995 to 1999, total suspended particles were measured with a β-absorption device (ESM-Andersen FH 62 I-N) at two fixed urban background sites within the city of Augsburg and scaled down by a factor of 0.83 to derive PM_{10} (von Klot et al., 2005). Afterwards, PM_{10} was directly assessed with the same devices and from 2001 on, additionally with a third monitor. Monitors were averaged with a modified APHEA procedure (Katsouyanni et al., 1996; von Klot et al., 2005). Both $PM_{2.5}$ and PNC were measured at a single urban background site located 1 km north of the city centre until 2003. The site was relocated to 1 km south of the city centre in the beginning of 2004. $PM_{2.5}$ was measured by a Tapered Element Oscillating Microbalance (TEOM model 1400A, Rupprecht and Patashnick, German distributor: MLU, Essen, Germany). PNC was obtained from 1999 to 2003 by a condensation particle counter (CPC 3022A, TSI, Aachen, Germany) and from 2004 to 2009 by a triple-instrument approach combining a Twin Differential Mobility Particle Sizer (TDMPS), and an Aerodynamic Particle Sizer (APS, model 3321, TSI Inc., USA) to measure particles between 10 and 2000 nm which is comparable to the size range of the CPC. For previous years, concentrations were predicted within a regularized linear prediction model based on the measurements for the year 2001 (Paatero et al., 2005). Measured values are indicated with PNC_m and the combined time series of measured and fitted values with PNC_{m+f}. Air temperature, relative humidity, and barometric pressure were measured at an urban background measurement station located 7 km (until 2000) and 5 km south of the city centre (from 2001 on). Until 2004, PM_{10} was only available on a 3-h basis. All other data were available on an hourly basis, and 24-h mean values were calculated if at least 75% of the hourly values were available.

Statistical analyses

Model building

Based on the literature (Forastiere et al., 2005; Zanobetti and Schwartz, 2005), we assumed a log-linear association between air pollutants and daily cases of MI and coronary deaths. We used generalized additive Quasi-Poisson models to accommodate a Poisson distribution with overdispersion. For each pollutant, a separate model was calculated for the exposure of the same day, the day before MI occurrence and up to four days before the event, and the average exposure over five days (mean concentration of same and previous four days). As potential confounders, we considered a global time trend to model long-term changes, temperature of the same and the average of the three previous days to cover the effects of hot and cold days, same day relative humidity and barometric pressure, and seasonal and weekday variations. To model nonlinear confounder effects, we used penalized regression splines to optimize the degree of smoothness. The optimal degree was then kept fix to allow a better comparability when entering the air pollutants. Model selection was based on the reduction of the generalized cross-validation criteria (GCV) and the absolute value of the sum of the partial autocorrelation function (Touloumi et al., 2004). Statistical significance was mandatory for season and weekdays which were included as indicator variables. The final model included a