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RESEARCH ARTICLE

Association between atmospheric pollutants and hospital admissions in Lisbon

A. M. J. Cruz · S. Sarmento · S. M. Almeida · A. V. Silva · C. Alves · M. C. Freitas · H. Wolterbeek

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Abstract Ambient air pollution is recognised as one of the potential environmental risk factors causing health hazards to the exposed population, demonstrated in numerous previous studies. Several longitudinal, ecological and epidemiological studies have shown associations between outdoor levels of outdoor atmospheric pollutants and adverse health effects, especially associated with respiratory and cardiovascular hospital admissions. The aim of this work is to assess the influence of atmospheric pollutants over the hospital admissions in Lisbon, by Ordinary Least Squares Linear Regression. The pollutants (CO, NO, NO_2, SO_2, O_3, PM_{10} and $PM_{2.5}$) were obtained from 13 monitoring stations of the Portuguese Environmental Agency, which provide hourly observations. Hospital admission data were collected from the Central Administration of the Health System and were compiled by age: <15, 15–64, >64 years old. The study period was 2006–2008. Results showed significant positive associations between the following: (1) the pollutants CO, NO, NO_2, SO_2, PM_{10} and $PM_{2.5}$ and circulatory diseases for ages between 15 and 64 years (0.5 % hospital admissions (HA) increase with 10 μg m^{-3} NO increase) and above 64 years (1.0 % stroke admission increase with 10 μg m^{-3} NO_2 increase); (2) the pollutants CO, NO, NO_2, SO_2, PM_{10} and $PM_{2.5}$ and respiratory diseases for ages below 15 years (up to 1.9 % HA increase with 10 μg m^{-3} pollutant increase); and (3) the pollutants NO, NO_2 and SO_2 and respiratory diseases for ages above 64 years (1.3 % HA increase with 10 μg m^{-3} CO increase).

Keywords Air pollutants · Hospital admissions · Health effects · Respiratory diseases · Cardiovascular diseases

Introduction

Links between air pollution, especially atmospheric particles and sulphur dioxide—SO_2, and the number of hospital admissions have been established in studies in North America (Barnett 1997; Lipfert 1997; Middleton et al. 2008; Pope et al. 2004, 2008; Roemer et al. 1998; Zanobetti and Schwartz 2005; Wilson et al. 2004), South America (Lumley and Sheppard 2000; Roberts 2005), Asia (Cameron and Trivedi 1998; Luvsan et al. 2012; Ritchie and Lehnen 2004), Australia (Hansen et al. 2012) and Europe (Almeida et al. 2014; Alves et al. 2010; Freitas et al. 2010; Pablo Dávila et al. 2013; Pascal et al. 2013; Neuberger et al. 2007; Ayres-Sampaio et al. 2014). However, still many efforts to clarify pollution-health associations are necessary, including the separation of the short-term and long-term health effects of individual air pollutants and those of complex pollutant mixtures. Additionally, more research is needed to infer specific regional links between air pollution and adverse health effects. As the composition of the air pollution mixture differs between locations, the health risks associated with the pollutant of interest

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A. M. J. Cruz (✉)
Escola Superior de Tecnologia e Gestão de Oliveira do Hospital, Instituto Politécnico de Coimbra, Rua General Santos Costa, 3400-124 Oliveira do Hospital, Portugal
e-mail: ana.cruz@estgoh.ipc.pt

S. Sarmento · S. M. Almeida · A. V. Silva · M. C. Freitas
Centro de Ciências e Tecnologias Nucleares, Instituto Superior Técnico, Universidade de Lisboa, Estrada Nacional 10, 139.7 km, 2695-066 Bobadela LRS, Portugal

A. M. J. Cruz · H. Wolterbeek
College of Applied Sciences, Department of Radiation, Radionuclides and Reactors, Section RIH, Technical University of Delft, Delft, The Netherlands

C. Alves
CESAM, Universidade de Aveiro, 3810-193 Aveiro, Portugal

may vary over time and space (Almeida et al. 2011; Shin et al. 2008).

Recent studies, in Spain, demonstrate that the levels of the finest PM fractions are important risk factors for daily cardiovascular mortality (Máté et al. 2010; Perez et al. 2012) and increase the number of hospital admissions (Linares and Díaz 2010; Linares et al. 2010). Including hospital admissions in studies related to the short-term effects of air pollution on human health is particularly valuable since such studies may refer to large parts of the population and, consequently, constitute the most direct way of accessing the cardiorespiratory health burdens imposed by the complex air pollution mixtures to the exposed communities (Delfino et al. 2009). According to this assumption, a lot of remarkable studies have been conducted in order to define the association among one or more pollutants and the number of daily hospital admissions (Atkinson et al. 2001; Kan and Chen 2004; Katsouyanni et al. 2001; Moshammer et al. 1987, 2006; Morawska et al. 2002; Neuberger et al. 2007, 2013). Apart from particulates, ozone (O_3) and a wider range of pollutants, including monoxide carbon (CO), nitrogen dioxide (NO_2) and SO_2, play a very significant role in the public health decline (Katsouyanni et al. 1997; McConnel et al. 2002). Furthermore, most models only estimate the short-run impact of air pollution and do not account for the possibility that a high number of hospital admissions on a certain day might be followed by an offsetting reduction in the number of hospital admissions spread over the following weeks and months (Maddison 2005). Recognising the importance of this issue, some researchers (Schwartz 2000a, 2001; Zeger et al. 1999) have thought to provide what they refer as 'harvesting resistant' estimates of the health effects of air pollution. The approach adopted by Schwartz (2001) involves analysing the association between 15-day moving averages of air pollution and mortality counts and hospital admissions. Adopting an alternative approach, Zanobetti et al. (2002) used polynomial lags to explore the short-term mortality displacement issue. Based on the above remarks, it is obvious that the question of how to best aggregate exposure and health variables over time and estimate the unique effect of single pollutants remains an open issue, given that there is a lot of speculation about the suitability of the applied methodologies, or the special conditions of the urban area under study.

Time-series analysis usually performed linear regression when data are normally distributed and, more recently, Poisson regression. Usually, models include only one pollutant. Additional covariates denote, on the one hand, temporal patterns in diseases (long-term trends, seasonality and weekday trends) and, on the other hand, meteorological indicators and seasonal indicators (Smith et al. 2000). Most time-series studies use days as the units of comparison which is the minimum time period for which hospitalisation data are recorded. Such data usually contains very low counts of health events, it is thus very noisy and it is necessary to adjust for weekly averages as well as consider potential lag effects. The daily data may consist of the actual daily values of health and pollution or some form of average over a number of days. Some studies have modelled single day health events with exposure averages over multiple days (Lipfert 1993; Lumley and Sheppard 2000; Roberts 2005; Sarmento et al. 2009; Smith et al. 2000) and a minority has modelled the health events also as averages over multiple days, while preserving the days as units of comparison (Schwartz 2000a, b).

Recent epidemiological studies have consistently shown positive associations between low-level exposure to air pollutants and health outcomes. In Portugal, very few studies have analysed the acute effect of air pollutants on public health (Almeida et al. 2011, 2014; Alves et al. 2010; Freitas et al. 2009). In this study, an attempt is made to find the short-term associations between hospital admissions for diseases: all circulatory, cardiac, ischaemic heart, stroke, all respiratory diseases, asthma and air pollutants: CO, nitrogen monoxide—NO, NO_2, SO_2, O_3, particles with aerodynamic equivalent diameter of 10 μm—PM_{10} and particles with aerodynamic equivalent diameter of 2.5 μm—$PM_{2.5}$, in the area of Lisbon, Portugal, for a 3-year period, from 2006 to 2008.

