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Association Between Ambient Air Pollution and Hospitalization for Respiratory Diseases in Perth, Australia

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Abstract

Background: Numerous studies continue to demonstrate that air pollution is a significant contributor for respiratory hospitalizations among adults and children.

Methods: We examined the associations between daily changes in exposure levels of selected air pollutants including nitrogen dioxide (NO_2), particulate matter with <10μm (PM_{10}) and <2.5μm in aerodynamic diameter ($PM_{2.5}$), and hospitalization for respiratory symptoms, respiratory infections and asthma in Perth, Western Australia for a 5-year period. A Poisson model allowing for autocorrelation and over-dispersion was applied in the analysis for each air pollutant and also carried out separately for different seasons, defined as "warm" (November to April) and "cold" (May to October) seasons.

Results: Daily hospital admissions due to respiratory infections were significantly related to daily changes in concentrations of NO_2, PM_{10} and $PM_{2.5}$. Significant lagged effects on hospitalization for asthma and respiratory symptoms were established for $PM_{2.5}$. Respiratory symptoms hospitalization was also significantly affected by NO_2 at longer lags.

Conclusions: Our findings have important public health implications as we established significant associations between air pollutants and respiratory hospitalization in a city where the average concentrations of NO_2, PM_{10} and $PM_{2.5}$ are below the national standards.

Keywords: Air Pollution; Hospital Admissions; Respiratory Symptoms; Australia

Introduction

Air quality is not a new concept, however, continues to be a significant health and environmental problem worldwide including in Australia. Studies from around the world continue to demonstrate relationships between air pollution and adverse health effects among adults and children. Some of the air pollutants considered as a major health concern in urban areas include particulate matter and gases such as ozone, oxides of nitrogen, carbon monoxide and sulphur dioxide [1].

Particulate matter (PM) consists of solid and liquid particles which vary in size, number, shape, surface area, chemical composition, solubility and origin [2]. PM can be separated into three major fractions based on size which is defined to a 50% cut point at a specific aerodynamic diameter. Coarse particles are defined as particles smaller than 10μm in aerodynamic diameter (PM_{10}), fine particles are smaller than 2.5μm ($PM_{2.5}$) and ultrafine particles (PM1.0) smaller than 0.1μm in aerodynamic diameter. In urban areas, the coarse particulates typically contain resuspended dust from roads and industrial activities, and biological material such as pollen grains and bacterial fragments. Fine particles are largely formed from combustion processes related to traffic, wood burning, power generation and industrial processes including smelters, cement plants, and paper mills. According to the World Health Organisation (WHO) [3] fine particles are considered more dangerous than

course particles because they consist of high proportion of various toxic metals and acids, and aerodynamically they can penetrate deeper into the respiratory tract when compared with PM_{10}. Numerous epidemiological and clinical studies have linked particulate air pollution to long term and short term effects of morbidity on respiratory, pulmonary and cardiovascular diseases [4-6]. Three independent Australian studies conducted in Perth, Melbourne and Brisbane found a significant relationship between exposure to particulate matter and hospital admissions for respiratory symptoms in adults [7-9]. The results are consistent with those found in Europe [10-13] and USA [14,15]. The Australian National Environment Protection Council (NEPC) [16] estimated that exposure to higher concentrations of $PM_{2.5}$ in Sydney, Melbourne, Brisbane and Perth caused a total of 1,611 premature deaths annually. Another air pollutant, studied extensively and associated with respiratory illnesses, including bronchitis, pneumonia, decreased resistance to pulmonary infections and decreased lung function is nitrogen dioxide (NO_2), an oxidised form of nitric oxide [17]. According to WHO [18], fuel combustion with emissions due mainly to motor vehicle exhausts, heating and power sources, is one of the main sources of NO_2.

Air Quality in Australia is regulated by the Protection Measure for Ambient Air Quality developed through the Environment Protection and Heritage Council. The Protection Measure sets air quality standards that are legally binding on each level of the Government. The standards relate to six criteria air pollutants including particulates, nitrogen dioxide, carbon monoxide, photochemical oxidants, sulphur dioxide, and lead.

The climate regime in Perth is a Mediterranean – style climate with hot, dry summers and cool, wet winters. The air quality in Perth is considered as acceptable, with the air pollutants' concentrations within the national standardswith some occasional episodes of poor air quality [19]. The major sources of air pollution in Perth include motor vehicles, domestic sources (principally wood heaters) and industries. Regional areas of Western Australia may also experience poor air quality at times, pollution being caused by bushfires and windblown dust, industrial facilities and hazard reduction burns.

