EXPANSION of the multi-city mortality and morbidity study

Environment Protection and Heritage Council

FINAL REPORT

Volume 2
Project results and conclusions

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Chapter 1 Study data

1.1 Study population

Daily health, air quality and weather data were collected for the years 1998-2001 in five cities in Australia (Brisbane, Canberra, Melbourne, Perth and Sydney) and two cities in New Zealand (Auckland, Christchurch) (Figure 1.1). These cities covered 53% of the Australian population and 44% of the New Zealand population, for a total population of just under 12 million people (Table 1.1). *Volume 3: Appendix 1* contains a full set of summary statistics for health, air pollutant and weather data in each city. The median income and population data were supplied by the Australian Bureau of Statistics and Statistics New Zealand.

Figure 1.1: Location of Australian and New Zealand cities used in the analysis

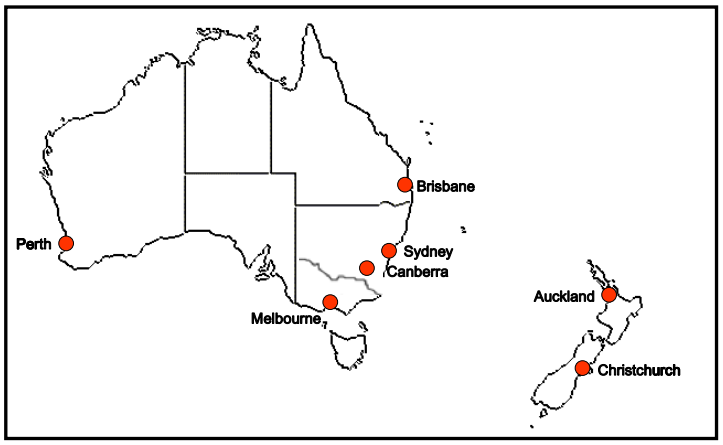


Table 1.1: Summary statistics for demographic data by city (years 1998-2001)

	Auckland	Brisbane	Canberra	Christchurch	Melbourne	Perth	Sydney
Demographic data							
Total population	1,158,891	1,627,535	311,518	316,224	3,366,542	1,339,993	3,997,321
Median weekly individual income $	400-499	300-399	500-599	300-399	400-499	300-399	400-499
Percentage of population <15 yrs %	22.9	21.0	21.2	19.3	19.8	20.7	20.2
Percentage of population >65 yrs %	10.0	11.0	8.3	13.7	12.1	11.3	11.9

1.2 Health outcomes

Health data were collected from state government health departments in Australia and the New Zealand Health Information Service (Ministry of Health) in New Zealand. The definition of the study regions for the collection of health data for Brisbane, Canberra, Melbourne and Perth came from the Australian Bureau of Statistics (ABS) statistical division (SD), but for Sydney the SD was modified to exclude areas that were not considered to be represented by the air quality monitoring network. For the New Zealand cities, the study regions were defined using the Statistics New Zealand divisions for the Auckland and Christchurch regions.

Disease groups were classified according to the International Classification of Diseases 9th Revision [ICD-9] (for the period January 1998–June 1998) and 10th revision [ICD-10] categories (for the period July 1998–December 2001). The following categories for cardiovascular admissions were examined in this study: cardiac, ischemic heart disease, stroke, arrhythmia, cardiac failure and myocardial infarction. The respiratory disease groups considered included total respiratory disease, asthma, chronic obstructive pulmonary disease and pneumonia and acute bronchitis (see Table 1.2 for the ICD codes).

Table 1.2: ICD-9 and ICD-10 codes for admission and mortality outcomes

	ICD-9	ICD-10
Admissions		
Cardiac	390-429	I00-I52, I97.0, I97.1, I98.1
Ischemic heart disease	410-413	I20, I21, I22, I24, I25.2
Stroke	430-438	I60-I66, I67 (excluding I67.0, I67.3), I68 (excluding I68.0), I69, G45 (excluding G45.3), G46
Arrhythmia	427	I46-I49
Cardiac failure	428	I50
Myocardial infarction	410	I21, I22
Total respiratory disease	460-519	J00-J99 (excluding J95.4 to J95.9), R09.1, R09.8
Asthma	493	J45, J46, J44.8
Chronic obstructive pulmonary disease	490-492, 494-496	J40-J44, J47, J67
Pneumonia and acute bronchitis	466, 480-486	J12-J17, J18.0, J18.1, J18.8, J18.9, J20, J21
Mortality		
Total mortality	all <800	A-R, Z35.5, Z35.8
Cardiovascular mortality	390-459	I00-I99 (excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0), G45 (excluding G45.3), G46, M30, M31, R58
Respiratory mortality	460-519	J00-J99 (excluding J95.4 to J95.9), R09.1, R09.8

1.2.1 Hospital admissions

Both respiratory and cardiovascular admissions were considered. Emergency attendances, scheduled admissions, transfers from other hospitals and admissions arranged through a general practitioner were excluded to minimise the delay and level of uncertainty associated with the period between onset of symptoms and day

of admission to hospital. Summary statistics on cardiovascular hospital admissions and respiratory hospital admissions are given in Table 1.3 and Table 1.4, respectively.

Responses to air pollution are age-dependent, and accordingly all analyses were stratified by age group. The age groups used were: less than one year of age, 1-4 years, 5-14 years, 15-64 years and greater or equal to 65 years. Asthma admissions in the first age group were not considered due to difficulties in accurate diagnosis of the condition in this age group.

Table 1.3: Summary statistics for cardiovascular admission rates per million population - by city and age group (years 1998-2001)

	Auckland	Brisbane	Canberra	Christchurch	Melbourne	Perth	Sydney
Daily admissions: mean (range)							
Total cardiovascular							
all ages	30.0 (4,53)	28.0 (13,46)	37.9 (0,109)	36.6 (6,79)	24.2 (13,37)	26.8 (10,44)	23.3 (14,36)
15-64 yrs	11.5 (3,31)	9.3 (2,19)	17.6 (0,67)	10.2 (0,32)	7.7 (2,14)	7.9 (1,17)	7.7 (3,14)
65+ yrs	18.1 (4,35)	18.6 (8,33)	20.0 (0,61)	26.2 (0,60)	16.3 (8,28)	18.8 (6,35)	15.5 (7,26)
Cardiac							
all ages	21.6 (2,44)	21.6 (9,37)	24.8 (0,64)	25.5 (0,63)	17.1 (9,29)	19.7 (6,35)	17.1 (9,26)
15-64 yrs	8.6 (1,24)	7.5 (1,17)	10.5 (0,42)	7.3 (0,28)	5.5 (1,11)	6.0 (0,14)	5.9 (2,11)
65+ yrs	12.8 (2,27)	14.0 (5,29)	14.1 (0,45)	18.1 (0,44)	11.5 (5,21)	13.7 (3,27)	11.1 (5,20)
Ischemic heart disease							
all ages	11.0 (0,28)	12.1 (4,23)	10.8 (0,42)	14.8 (0,41)	8.9 (4,15)	10.1 (2,19)	8.0 (4,14)
15-64 yrs	4.5 (0,14)	4.5 (0,11)	4.8 (0,26)	4.5 (0,19)	3.2 (1,6)	3.4 (0,9)	3.1 (0,7)
65+ yrs	6.5 (0,18)	7.6 (1,18)	6.1 (0,26)	10.4 (0,35)	5.7 (2,11)	6.7 (1,15)	4.9 (2,10)
Stroke							
all ages	5.1 (0,13)	4.2 (0,10)	3.6 (0,22)	7.4 (0,32)	4.6 (1,10)	4.3 (0,12)	4.1 (1,9)
15-64 yrs	1.6 (0,7)	0.9 (0,6)	1.0 (0,10)	1.8 (0,13)	1.1 (0,3)	1.0 (0,5)	1.0 (0,3)
65+ yrs	3.5 (0,10)	3.2 (0,9)	2.6 (0,16)	5.5 (0,22)	3.5 (1,10)	3.4 (0,9)	3.1 (1,7)
Arrhythmia							
all ages	4.6 (0,13)	3.5 (0,10)	4.5 (0,19)	4.0 (0,22)	2.9 (0,8)	3.3 (0,9)	3.2 (1,7)
15-64 yrs	2.0 (0,9)	1.3 (0,6)	2.0 (0,19)	1.3 (0,13)	1.0 (0,4)	1.1 (0,6)	1.2 (0,4)
65+ yrs	2.5 (0,9)	2.1 (0,7)	2.5 (0,16)	2.7 (0,16)	1.8 (0,5)	2.2 (0,7)	1.9 (0,5)
Cardiac failure							
all ages	3.5 (0,11)	3.7 (0,12)	2.8 (0,16)	4.1 (0,22)	3.7 (1,9)	4.2 (0,13)	3.4 (1,10)
15-64 yrs	0.8 (0,5)	0.5 (0,3)	0.4 (0,6)	0.4 (0,9)	0.5 (0,3)	0.5 (0,4)	0.4 (0,2)
65+ yrs	2.7 (0,10)	3.2 (0,10)	2.3 (0,13)	3.7 (0,22)	3.3 (1,7)	3.7 (0,12)	2.9 (0,9)
Myocardial infarction							
all ages	3.8 (0,13)	3.8 (0,10)	2.5 (0,19)	6.5 (0,28)	3.1 (1,7)	3.7 (0,11)	2.8 (1,6)
15-64 yrs	1.5 (0,7)	1.4 (0,5)	1.2 (0,10)	1.8 (0,13)	1.2 (0,4)	1.3 (0,5)	1.1 (0,3)
65+ yrs	2.3 (0,9)	2.4 (0,7)	1.4 (0,13)	4.7 (0,25)	1.9 (0,6)	2.4 (0,9)	1.7 (0,4)

Table 1.4: Summary statistics for respiratory admission rates per million population - by city and age group (years 1998-2001)