Data

Lisbon is the capital and largest city of Portugal, with a population of 564,657 within its administrative boundaries on a land area of 84.8 km^2. Lisbon is the westernmost large city located in Europe, as well as the westernmost capital city and the only one along the Atlantic coast (Almeida et al. 2010). The urban area of Lisbon extends beyond the administrative city limits and this study includes the neighbour municipalities of Loures, Odivelas, Amadora, Oeiras and Cascais, with a total area of 448 km^2 and a resident population of 1,378,868 inhabitants (average over 2006–2008) (Fig. 1).

The industrial area includes textiles, chemicals, steel, oil, cement, sugar refining, shipbuilding, soap and flour production (Almeida et al. 2007, 2013a; Farinha et al. 2004). In the city, traffic is the main source of atmospheric pollution (Almeida et al. 2009a, b). Due to the geographic position of Lisbon and to the dominant western wind regime, influenced by the presence of the semi-permanent Azores high-pressure and the Icelandic low-pressure systems over the North Atlantic Ocean, the expected high levels of pollutants are uncommon. The transport of maritime air mass is usually associated with cleaner air masses from the Atlantic Ocean and with better dispersion conditions of pollutants coming from the industrial areas (Almeida et al. 2013b). Nevertheless, under adverse meteorological conditions, low dispersion conditions and thermic inversions, high concentrations of air pollutants (AP) can be registered.

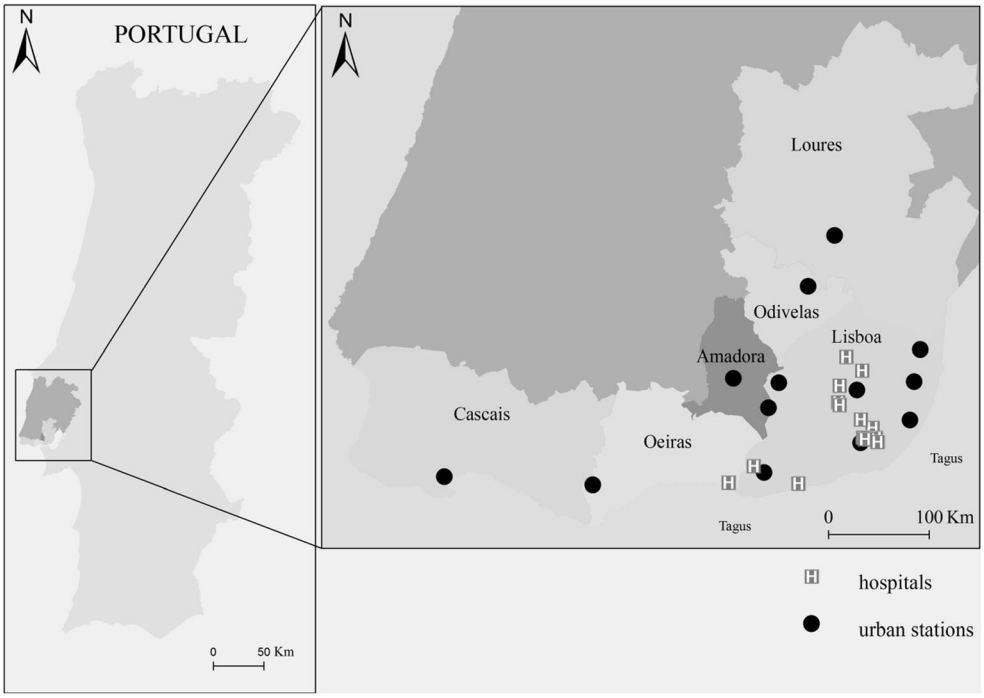


Fig. 1 The studied area: spatial distribution of the main hospitals and air quality monitoring stations

In this study, the measuring stations to be considered are in municipalities whose population is served by the hospitals where the hospital admissions data (HA) were collected.

Air pollutants

Data on regulated AP (CO, NO, NO_2, SO_2, O_3, PM_{10} and $PM_{2.5}$) were obtained for all the monitoring urban stations in the studied area: in Loures, Odivelas, Cascais and Oeiras one station each, in Amadora two stations and Lisbon seven stations. All the stations are considered as background (areas not directly influenced by traffic) stations except for the one in Odivelas, Cascais and three in Lisbon with intense traffic. This monitoring network is assumed to represent the exposure of the population served by the 13 hospitals from which health data were collected.

The data, provided by the Portuguese Environmental Agency (QUALAR network), consisted of hourly measurements, over the years 2006–2008. Daily averages were calculated for each pollutant in each of the 13 monitoring stations following the recommendations of Directive 2008/50/CE of 21 May 2008. Daily values were calculated as the 24-h average for SO_2, PM_{10} and $PM_{2.5}$, as the daily 1-h maximum for NO and NO_2; and as 1-h maximum of octo-hourly moving averages for CO and O_3. Daily values were calculated only when at least 75 % of the hourly values on a particular day were non-missing.

The daily values of each pollutant were then averaged over the 13 monitoring stations as a criteria to obtain a daily single value of AP which may represent a daily pollutant measure for the whole studied area.

Hospital admissions

The HA database was provided by the Central Administration of the Health System (ACSS). It consists of daily counts of hospitals admissions, over the years 2006–2008 in 13 public hospitals, all in Lisbon municipalities (Fig. 1). In HA database, it was observed that hospital admission counts tend to be lower on weekends as compared to working days, as already concluded by Sarmento et al. (2009), suggesting that either weekend admissions are registered on Mondays only or patients wait for Monday to go to hospital if the symptoms are not so severe or the number of doctors in hospital is reduced during weekend and people may wait to Monday to be assisted. This is visible in Fig. 2 which shows a HA week study for the diseases under study from 2006 to 2008.

Daily HA database were aggregated by age group (<15, 15–64, >64 years old). These age groups were adopted for consistency with former studies in Portugal (Sarmento et al. 2009; Almeida et al. 2014). The studied causes of HA classified according to the World Health Organisation's International Statistical Classification of Diseases by the, 9th Revision (ICD-9) and 10th Revision (ICD-10) endorsed by the Forty-third World Health Assembly in May 1990, came into use in WHO Member States since 1994 (WHO 2014): all circulatory (ICD-9, 390–459; ICD-10, I00–I99), cardiac (ICD-9, 390–429; ICD-10, I30-I52), ischaemic heart (ICD-9, 410–414; ICD-10, I20-I25), stroke (ICD-9, 430–438; ICD-10,

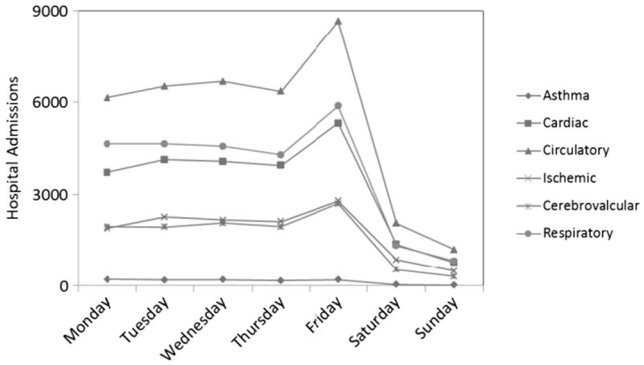


Fig. 2 Weekly study of hospital admissions (HA), for all diseases, from 2006 to 2008

I60–I69); all respiratory (ICD-9, 460–519; ICD-10, J00–J99), asthma (ICD-9, 493; ICD-10, J45).