This paper evaluates the relationship between hospital admissions for respiratory illnesses and daily changes in $PM_{2.5}$, PM_{10} and NO_2 during a five year period (2004-2008) in Perth, Western Australia.

Methodology

We conducted a time-series analysis to examine the impact of daily changes in $PM_{2.5}$, PM_{10} and NO_2 on daily hospitalization for respiratory symptoms, respiratory infections and asthma in Perth, Western Australia for all age groups. The study included a period of five years and according to the Australian Demographic Statistics (ABS) the total population in Perth during the study period was 2,296,411.

Morbidity data

Daily hospital admissions for respiratory illnesses to all hospitals in Perth metropolitan area for the period between 2004 and 2008 were obtained through the Health Department, Western Australia. The standard diagnostic tool, International Classification of Diseases (ICD), was applied in this study. The retrospective data for daily hospitalization for acute respiratory infections (ICD codes J00-J22), respiratory symptoms (ICD codes J23-J99, excluding J45) and asthma (ICD code J45) were provided as an overall daily estimate for all age groups. Respiratory health diagnoses were based on primary discharge information obtained from the patients' charts. All transfers from other hospitals were subsequently excluded. Residents living outside the metropolitan area were also excluded from the analysis. Hospital admissions for diseases related to the digestive system (ICD codes K00-K93) were also obtained for the study period and used as a control confounding factor as no research demonstrated a relationship between gastrointestinal disease and air pollution.

Air Quality Data

Currently there are twelve air quality monitoring stations established across Perth metropolitan areas to assess the state of air quality against the National Environment Protection Measure (NEPM) standards. Due to missing data from some stations, three monitoring stations with the most completed data, including Duncraig, Caversham and South Lake located north, middle and south of Perth CBD (Central Business District), were averaged to represent the air quality for metropolitan Perth area.

The retrospective data for daily concentrations (24 hr average) of PM_{10}, $PM_{2.5}$, and NO_2 during the study period between 2004 and 2008 were provided by the Department of Environment and Conservation (DEC), Western Australia, however, daily average concentrations of $PM_{2.5}$ were available only from the meteorological station in Duncraig. All data were collected by direct measurements as Tapered Element Oscillating Microbalance was used to obtain continuous readings of PM and the chemiluminescence method was applied for collecting the NO_2 data.

Statistical Analysis

Descriptive statistics were generated for relevant variables by using the IBM SPSS Statistics for Windows, Version 22 (IBM Corp. Released 2010 Armonk, NY).

To explore the relationship between hospital admissions for asthma, respiratory symptoms, and respiratory infections collected over the 5-year period starting from 01 January 2004 to 31 December 2008 and daily concentrations (24 hr average) of PM_{10}, $PM_{2.5}$, and NO_2, a Poisson model allowing for autocorrelation and over-dispersion among count data, implemented by Stata routine Arpois[20] , was applied for each air pollutant and successfully applied in other similar studies[21-23]. The

autocorrelation among the time series data was controlled by including different order of autoregressive terms depending on different models. Ljung–Box portmanteau (Q) test was used to assess whether any autocorrelation remained in the regression residuals. Seasonality was controlled by inclusion of the function for k = 0.5, 1, 2, 3, 4, 5, 6 and t = 1,2, ..., 1827 (the number of study days). Long-term trends were controlled by the inclusion of the number of the day and its square. Linear or quadratic trend (if appropriate), indicator of years, week days, and public holidays, humidity, and temperature (lag of humidity or temperature were included only if the model was improved) and their squares (if appropriate), and the admission number of digestive disorder were included in the model as potential confounding factors.

Since the health effects of PM_{10}, $PM_{2.5}$, and NO_2 are in a time dependent fashion, hospital admissions for asthma, respiratory infection and respiratory symptoms might not be due to exposure to air pollution on the day of the admission but over the preceding days as well. In the Poisson modeling, we therefore examined the effect of legging exposure for 0,1,2, 3 and 4 days (lag0, lag1, lag2, lag3 and lag4 days, respectively). Consecutive cumulative lag effects were also considered in the modeling (lag0-1: average of lag0 and lag1; lag0-2: average of lag 0 to lag2; lag0-3: average of lag0 to lag3; lag0-4: average of lag0 to lag4).

Since there was no information available for asthma hospital admissions for the period between 2004 and 2006, the Poisson modeling for asthma was conducted only for the period of two years, 2007 and 2008.

To examine if a season might have an impact on the relationship between air pollutants and hospital admissions, the Poisson modeling was carried out separately for different seasons, defined as "warm" (November to April) and "cold" (May to October) seasons.