	Auckland	Brisbane	Canberra	Christchurch	Melbourne	Perth	Sydney
Daily admissions: mean (range)							
Total respiratory							
all ages	30.1 (4,84)	23.8 (7,62)	27.8 (0,87)	36.7 (0,114)	20.7 (8,39)	24.6 (7,60)	23.3 (10,47)
0 yrs	4.6 (0,29)	2.1 (0,9)	2.1 (0,22)	4.8 (0,28)	1.4 (0,6)	2.4 (0,16)	2.1 (0,10)
1-4 yrs	4.7 (0,19)	4.2 (0,12)	4.9 (0,22)	8.1 (0,35)	3.1 (0,9)	4.7 (0,13)	4.5 (0,13)
5-14 yrs	2.1 (0,11)	2.1 (0,9)	3.8 (0,32)	3.1 (0,22)	1.6 (0,7)	2.3 (0,10)	2.1 (0,9)
15-64 yrs	10.3 (1,28)	7.5 (1,23)	10.0 (0,48)	9.8 (0,63)	6.4 (1,14)	6.3 (1,18)	6.4 (2,15)
65+ yrs	8.4 (1,25)	7.9 (1,23)	6.9 (0,29)	10.9 (0,51)	8.2 (2,19)	8.9 (1,24)	8.1 (2,21)
Asthma							
all ages	5.5 (0,18)	6.1 (0,23)	4.3 (0,26)	6.7 (0,38)	4.7 (0,15)	5.6 (0,16)	5.9 (1,20)
1-4 yrs	1.6 (0,8)	1.6 (0,7)	1.4 (0,13)	2.3 (0,19)	1.3 (0,6)	1.9 (0,8)	1.9 (0,10)
5-14 yrs	1.0 (0,7)	1.3 (0,7)	1.0 (0,16)	1.3 (0,13)	0.9 (0,7)	1.3 (0,7)	1.2 (0,8)
15-64 yrs	2.6 (0,11)	2.5 (0,11)	1.3 (0,13)	2.5 (0,22)	1.9 (0,6)	1.7 (0,10)	2.1 (0,7)
65+ yrs	0.3 (0,3)	0.6 (0,4)	0.5 (0,6)	0.4 (0,6)	0.6 (0,3)	0.6 (0,5)	0.6 (0,3)
COPD*							
all ages	5.0 (0,19)	4.5 (0,14)	3.5 (0,19)	7.6 (0,38)	4.2 (0,10)	4.9 (0,15)	4.4 (1,11)
15-64 yrs	1.6 (0,8)	1.1 (0,5)	0.7 (0,10)	2.3 (0,22)	0.9 (0,5)	0.9 (0,5)	1.0 (0,5)
65+ yrs	3.4 (0,15)	3.4 (0,12)	2.7 (0,16)	5.3 (0,35)	3.1 (0,8)	4.0 (0,12)	3.4 (0,10)
Pneumonia and acute bronchitis							
all ages	11.8 (0,47)	6.8 (1,24)	7.1 (0,39)	10.3 (0,47)	6.6 (1,16)	6.6 (0,25)	6.7 (1,20)
0 yrs	3.6 (0,25)	1.4 (0,7)	1.3 (0,19)	2.8 (0,28)	0.9 (0,6)	1.5 (0,14)	1.4 (0,8)
1-4 yrs	1.9 (0,13)	1.0 (0,5)	1.3 (0,16)	1.6 (0,16)	0.6 (0,3)	0.9 (0,7)	1.0 (0,5)
15-64 yrs	2.7 (0,12)	1.7 (0,6)	1.7 (0,13)	2.0 (0,16)	1.7 (0,5)	1.5 (0,7)	1.4 (0,5)
65+ yrs	3.1 (0,12)	2.5 (0,13)	2.3 (0,16)	3.5 (0,25)	3.2 (0,9)	2.4 (0,9)	2.5 (0,8)

* *COPD=Chronic obstructive pulmonary disease*

1.2.2 Mortality

Mortality outcomes considered were: all-cause mortality excluding accidental and other external causes of death, total cardiovascular mortality and total respiratory mortality (Table 1.5). All analyses were stratified by the age groups 15-64 years, 65-74 and greater or equal to 75 years.

Table 1.5: Summary statistics for mortality rates per million population - by city and age group (years 1998-2001)

	Auckland	Brisbane	Canberra	Christchurch	Melbourne	Perth	Sydney
Daily mortality: mean (range)							
Total all cause*							
All ages	15.9 (4,42)	15.4 (6,34)	10.7 (0,77)	21.3 (0,63)	16.5 (5,27)	15.0 (4,30)	14.2 (8,23)
15-64 yrs	3.2 (0,10)	2.7 (0,9)	2.4 (0,19)	3.1 (0,16)	2.6 (0,6)	2.5 (0,9)	2.4 (1,5)
65+ yrs	12.4 (3,36)	12.5 (4,29)	8.1 (0,61)	18.0 (0,57)	13.6 (4,23)	12.3 (4,27)	11.6 (5,20)
Respiratory							
All ages	1.4 (0,8)	1.3 (0,7)	0.8 (0,13)	1.9 (0,13)	1.4 (0,4)	1.3 (0,6)	1.3 (0,5)
15-64 yrs	0.1 (0,3)	0.1 (0,2)	0.1 (0,6)	0.2 (0,6)	0.1 (0,1)	0.1 (0,1)	0.1 (0,2)
65+ yrs	1.3 (0,8)	1.2 (0,6)	0.7 (0,10)	1.7 (0,13)	1.3 (0,4)	1.2 (0,5)	1.2 (0,4)
Cardiovascular							
All ages	6.7 (1,18)	6.8 (1,23)	4.3 (0,32)	9.7 (0,44)	6.6 (3,12)	5.9 (0,13)	6.2 (2,12)
15-64 yrs	0.9 (0,4)	0.7 (0,4)	0.6 (0,6)	0.9 (0,13)	0.6 (0,3)	0.6 (0,3)	0.6 (0,2)
65+ yrs	5.7 (0,16)	6.0 (1,23)	3.7 (0,32)	8.8 (0,44)	6.0 (2,11)	5.3 (0,13)	5.5 (2,12)

** Total all cause mortality excluding accidental other external causes of death*

1.3 Air quality variables

The major air pollutants considered are the gases — nitrogen dioxide (NO_2), ozone (O_3) and carbon monoxide (CO) — and particles or particulate matter (PM). Most of these pollutants are the products of combustion processes (such as motor vehicle engines, industrial operations, home heating) and are emitted directly into the atmosphere (such as NO_2, CO and some PM). These pollutants may be general indicators of mobile-source emissions, including particles from mobile sources, rather than direct toxic agents.

Other (secondary) pollutants can be formed in the atmosphere by chemical interactions among pollutants emitted into the atmosphere and normal atmospheric constituents (for example, in photochemical smog production, which produces more ozone, and PM such as secondary nitrates and sulfates). A summary of the major air pollutants and their sources is shown in Table 1.6, and a summary of the types of health effects associated with air pollution exposure is shown in Table 1.7.

Table 1.6: Air pollutants used here and their sources

Pollutant	Source
Nitrogen oxides (NO, NO_2)	Combination of nitrogen and oxygen during high temperature combustion
Dust, particulates, soot	Combustion and mining processes, land clearing
Ozone (O_3)	Formed in the atmosphere through photochemical reactions of nitrogen oxides and hydrocarbons emitted from motor vehicles and industry
Carbon monoxide (CO)	Combustion, particularly motor vehicles

Table 1.7: Health effects and populations 'at risk'

Pollutant	Health effects	Population at risk
Nitrogen dioxide	Hospital admissions for respiratory disease; decreases in lung function	Sufferers of respiratory disease, such as children with asthma; those with recent viral infection
Particulates	Mortality due to cardiovascular and respiratory diseases; hospital admissions due to respiratory disease; decreases in lung function	Elderly people with respiratory and cardiovascular diseases; people with respiratory diseases, such as children with asthma
Ozone	Mortality due to respiratory and cardiovascular diseases; hospital admissions due to respiratory diseases; decreases in lung function	Elderly people; people with respiratory diseases
Carbon monoxide	Increase in illness due to ischemic heart diseases	People with ischemic heart conditions

The pollutants considered were $PM_{2.5}$ ($\mu g.m^{-3}$), PM_{10} ($\mu g.m^{-3}$), nitrogen dioxide (NO_2) (ppb), ozone (O_3) (ppb), and carbon monoxide (CO) (ppm). A range of pollutant averaging periods were selected based on the National Environment Protection Measure (NEPM) reporting requirements: daily 24-hour averages for $PM_{2.5}$, PM_{10} and NO_2; daily 1-hour maxima for NO_2 and O_3; daily average 8-hour maxima for CO and O_3; and a daily average 4-hour maxima for O_3. Air quality data were provided by the environmental protection agency in each jurisdiction. Selection of sites was determined by each state in consultation with the relevant environment authorities. Individual sites were included based on the representativeness of each site of the daily outdoor air quality in that city. The air pollutant indicator used for analysis was calculated by averaging data from the selected sites within each city network.

The number of monitors used for the city average of each individual pollutant varied among and within cities (Table 1.8). Some air pollutants were unavailable in some cities, and in some cases where an air pollutant was measured it was unavailable on a daily basis. For example, where hi-volume samplers were used, particulate readings were only available every sixth day. The latter included PM_{10} and $PM_{2.5}$ in Auckland and PM_{10} in Canberra.

Table 1.8: Number of monitors included in the average for each pollutant in each city

	NO_2	CO	O_3	PM_{10}	$PM_{2.5}$
Auckland	2	3	0	6*	1*
Brisbane	7	1	7	4	1
Canberra	1	1	0	1*	0
Christchurch	1	2	0	2	0
Melbourne	8	3	8	4	2
Perth	5	3	3	1	2
Sydney	13	4	12	11	3

** hi-vol data (recorded once every six days)*

The number of air quality monitoring sites included in the network average for each city was one (Canberra), three (Christchurch), five (Perth), eight (Auckland, Brisbane, Melbourne) and thirteen (Sydney). The number of monitors used for the network average of each individual pollutant varied within each city: for example, there was an average of six monitors for NO_2 in each city, and an average of 2.7 monitors for CO. In general, the more sites used in a network average, the more representative the measure of the exposure of the population in that city. Summary statistics for air pollutant data are given in Table 1.9 for each city. See Table 6.2.1 for details of the numbers of observations available for each air pollutant, within each city.

1.4 Meteorological variables

Daily temperature, dew point temperature, relative humidity, barometric pressure and rainfall were collected from the Australian Bureau of Meteorology and from the New Zealand National Climate Database. Summary statistics for weather variables in each city are given in Table 1.9.