Methodology

In an attempt to better understand the effect of the pollutants on HA for the different diseases, four different ways of aggregating atmospheric pollutants and hospital admission variables were considered over time. Studies on human health effects usually use daily data; although, recent epidemiological studies have been using exposures in the form of moving averages, distributed lags or data aggregated over several days or even weeks (Schwartz 2000b; Sarmento et al. 2009).

In this study, we followed the methodology applied by Sarmento et al. (2009) and Almeida et al. (2014). The AP and HA daily databases were joined in into one (DAY database—*N* equals 1,096), and from it, we generated other three differently aggregated databases:

WEEK (both AP an HA are expressed as 7-day averages). The number of observations is thus seven times less than the number of observations in the DAY database (*N* reduced from 1,096 to 156);

O&MA (HA are expressed with the original daily values, AP are expressed as a prior 7-day moving average, this is the seventh HA value connects with the average of the first seven AP values; the HA eighth value connects with the average of the AP seven values, from the second to the eight—moving average—from AP and so on. The number of observations is thus equal to the number of observations in the DAY database minus 6);

MA&MA connects the average from the 8th to the 14th HA values with the average from the 1st to the 7th AP value. The next value results from the average from the 9th to the 15th HA value with the average from the 2nd to the 8th AP value, and so on. The number of observations is thus equal to the number of observations in the DAY database minus 12.

WEEK and MA&MA databases aimed to control the effect of weekday on HA, to mitigate the influence of potential peaks on the evaluation of associations, and to account for lagged health responses which was considered to be 7 days (Sarmento et al. 2009; Almeida et al. 2014).

Averages of 7 days were selected in agreement with Sarmento et al. (2009) and Almeida et al. (2014) because 7 days is still considered an acute time scale for associations (WHO 1999, 2006), and averages of 7 days in both AP and HA can adjust for biases arising from weekly fluctuations.

To control the seasonal variability of AP data, 12 dummy variables (one per month) were added. The dummy variables took either the zero value or the average value of the daily observations of each month (considering all the years). For instance, the dummy variable for January assumed the value of zero for all observations, except for January, for which the month averages were considered; the same applies for February, March and so on.

Weekend effects that were over HA data were controlled, in models DAY and O&MA (not necessary in WEEK and MA&MA because already week aggregated), by including a weekend dummy, zero on weekends and 1 on weekdays.

Ordinary Least Squares Linear Regression (OLS) was applied to DAY, WEEK, O&MA and MA&MA databases and was performed with Excel 2007 and SPSS 17.0 software packages. In OLS, HA and AP were the dependent and independent variables, respectively. OLS was applied to be consistent with similar former studies in Portugal (Sarmento et al. 2009; Almeida et al. 2014).

Pearson correlation was applied to verify linearity of models following the requirements of OLS: (1) the normality of residuals was observed in these histograms; (2) the constant variance of residuals was checked using appropriate graph (scatter plot between standard and variable waste); (3) zero waste covariance was verified by the Durbin-Watson test.

The associations obtained with OLS and Poisson regression for the WEEK database were compared.

Results and discussion

All AP present a seasonal pattern with high winter levels and low summer levels, except O_3 (not shown). The summer higher values of O_3 could be explained by the adequate weather conditions for its formation—sunlight, warm temperatures and high emission of precursor pollutants (nitrogen oxides and volatile organic compounds) lead to high levels of this atmospheric oxidant during the summer season (Karr et al. 2007; Alves et al. 2010).

The AP descriptive statistics is shown in Table 1. Comparing with the Europe Union air quality guidelines suggested in Directive 2008/50/EC, it is possible to verify that, over the period 2006–2008: (1) CO average annual concentration did not exceed the maximum daily 8-h average (10 mg m^{-3}); (2) NO_2 and PM_{10} average annual concentrations exceeded the annual limit of 40 μg m^{-3}; (3) SO_2 concentrations were very low over the studied period as compared to the daily average legal value (125 μg m^{-3}—not to be exceeded more than 3 days per year); (4) O_3 concentrations were under the maximum of octo-hourly of 120 μg m^{-3} (not to be exceeded more than 25 days per year); (5) daily averages of PM_{10} concentrations exceeded the limit value of 50 μg m^{-3} in 2006 (58 times) and 2007 (37 times), although PM_{10} concentrations decreased to values under the daily limit established by the Directive; (6) $PM_{2.5}$ annual concentrations were under the annual limit established by the Directive (25 μg m^{-3}). Comparing AP descriptive statistics with Alves et al. (2010) in 1999–2004 for the same area, all AP decreased, except O_3 and NO_2; the standard deviations were in the same order of magnitude. Average and median values are similar indicating a reasonable normality of the data. Histograms are included in the Table 1. Differences between minima and maxima are quite large, as it is expected in AP values; consequently, standard deviations are high too. Table 1 also shows the statistical values for winter and summer. Only summer and winter are shown because these seasons have the extreme climacteric conditions considering temperature and humidity. The seasonality is evident.

Statistics for the daily HA in the studied area is presented in Table 2. Except for asthma, higher values were recorded for people >64 than for <64 years old. For asthma, annual HA do not appear to vary much with age. Averages and medians are mostly different for circulatory and cardiac diseases, which show bimodal histograms. For these diseases, either there are very low HA values or very high HA values. This may already give an indication of high associations with AP peaks. Stroke and respiratory diseases are also bimodal but not so sharp as the previous ones. Ischaemic heart and asthma diseases may be considered normal. Table 2 also shows the statistical values for winter and summer. Seasonal variation appears to be evident.

Table 3 shows the associations that were significant ($p<0.05$) at least for one model (DAY, WEEK, O&MA, MA&MA) after applying OLS, as well as their semi-elasticity, expressed in percent of variation in HA per 10 μg m^{-3} increase in the atmospheric pollutants. They are 37 out of a total of 126 (6 HA × 3 age groups × 7 AP) associations. It was observed that statistical significance and semi-elasticity were highly sensitive to the study design issues defined by the four models.

According to Lipfert (1993, 1997), the semi-elasticity at the average of both atmospheric pollutant and hospital admission was calculated from the risk difference provided by the slope of the linear regression. The use of the semi-elasticity makes it easier to compare our results, obtained from a linear model, with those obtained from time-series studies using Poisson (Cameron and Trivedi 1998) and log-linear models, which use the Risk Ratio as the measure of effect.

Comparing the results of OLS and Poisson for the WEEK database, we obtained similar results even considering that OLS get more associations than the Poisson regression. For instance, the increase of 10 μg m^{-3} in Lisbon of the PM_{10} was associated with an increase in circulatory diseases, for 15–64 years of the 0.6 % with OLS while Poisson was 1 % and an increase in cardiac diseases, for 15–64 years, 0.7 and 1 %, respectively. The increase of 10 μg m^{-3} of the $PM_{2.5}$ was associated with an increase in stroke diseases, over 64 years, is respectively 0.9 and 0.6 % and an increase in cardiac diseases, and for 15–64 years of the 0.6 and 0.3 %, respectively. Sarmento et al. (2009) has already concluded the same for O&MA and MA&MA databases.

Comparing the models applied, more numerous statistically significant associations with MA&MA were observed, followed by the DAY models, decreasing to about half in WEEK and O&MA models. The fact that the WEEK model yields a lower number of significant associations can simply be due to a much smaller number of observations (N=156) than in all other models (N around 1,096).