The results are presented as the average percentage (%) change in hospital admissions and associated 95 percent confidence intervals (95% CI) for each interquartile range increase in the relevant air pollutant.

Results

Air Quality

Summary statistics for the concentrations of air pollutants, meteorological variables and daily respiratory hospitalizations are presented in Table 1. It can be seen that the mean concentrations of all pollutants were well below the national standards. Significantly ($p<0.01$) lower concentrations of PM_{10} and $PM_{2.5}$ were measured in 2008 compared to the previous years, with similar trends recorded for NO_2 although

Year	Statistics	Concentrations of air pollutants			Meteorological measures		Number of hospital admissions		
		PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	NO_2(ppb)	T^0C	RH (%)	Asthma	Respiratory Symptoms	Respiratory Infections
2004	Mean(SD) Median Min-Max	17.52 (6.12) 16.7 5.66-47.07	7.93 (3.07) 7.30 3-24.4	7.13 (3.17) 6.65 1.1 -18.4	18.20 (5.24) 17.55 8.6-33.0	62.68 (16.18) 64.10 20.8-97.7	No sufficient data	17.01 (5.7) 16.5 5.0-37.0	34.27 (12.85) 32.0 8.0-72.0
2005	Mean(SD) Median Min-Max	16.73 (7.49) 14.99 2.93-55.23	7.82 (3.35) 7.2 3-40.5	7.15 (3.34) 6.73 1.3 – 17.4	17.38 (4.78) 16.8 7.7-31.7	67.12 (14.02) 68.8 17.9-96.6	No sufficient data	18.18 (6.32) 17.0 2.0-36.0	35.69 (14.9) 32.0 8.0-87.0
2006	Mean(SD) Median Min-Max	17.42 (6.35) 16.5 3.85-37.29	8.33 (3.04) 7.7 3.3-27.38	7.14 (3.45) 6.9 1 – 17.9	18.22 (4.88) 17.60 7.9-30.80	61.77 (14.37) 63.7 19.1-92.6	No sufficient data	16.59 (5.42) 16.0 2.0-32.0	32.46 (11.59) 30.0 9.0-71.0
2007	Mean(SD) Median Min-Max	16.41 (6.40) 15.08 4.83-48.32	7.47 (2.25) 7. 10 2.91-19.10	6.70 (3.13) 6.33 1.1-16.2	18.05 (4.80) 17.05 10.3-33.5	63.05 (14.87) 65.9 15.2-95.7	7.85 (3.80) 8.00 1.0-24.0	43.60 (20.5) 46.0 7.0-94.0	29.62 (12.87) 26.0 5.0-71.0
2008	Mean(SD) Median Min-Max	15.24 (6.16) 13.79 5. 94-46.16	7.50 (2.95) 6.74 3.48-36.61	6.75 (3.34) 6.18 1.1-14.7	18.02 (5.13) 17.3 8.1-32.0	62.05 (14.89) 63.7 24.0-91.8	7.55 (3.72) 7.00 0-20.00	46.50 (21.48) 48.5 6.0-96.0	30.94 (11.2) 30.0 9.0-61.0
Overall	Mean(SD) Median Min-Max	16.69 (6.57) 15.41 2.93-55.23	7.81(2.96) 7.203 2.91-40.50	6.98[a] (3.28) 6.53 1.0-18.4	17.97 (4.97) 17.2 7.7-33.5	63.32 (14.99) 65.4 15.2-97.7	7.70 (3.76) 7 0-24	28.38 (19.57) 20 2-96	32.60 (12.93) 30 5-87

Table 1. Summary descriptive statistics of air pollutants concentrations, meteorological measures, and number of hospital admissions by year, Perth WA

the differences were not statistically significant. The overall average temperature and relative humidity were 17.90C and 63.4%, respectively and there were no significant changes in both meteorological parameters over the study period.

As mentioned earlier, the asthma data was incomplete for the study period but when compared with other hospital data recorded for 2007 and 2008, most people were hospitalized with respiratory symptoms followed by those with respiratory infections and asthma. With regards to seasonal differences in respiratory hospitalizations, more people were admitted to a hospital during the cold season when compared with the warmer periods (Table 2). Significantly higher ($p<0.05$) concentrations of NO_2 were recorded during the cold months which is in contrast with the PM_{10} and $PM_{2.5}$ concentrations (Table 2).