Table 1.9: Summary statistics for daily air pollutant and weather data (years 1998-2001)

	Auckland mean (range)	Brisbane mean (range)	Canberra mean (range)	Christchurch mean (range)	Melbourne mean (range)	Perth mean (range)	Sydney mean (range)
Daily pollutant levels							
24h $PM_{2.5}$ ($\mu g.m^{-3}$)	11.0* (2.1-37.6)	9.7 (3.2-122.8)	-	-	8.9 (2.8-43.3)	8.1 (1.7-29.3)	9.4 (2.4-82.1)
Number of monitors	1	1	0	0	2	2	3
24h PM_{10} ($\mu g.m^{-3}$)	18.8* (3.2-101.4)	16.5 (3.8-50.2)	-	20.6 (1.3-156.3)	16.6 (3.1-71.1)	16.5 (4.4-68.9)	16.6 (3.7-104.7)
Number of monitors	6	4	0	2	4	1	11
1h NO_2 (ppb)	19.1 (4.2-86.3)	17.3 (4-44.1)	17.9 (0-53.7)	15.7 (1.2-54.6)	23.2 (4.4-62.5)	21.3 (4.4-48)	22.6 (5.2-51.4)
24h NO_2 (ppb)	10.2 (1.7-28.9)	7.6 (1.4-19.1)	7.0 (0-22.5)	7.1 (0.2-24.5)	11.7 (2-29.5)	9.0 (2-23.3)	11.5 (2.5-24.5)
Number of monitors	2	7	1	1	8	5	13
8h CO (ppm)	2.1 (0.2-7.9)	1.7 (0-7)	0.9 (0-5.8)	0.5 (0-5.4)	1.0 (0.1-8)	1.0 (0.1-4)	0.8 (0-4.5)
Number of monitors	3	1	1	2	3	3	4
1h O_3 (ppb)	-	31.5 (7-92.3)	-	-	23.8 (1.7-85.4)	33.6 (13-85)	31.7 (3.2-126.7)
4h O_3 (ppb)	-	28.9 (5.4-75.2)	-	-	21.8 (1.3-73.1)	31.3 (10.6-72.8)	28.9 (2.2-105.1)
8h O_3 (ppb)	-	25.5 (3.7-58.4)	-	-	19.0 (0.8-63)	28.5 (8-64)	24.9 (1.4-86.8)
Number of monitors	0	7	0	0	8	3	12
Weather							
Temperature (°C)	15.7 (6.3-24.1)	20.0 (9.5-30.4)	13.7 (1-28)	11.6 (0-27.2)	15.3 (5.9-31.8)	18.2 (8.2-32.3)	17.8 (8.5-30.1)
Relative humidity (%)	79.1 (52.1-100)	72.4 (29.3-96.3)	69.9 (24.1-97)	75.9 (31-99)	68.7 (25.1-95.5)	67.8 (28-98.5)	70.6 (26.3-97.1)

** There were no TEOM data available for Auckland for the study period, the PM data available for Auckland was hi-vol data recorded once every six days.*

1.5 Validation of air quality and meteorological variables

A strict data preparation protocol was followed to maximise the validity of the data used for analysis. All data including morbidity, mortality, pollutant and weather data underwent extensive screening and manipulation. A thorough examination of each variable was conducted using time-series plots and univariate statistics before that variable was considered eligible for the analysis. Any unusual events, outliers or strange patterns in the data were discussed with the suppliers of the data and resolved, which in some cases led to the resupply of the data due to errors in its extraction. Final data sets were prepared for each city that included all validated data.

1.6 Correlations among air quality and meteorological variables

1.6.1 Overall

Correlations among pollutants and meteorological variables were calculated using the Pearson correlation coefficient. Correlations theoretically range between 1 and –1, where 1 indicates perfect positive correlation, 0 indicates no correlation, and –1 indicates perfect negative correlation. Correlations above an absolute value of 0.8 indicate a strong correlation, those between 0.4 and 0.8 indicate a moderate correlation, and those between 0.2 and 0.4 indicate a weak correlation. Tests of significance establish that a correlation is, or is not, different from zero, within the bounds of chance; they do not establish that a correlation is large or that the association is causative.

As in many other cities, the results showed usually moderate to weak (but statistically significant) correlations between some pollutants, the exception generally being $PM_{2.5}$ and PM_{10}, as the former is usually a significant component of the latter (Table 1.10). There are also moderate to weak (but often statistically significant) correlations between pollutant concentrations and weather variables such as temperature and humidity. Correlations between ozone concentrations and temperature are as expected, because photochemical smog production occurs in the higher temperature periods.

Monitored results for ozone for Auckland and Canberra were considered problematical by the agencies concerned so these were not used.

Table 1.10: Correlation matrix of air pollutants (average 24-hour) and weather variables by city [a]

(A) Auckland

	Relative humidity (RH)	PM_{10}	NO_2	CO
Temp (°C)	-0.17	-0.21	-0.66	-0.50
RH (%)		-0.02[b]	0.29	0.22
PM_{10}			0.25	0.38
NO_2				0.53

(B) Brisbane

	RH	$PM_{2.5}$	PM_{10}	NO_2	CO	O_3
Temp	0.15	-0.07[b]	0.01	-0.60	-0.31	0.12
RH		-0.05[b]	-0.26	0.05	0.24	-0.25
$PM_{2.5}$			0.67	0.34	0.20	0.32
PM_{10}				0.36	0.21	0.40
NO_2					0.64	0.11
CO						-0.10

(C) Canberra

	RH	PM_{10}	NO_2	CO
Temp	-0.57	-0.34	-0.28	-0.58
RH		0.20	0.28	0.42
PM_{10}			0.59	0.76
NO_2				0.55

(D) Christchurch

	RH	PM_{10}	NO_2	CO
Temp	-0.49	-0.46	-0.55	-0.58
RH		0.09 [b]	0.10	0.16
PM_{10}			0.57	0.88
NO_2				0.69

(E) Melbourne

	RH	$PM_{2.5}$	PM_{10}	NO_2	CO	O_3
Temp	-0.66	-0.04 [b]	0.35	-0.28	-0.40	0.68
RH		0.15	-0.27	0.31	0.37	-0.57
$PM_{2.5}$			0.77	0.68	0.61	-0.02
PM_{10}				0.38	0.27	0.27
NO_2					0.68	-0.15
CO						-0.36

(F) Perth

	RH	$PM_{2.5}$	PM_{10}	NO_2	CO	O_3
Temp	-0.62	0.08 [b]	0.34	-0.32	-0.31	0.37
RH		0.03 [b]	-0.22	0.26	0.39	-0.23
$PM_{2.5}$			0.78	0.51	0.54	0.25
PM_{10}				0.21	0.30	0.32
NO_2					0.73	0.18
CO						0.02

(G) Sydney

	RH	$PM_{2.5}$	PM_{10}	NO_2	CO	O_3
Temp	-0.05 [b]	0.05 [b]	0.34	-0.35	-0.44	0.56
RH		0.02 [b]	-0.07	0.16	0.18	-0.37
$PM_{2.5}$			0.86	0.45	0.40	0.30
PM_{10}				0.29	0.22	0.45
NO_2					0.70	-0.08
CO						-0.33

[a] *All coefficients statistically significant at p=0.0001 unless indicated*
[b] *Not statistically significant*
RH=Relative humidity, Temp=temperature, PM=particulate matter, NO_2=nitrogen dioxide, CO=carbon monoxide, O_3=ozone

Table 1.10 shows that where moderate correlation does occur between the pollutants they arise for pollutants connected to similar human combustion emission sources, such as motor vehicle exhausts. Correlated pollutants might therefore serve as indicators of particles from mobile sources rather than as direct toxic agents. This point is examined in more detail in the summary and discussion section of this document.

These correlations are often moderate in magnitude but suggest that it may be difficult to identify the independent contribution of individual pollutants to any association with health outcomes.

1.6.2 By season

Correlation between pollutants for warm and cool seasons are expected to differ. Photochemical smog events in summer lead to increases in ozone and NO_2 at the same time (but not the same place in the region). Significant additional combustion sources, such as fires for home heating (especially in Christchurch) occur in winter. Exhaust emissions from motor vehicles are higher in winter because of 'cold starts'.

The correlation between pollutants is usually above 0.5 (especially in the cool periods), and in the range 0.5 - 0.7 all year round in all cities (Table 1.11).

Table 1.11: Correlations between 8-hour CO and 24-hour NO_2 by city and season

City	All year	Cool period	Warm period
Auckland	0.53	0.47	0.39
Brisbane	0.64	0.64	0.54
Canberra	0.55	0.52	0.52
Christchurch	0.69	0.65	0.57
Melbourne	0.68	0.65	0.71
Perth	0.73	0.67	0.73
Sydney	0.70	0.65	0.73

It is clear that the correlations between particles and the gases CO and NO_2 are strongest in winter (Table 1.12), and it is noted the emission sources are very similar then (motor vehicle exhausts, home fires) for those with positive correlations, i.e. particles, particularly $PM_{2.5}$, and the gases CO and NO_2.

Table 1.12: Correlations between average 24-hour $PM_{2.5}$ and PM_{10} and maximum 8-hour CO and average 24-hour NO_2, by city and season

City	All year NO_2	Cool period NO_2	Warm period NO_2	All year CO	Cool period CO	Warm period CO
Auckland						
PM_{10}	0.25	0.37	0.04	0.38	0.49	0.05
Brisbane						
$PM_{2.5}$	0.34	0.44	0.18	0.20	0.24	0.09
PM_{10}	0.36	0.33	0.23	0.21	0.17	0.16
Canberra						
PM_{10}	0.59	0.62	0.47	0.76	0.82	0.32
Christchurch						
PM_{10}	0.57	0.57	0.20	0.88	0.89	0.39
Melbourne						
$PM_{2.5}$	0.68	0.77	0.62	0.61	0.71	0.47
PM_{10}	0.38	0.66	0.43	0.27	0.55	0.25
Perth						
$PM_{2.5}$	0.51	0.65	0.46	0.54	0.66	0.47
PM_{10}	0.21	0.36	0.30	0.30	0.52	0.30
Sydney						
$PM_{2.5}$	0.45	0.73	0.35	0.40	0.62	0.32
PM_{10}	0.29	0.62	0.30	0.22	0.48	0.26

Correlations between ozone and other pollutant concentrations are generally only positive in the warm period and not in the cool period (Table 1.13). During photochemical smog events (a feature of the warm season), the concentrations of ozone, particles (especially $PM_{2.5}$) and NO_2 increase (Table 1.13). The exception is Brisbane with its wet summers and high sunshine winters when 'cool' period smog events are possible. Perth and Melbourne, with their hot dry summers and cold wet winters, show the cool and warm period differences most strongly.