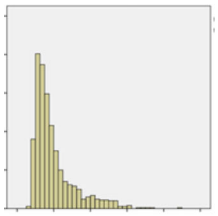
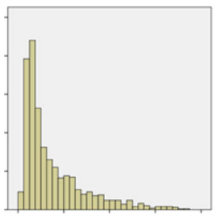
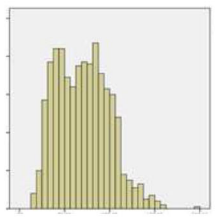
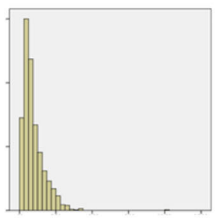
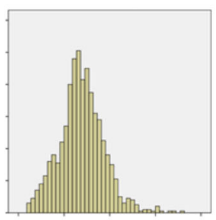
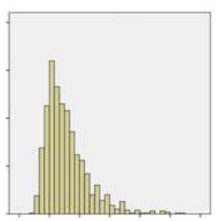
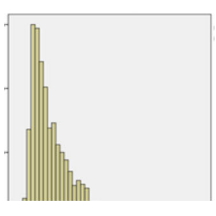
The results in Table 3 indicate positive significant associations: (1) between CO, NO, NO_2, SO_2, PM_{10}, $PM_{2.5}$ and respiratory diseases for ages below 15 years; (2) CO and stroke and ischaemic above 64 years; NO and cardiac, circulatory, and ischaemic diseases for ages between 15 and 64 years; (3) cardiac, stroke, circulatory, ischaemic and respiratory above 64 years; NO_2 and ischaemic diseases for ages between 15 and 64 years, and stroke, circulatory and respiratory diseases above 64 years; (4) SO_2 and cardiac, circulatory and ischaemic diseases ages between 15 and 64 years, and stroke, circulatory, ischaemic and respiratory diseases above 64 years; (5) PM_{10} and cardiac, stroke, circulatory and ischaemic diseases for ages between 15 and 64 years, and stroke and ischaemic diseases above 64 years; (6) $PM_{2.5}$ and cardiac and circulatory diseases for ages between 15 and 64 years, and stroke and ischaemic diseases above 64 years.

The most relevant results (p=0.000 and highest E (%)) in Lisbon were obtained in MA&MA model, in which the increase of 10 μg m^{-3} in NO_2 and PM_{10} concentrations was associated with an increase in HA of about 1.9 % due to respiratory causes, for individuals under 15 years.

The association between NO and HA due to respiratory diseases for individuals under 15 years was significant for all applied models. Results showed that an increase of 10 μg m^{-3} in this pollutant concentration is associated with an increase in HA of 1.2, 1.8, 1.8 and 0.9 %, for the different models. This shows that roughly 1 to 2 % effect might be supported.

The significance of the associations between other AP and HA due to the different diseases depends of the applied model.

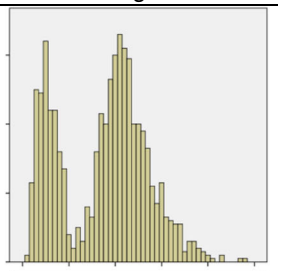
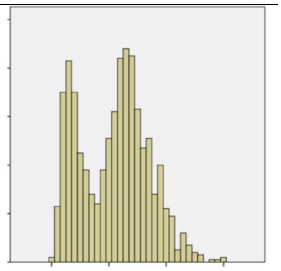
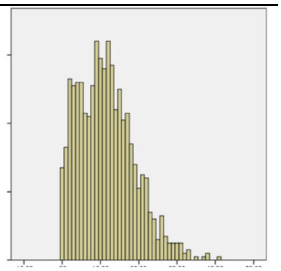
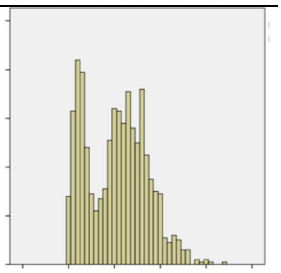
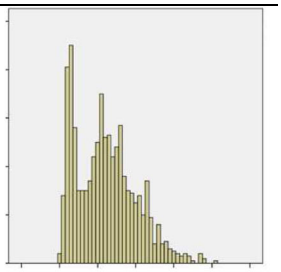
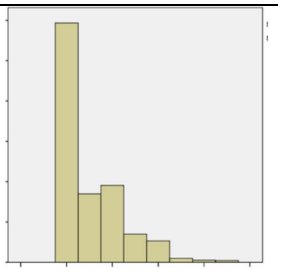
Table 1 Summary statistics of AP daily average concentrations (μg m^{-3}) for the period 2006–2008

Pollutants	Year	Average	Median	SD	Min.	Max.	Histogram
CO	2006	522.82	420.55	303.29	213.64	2,229.93	
	Winter/summer station	796.24 363.93	728.06 328.56	360.51 151.67	269.05 213.64	2,229.93 1,305.45	
	2007	516.16	419.29	294.48	184.63	1,688.17	
	Winter/summer station	810.30 311.36	744.37 288.01	328.92 83.05	309.62 184.63	1,688.17 717.04	
	2008	449.86	373.02	245.21	170.51	1,549.73	
	Winter/summer station	677.46 282.03	635.17 267.80	277.53 62.00	274.66 170.51	1,549.73 484.48	
	2006–2008	496.24	401.78	283.79	170.51	2,229.93	
	Winter/summer station	761.10 319.11	707.22 294.23	328.58 111.08	269.05 170.51	2,229.93 1,305.45	
NO	2006	78.61	51.57	69.38	6.83	351.57	
	Winter/summer station	134.57 39.75	113.00 30.06	83.58 27.94	19.95 6.83	351.57 140.54	
	2007	80.05	50.78	71.34	10.67	346.49	
	Winter/summer station	138.62 38.62	122.14 31.54	84.01 27.42	12.84 10.67	346.49 197.20	
	2008	66.62	40.58	63.30	7.98	367.67	
	Winter/summer station	115.78 27.31	98.48 24.83	77.91 13.79	12.82 7.98	367.67 101.19	
	2006–2008	75.08	46.40	68.29	6.83	367.67	
	Winter/summer station	129.62 35.22	112.85 28.55	82.25 24.55	12.82 6.83	367.67 197.20	
NO_2	2006	70.17	68.94	28.32	17.72	158.22	
	Winter/summer station	90.02 56.12	91.37 46.35	23.91 28.06	38.19 17.72	158.22 153.71	
	2007	73.87	72.25	30.75	18.28	194.60	
	Winter/summer station	93.17 50.93	90.79 46.62	26.71 23.66	25.81 18.28	194.60 153.62	
	2008	67.49	66.18	28.94	14.67	148.29	
	Winter/summer station	89.44 44.24	87.74 38.35	24.47 18.88	31.62 14.67	148.29 104.99	
	2006–2008	70.51	69.32	29.44	14.67	194.60	
	Winter/summer station	90.87 50.43	90.67 42.50	25.04 24.26	25.81 14.67	194.60 153.71	
SO_2	2006	2.46	2.01	1.81	0.09	20.26	
	Winter/summer station	2.97 2.30	2.80 1.98	2.32 1.51	0.09 0.41	20.26 6.80	
	2007	2.09	1.72	1.40	0.21	8.26	
	Winter/summer station	2.64 1.77	2.28 1.44	1.64 1.37	0.21 0.35	8.06 8.26	
	2008	1.23	0.99	0.92	0.15	5.39	
	Winter/summer station	1.67 0.85	1.36 0.71	1.12 0.57	0.35 0.15	5.34 4.35	
	2006–2008	1.93	1.48	1.51	0.09	20.26	
	Winter/summer station	2.42 1.64	1.94 1.12	1.84 1.36	0.09 0.15	20.26 8.26	
O_3	2006	68.93	67.06	27.04	13.63	181.19	
	Winter/summer station	47.92 88.30	49.63 80.67	17.18 27.79	15.43 36.58	85.57 181.19	
	2007	70.20	70.85	24.56	10.74	145.62	
	Winter/summer station	45.78 81.29	43.60 78.31	17.58 18.88	10.74 45.60	83.84 145.62	
	2008	69.15	68.25	21.64	12.19	153.09	
	Winter/summer station	49.79 80.70	52.97 77.28	13.89 20.89	12.19 39.28	80.12 153.09	
	2006–2008	69.43	68.30	24.49	10.74	181.19	
	Winter/summer station	47.84 83.43	49.57 78.37	16.33 23.04	10.74 36.58	85.57 181.19	
PM_{10}	2006	34.87	30.98	16.50	10.72	106.55	
	Winter/summer station	39.25 34.69	35.44 28.61	15.43 19.91	13.01 10.72	86.67 106.55	
	2007	32.69	30.50	13.80	10.02	107.77	
	Winter/summer station	39.30 25.96	38.18 24.78	17.06 9.16	11.80 10.02	107.77 57.11	
	2008	26.78	24.26	10.62	9.98	81.97	
	Winter/summer station	31.24 22.58	27.96 20.67	13.00 7.81	13.59 9.98	81.97 50.55	
	2006–2008	31.44	27.90	14.25	9.98	107.77	
	Winter/summer station	36.58 27.74	32.90 24.20	15.67 14.34	11.80 9.98	107.77 106.55	
$PM_{2.5}$	2006	16.13	12.60	10.40	2.66	62.13	
	Winter/summer station	21.44 15.04	19.03 12.60	11.85 9.95	5.71 2.66	62.13 46.45	
	2007	16.09	13.62	10.19	3.05	72.14	
	Winter/summer station	22.02 10.83	19.51 8.64	12.62 6.19	5.25 3.05	72.14 35.78	
	2008	11.89	9.67	7.17	2.96	49.65	
	Winter/summer station	16.02 8.82	14.04 7.29	8.83 4.46	4.88 2.96	49.65 23.51	
	2006–2008	14.70	11.79	9.57	2.66	72.14	

