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Evaluation of short-term mortality attributable to particulate matter pollution in Spain

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ABSTRACT

According to the WHO, 3 million deaths are attributable to air pollution due to particulate matter (PM) world-wide. However, there are no specific updated studies which calculate short-term PM-related cause specific mortality in Spain. The objective is to quantify the relative risks (RRs) and attributable risks (ARs) of daily mortality associated with PM_{10} concentrations, registered in Spanish provinces and to calculate the number of PM-related deaths. We calculated daily mortality due to natural (ICD-10: A00 R99), circulatory (ICD-10: I00 I99) and respiratory causes (ICD-10: J00 J99) for each province across the period 2000–2009. Mean daily concentrations of PM_{10}, NO_2 and O_3 was used. For the estimate of RRs and ARs, we used generalised linear models with a Poisson link. A meta-analysis was used to estimate RRs and ARs in the provinces with statically significant results. The overall RRs obtained for these provinces, corresponding to increases of 10 μ g/m^3 in PM_{10} concentrations were 1.009 (95% CI: 1.006 1011) for natural, 1.026 (95% CI: 1.019 1033) for respiratory, and 1.009 (95% CI: 1.006 1012) for circulatory-cause mortality. This amounted to an annual overall total of 2683 deaths (95% CI: 852 4354) due to natural, 651 (95% CI: 359 1026) due to respiratory, and 556 (95% CI: 116 1012) due to circulatory causes, with 90% of this mortality lying below the WHO guideline values. This study provides an updated estimate of the effect had by this type of pollutant on causes of mortality, and constitutes an important basis for reinforcing public health measures.

1. Introduction

Particulate matter (PM) consists of a complex mix of solid and liquid particles of organic and inorganic substances suspended in the air. The main components of PM are sulphates, nitrates, ammonium, sodium chloride, "black carbon", mineral dust, organic matter and water (Querol et al., 2012). The respirable particles, PM_{10} (aerodynamic diameter of less than 10 microns) and $PM_{2.5}$ (aerodynamic diameter of less than 2.5 microns), are the types of PM which have the greatest health impact (WHO, 2013). According to a recent WHO study (WHO, 2016), in 2012 some 3 million deaths world-wide were estimated to be attributable to PM-related air pollution, with 193,000 of these occurring in Europe and 7000 in Spain.

The health effects of PM are especially well documented, with a distinction being drawn between two types of effects, short- and long-term. Cohort studies designed to detect the long-term effects on population health, link exposure to PM to an increased risk of death (Dockery et al., 1993; Pope et al., 1995; Miller et al., 2007; Beelen et al., 2008a; Ostro et al., 2010), even for very low $PM_{2.5}$ concentrations (Crouse et al., 2012). Although the principal causes of mortality associated with long-term exposure to PM are some types of cancer (Beelen et al., 2008b), recently the International Agency for Research on Cancer (IARC) classified $PM_{2.5}$ as a carcinogen (Loomis et al., 2013); equally notable are its effects on cardiovascular (Brook et al., 2010; Dominici et al., 2006) and respiratory causes (Dominici et al., 2006; Guaita et al., 2011; Kim et al., 2012), with clearly established physiopathological mechanisms (Brook et al., 2010; Rückerl et al., 2011). Recent studies suggest other types of health outcomes, in which PM is associated with other types of diseases (Rückerl et al., 2011). Hence, PM has been found to have an effect on diabetes (Brook et al., 2008), neurological development in children (Freire et al., 2010) and neurological disorders in adults (Ranft et al., 2009).

There are also numerous studies which associate short-term exposure to PM –both PM_{10} and $PM_{2.5}$- with morbidity and

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mortality due to respiratory and cardiovascular causes (Dominici et al., 2006; EPA, 2009; Maté et al., 2010; Brook et al., 2010; Guaita et al., 2011; Rückerl et al., 2011), with a greater effect being detected in older adults (Zanobetti et al., 2000; Aga et al., 2003; Jiménez et al., 2009, 2010), though there are also effects on the childhood population (Schwartz and Neas, 2000; Barnett et al., 2005; Linares and Díaz 2009; Díaz et al., 2004). Furthermore, PM levels have been associated in the short term with adverse birth variables, including foetal mortality (Arroyo et al., 2016).

Unlike the USA, few multi-city studies have been undertaken in Europe (Samoli et al., 2013). Some of the major studies undertaken in Europe to address the effect of PM on morbidity and mortality have been the APHEA project (Katsouyanni et al., 2001; Atkinson et al., 2001), which focused on 29 towns/cities with the main aim of ascertaining the short-term effect of PM on health, and the APHEIS project (Boldo et al., 2006; Ballester et al., 2008), which analysed the long-term health impact of $PM_{2.5}$ in 23 European towns/cities. More recently, the MEDPARTICLES project sought to analyse the short term effect of PM on morbidity and mortality in 12 towns/cities in Mediterranean Europe (Samoli et al., 2013, 2014; Basagaña et al., 2015).