Table 1.13: Correlations between average 24-hour $PM_{2.5}$, PM_{10}, NO_2, maximum 8-hour CO and temperature and maximum 8-hour O_3, by city and season

City	All year O_3	Cool period O_3	Warm period O_3
Brisbane			
$PM_{2.5}$	0.32	0.42	0.39
PM_{10}	0.40	0.28	0.48
NO_2	0.11	-0.11	0.26
CO	-0.10	-0.20	-0.05
Temperature	0.12	0.41	0.23
Melbourne			
$PM_{2.5}$	-0.02	-0.51	0.43
PM_{10}	0.27	-0.30	0.39
NO_2	-0.15	-0.62	0.52
CO	-0.36	-0.56	0.29
Temperature	0.68	0.46	0.73
Perth			
$PM_{2.5}$	0.25	-0.07	0.48
PM_{10}	0.32	0.14	0.39
NO_2	0.18	-0.19	0.52
CO	0.02	-0.19	0.29
Temperature	0.37	0.17	0.54
Sydney			
$PM_{2.5}$	0.30	-0.16	0.54
PM_{10}	0.45	0.04	0.59
NO_2	-0.08	-0.30	0.33
CO	-0.33	-0.57	0.13
Temperature	0.56	0.51	0.57

The correlation between ozone and temperature would be expected to be strongest in the warm periods as well, as it is shown for Melbourne and Perth. The results for Brisbane show the influence of wet summers, and Sydney shows a similar correlation for both periods.

The magnitude of these correlations indicate that it may be difficult to use multi-pollutant models to separate associations found for different air pollutants and health outcomes, especially in the warm period. This is particularly the case for Melbourne and Perth, the two cities where the relationship between the particles and gases in summer is the strongest.

The correlations between CO and ozone in the warm period are weaker than those between other pollutants and ozone (Table 1.13), as might be expected given CO concentrations are not affected by photochemical smog production.

1.7 Standardising effect estimates

In regression models the effect of an air pollutant on a health outcome is measured as an increase in the effect (for example, risk of death) per unit of the pollutant in question; for example one unit of $PM_{2.5}$ (1 $\mu g.m^{-3}$) or one unit of PM_{10} (1 $\mu g.m^{-3}$). A change of 1 unit of $PM_{2.5}$ does not represent the same increment as 1 unit of PM_{10}, in comparison to the range of exposure of the respective variables: Brisbane daily $PM_{2.5}$ levels ranged from 3.2 to 122.8 $\mu g.m^{-3}$ while PM_{10} levels ranged from 3.8 to 50.2 $\mu g.m^{-3}$ ozone varies from 7 to 92.3 ppb (Table 1.9). Similar problems arise in comparing effects of particles (per 1 $\mu g.m^{-3}$) with effects of gases (per 1 ppb, for example). In addition, because a single unit increment is small in comparison with the range, the corresponding health increment may be small and appear insignificant even though it is statistically significant.

The solution to this is to standardise the effect estimates to represent a comparable (large) change across the distribution of the air pollutant. The measure chosen as the standardised increment in the air pollutant is the inter-quartile range (IQR). The IQR is the difference between the 25th and 75th percentile cut-offs for that pollutant. It can be thought of as the difference in air pollution levels for a moderately bad day (only 25 per cent of days have higher levels) and a moderately good day (only 25% of days have lower levels) for that pollutant. Effectively, IQR represents the change in air pollution in moving from the middle of the lower half of the distribution to the middle of the upper half of the distribution.

The same IQRs were used across cities to standardise the results by city also. For each pollutant, the standard IQR was estimated by taking the mean IQR across cities. The mean IQRs used are shown in Table 1.14.

Table 1.14 also interprets the IQRs, giving the percentages they represent of the mean over all available cities (Table 1.9), the inter-city range in means (difference between highest and lowest city mean), and the national standard, where relevant. With the exception of 8-hour CO, the IQRs represent about a shift of 30-40% in the mean concentration, and an amount one to two times the inter-city range. They represent 7.5 to 15.2 % of the national standard.

Table 1.14: Mean inter-quartile range (IQR) for each pollutant

Pollutant	IQR	Unit	IQR as % of mean over cities	Inter-city range as % of IQR	IQR as % of standard
24-hour $PM_{2.5}$	3.8	$\mu g.m^{-3}$	41.3	76.3	15.2%
24-hour PM_{10}	7.5	$\mu g.m^{-3}$	44.9	54.7	15.0%
24-hour NO_2	5.1	ppb	31.0	92.2	
1-hour NO_2	9.0	ppb	42.9	83.3	7.5%
8-hour CO	0.86	ppm	116	186.0	9.6%
8-hour O_3	8.8	ppb	36.7	108.0	
4-hour O_3	9.1	ppb	36.4	104.4	11.4%
1-hour O_3	9.8	ppb	35.0	100.0	9.8%

1.8 Reporting of results

The **magnitude of effect estimates** are reported in two ways:

a) The average or mean increase (%) in daily health outcomes counts *per unit increase in pollutant concentration*, e.g. per 1 ppb increase in ozone.

b) The average or mean increase (%) in daily health outcomes counts that would be expected to occur when the air pollution concentrations increase by an amount equal to the IQR for the pollutant (e.g. by 3.8 $\mu g.m^{-3}$ in $PM_{2.5}$).

For example, the estimated per cent increase in all cardiac hospital admissions per day for adults in the age group, 65 years and greater, *per unit increase in pollutant concentration* are: **0.6%** per 1ppb of 24-hour average NO_2, **3.3%** per 1 ppm of maximum 8-hour CO, and **0.5%** per 1 $\mu g.m^{-3}$ of 24-hour average $PM_{2.5}$. The results might suggest that CO is much more important than the other two pollutants in terms of cardiac admissions. Using IQRs from Table 1.14 we see that the estimated per cent increase in cardiac admissions per day, per IQR increase is 0.6%×5.1 = 3.1% , 3.3%×0.86 = 2.8%, 0.5%×3.8 = 1.9% for 24-hour average NO_2, maximum 8-hour CO, and 24-hour average $PM_{2.5}$ respectively. Thus, 24-hour average NO_2 actually has the largest effect, and the strengths of the effects are more similar than the per-unit effects suggest.

An additional consideration in interpreting effect estimates is the meaning of an IQR change, in terms of the current pattern of air pollution. Is it plausible that an air pollutant concentration could change by that amount? Do different cities have different concentrations to the extent of an IQR? For example, the IQR for 24-hour average $PM_{2.5}$ is 3.8 $\mu g.m^{-3}$, or about 41% of the observed mean (Table 1.14). Thus, if mean $PM_{2.5}$ levels were to increase by 41%, on average, we would predict an increase of 1.9% in daily cardiac admissions in the elderly. Mean 24-hour average $PM_{2.5}$ vary from 8.1 (Perth) to 11.0 (Auckland) a difference equal to 76% of the IQR.

Uncertainty in the estimates

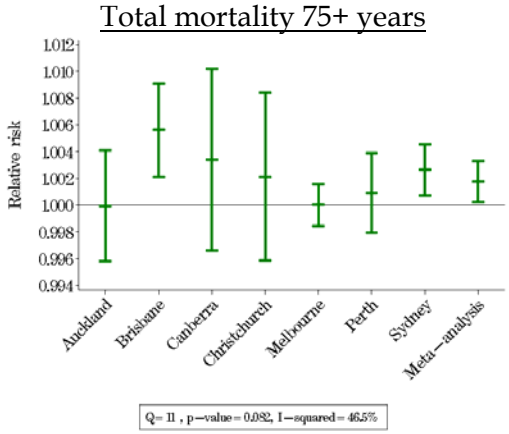
Effect estimates are based on data collected over a given time, in a finite number of places. In considering how well these represent effects generally over all similarly exposed populations at any time, we need to quantify the uncertainty in the estimates. These will depend on the number of observed events; for example, estimates based on Perth data will be less precise than estimates based on Sydney data.

Uncertainty in the estimates is incorporated using 95% confidence intervals. These are ranges around the point estimates of effects, constructed according to statistical principles so that the range has a 95% probability of including the 'true' value of the effect, given that our observed effect is assumed to contain a component of error.

For example, in Figure 1.2, the plots show the estimated relative risks of all cause death (middle cross-bars of each vertical line) associated with a 1 ppb increase in exposure to NO_2. A relative risk of 1 represents no increase in mortality counts, a relative risk of 1.01 represents a predicted 1% increase in mortality counts if NO_2 concentrations went up by 1 ppb; for example, Figure 1.2 also shows the 95%

confidence intervals (the upper and lower cross-bars of the vertical lines) for these relative risks. The confidence intervals for the larger cities, Sydney and Melbourne, are much narrower than those for Canberra and Christchurch, for instance, reflecting greater confidence in these estimates. Given that the 95% confidence intervals for the estimates for Brisbane and Sydney exclude the value 1, corresponding to zero increase, it is conventional to conclude that the findings are consistent with a 'true' association between the pollutant and mortality. Conversely, the confidence intervals for Canberra, Christchurch, and Perth include the value 1, and it would be concluded that this is consistent with a 'true' relative risk of 1, corresponding to zero increase.

Figure 1.2: City specific and meta-analysis relative risks (and 95% confidence intervals) for an increase in total daily mortality for adults (aged 75 years and greater) associated with a 1 ppb increase in 1-hour maximum NO_2 (lag 0-1)



Confidence intervals also assist in interpreting differences between results for different outcomes. For example, the mean estimate for the per cent increase in total mortality counts of adults in the age group 75 years and greater, per1 ppb increase in maximum 1-hour NO_2 levels, is 0.2% with a 95% CI of (0.1-0.8%), while it is 0.4% (0.1-0.8%) for respiratory mortality for the same age group (Table 1.15). The confidence interval for increases in respiratory mortality is as wide as the one for all cause mortality, because of the lower number of events, despite the greater effect size, suggesting that imprecision in the estimates is a plausible explanation of the observed difference.