In histograms, *x* represents the daily averages AP concentrations and *y* their frequencies

SD standard deviation of the average

Table 2 Summary statistics of daily HA values for the period 2006–2008

Disease	Age	Year	Count.	Average	Med.	SD	Min.	Max.	Histogram
All Circulatory	All ages	2006–2008	3,7622	11.44	6	13.72	0	75	
		Winter/summer	83 59	0.23 0.16	0 0	0.92 0.40	0 0	13 2	
	< 15	2006–2008	186	0.17	0	0.41	0	2	
		Winter/summer	61 59	0.17 0.16	0 0	0.42 0.40	0 0	2 2	
	15–64	2006–2008	9580	8.74	9	5.60	0	30	
		Winter/summer	3,285 3,059	9.10 8.29	9 9	6.01 5.16	0 0	28 23	
	≥ 64	2006–2008	27856	25.42	28	14.27	1	75	
		Winter/summer	10,026 8,762	27.77 23.75	31 26	15.42 13.25	1 2	74 54	
Cardiac	All ages	2006–2008	23,314	7.09	4	8.67	0	50	
		Winter/summer	49 38	0.13 0.10	0 0	0.67 0.32	0 0	10 2	
	< 15	2006–2008	115	0.10	0	0.32	0	2	
		Winter/summer	33 38	0.09 0.10	0 0	0.29 0.32	0 0	1 2	
	15–64	2006–2008	5,902	5.39	5	3.67	0	21	
		Winter/summer	2,080 1,844	9.10 8.29	9 9	6.01 5.16	0 0	28 23	
	≥ 64	2006–2008	17297	15.78	17	9.21	0	50	
		Winter/summer	6,300 5,381	17.45 14.58	18 15	10.03 8.35	0 0	48 34	
Ischaemic Heart	All ages	2006–2008	12610	3.84	3	3.72	0	25	
		Winter/summer	545 464	1.50 1.26	1 1	1.45 1.25	0 0	7 6	
	< 15	2006–2008	1,456	1.33	1	1.33	0	8	
		Winter/summer	535 464	1.48 1.26	1 1	1.44 1.25	0 0	7 6	
	15–64	2006–2008	3,818	3.48	3	2.59	0	17	
		Winter/summer	1,406 1,160	3.89 3.14	4 3	2.83 2.36	0 0	17 11	
	≥ 64	2006–2008	7,336	6.69	6	4.29	0	25	
		Winter/summer	2,756 2,199	7.63 5.96	7 6	4.81 3.68	0 0	25 23	
Stroke	All ages	2006–2008	11,477	3.49	1	4.62	0	27	
		Winter/summer	16 7	0.04 0.02	0 0	0.25 0.14	0 0	3 1	
	< 15	2006–2008	31	0.03	0	0.17	0	2	
		Winter/summer	12 7	0.03 0.02	0 0	0.19 0.14	0 0	2 1	
	15–64	2006–2008	2,741	2.50	2	2.12	0	11	
		Winter/summer	919 921	2.55 2.50	2 2	2.22 2.01	0 0	9 9	
	≥ 64	2006–2008	8,705	7.94	8	5.18	0	27	
		Winter/summer	3,097 2,795	8.58 7.57	9 8	5.58 4.92	0 0	27 22	
All Respiratory	All ages	2006–2008	26,180	7.96	5	8.65	0	55	
		Winter/summer	1,778 667	4.88 1.81	4 1	3.50 1.67	0 0	18 9	
	< 15	2006–2008	3366	3.07	2	2.88	0	18	
		Winter/summer	1,764 667	4.89 1.81	4 1	3.50 1.67	0 0	18 9	
	15–64	2006–2008	5,734	5.23	5	4.10	0	24	
		Winter/summer	2,327 1,611	6.45 4.37	6 4	4.69 3.41	0 0	24 17	
	≥ 64	2006–2008	17,080	15.58	15	10.48	0	55	
		Winter/summer	7,037 4,569	19.49 12.38	19 13	12.42 7.45	0 0	54 31	
Asthma	All ages	2006–2008	1,046	0.32	0	0.63	0	4	
		Winter/summer	139 68	0.38 0.18	0 0	0.61 0.45	0 0	3 2	
	< 15	2006–2008	326	0.30	0	0.55	0	3	
		Winter/summer	139 68	0.39 0.18	0 0	0.61 0.45	0 0	3 2	
	15–64	2006–2008	430	0.39	0	0.69	0	4	
		Winter/summer	182 112	0.50 0.30	0 0	0.78 0.62	0 0	4 4	
	≥ 64	2006–2008	290	0.26	0	0.54	0	3	
		Winter/summer	136 67	0.38 0.18	0 0	0.65 0.44	0 0	3 2	

In histograms, *x* represents HA values and *y* their frequencies

SD standard deviation of the average

Table 3 Statistically significant ($p<0.05$) positive relationships between atmospheric pollutants and hospital admissions, at least in one of the four models (DAY, WEEK, O&MA, MA&MA)