Hospital admissions for respiratory infections were also significantly associated with exposures to PM_{10} and $PM_{2.5}$, indicating that for every 8.06 $\mu g/m^3$ increase in the concentration of PM_{10} and 3.10 $\mu g/m^3$ in $PM_{2.5}$, the number of people admitted to hospitals with respiratory infections increased by 1.87% (95% CI: 0.46-3.31) and by 1.90% (95% CI: 0.91-2.91%), respectively. In addition, exposures to NO_2, significantly increased the respiratory infections hospitalization by 2.17% at leg1. Furthermore, significant cumulative lag health impacts were found at longer lags for NO_2 (at lag0-1, lag0-2, lag0-3, and lag0-4) and $PM_{2.5}$ (at lag0-1 and lag0-2) but no lag effect was determined for PM_{10} (Table 3).

Association Between Air Pollution And Number of Hospital Admissions for Respiratory Symptoms, 2004-2008

	Mean (SD)	Median	Min	Max	IQR
Cold period (n =920)					
Concentration					
NO_2 (ppb)	8.16 (3.26)	8.23	1.1	17.9	4.8
PM_{10} ($\mu g/m^3$)	14.00 (4.64)	13.37	5.01	38.98	5.75
$PM_{2.5}$ ($\mu g/m^3$)	7.50 (2.74)	6.95	2.91	27.38	3.10
Admission					
Respiratory infections	41.23 (11.59)	40	12	87	16
Respiratory symptoms	31.56 (20.02)	23	5	94	27
Asthma (2007 ~ 2008)	9.62 (3.57)	9	2	24	5
Warm period (n =907)					
Concentration					
NO_2 (ppb)	5.78 (2.84)	5.23	1.0	18.4	3.5
PM_{10} ($\mu g/m^3$)	19.42 (7.09)	18.70	2.93	55.23	8.35
$PM_{2.5}$ ($\mu g/m^3$)	8.13 (3.14)	7.50	3.00	40.50	3.20
Admission					
Respiratory infections	23.83 (6.92)	23	5	53	9
Respiratory symptoms	25.15 (18.56)	17	2	96	18
Asthma (2007 ~ 2008)	5.75 (2.82)	5	0	15	4

Table 2 . Seasonal differences in concentrations of air and daily respiratory hospitalizations (2004-2008), Perth WA

Significant ($p<0.05$) but weak correlations between air pollutants (24h exposure) and meteorological variables were established. For the entire study period, course and fine particulates were significantly correlated with each other ($r=0.324$) and NO_2 was significantly correlated with $PM_{2.5}$ ($r = 0.24$) but not with PM_{10}. Particulate air pollution and NO_2 were significantly ($p<0.05$) correlated with temperature and relative humidity.

Association Between Air Pollution and Number of Hospital Admissions for Respiratory Infections, 2004-2008

All pollutants showed significant associations with respiratory infections hospitalization with the greatest effect seen for NO_2. With every 5.0 ppb increase in NO_2 the number of hospital admissions for respiratory infections increased by almost 4% on average at lag0 (95% CI: 2.31-5.71) (Table 3).

The Poisson models failed to establish significant associations between hospitalization for respiratory symptoms and daily changes in concentrations of NO_2, $PM_{2.5}$ and PM_{10} on the day of the admission (lag0), however, there were several significant lag effects. The number of patients admitted to a hospital with respiratory symptoms increased by 2.22% (95% CI: 0.11%, 4.37%) and 3.42% (95% CI: 1.27%, 5.61%) for every 0.5ppb increase in NO_2 exposure at lag1 and lag4, respectively. Significant cumulative lag effects of NO_2 were found for hospital admissions of respiratory symptoms at lag0-1, lag0-2, lag0-3, and lag0-4. (Table 3). The current study also showed significant lag effects of exposure to $PM_{2.5}$ on respiratory symptoms hospitalization but failed to demonstrated such effects for PM_{10} (Table 3).