Figure 1.2 shows the combined (meta-analysis) estimate. It has a relatively narrow confidence interval, as it is based on all cities combined and therefore has less uncertainty, showing the advantage of meta-analysis. We note that the 95% confidence interval just excludes 1, indicating the overall association between mortality and 1-hour maximum NO_2 (lag1) to be significant. One disadvantage of meta-analysis is that it suppresses among-city variation. This is measured by the I^2 statistic which show the variability among estimates compared to within estimates.

Table 1.15: Significant increases in mortality (and 95% confidence intervals) associated with maximum 1-hour NO_2 (lags 0-1) for all seven cities (case-crossover meta-analysis)

Health outcome	Age group (years)	Per unit % increase (95% CI)	I^2 statistic
Respiratory	75+	0.4 (0.1,0.8)	0.0
Total all cause	75+	0.2 (0.0,0.3)	46.5

Statistical significance of effects

Two types of significance tests are used. The first deals with the primary purpose of the analysis: to determine whether there is evidence of a pollutant-health outcome relationship, whether in an individual city or in the pooled data for all cities. These can be derived from standard regression model analyses and are based upon an estimate and its standard error. A virtually equivalent ascertainment of significance can be obtained by identifying whether the 95% confidence interval includes (not significant) or excludes (significant) the null value.

Secondly, we usually want to determine whether observed variation among cities exceeds that expected by chance. Here we carry out a test of heterogeneity, again derived from a regression model which includes a city by health effect factor. The seven cities in this study differ in size by an order of magnitude. The two small cities, Canberra and Christchurch, will therefore have the least precision for all the city-specific associations. Due to this lack of precision, the statistical test for heterogeneity of the city-specific associations will be sensitive mainly to the five larger cities and so attention should be focused on these in terms of addressing explanations.

Magnitude of effects vs statistical significance

In much scientific literature, statistical tests of significance are needed and/or expected before a 'finding' can be claimed. Their role is to eliminate chance (within limits) as an explanation for an observed association. They do not identify whether an association is large or important from a clinical or public health point of view. They do not establish causality. This report places some emphasis on significance, in its role of eliminating chance findings. This is particularly important when large numbers of associations are examined: if 5% is used as the cut-off for statistical significance (the usual practice), then if 100 independent tests are performed it would be expected that 5 would be false positives (declaring an association significant when it is not).

Negative (non-significant) findings may be important, but usually only when the study has had sufficient power to detect an effect whose magnitude is of interest. They may only indicate that the study has insufficient power to detect the effect, or that other factors may have attenuated a 'real' effect.

However, effect estimates, with confidence intervals, are also important to present, with some scrutiny of the consistency of patterns when one is dealing with a large set of results. An added complication for the present study is the relatively high correlation among pollutants, which means that some statistical tests and point estimates of effects are themselves correlated, with some redundancy of information.

It would be scientifically difficult to claim a positive finding without calculating and presenting P-values for significance, no matter what the consistency of effect estimates (the appearance of which may be driven by correlations among pollutants). The use of significance tests in this report reflects that reality.

So the approach used in this report is two-fold: first significance tests are used to evaluate the role of chance. As well, the pattern of effect estimates and their confidence intervals is presented and considered. Effect estimates are declared significant using the conventional 5% level of significance, without adjustment downwards for multiple comparisons. Given the large number of tests performed and the practice of adjusting P-values for multiple comparisons this is probably over-inclusive.

Chapter 2 Nitrogen dioxide

Nitrogen dioxide is monitored in all the cities under study here: Auckland (2 monitors), Brisbane (7 monitors), Canberra (1 monitor), Christchurch (1 monitor), Melbourne (8 monitors), Perth (5 monitors), and Sydney (13 monitors). As the number of monitors used in each city ranges from 1 to 13, the representativeness of the network varies. The project team used the data supplied by the government agencies in each city. Detailed results are supplied in *Volume 3: Appendix 1.*

The results are reported as the percentage increase in the daily health outcome data for 1998 to 2001 either due to an increase in the daily maximum 1-hour concentrations of NO_2, or due to an increase in the 24-hour average concentrations of NO_2.

Summarised in Table 2.0.1 are the statistically significant meta-analysis estimates for health impacts associated with NO_2. Given are the associations for the short-term exposure (an average of the NO_2 concentration on the same day as the health effect, and the day before) to NO_2 on morbidity and mortality health outcomes, for those cases when the estimates for all cities in Australia and New Zealand can be reasonably combined to derive such estimates. All individual city and meta-analysis estimates for short-term associations are shown in *Volume 3: Appendix A3.1.*

Table 2.0.2 summarises the associations for long-term exposure (an average of the NO_2 concentration on the same day as the health effect, and all forty-days before). These results indicate there is no 'harvesting' effect in these results, with the long-term effects showing larger impacts than the short-term effects. All individual city and meta-analysis estimates for 40-day associations are shown in *Volume 3: Appendix A5.1.*

Table 2.0.1: Significant increases in morbidity and mortality (and 95% confidence intervals) associated with a one-unit (ppb) increase in NO_2 (lags 0-1) for all seven cities (case-crossover meta-analysis)

Health outcome	Age group (years)	Averaging period	% Increase (95% CI)	I^2 statistic
Hospital admissions				
All cardiovascular	15-64	24-h av.	0.3 (0.1,0.5)	0.0
		1-h max.	0.1 (0.0,0.2)	0.0
	65+	24-h av.	0.6 (0.4,0.8)	18.4
		1-h max.	0.3 (0.2,0.4)	3.5
Cardiac	15-64	24-h av.	0.4 (0.2,0.7)	0.0
	65+	24-h av	3.4 (1.9, 4.9)	54.1*
		1-h max.	0.6 (0.4,0.9)	34.0
Ischemic heart disease	65+	24-h av.	0.5 (0.2,0.8)	19.7
		1-h max.	0.3 (0.2,0.4)	0.0
Myocardial infarction	65+	24-h av.	0.8 (0.2,1.5)	38.2
		1-h max.	0.4 (0.2,0.7)	19.7
Cardiac failure	15-64	24-h av.	0.9 (0.0,1.8)	0.0
	65+	24-h av.	1.3 (0.4,2.2)	61.3*
		1-h max.	0.7 (0.4,1.0)	50.0
Arrhythmia	15-64	24-h av.	1.0 (0.4,1.5)	0.0
		1-h max.	0.4 (0.1,0.7)	0.0
All respiratory	1-4	1-h max.	0.3 (0.1,0.5)	46.9
	5-14	24-h av.	1.1 (0.3, 1.9)	54.0*
		1-h max.	0.5 (0.2, 0.9)	52.0
	15-64	1-h max.	0.1 (0.0,0.3)	0.0
Mortality				
Cardiovascular	All ages	1-h max.	0.2 (0.0,0.3)	0.0
	75+	1-h max.	0.2 (0.0,0.3)	14.3
Respiratory	All ages	1-h max.	0.4 (0.1,0.7)	0.0
	75+	1-h max.	0.4 (0.1,0.8)	0.0
Total all cause	All ages	1-h max.	0.2 (0.0,0.3)	51.5
	0-74	1-h max.	0.2 (0.0,0.3)	0.0
	75+	1-h max.	0.2 (0.0,0.3)	46.5

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.0.2: Significant forty-day increases in mortality (and 95% confidence intervals) associated with a one IQR increase in average 24-hour NO_2

Mortality	Age group (years)	Per unit IQR % increase (95% CI) for 40 days	Per unit IQR % increase (95% CI) for 0-1 days
Cardiovascular	All ages	2.3 (1.1,3.5)	1.0 (-0.2, 2.2)
Respiratory	All ages	5.1 (2.4,7.9)	2.3 (-0.3, 5.1)
Total all cause	All ages	1.7 (0.9,2.5)	1.1 (-0.2, 2.4)

Only Australian cities

The results from the meta-analysis can sometimes indicate differences between the countries. When the meta-analysis was conducted on only the five Australian cities, there were sometimes significant results that did not occur when all seven cities (including the two New Zealand cities) were considered. Shown in Table 2.0.3 are the additional significant results when only the five Australian cities were considered.

The results were also the same for all Australian cities.

Table 2.0.3: Significant increases in morbidity (and 95% confidence intervals) associated with a one-unit (ppb) increase in NO_2 (lags 0-1) for all (5) Australian cities – results only presented for those not identified in Table 2.0.1

Health outcome	Age group (years)	Averaging period	Per unit % increase (95% CI)	I^2 statistic
Hospital admissions				
Cardiac failure	65+	24-h av.	1.0 (0.8,1.3)	0.0
		1-h max.	0.8 (0.6,1.1)	28.6
Respiratory	< 1	24-h av.	0.6 (0.3,0.9)	0.0
		1-h max.	0.5 (0.2,0.8)	29.7
	5-14	24-h av.	0.5 (0.2,0.8)	32.6
		1-h max.	0.4 (0.1,0.8)	46.4
Asthma	1-4	24-h av.	0.4 (0.0,0.7)	12.3
		1-h max.	0.3 (0.0,0.6)	0.0
	15-64	24-h av.	0.3 (0.1,0.6)	6.1
Pneumonia & acute bronchitis	< 1	24-h av.	0.6 (0.3,1.0)	7.2

*

However, it is not possible in statistical analyses like these to identify any causes for the increases; only significant associations can be noted. It is also possible that the increases identified here with exposure to NO_2 may not be due to NO_2, but to other pollutants which are correlated with NO_2 as they arise from the same emissions source, such as motor vehicle exhausts.

When significant increases were found for exposure to outdoor concentrations of CO, NO_2, and particles, it was not possible to separate them (that is, the increases are not additive, but may be referring to the same impact, such as from a mixture of air pollutants from motor vehicle exhausts).

2.1 Mortality

2.1.1 Short-term averages for air pollutants

Two different age groups were examined: all ages and 75 years and greater. Table 2.0.1 shows that the short-term averages (average of same day and day before: 0-1) of outdoor concentrations of NO_2 are associated with significant increases in mortality for all causes, and for cardiovascular and respiratory disease, but only for increases in maximum 1-hour NO_2 concentrations. All individual city and meta-analysis estimates for short-term associations with mortality are shown in *Volume 3: Appendix A3.1*, Tables CM.30 to CM.38.