Pollutant	Disease	Age	DAY *p* (B)	WEEK	O&MA	MA&MA	DAY *E* (%)[a]	WEEK	O&MA	MA&MA
CO	Stroke	>64	–	0.045	–	0.000		0.919	–	0.940
	Ischaemic	>64	–	–	0.044	0.023	–	–	1.167	0.450
	Respiratory	<15	–	0.031	0.003	0.000	–	2.104	2.390	1.311
NO	Cardiac	15–64	0.006	–	–	–	0.639	–	–	–
		>64	0.013	–	–	–	0.414	–	–	–
	Stroke	>64	0.045	–	0.017	0.000	0.407	–	0.878	0.674
	Circulatory	15–64	0.020				0.469	–	–	–
		>64	0.011		0.045	0.000	0.377	–	0.543	0.277
	Ischaemic	15–64	0.016	–	–	0.009	0.655	–	–	0.463
		>64	0.026	–	–	0.029	0.483	–	–	0.291
	Respiratory	<15	0.000	0.007	0.001	0.000	1.264	1.756	1.786	0.924
		>64	0.050	–	–	0.010	0.361	–	–	0.309
NO_2	Stroke	>64	0.016	–	–	0.000	1.007	–	–	0.862
	Circulatory	>64	0.005	–	–	–	0.861	–	–	–
	Ischaemic	15–64	0.000	–	–	–	2.031	–	–	–
	Respiratory	<15	0.006	0.014	0.006	0.000	1.728	2.860	2.713	1.945
		>64	0.006	–	–	0.000	1.037	–	–	0.847
SO_2	Cardiac	15–64	0.019	–	–	0.046	0.538	–	–	0.212
	Stroke	>64	0.037	0.010	–	0.000	0.416	0.607	–	0.428
	Circulatory	15–64	–	–	–	0.000	–	–	–	0.320
		>64	–	0.028	–	–	–	0.341	–	–
	Ischaemic	15–64	0.017	0.016	0.002	0.000	0.638	0.918	1.174	0.881
	Ischaemic	>64	0.006	0.002	0.001	0.000	0.580	0.878	0.964	0.639
	Respiratory	<15	0.002	0.002	0.003	0.000	0.900	1.557	1.267	0.913
		>64				0.001	–	–	–	0.300
PM_{10}	Cardiac	15–64	0.019	–	–	0.009	0.877	–	–	0.454
	Stroke	15–64	–	–	–	0.002	–	–	–	0.769
		>64	0.014	0.008	0.021	0.000	0.792	1.043	1.076	0.761
	Circulatory	15–64	0.047			0.000	0.640	–	–	0.597
	Ischaemic	15–64	0.002	0.021	0.044	0.009	1.374	1.478	1.264	0.582
		>64	0.005	0.016	0.030	0.021	0.964	1.152	1.073	0.388
	Respiratory	<15	–	0.046	0.003	0.000	–	1.687	2.077	1.922
$PM_{2.5}$	Cardiac	15–64	–	–	–	0.018	–	–	–	0.307
	Stroke	>64	–	0.003	0.009	0.000	–	0.861	0.912	0.706
	Circulatory	15–64	–	–	–	0.006	–	–	–	0.308
	Ischaemic	>64	–	0.019	–	0.043	–	0.833	–	0.255
	Respiratory	<15	–	0.039	0.002	0.000	–	1.291	1.585	1.195

This table shows the statistical significance of the association (*p*) and semi-elasticity (*E*) in percent

[a] For each 10 μg m^{-3} of atmospheric pollutants a certain percent of hospital admissions changes

Wong et al. (2002) used Poisson regression to compare HA and air quality between Hong Kong and London. They found that respiratory admissions (>64 years of age) were related to NO_2 and SO_2 in both cities. Significant associations were also found between PM_{10} and SO_2 for cardiac admissions. Wilson et al. (2004) concluded that air pollution has significant and remarkably similar effects in both cities, regardless of differences in social, lifestyle and environmental factors, suggesting that the association is casual.

In Valencia (Spain), Ballester et al. 2001 shown a rise in SO_2 levels of 10 μg m^{-3} was significantly associated with an increment of 3.0 and 3.6 % in the expected number of all cardiovascular and heart diseases admissions, respectively. A similar increase in NO_2 was associated with an

increment of 3.6 % in the expected number of admissions for stroke diseases.

Alves et al. (2010) found that, in Lisbon, the risk for circulatory diseases increased by 0.8, 0.5 and 2.2 per 10 μg m^{-3} increase in NO_2 for the under 15, 15–64 and over 64 age groups, respectively. A 10 μg m^{-3} increase in the same atmospheric pollutant increases the risk due to respiratory diseases for the <15 and >64 age groups by 1.2 and 2.0 %, respectively. The results obtained in Lisbon are in the same range of values reported by other studies carried out in the USA and Europe. The association between PM_{10} and $PM_{2.5}$ and the HA due to stroke and ischaemic diseases for individuals over 64 years was significant for all models. Results showed that an increase of 10 μg m^{-3} in PM_{10} is associated with an increase in that typology of HA between 0.7 and 1.0 % for stroke and from 0.4 to 1.0 % for ischaemic diseases; the same increment in $PM_{2.5}$ is associated with an increase of that typology of HA that ranged between 0.3 and 0.9 % for stroke and between 0.2 and 0.8 % for ischaemic diseases.

The studies reviewed here encompass a variety of methodologies to investigate the short-term impact of atmospheric pollutants and public health. Each of the studies reported a significant association with at least one atmospheric pollutant. For respiratory admissions, Schwartz (1997) summarised a selection of US studies and reported increases in respiratory admissions of 1.3 % for 10 μg m^{-3} increases in PM_{10}. In 26 US communities, and for a 10 μg m^{-3} increase in $PM_{2.5}$ concentration, Zanobetti et al. (2009) found an increase of 2.07 % in respiratory admissions. The United States Environmental Protection (US EPA) reviewed the evidence for health effects of particles, including admission time-series studies, and tabulated results from a large number of works. The US EPA reports that particle effects ranged from approximately 1 to 5 % for 10 μg m^{-3} increases (Chapman et al. 1996). Results obtained for eight European cities within the APHEA project showed an increase in respiratory admissions of 0.9 % for 10 μg m^{-3} increases in PM_{10}, for individuals over 64 years (Atkinson et al. 2001). Wellenius et al. (2006) found in six US cities that a 10 μg m^{-3} increase in PM_{10} was associated with an increase of 0.72 % in HA due to cardiac diseases. In Hong Kong, Hong et al. (2013) obtained, for an increase of 10 μg m^{-3} in $PM_{2.5}$ levels, an increase of 1.86 % in HA due to circulatory diseases.

In Lisbon, no significant correlations between PM_{10} and HA due to respiratory diseases for individuals between 15 and 64 years and over 64 years was found in this study; in Setúbal (Almeida et al. 2014), the increase of 10 μg m^{-3} in PM_{10} was associated with an increase in HA of 1.6 % for respiratory diseases for individuals under 15 years as observed in our study by the WEEK model. Our results point out 1.6 to 2.1 % for 10 μg m^{-3} increases of PM_{10} and 1.3 to 1.6 % for 10 μg m^{-3} increases of $PM_{2.5}$. These increases are for below 15 years only (the significant one).

Conclusions

In this study, we have studied potential AP and HA associations in Lisbon urban area, from 2006 to 2008. AP values in this study presented higher values during winter than in summer, except ozone which was the other way around; these are expected observations. Only NO_2 and PM_{10} exceeded the annual limit as given by Directive 2008/50/EC of European Union. AP averaged over the stations present normality in all cases; HA averaged over the hospitals are bimodal for the studied diseases, except asthma.

Four models were applied using OLS, following previous studies, considering the databases day AP-day HA, day AP-week HA, week AP-day HA and week AP-week HA. The later was the one leading to a larger number of significant associations. The increase of HA with the increase of 10 μg m^{-3} in AP, given by the different models, is of the same order of magnitude; however, the week-week models gave in general lower values, most probably due to the smoothing of both AP and HA data. The increases go up to 3 %, considering all the models. It may be concluded that any of the models can be adopted because they lead to similar conclusions. However, since the week-week models produces a larger number of associations might be suggested the use of this one.