	NO_2	PM_{10}	$PM_{2.5}$
Respiratory infection			
Lag0	**3.99 (2.31, 5.71)***	**1.87 (0.46, 3.31)***	**1.90 (0.91, 2.91)***
Lag1	**2.17 (0.50, 3.86)***	-0.30 (-1.68, 1.11)	0.64 (-0.36, 1.65)
Lag2	0.18 (-1.46, 1.84)	-0.89 (-2.28, 0.30)	-0.25 (-1.15, 0.76)
Lag3	-0.43 (-2.06, 1.22)	-1.11 (-2.51, 0.30)	-0.52 (-1.53, 0.50)
Lag4	-1.03 (-2.22, 0.61)	-1.39 (-2.78, 0.02)	-0.86 (-1.87, 0.15)
Lag 0-1	**4.01 (2.22, 5.84)***	1.07 (-0.47, 2.62)	**1.71 (0.63, 2.80)***
Lag 0-2	**3.51 (1.59, 5.47)***	0.38 (-1.22, 2.00)	**1.26 (0.13, 2.42)***
Lag 0-3	**3.03 (1.00, 5.10)***	-0.21 (-1.86, 1.47)	0.91 (-0.27, 2.10)
Lag 0-4	**2.48 (0.31, 4.69)***	-0.66 (-2.37, 1.07)	0.48 (-0.74, 1.72)
Respiratory symptoms			
Lag0	**1.91 (-0.16, 4.03)#**	1.32 (-0.42, 3.08)	0.82 (-0.48, 2.13)
Lag1	**2.22 (0.11, 4.37)***	0.09 (-1.62, 1.82)	0.28 (-1.00, 1.57)
Lag2	1.17 (-0.91, 3.30)	0.44 (-1.29, 2.20)	0.69 (-0.59, 1.99)
Lag3	0.98 (-1.10, 3.10)	0.61 (-1.14, 2.39)	0.95 (-0.40, 2.31)
Lag4	**3.42 (1.27, 5.61)***	0.67 (-1.07, 2.44)	**1.52 (0.18, 2.88)***
Lag 0-1	**2.73 (0.51, 4.99)***	0.90 (-0.99, 2.82)	0.72 (-0.67, 2.14)
Lag 0-2	**2.92 (0.53, 5.36)***	0.90 (-1.07, 2.92)	0.96 (-0.50, 2.45)
Lag 0-3	**3.08 (0.54, 5.68)***	1.02 (-1.04, 3.13)	1.29 (-0.25, 2.86)
Lag 0-4	**4.37 (1.61, 7.21)***	1.18 (-0.97, 3.39)	**1.84 (0.22, 3.49)***
Asthma (2007 ~ 2008)			
Lag0	3.71 (-1.36, 9.04)	-0.43 (-4.94, 4.30)	2.10 (-1.44, 5.75)
Lag1	2.42 (-2.61, 7.71)	1.26 (-3.30, 6.04)	**3.91 (0.35, 7.59)***
Lag2	2.41 (-2.60, 7.67)	2.75 (-1.84, 7.56)	3.30 (-0.25, 6.96)
Lag3	-2.28 (-7.07, 2.76)	0.50 (-4.00, 5.22)	-0.80 (-4.30, 2.83)
Lag4	0.19 (-4.71, 5.35)	2.00 (-2.48, 6.69)	-0.25 (-3.64, 3.27)
Lag 0-1	3.78 (-1.61, 9.47)	1.02 (-4.05, 6.35)	**4.05 (0.13, 8.12)***
Lag 0-2	4.68 (-1.11, 10.82)	2.25 (-3.13, 7.91)	**4.95 (0.79, 9.28)***
Lag 0-3	2.91 (-3.20, 9.41)	2.17 (-3.44, 8.11)	3.89 (-0.43, 8.40)
Lag 0-4	2.62 (-4.01, 9.71)	2.92 (-3.01, 9.22)	3.55 (-0.97, 8.28)

*Statistically significant at 5% level

Statistically significant at 10% level

Table 3. Percentage change (95% CI) in hospital admission (respiratory infection, respiratory symptoms, and asthma) per interquartile range increases in air pollutants at different lags for WA 2004-2008

Association Between Air Pollution and Number of Hospital Admissions for Asthma, 2007-2008

Due to lack of complete asthma data, the Poisson modelling for asthma hospitalization was developed only for two year period, between January 2007 and December 2008. The study did not establish significant associations between asthma hospitalization and daily changes in concentrations of NO_2 and PM_{10} (Table 3). However, we established lag effects of exposure to $PM_{2.5}$ as the risk for asthma hospitalization increased by 3.91% (95% CI: 0.35%, 7.59%) for each unit increase in IQR of $PM_{2.5}$. at lag 1.

Seasonal Difference in Respiratory Hospitalization

The Poison modeling was also applied to assess if seasonality had an impact on the relationship between respiratory hospital admissions and air pollution. The results are presented in Table 4 and showed no significant association between exposure to air pollutants and number of people admitted to hospitals with respiratory symptoms, except at lag4 for exposure to NO_2 during both seasons. Significant associations, however, were established for hospital admissions with respiratory infections and asthma which are discussed in detail below.

Cold Period (May to October)

The association between hospital admissions for respiratory infections and exposure to NO_2 during the cold season followed similar pattern for all year around (Supp - Figure 1).