Typical significant mean increases in daily death counts (associated with a *one-unit* increase of maximum 1-hour NO_2, average lags 0-1) in different disease categories were as follows:

- 0.2% increase in total mortality, and in mortality due to cardiovascular disease, both for all ages and the age group 75 years and greater, and these results are similar for all cities

- 0.4% increase in mortality due to respiratory disease, both for all ages and the age group 75 years and greater, and these results are similar for all cities.

As deaths due to cardiovascular disease are often of the order of five times greater than those due to respiratory disease, and deaths in all categories more than ten times greater, then the largest increases in deaths are in the total deaths in the elderly, half of which are due to cardiovascular disease, even though the per unit increase is larger in the respiratory disease category.

The mortality impacts of NO_2 for the short term (average of same day as, and day before, death) are shown in Figures 2.1.1 to 2.1.2 for total deaths (non-accidental, all causes), deaths due to cardiovascular disease and deaths due to respiratory disease. The results are shown for two age groups: all age groups and the oldest (75 years and greater). It is clear that the larger impacts usually occur in the elderly, the frailer group.

There are some differences between cities for associations between all total all cause mortality and maximum 1-hour NO_2 (but not statistically significant, as confirmed by the statistical test) with an average estimate positive. The results for Brisbane are the reason for heterogeneity between the cities, with its estimate higher than the other pollutants. The test for heterogeneity shows a p-value of 0.054. However, when Brisbane is removed from the multi-city analysis, the pooled estimate remains positive and significant, and there is no evidence to suggest there is a significant decrease in the resulting estimate compared with that for all the cities (see Table 2.2.1). Therefore, the assumption that the pooled estimate can be applied to effects in all cities is valid.

Figure 2.1.1: City specific and meta-analysis relative risks (and 95% confidence intervals) for mortality associated with a one-unit (ppb) increase in 24-hour NO_2 (average lag 0-1)

Figure 2.1.1.A: Total all cause mortality

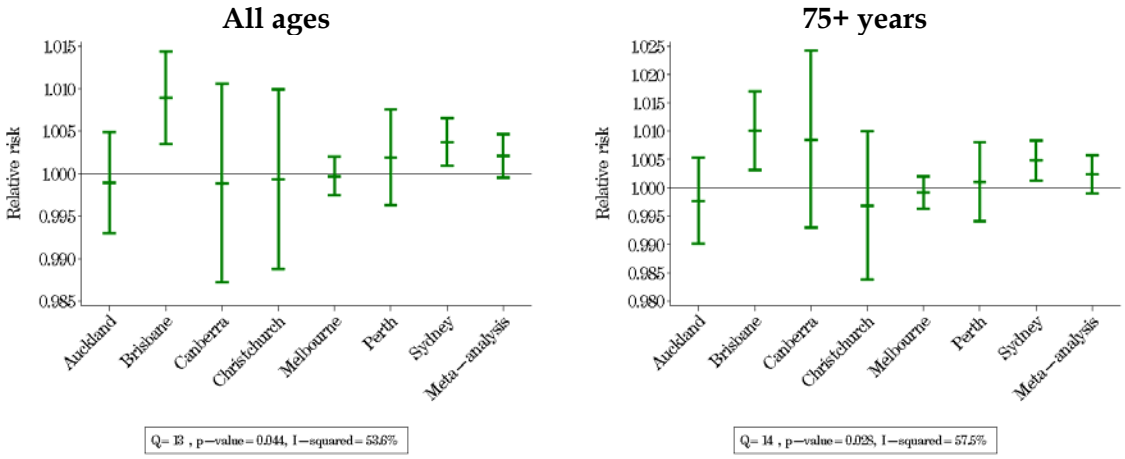


Figure 2.1.1.B: Respiratory mortality

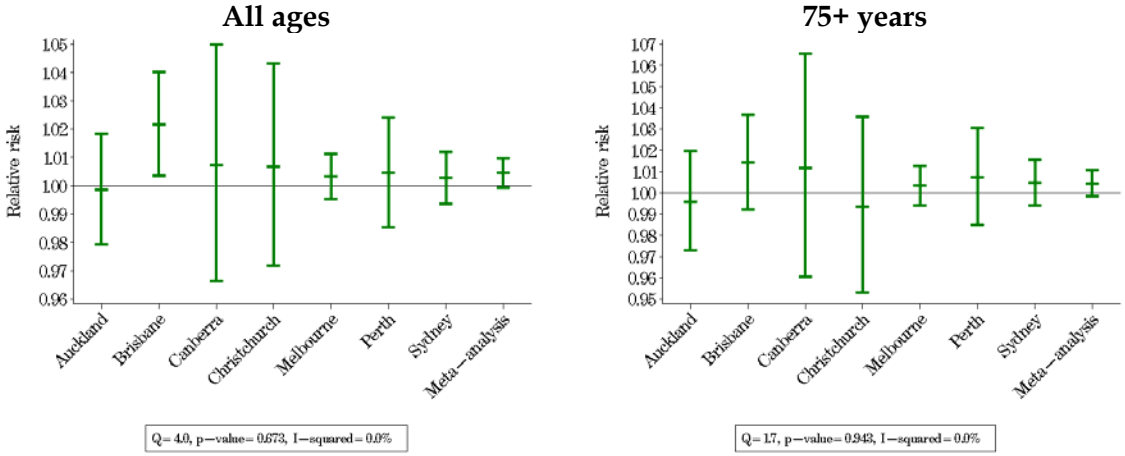


Figure 2.1.1.C: Cardiovascular mortality

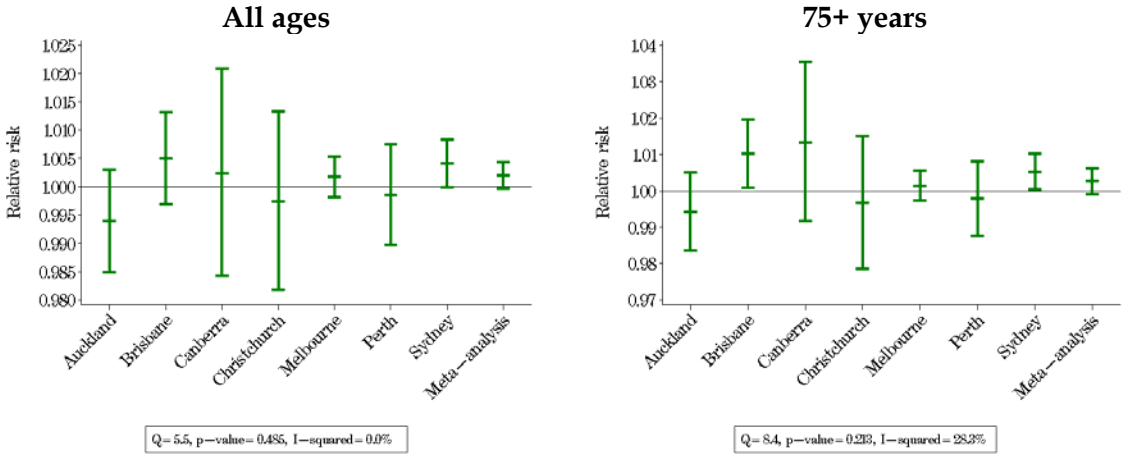


Figure 2.1.2: City specific and meta-analysis relative risks (and 95% confidence intervals) for mortality associated with a one-unit (ppb) increase in 1-hour NO_2 (average lag 0-1)