Three ranges of age were studied: <15, between 15 and 64, >64.

For <15, all models showed significant associations between respiratory diseases and all AP except the day-day model for PM_{10} and $PM_{2.5}$. For this age range, no more associations were found between AP and the other studied diseases. For the other age ranges, associations depended of the disease and the pollutant.

For >64, similar associations as for other authors were found between respiratory diseases and NO_2, SO_2 and PM_{10}. This age range is sensitive to all diseases associated with almost all pollutants. For stroke, 1 % increase when NO_2 increased of 10 μg m^{-3} was found in this work: other authors pointed out 3.6 %. No association between PM_{10} and respiratory disease was observed, as other authors have found.

For individuals between 15 and 64, HA due to cardiac, stroke, circulatory and ischaemic diseases are associated to most of the pollutants. No association was found between respiratory disease hospital admissions and the pollutants. Also no association was found between NO_2 and HA by circulatory diseases as pointed out by other authors as 0.5 % increase in HA when the pollutant increase 10 μg m^{-3}; this value was obtained in this work with NO.

The followed methodology was validated by comparison with other authors, with comparison of some data with Poisson regression, and it was sustained by previous publications.

References

Almeida SM, Farinha MM, Ventura MG, Pio CA, Freitas MC, Reis MA, Trancoso MA (2007) Measuring air particulate matter in large urban areas for health effect assessment. Water Air Soil Pollut 179:43–55

Almeida SM, Freitas MC, Repolho C, Dionísio I, Dung HM, Caseiro A, Alves C, Pio CA, Pacheco AMG (2009a) Characterizing air particulate matter composition and sources in Lisbon, Portugal. J Radioanal Nucl Chem 281:215–218

Almeida SM, Freitas MC, Repolho C, Dionísio I, Dung HM, Pio CA, Alves C, Caseiro A, Pacheco AMG (2009b) Evaluating children exposure to air pollutants for an epidemiological study. J Radioanal Nucl Chem 280(2):405–409

Almeida SM, Canha N, Silva A, Freitas MC, Pegas P, Alves C, Evtyugina M, Pio AC (2010) Children exposure to atmospheric particles in indoor of Lisbon primary schools. Atmos Environ 45(2011):7594–7599

Almeida SP, Casimiro E, Calheiros J (2011) Short-term association between exposure to ozone and mortality in Oporto, Portugal. Environ Res 111:406–410

Almeida SM, Freitas MC, Reis M, Pinheiro T, Felix PM, Pio CA (2013a) Fifteen years of nuclear techniques application to suspended particulate matter studies. J Radioanal Nucl Chem 297(3):347–356

Almeida SM, Silva AI, Freitas MC, Dung HM, Caseiro A, Pio AC (2013b) Impact of maritime air mass trajectories on the western European coast urban aerosol. J Toxicol Environ Health A 76(4–5):252–262

Almeida SM, Silva AV, Sarmento S (2014) Effects of exposure to particles and ozone on hospital admissions for cardiorespiratory diseases in Setúbal, Portugal. J Toxicol Environ Health A 77(14–16):837–848

Alves CA, Scotto MG, Freitas MC (2010) Air pollution and emergency admissions for cardiorespiratory diseases in Lisbon (Portugal). Quim Nova 33(2):337–344

Atkinson RW, Anderson HR, Sunyer J, Ayres J, Baccini M, Vonk JM et al (2001) Acute effects of particulate air pollution on respiratory admissions: results from APHEA 2 project. Air pollution and health: a European approach. Am J Respir Crit Care Med 164:1860–1866

Ayres-Sampaio D, Teodoro AC, Sillero N, Santos C, Fonseca J, Freitas A (2014) An investigation of the environmental determinants of asthma hospitalizations: an applied spatial approach. Appl Geogr 47:10–19

Ballester F, Tenías JM, Pérez-Hoyos S (2001) Air pollution and emergency hospital admissions for cardiovascular diseases in Valencia, Spain. J Epidemiol Community Health 55:57–65. doi:10.1136/jech.55.1.57, Public health policy and practice

Barnett R (1997) Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. Environ Res 72:24–31

Cameron CA, Trivedi P (1998) Regression analysis of count data. Econometric Society Monograph. 30. p. 89

Chapman R, Grant LD, Hasselblad V, Ito K, Kalkstein L, Kotchmar D, Lipfert FW, Marcus A, Thurston GD (1996) Air quality criteria for particulate matter. EPA/600/P-95/001cF. U.S. Environmental Protection Agency

Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M (2009) The relationship of respiratory and cardio vascular hospital admissions to the southern California wildfires of 2003. Occup Environ Med 66(3):189–197

European Commission (2008) Official Journal of the European Union. Directive 2008/50 CE of the European Parliament and Council of 21 May 2008 on ambient air quality and cleaner air for Europe. 11 June 2008. L152/1–L152/44

Farinha MM, Freitas MC, Almeida SM (2004) Air quality control monitoring at an urban and industrialized area. J Radioanal Nucl Chem 259(2):203–207

Freitas MC, Almeida SM, Pacheco AMG, Dionísio I, Repolho C, Caseiro A, Pio CA, Alves C (2009) Characterization of the seasonal exposure to air pollutants for a respiratory epidemiological study. J Radioanal Nucl Chem 281:219–223

Freitas MC, Pacheco AMG, Verburg TG, Wolterbeek HT (2010) Effect of particulate matter, atmospheric gases, temperature, and humidity on respiratory and circulatory diseases' trends in Lisbon, Portugal. Environ Monit Assess 162:113–121

Hansen A, Bi P, Nitschke M, Pisaniello D, Ryan P, Sullivan T, Barnett AG (2012) Particulate air pollution and cardiorespiratory hospital admissions in a temperate Australian city: a case-crossover analysis. Sci Total Environ 416(1):48–52

Hong Q, Ignatius TY, Xiaorong W, Linwei T, Lap AT, Tze WW (2013) Differential effects of fine and coarse particles on daily emergency cardiovascular hospitalizations in Hong Kong. Atmos Environ 64: 296–302, ISSN 1352-2310

Kan H, Chen B (2004) Particulate air pollution in urban areas of Shanghai, China: health-based economic assessment. Sci Total Environ 322(1–3):71–79

Karr C, Lumley T, Schreuder A, Davis R, Larson T, Ritz B, Kaufman J (2007) Effects of subchronic and chronic exposure to ambient air pollutants on infant bronchiolitis. Am J Epidemiol 165:553–560

Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, Rossi G, Wojtyniak B, Sunyer J, Bacharova L, Schouten JP, Ponka A, Anderson HR (1997) Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. Air Pollution and Health: a European Approach. BMJ 314(7095):1658

Katsouyanni K, Touloumi G, Samoli E, Gryparis A, Le Tertre A, Monopolis Y et al (2001) Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. Epidemiology 12:521–531

Linares C, Díaz J (2010) Short-term effect of concentrations of fine particulate matter on hospital admissions due to cardiovascular and respiratory causes among the over-75 age group in Madrid, Spain. Public Health 124(1):28–36, ISSN 0033-3506

Linares C, Tobías A, Díaz J (2010) Is there new scientific evidence to justify reconsideration of the current WHO guidelines for particulate matter during dust intrusions? Sci Total Environ 408(10):2283–2284. doi:10.1016/j.scitotenv.2010.02.005, ISSN 0048-9697