Exposure to NO_2 increased asthma hospitalizations by 6.10% (95% CI: 0.02-12.56) (Table 4). Significant lag effects of NO_2 exposures were observed for hospital admissions with asthma with the most significant effects at lag0-1 (6.77%) and lag0-2 (6.86%).

With regards to hospital admissions for respiratory

	Lag0	Lag1	Lag2	Lag3	Lag4	Lag 0-1	lag 0-2	lag 0-3	Lag 0-4
Respiratory infections									
NO_2									
Cold	**4.15* (2.22, 6.12)**	**2.38* (0.45, 4.35)**	0.31 (-1.60, 2.24)	0.09 (-1.83, 2.04)	0.32 (-1.62, 2.30)	**4.44* (2.30, 6.64)**	**3.62* (1.54, 5.75)**	**3.32* (1.18, 5.51)**	**3.16* (0.93, 5.45)**
Warm	**2.92* (0.49, 5.41)**	1.92 (-0.44, 4.34)	-0.22 (-2.40, 2.00)	-0.63 (-2.86, 1.66)	-0.24 (-2.61, 2.19)	**3.20* (0.53, 5.93)**	**2.83# (-0.07, 5.82)**	2.08 (-0.83, 5.00)	1.64 (-1.41, 4.79)
PM_{10}									
Cold	0.88 (-0.71, 2.49)	-0.23 (-1.80, 1.36)	-0.58 (-2.14, 1.01)	-0.72 (-2.28, 0.85)	-0.91 (-2.45, 0.66)	0.51 (-1.12, 2.17)	0.11 (-1.58, 1.84)	-0.26 (-2.03, 1.54)	-0.69 (-2.49, 1.16)
Warm	**2.41* (0.25, 4.44)**	-0.76 (-2.77, 1.29)	-0.89 (-3.03, 1.30)	-0.50 (-2.65, 1.70)	-0.74 (-2.77, 1.33)	1.06 (-1.40, 3.58)	0.26 (-2.07, 2.64)	0.31 (-2.10, 2.77)	0.33 (-2.10, 2.82)
$PM_{2.5}$									
Cold	**1.62* (0.24, 3.02)**	1.08 (-0.28, 2.47)	0.11 (-1.24, 1.47)	0.07 (-1.30, 1.46)	-0.39 (-1.75, 0.99)	**1.75* (0.29, 3.22)**	1.41# (-0.01, 2.87)	1.28 (-0.19, 2.77)	0.94 (-0.52, 2.42)
Warm	**2.48* (0.83, 4.16)**	-0.35 (-1.94, 1.27)	-0.90 (-2.60, 0.83)	-0.33 (-2.06, 1.44)	-0.38 (-2.10, 1.38)	1.47 (-0.32, 2.75)	0.72 (-1.26, 2.67)	0.52 (-1.59, 2.67)	0.30 (-1.85, 2.51)
Respiratory symptoms									
NO_2									
Cold	0.96 (-1.42, 3.40)	2.07 (-0.40, 4.61)	0.62 (-1.79, 3.09)	0.48 (-1.96, 2.99)	**2.77* (0.26, 5.35)**	1.97 (-0.70, 4.71)	1.83 (-0.79, 4.51)	1.77 (-0.93, 4.55)	2.65 (-0.21, 5.59)
Warm	1.94 (-1.07, 5.03)	0.44 (-2.42, 3.39)	0.86 (-1.99, 3.79)	1.79 (-1.21, 4.87)	**2.93* (0.08, 6.02)**	1.57 (-1.65, 4.91)	1.81 (-1.67, 5.40)	2.46 (-1.12, 6.18)	**4.04* (0.08, 8.15)**
PM_{10}									
Cold	1.40 (-0.69, 3.53)	0.19 (-1.89, 2.31)	-0.20 (-2.25, 1.89)	-0.31 (-2.34, 1.77)	0.39 (-1.65, 2.47)	0.94 (-1.21, 3.14)	0.65 (-1.59, 2.94)	0.42 (-1.92, 2.81)	0.56 (-1.86, 3.04)
Warm	-0.18 (-2.81, 2.51)	-0.67 (-3.13, 1.85)	1.03 (-1.65, 3.19)	1.76 (-0.94, 4.53)	1.25 (-1.29, 3.86)	-0.35 (-3.35, 2.75)	0.22 (-2.72, 3.25)	0.92 (-2.16, 4.10)	1.30 (-1.79, 4.48)
$PM_{2.5}$									
Cold	1.48 (-0.43, 3.43)	0.55 (-1.32, 2.46)	0.04 (-1.78, 1.90)	0.21 (-1.68, 2.13)	1.72 (-0.18, 3.65)	1.29 (-0.72, 3.33)	0.98 (-0.97, 2.96)	0.93 (-1.08, 2.99)	1.43 (-0.60, 3.50)
Warm	-0.03 (-2.09, 2.07)	-0.40 (-2.36, 1.61)	1.44 (-0.63, 3.55)	2.26* (0.04, 4.52)	1.01 (-1.15, 3.22)	1.81 (-0.44, 4.21)	-0.34 (-2.50, 1.87)	1.66 (-1.08, 4.49)	2.27 (-0.55, 5.18)
Asthma (2007 ~2008)									
NO_2									
Cold	**6.10* (0.02,12.56)**	5.42 (-0.78,12.01)	3.40 (-2.74,9.94)	-3.13 (-8.76,2.86)	1.78 (-4.25,8.18)	**6.77* (0.04,13.95)**	**6.86* (0.11,14.07)**	4.68 (-2.31,12.17)	5.11 (-2.41,13.21)
Warm	-1.58 (-8.79,6.20)	-2.95 (-10.13,4.80)	2.55 (-4.41,10.02)	3.68 (-3.32,11.20)	2.41 (-4.79,10.16)	-3.35 (-11.05,5.01)	-1.21 (-9.65,8.01)	0.93 (-7.62,10.27)	2.02 (-7.22,12.19)
PM_{10}									
Cold	0.54 (-4.84, 6.23)	2.51 (-2.99,8.32)	3.87 (-1.59,9.62)	-2.42 (-7.66,3.11)	-1.96 (-7.15,3.51)	1.78 (-3.94,7.85)	3.28 (-2.90,9.85)	1.86 (-4.69,8.86)	0.80 (-6.13,8.25)
Warm	0.26 (-6.27,7.25)	1.33 (-5.07,8.16)	1.48 (-4.85,8.23)	4.80 (-2.07,12.06)	**7.69* (1.07,14.74)**	1.72 (-6.18,10.27)	2.53 (-5.15,10.83)	4.22 (-3.66,12.73)	6.25 (-1.65,14.78)
$PM_{2.5}$									
Cold	3.74 (-1.83,9.62)	4.98 (-0.59,10.85)	**5.94* (0.43,11.75)**	-2.46 (-7.75,3.14)	0.16 (-5.27,5.90)	5.40 (-0.57,11.72)	**6.78* (0.85,13.07)**	5.09 (-1.12,11.69)	4.78 (-1.61,11.58)
Warm	1.91 (-3.29,7.45)	3.89 (-1.30,9.36)	1.74 (-3.31,7.06)	1.86 (-3.15,7.14)	1.07 (-3.62,6.00)	4.64 (-1.33,10.97)	5.16 (-1.66,12.45)	5.72 (-1.43,13.38)	5.54 (-1.50,13.09)