In Spain, the only multicentre study to analyse the association at short term between morbidity and mortality and chemical air pollution, with data obtained from *in situ* measurements, has been the EMECAM-EMECAS. This study was conducted in 16 Spanish towns/cities using pre-1997 data and total suspended particles (TSP) as the PM indicator (Ballester et al., 2002, 2006). More recently, the SERCA Project was implemented in Spain (Boldo et al., 2011), with the aim of ascertaining $PM_{2.5}$—related mortality for Spain as a whole, though this project also displays limitations. On the one hand, the $PM_{2.5}$ values were the result of modelling based on emission sources, i.e., estimated $PM_{2.5}$ concentrations; and, at an epidemiological level, the dose-response functions used were mainly extrapolated from cohort studies (long term effect) undertaken in the USA. In view of the lack of updated multi-city studies based on measured exposure values, with dose-response functions specifically calculated for each city, we present the following study, whose aim was to analyse the impact of PM on short-term mortality in Spain. Using a times series analysis, based on aggregated natural-, circulatory- and respiratory-cause mortality data for all age groups and air pollution data for each of Spain's provinces across the period 2000–2009, relative risks and PM-related risks were calculated for each province, thereby yielding a closer approximation of the impact of PM on the overall Spanish population and making this the European study with one of the widest coverages at the level of the cities analysed to have ever evaluated the short-term impact of PM on daily mortality due to different causes.

2. Material and methods

2.1. Variables used in the analysis

Dependent variable: as the dependent variable, we used daily mortality due to natural (all causes except accidents) (ICD-10: A00-R99), circulatory (ICD-10: I00I99) and respiratory causes (ICD-10: J00-J99) registered in 52 Spanish provinces across the period 2000–2009. In the case of Madrid, the data corresponded exclusively to the Madrid metropolitan area. These data were furnished by the National Statistics Institute (*Instituto Nacional de Estadística/INE*).

Independent variable: the principal independent variable was mean daily PM_{10} concentrations ($\mu g/m^3$) recorded at monitoring stations in each provincial capital from 2000 to 2009. All measurements were made using the gravimetric method or an equivalent method (beta-attenuation). All data were validated and comparable, were supplied by the Ministry of Agriculture & Environment (*Ministerio de Agricultura, Alimentación y Medio Ambiente/MAGRAMA, 2016*). For the cities of Madrid, Las Palmas de Gran Canaria and Santa Cruz de Tenerife, $PM_{2.5}$ values were available in addition to those for PM_{10}.

Control variables: in the analysis, we controlled for the different variables related to the designated study objective, namely:

- other pollutants: we controlled for mean daily concentrations ($\mu g/m^3$) of NO_2 and O_3. These pollutants were measured at the same stations as those which obtained the PM_{10} values, and were likewise supplied by the MAGRAMA.
- meteorological variables: we considered the daily maximum temperatures (T_{max}) and minimum temperatures (T_{min}) at each reference observatory situated in each provincial capital. These data were furnished by the State Meteorological Agency (*Agencia Estatal de Meteorología/AEMET*).
- other control variables: we controlled for the presence or absence of influenza epidemics. This variable was introduced dichotomously, with a value = 1 when there was an epidemic and a value = 0 when there was no epidemic. This information was supplied by the National Centre of Epidemiology at the Carlos III Institute of Health.

Similarly, we took into account the trend of the series, day of the week, and annual, six-monthly and three-monthly seasonalities were taken into account, using the sine and cosine functions of the periods of 365, 180 and 90 days respectively. In addition we also controlled for the autoregressive nature of the dependent variable.

2.2. Transformation of variables

Lagged variables: multiple studies have shown that the effect of air pollution on short-term mortality may not be immediate, but that this effect can occur up to 5 days afterwards in the case of PM and NO_2 (Díaz et al., 1999; Maté et al., 2010) and up to 9 days afterwards in the case of O_3 (Díaz et al., 1999). In the case of temperatures, the lagged effect on mortality can be delayed up to 4 days in the case of heat (Alberdi et al., 1998; Díaz et al., 2002, 2015) and up to 13 days in the case of cold (Alberdi et al., 1998; Carmona et al., 2016). To take these impacts into account, we created the corresponding lagged variables for each of the above-mentioned variables.

Non-linear control variables: previous studies have shown that the functional relationship between ozone and mortality is not linear, displaying a U-shaped pattern, with the right-hand side of the U corresponding to the increase in mortality associated with high ozone values (O_{3h}) (Díaz et al., 1999). The minimum value of the quadratic function (U), i.e., the threshold ozone value ($O_{threshold}$), varies from one city to another and was determined for each provincial capital in previous studies (Ortiz et al., 2016). The variable O_3 in each province included in the modelling was therefore parameterised as follows:

$O_3 = 0$ if $O_3 < O_{threshold}$

$O_{3h} = O_3 - O_{threshold}$ if $O_3 > O_{threshold}$

Similarly, it is widely known that temperature displays a U-shaped relationship with mortality (Alberdi et al., 1998), in which the left-hand side corresponds to the effect of low temperatures and the right-hand side to the effect of high temperatures. This effect of heat and cold on mortality is exacerbated in so-called heat and cold waves. Determination of the threshold temperatures