Lipfert FW (1993) A critical review of studies of the association between demands for hospital services and air pollution. Environ Health Perspect 101(suppl2):229–268

Lipfert FW (1997) Air pollution and human health: perspectives for the '90s and beyond. Risk Anal 17:137–146

Lumley T, Sheppard L (2000) Assessing seasonal confounding and model selection bias in air pollution epidemiology using positive and negative control analyses. Environmetrics 11:705–717

Luvsan M-E, Ruei-Hao S, Tseden P, Lkhagvasuren B, Barkhasragchaa B, Chang-Chuan C (2012) The influence of emission sources and meteorological conditions on SO_2 pollution in Mongolia. Atmos Environ 61:542–549, ISSN 1352-2310

Maddison D (2005) Air pollution and hospital admissions: an ARMAX modelling approach. J Environ Econ Manag 49(1):116–131, ISSN 0095-0696

Máté B, Roland B, Zsófia AG, Brigitta T, Márk M (2010) Age dependent correlation between the CNV amplitude and heart rate change. Int J Psychophysiol 77(3):258, ISSN 0167-8760

McConnel R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM (2002) Asthma in exercising children exposed to ozone: a cohort study. Lancet 359(9304):386–391

Middleton N, Yiallouros P, Kleanthous S,Kolokotroni O, Schwartz J, Dockery D, Demokritou P, Koutrakis P (2008) A 10-year time-series analysis of respiratory and cardiovascular morbidity in Nicosia, Cyprus: the effect of short-term changes in air pollution and dust storms. Environmental Health. BioMed Central Ltd, Middleton

Morawska L, Jayaratnea ER, Mengersenb K, Jamriskaa M, Thomasa S (2002) Differences in airborne particle and gaseous concentrations in urban air between weekdays and weekends. Atmos Environ 36: 4375–4383

Moshammer H, Hutter HP, Hauck H, Neuberger M et al (1987) Grippe, Luftverunreinigung und Mortalität in Wien. Forum Städtehygiene 38:7–11

Moshammer H, Hutter HP, Hauck H, Neuberger M (2006) Low levels of air pollution induce changes of lung fuction in a panel of school children. Eur Respir J 27:1138–1143

Neuberger M, Daniel R, Moshammer H (2007) Extended effects of air pollution on cardiopulmonary mortality in Vienna. Atmos Environ 41:8549–8556

Neuberger M, Moshammer H, Rabczenko D (2013) Acute and subacute effects of urban air pollution on cardiopulmonary emergencies and mortality: time series studies in Austrian cities. Int J Environ Res Public Health 10(10):4728–4751

Pablo Dávila FL, Soriano LR, Sánchez-Llorente JM, Nájera-López A (2013) Effects of weather types on hospital admissions for respiratory diseases in Castilla-La Mancha, Spain. Atmósfera 26(1):95–107

Pascal M, Corso M, Channel O, Declercq C, Badaloni C, Cesaroni G, Henschel S, Meister K, Haluza D, Martin-Olmedo P, Medina S, on behalf of the Aphekom group (2013) Assessing the public health impacts of urban air pollution in 25 European cities: results of the Aphekom project. Sci Total Environ 449:390–400

Perez L, Tobías A, Querol X, Pey J, Alastuey A, Díaz J, Sunyere J (2012) Saharan dust, particulate matter and cause-specific mortality: a case–crossover study in Barcelona (Spain). Environ Int 48:150–155

Pope CA III, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ (2004) Cardiovascular mortality and long term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109:71–77

Pope CA III, Renlund DG, Kfoury AG, May HT, Horne BD (2008) Relation of heart failure hospitalization to exposure to fine particulate air pollution. Am J Cardiol 102(9):1230–1234. doi:10.1016/j.amjcard.2008.06.044

Ritchie IM, Lehnen RG (2004) Internet J Pulm Med 4:N1

Roberts S (2005) Using moving total mortality counts to obtain improved estimates for the effect of air pollution on mortality. Environ Health Perspect 113:1148–1152

Roemer W, Hoek G, Brunekreef B, Haluszka J, Kalandidi A, Pekkanen J (1998) Daily variations in air pollution and respiratory health in a multicentre study: the PEACE project. Pollution Effects on Asthmatic Children in Europe. Eur Respir J 12:1354–1361

Sarmento S, Verburg TG, Almeida SM, Freitas MC, Wolterbeek HT (2009) Robustness of different regression modelling strategies in epidemiology: a time-series analysis of hospital admissions and air pollutants in Lisbon (1999–2004). Environmetrics 22:86–97

Schwartz J (1997) Health effects of air pollution from traffic: ozone and particulate matter. In: Fletcher T, McMichael AJ (eds) Health at the crossroads: transport policy and urban health. Wiley, Chichester, pp 61–82

Schwartz J (2000a) Assessing confounding, effect modification and thresholds in the association between ambient particles and daily deaths. Environ Health Perspect 108:563–568

Schwartz J (2000b) Harvesting and long-term exposure effects in the relation between air pollution and mortality. Am J Epidemiol 151: 440–448

Schwartz J (2001) Is there harvesting association of airborne particles with daily deaths and hospital admissions? Epidemiology 12:55–61

Shin H, Stieb AM, Jessiman B, Goldberg MS, Brion O, Brook J, Ramsay T, Burnett RT (2008) A temporal, multicity model to estimate the effects of short-term exposure to ambient air pollution on health. Environ Health Perspect 116(9):1147–1153

Smith RL, Davis JM, Sacks J, Speckman P, Styer P (2000) Regression models for air pollution and daily mortality: analysis of data from Birmingham, Alabama. Environmetrics 11:719–743

Wellenius GA, Schwartz J, Murray AM (2006) Particulate air pollution and hospital admissions for congestive heart failure in seven United States cities. Am J Cardiol 97(3):404–408, ISSN 0002-9149

WHO (1999) Monitoring ambient air quality for health impact assessment, WHO Regional Series, European Series, No. 85. World Health Organization, Copenhagen

WHO (2006) WHO air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. WHO/SDE/PHE/EH/06.02. Available at http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf

WHO (2014) International Classification of Diseases (ICD), Available at http://www.who.int/classifications/icd/en/

Wilson A, Salloway JC, Wake CP, Kelly T (2004) Air pollution and the demand for hospital services: a review. Environ Int 30(8):1109–1118

Wong CS, Waser NAD, Whitney FA, Johnson WK, Page JS (2002) Time-series study of the biogeochemistry of the North East subarctic Pacific: reconciliation of the Corg/N remineralization and uptake ratios with the Redfield ratios. Deep-Sea Res II Top Stud Oceanogr 49(24–25):5717–5738, ISSN 0967-0645

Zanobetti A, Schwartz J (2005) The effect of particulate air pollution on emergency admissions for myocardial infarction: a multicity case-crossover analysis. Environ Health Perspect 8(113):978–982

Zanobetti A, Schwartz J, Samoli E, Gryparis A, Touloumi G, Atkinson R, Le Tertre A, Bobros J, Celko M, Goren A, Forsberg B, Michelozzi P, Rabczenko D, Aranguez Ruiz E, Katsouyanni K (2002) The temporal pattern of mortality responses to air pollution: a multicity assessment of mortality displacement. Epidemiology 13:87–93

Zanobetti A, Franklin M, Koutrakis P, Schwartz J (2009) Fine particulate air pollution and its components in association with cause-specific emergency admissions. Environ Heal 8:58

Zeger SL, Dominici F, Samet JM (1999) Harvesting-resistant estimates of pollution effects on mortality. Epidemiology 89:171–175