*Statistically significant at 5% level

Statistically significant at 10% level

Table 4. Percentage change (95%CI) in hospital admission for respiratory infections, respiratory symptoms, and asthma by cold (May to October) and warm (November to April) periods per interquartile increase in air pollutants at different lags, WA 2004 – 20088.

symptoms, a significant effect was found only for NO_2 exposures indicating that for every 0.5 ppb increase in the concentration of NO_2 the respiratory symptoms hospitalization increased by 2.77% (95% CI: 0.26-5.35).

Exposure to $PM_{2.5}$ increased respiratory infections hospitalization by 1.62% (95%CI: 0.24-3.02) but no effect was established for exposure to PM_{10} during this period (Table 4). Significant lag effects were established for asthma hospitalization and exposure to $PM_{2.5}$ (5.94% at lag2 and 6.78% at lag0-2) (Table 4; Supp-Figure 3).

Warm Period (November to April)

During the warm period, the exposure to air pollutants had a significant impact on hospital admissions for respiratory infections. For every unit increase in IQR of NO_2 (3.5 ppb), PM_{10} (8.35 μg/m3) and of $PM_{2.5}$ (3.2 μg/m3) hospital admissions for respiratory infections increased by 2.92% (95% CI: 0.49-5.41), 2.41% (95% CI: 0.25- 4.44), and 2.48% (95% CI: 0.83-4.16), respectively (Table 4, Supp-Figure 1). Significant increase in respiratory infections hospitalization was also established for exposure to NO_2 at lag 0-1 (Table 4).

A significant 4-day lag effect of NO_2 was found for respiratory symptoms hospitalization with an increase of 2.93% (95% CT: 0.08-6.02) (Table 4). Furthermore, a significant effect of cumulative lagging exposures to NO_2 was established for respiratory symptoms hospitalization with 4.04 % increase at lag 0-4 but the study failed to demonstrate any significant NO_2 impact on asthma hospitalization during the warm period (Supp-Figure 2).