Figure 2.1.2.A: Total all cause mortality

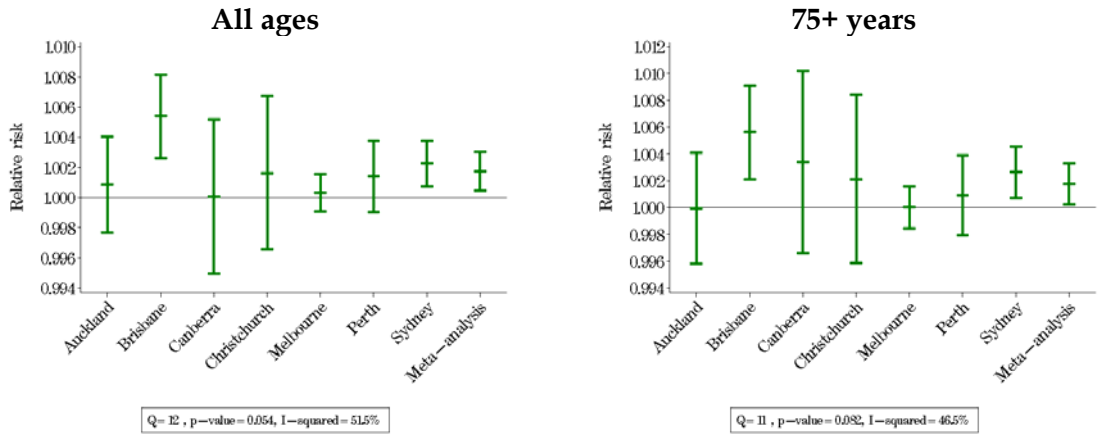


Figure 2.1.2.B: Respiratory mortality

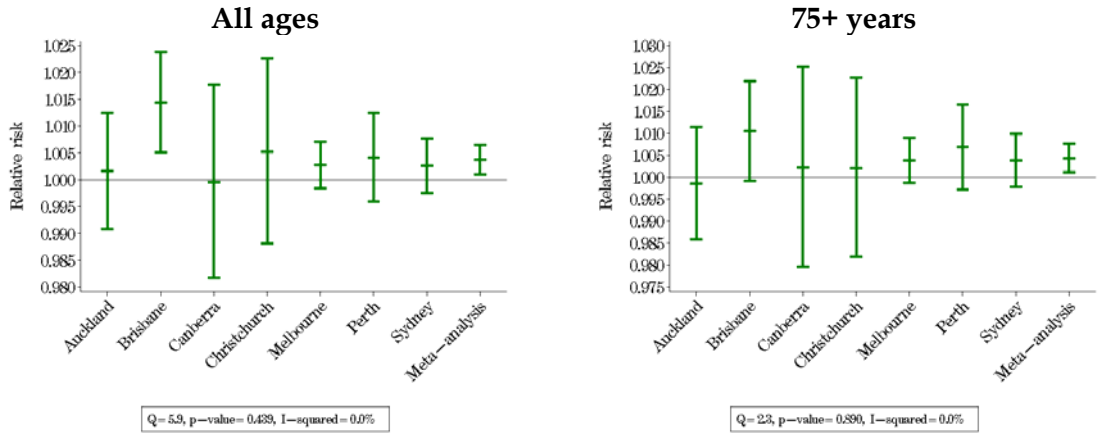
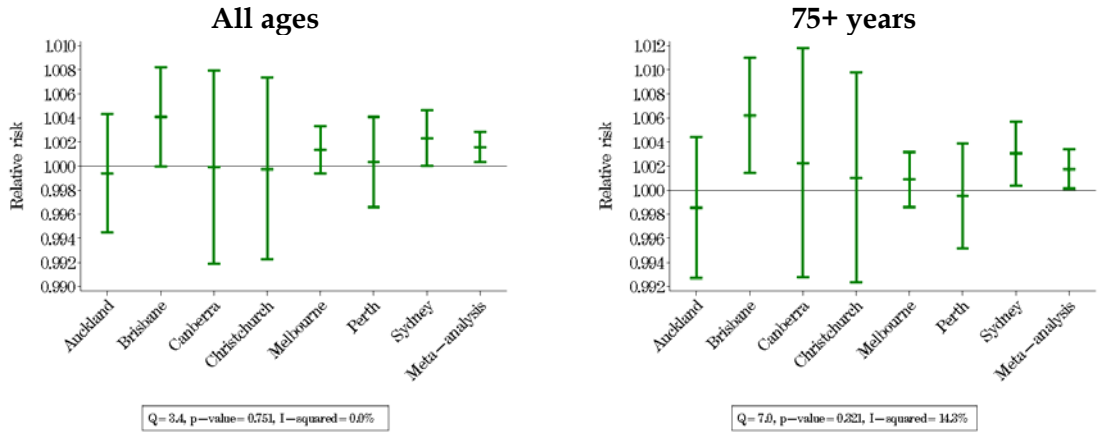


Figure 2.1.2.C: Cardiovascular mortality



Differences between cities can be seen in Figures 2.1.1 to 2.1.2. For cardiovascular and respiratory mortality, there appears to be little difference in the results for the cities, but Table 2.1.1 shows that, for total mortality, the increases are more homogeneous (lower I-squared statistic), but lower when Brisbane is left out. Such a result is apparent in Figure 2.1.1.A.

Table 2.1.1: Differences between cities using a leave-one-city-out sensitivity analysis for total mortality (all ages) associated with an IQR increase in NO_2 (average lag 0-1) (case-crossover meta-analysis)

	1-hour NO_2			24-hour NO_2		
City left out	Inc	95%CI	I^2	Inc	95%CI	I^2
Auckland	1.7	0.4, 3.0	59.1*	1.3	-0.1, 2.8	58.4*
Brisbane	1.0	0.3, 1.8	0.0	0.6	-0.3, 1.5	12.1
Canberra	1.7	0.4, 2.9	58.6*	1.2	-0.3, 2.6	60.6*
Christchurch	1.6	0.3, 2.9	59.6*	1.2	-0.3, 2.6	60.7*
Melbourne	2.0	0.8, 3.2	27.6	1.6	0.1, 3.0	34.1
Perth	1.6	0.3, 3.0	59.6*	1.1	-0.4, 2.6	61.3*
Sydney	1.5	0.0, 2.9	54.3*	0.8	-0.7, 2.4	50.4
Australian only	1.7	0.3, 3.2	67.3*	1.4	-0.2, 3.0	66.0*
NZ only	1.0	-1.4, 3.4	0.0	-0.5	-3.1, 2.2	0.0
Overall	1.6	0.4, 2.7	51.5	1.1	-0.2, 2.4	53.6*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.1.2 'Harvesting'?

Table 2.0.2 shows that the associations for the cumulative impacts of outdoor NO_2 concentrations **forty days** prior to the health outcome are much higher than those for short-term exposures. For example, a *one-unit* increase (ppb) in 24-hour average NO_2 was associated with a significant mean increase in daily mortality of 1.7% (CI of 0.9% to 2.5%) for all causes and all ages, compared to an insignificant increase of 0.2% (CI of -0.0% to 0.5%) for short-term, and there is evidence that this difference is significant. These results do not support the 'harvesting' effect hypothesis.

The I-squared statistic indicates that the harvesting results are the same for all cities.

Table 2.1.2: Per cent changes (and 95% confidence interval) in total mortality (all ages) over 40-day periods* associated with 24-hour NO_2

	24-hour NO_2	
City	Inc	95%CI
Auckland	1.7	-0.5, 4.0
Brisbane	2.7	0.7, 4.7
Canberra	-3.0	-7.9, 2.1
Christchurch	1.9	-1.2, 5.0
Melbourne	1.1	-3.6, 6.1
Perth	3.0	0.5, 5.6
Sydney	1.3	0.1, 2.5
I-squared (%)	0.0	- -
Meta-analysis	1.7	0.9, 2.5
Meta-analysis IQR	8.8	4.6, 13.0

Table 2.1.3: Per cent changes (and 95% confidence interval) in respiratory mortality (all ages) over 40-day periods* associated with 24-hour NO_2

	24-hour NO_2	
City	Inc	95%CI
Auckland	4.0	-3.1, 11.5
Brisbane	8.0	1.3, 15.2
Canberra	-2.0	-17.1, 16.0
Christchurch	1.9	-7.5, 12.3
Melbourne	11.7	-4.6, 30.9
Perth	7.4	-1.1, 16.6
Sydney	4.4	0.4, 8.6
I-squared (%)	0.0	- -
Meta-analysis	5.1	2.4, 7.9
Meta-analysis IQR	26.3	12.2, 40.7

Table 2.1.4: Per cent changes (and 95% confidence interval) in cardiovascular mortality (all ages) over 40-day periods* associated with 24-hour NO_2

	24-hour NO_2	
City	Inc	95%CI
Auckland	1.4	-1.9, 4.9
Brisbane	3.4	0.3, 6.5
Canberra	-1.8	-8.9, 5.8
Christchurch	2.2	-2.0, 6.5
Melbourne	1.6	-5.8, 9.5
Perth	3.6	-0.5, 7.8
Sydney	2.2	0.4, 4.0
I-squared (%)	0.0	- -
Meta-analysis	2.3	1.1, 3.5
Meta-analysis IQR	11.8	5.5, 18.2

* *Estimates from a random effects meta-analysis of cities using distributed lag models, individual city results are shown for a one-unit increase in pollutant and meta-analysis results are shown for an inter-quartile range (IQR) increase where indicated.*

2.1.3 Seasonal differences

Separate analyses were carried out for the warm (November - April) and cool (May - October) periods to identify if there are different effects in each period (due to different emission patterns, atmospheric chemistry). Individual city seasonal results for mortality are shown in *Volume 3: Appendix A3.4*, Tables WC.30 to WC.38. Meta-analysis and results from the leave-one-city-out analysis for mortality are shown in *Volume 3: Appendix A3.5*, Tables WCL.30 to WCL.38.

In the seasonal analysis, no significant differences were found between the cities. The differences found between the cool and warm periods can be summarised as follows:

- the increases in mortality for all ages due to all causes, all cardiovascular disease, and all respiratory disease, were only significant for the cool period
- the increases in mortality for adults aged 75 years and greater, due to all causes, were only significant for the cool period.

It might be expected that some pollutant effects may be different in different seasons. For example, ozone peaks during the summer smog episodes in general (although Brisbane may also have winter smog events). There do appear often to be larger warm period impacts in Brisbane, and sometimes in the other cities, but there are more likely to be larger impacts in the cool period, especially in Melbourne and Christchurch.

Significant seasonal associations found between mortality and NO_2 for both cool and warm seasons are shown in Table 2.1.5.

Table 2.1.5: Significant seasonal increases in mortality associated with an IQR increase in NO_2 (average lag 0-1) (case-crossover meta-analysis)

Mortality (age group)		1-hour NO_2			24-hour NO_2		
		Inc	95%CI	I^2	Inc	95%CI	I^2
Cardiovascular (all ages)	cool	1.7	0.0, 3.4	0.0	1.1	-0.7, 3.0	11.9
	warm	0.4	-1.4, 2.2	0.0	-0.5	-2.6, 1.8	0.0
Respiratory (all ages)	cool	4.3	0.7, 8.1	0.0	2.8	-0.7, 6.5	0.0
	warm	1.4	-2.7, 5.7	0.0	-0.4	-7.0, 6.6	22.4
All cause (75+ years)	cool	1.5	0.1, 2.9	0.0	1.1	-0.4, 2.7	12.5
	warm	1.6	-0.6, 4.0	49.6	0.8	-2.0, 3.6	45.4
All cause (all ages)	cool	1.5	0.3, 2.7	10.3	0.9	-0.4, 2.3	26.1
	warm	1.1	-0.4, 2.7	32.2	0.4	-1.6, 2.5	40.5

2.2 Hospital admissions

Typical significant mean increases in daily counts for hospital admissions (associated with a *one-unit* increase of maximum 1-hour NO_2) in different disease categories were as follows:

- for adults aged 65 years and greater, there was an estimated 0.3% increase in admissions for all cardiovascular disease, 0.6% increase for all cardiac disease, 0.3% increase for ischemic heart disease, 0.4% increase for myocardial infarction, and 0.7% increase for cardiac failure

- for adults aged between 15 and 64 years, there was an estimated 0.4% increase in admissions for arrhythmia, and 0.1% increase for all cardiovascular disease

- for all respiratory disease, there was an estimated 0.3% increase in admissions for children in the age group 1 to 4 years, and 0.1% increase for the age group 15 to 64 years.

The statistical tests for heterogeneity indicated these results were the same magnitude for all cities, with the possible exception of the results for cardiac failure.

2.2.1 Hospital admissions due to cardiovascular disease

The following categories for cardiovascular admissions were examined in this study (see Table 1.2 for the ICD codes):

- total cardiovascular disease
- stroke
- cardiac disease
- ischemic heart disease
- myocardial infarction
- cardiac failure
- arrhythmia.

In the following section, the associations between cardiac admissions for two age groups (15-64 years, 65 years and greater) and ambient concentrations of NO_2 in the short term (average of the same day and day before – lag [0,1]) are presented for these health outcomes, and the daily maximum 1-hour and 24-hour average NO_2 concentrations are used.