Discussion

The study demonstrated that ambient levels of PM_{10}, $PM_{2.5}$, and NO_2, which are primarily generated from combustion processes, industrial activities and wood burning, were associated with the risk of hospital admissions for respiratory illnesses in Perth, Western Australia. Similar results were reported in a Perth study conducted earlier (1992-1998) by Hinwood and colleagues[7]. Hinwood found that daily variations in NO_2 concentrations were associated with respiratory disease hospitalization among the 65 years and above age group. In a more recent study from Perth [6] and conducted between 2002-2008, emergency department admissions for asthma among children, aged 0-4 years, were also significantly associated with daily changes in NO_2. The findings were not expected given the low concentrations of NO_2 measured in Perth, compared with other cities including Hong Kong [24], Palermo in Italy [25] and Ontario in Canada [26]. The average NO_2 concentration measured in our study was 6.98 ppb which is consistent with the concentration of 6.28 ppb reported by the study of Pereira and colleagues[6] but lower than the average concentration of 10.3 ppb reported in the study of Hinwood [25] conducted more than ten years ago. Despite the apparent low exposure levels, the results presented herein indicate that current NO_2 levels in Perth still have a significant impact on daily hospitalization for respiratory diseases. Our findings are consistent with the findings of another Australian study conducted earlier in Sydney by Morgan[27]. Inconsistent results, however, were reported by a study conducted in Brisbane, Australia[8]. Petroeschevsky and colleagues failed to establish association between NO_2 and hospitalization for respiratory illnesses but showed a significant impact of particulate air pollution on respiratory hospitalizations. Similar results were reported by other Australian studies of Morgan[27] and Voigt[28]. In our study we demonstrated that even at $PM_{2.5}$ .concentrations of 7.2 μg/m3, which is more than twice less than the reported concentration in the study of Hinwood [7] (18.4 μg/m3), fine particulate air pollution in Perth still have a significant impact on respiratory hospitalization. Significant impacts on hospital admissions for respiratory symptoms and asthma were observed for longer lagging exposures to fine air particulate pollution ($PM_{2.5}$) but failed to show any significant effects of PM_{10}. Previous studies have also failed to show consistent effects of PM_{10} on asthma hospitalization. A study from Birmingham, England [29] showed no significant effect of PM_{10} on asthma hospitalization although the high concentrations measured for PM_{10} (130.9 μg/m3). In contrast, effects of PM_{10} on asthma hospitalization were found in a study in Seattle[30], in Utah Valley[31] and in California[32]. Similar findings were also reported in other countries including Canada, Europe and Hong Kong[24,26,33-36].

We can't provide a clear explanation for such discrepancy regarding the respiratory health effects of NO_2 and air particulate matter but reasons may include population characteristics, climate conditions, and also the complex mixture and different elemental composition of air particulate matter.

The present study has limitations which are similar to other studies as it unable to distinguish between mixtures of air pollutants and the confounding effect of co-pollutants. Moreover, due to the correlation between air pollutants and in particular between NO_2 and $PM_{2.5}$, it is difficult to determine the contribution of each pollutant on health effects[37]. Another limitation is that exposure to air pollutants was assessed at aggregated levels by using data from ambient monitoring stations rather than at individual levels, however, any measurement error is most likely to be non-differential and produces conservative estimates of associations[38]. We acknowledge that age of study population may have an impact on the study outcomes, however, due to the limited access to data we couldn't characterise the relationship between air pollution and respiratory hospital admissions for different age groups which is considered as a study limitation. Future studies may be conducted if age groups are made available.

Despite the study limitations, there are number of strengths to this study which include the relatively long time series variables available for up to 5 years, the controlling for long-term time trends, meteorological parameters (temperature, humidity) and other confounding effects (years, seasons, day of week, and public holidays) considered in the Poisson modelling.

In summary, we provide supporting evidence that the ambient

air pollution, in particular air particulate matter and nitrogen dioxide, has still significant impact on respiratory hospitalizations in Perth, WA. The results have important public health implications as the established associations are for a city where the concentrations of $PM_{2.5}$, PM_{10} and NO_2 are relatively low and meet the National Air Quality standards.

Conclusion

This study, similar to previous studies, suggests that, urban air pollution may be responsible for the increase of respiratory hospitalization in Perth, Western Australia. Motor vehicles are one of the major sources of ambient NO_2 and PM, and the findings of the study reinforce the need for public policy measures to better control air pollution.

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