2.2.1.1 Cardiovascular disease

The associations with total cardiovascular admissions are shown in Figure 2.2.1 and Figure 2.2.2. There are significant associations for both age groups, and there are no significant differences between the city results.

Figure 2.2.1: City specific and meta-analysis relative risks (and 95% confidence intervals) for total cardiovascular admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

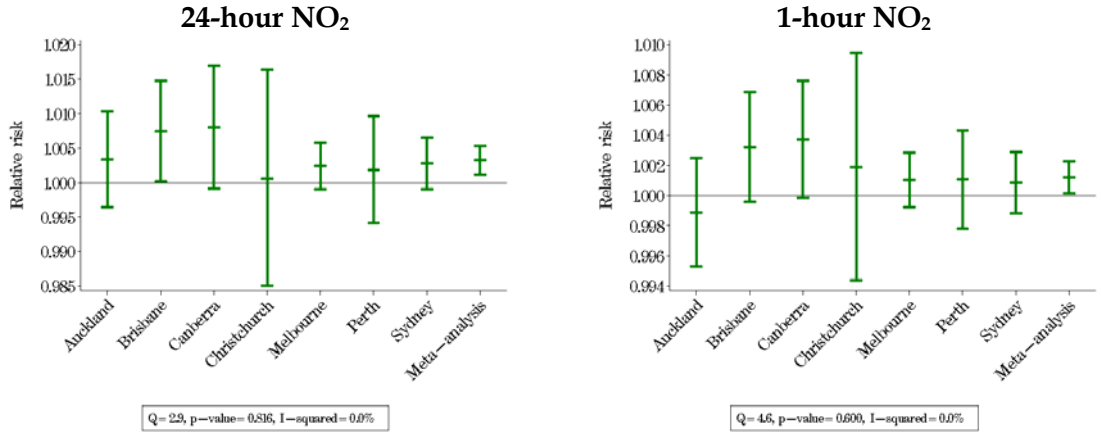
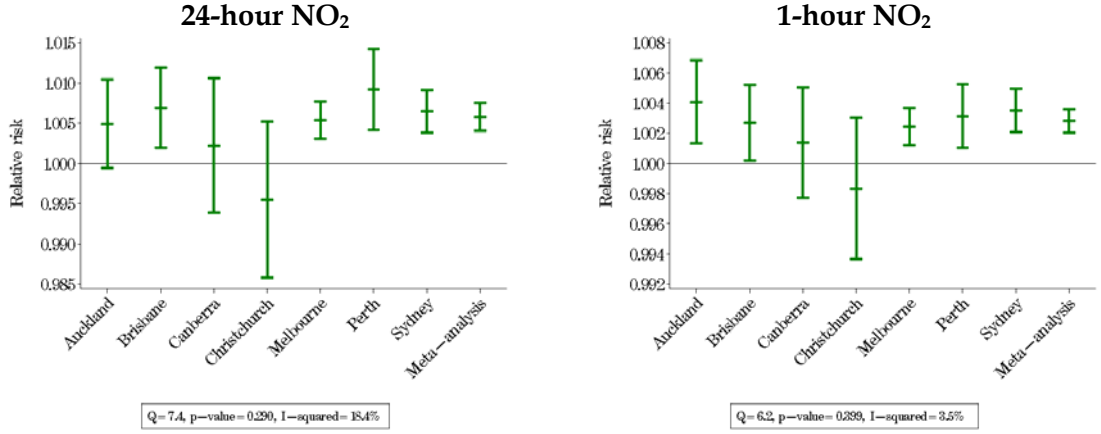


Figure 2.2.2: City specific and meta-analysis relative risks (and 95% confidence intervals) for total cardiovascular admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Table 2.2.1 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis for the older age group. The associations are statistically significant and positive for the Australian cities as a group, and for both age groups.

Table 2.2.1: Differences between cities using a leave-one-city-out sensitivity analysis for cardiovascular admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1) (case-crossover meta-analysis)

	1-hour NO_2			24-hour NO_2		
City left out	Inc	95%CI	I^2	Inc	95%CI	I^2
Auckland	2.4	1.7, 3.2	6.4	3.0	2.0, 4.1	30.9
Brisbane	2.5	1.7, 3.4	19.5	2.9	1.9, 4.0	30.1
Canberra	2.6	1.8, 3.4	10.8	3.1	2.1, 4.1	24.3
Christchurch	2.7	2.0, 3.4	0.0	3.2	2.4, 3.9	0.0
Melbourne	2.7	1.8, 3.7	11.5	3.0	1.8, 4.3	30.0
Perth	2.5	1.6, 3.4	18.3	2.8	2.0, 3.7	9.2
Sydney	2.3	1.5, 3.1	0.3	2.8	1.6, 4.1	28.7
Australian only	2.6	1.8, 3.3	0.0	3.2	2.4, 4.0	0.0
NZ only	1.5	-3.0, 6.1	76.6*	0.7	-3.5, 5.0	63.2*
Overall	2.6	1.8, 3.3	3.5	3.0	2.1, 3.9	18.4

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.1.2 Stroke

The associations with hospital admissions due to stroke are shown in Figure 2.2.3 and Figure 2.2.4. There are no statistically significant associations and there are no significant differences between the cities for maximum 1-hour NO_2 (confirmed by the statistical tests).

For 24-hour average NO_2, there are differences between the cities for the 15-64 years age group (but not for the 65+ years age group), mainly due to Brisbane and Canberra showing significant increases, but the multi-city estimates remain insignificant when Canberra or Brisbane is removed and then there is no evidence of heterogeneity. Therefore, the insignificant result is only applicable to Auckland, Christchurch, Melbourne, Perth and Sydney.

Figure 2.2.3: City specific and meta-analysis relative risks (and 95% confidence intervals) for stroke admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

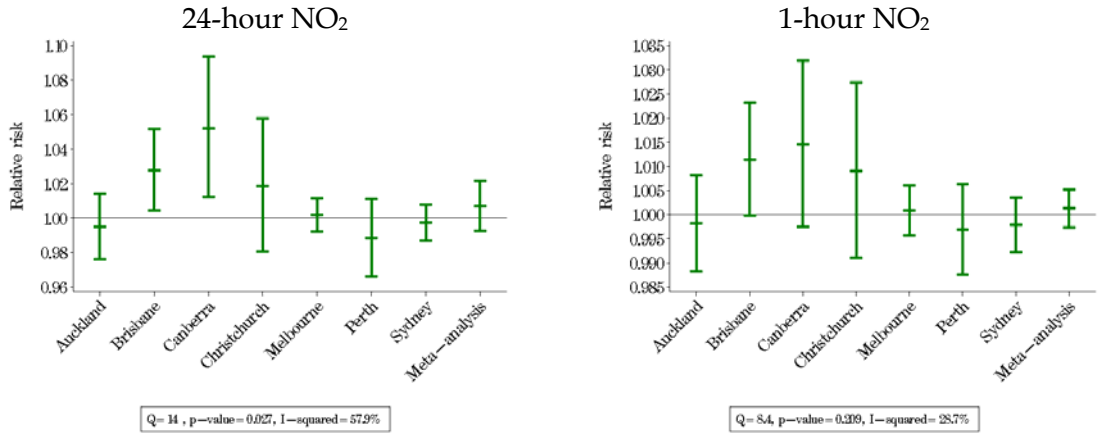
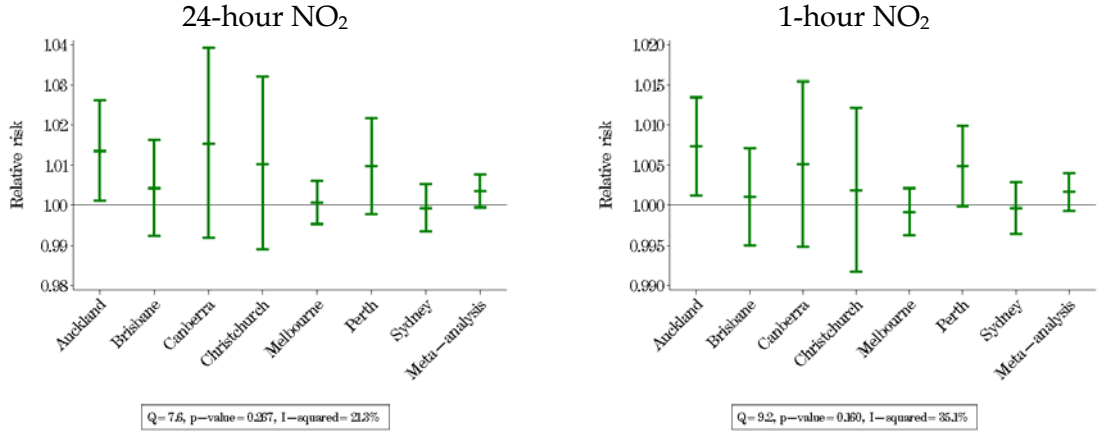


Figure 2.2.4: City specific and meta-analysis relative risks (and 95% confidence intervals) for stroke admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Table 2.2.2 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis. The impacts are statistically significant for the (two) New Zealand cities.

Table 2.2.2: Differences between cities using a leave-one-city-out sensitivity analysis for stroke admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2		
City left out	Inc	95%CI	I^2
Auckland	0.5	-1.2, 2.1	0.0
Brisbane	1.8	-0.7, 4.3	45.9
Canberra	1.4	-0.8, 3.7	42.1
Christchurch	1.6	-0.7, 4.0	45.8
Melbourne	2.3	-0.1, 4.8	25.4
Perth	0.9	-1.2, 3.0	25.9
Sydney	2.3	-0.4, 5.0	38.5
Australian only	0.6	-1.3, 2.5	16.7
NZ only	5.4	0.6, 10.5	0.0
Overall	1.5	-0.6, 3.7	35.1

2.2.1.3 Cardiac admissions

The associations with hospital admissions due to all cardiac disease are shown in Figure 2.2.5 and Figure 2.2.6. There are significant associations for both age groups (especially the older one). There is evidence of heterogeneity for the cities for the results for 24-hour average NO_2 in the older age group but not for the younger age group (confirmed by the statistical tests).

Figure 2.2.5: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

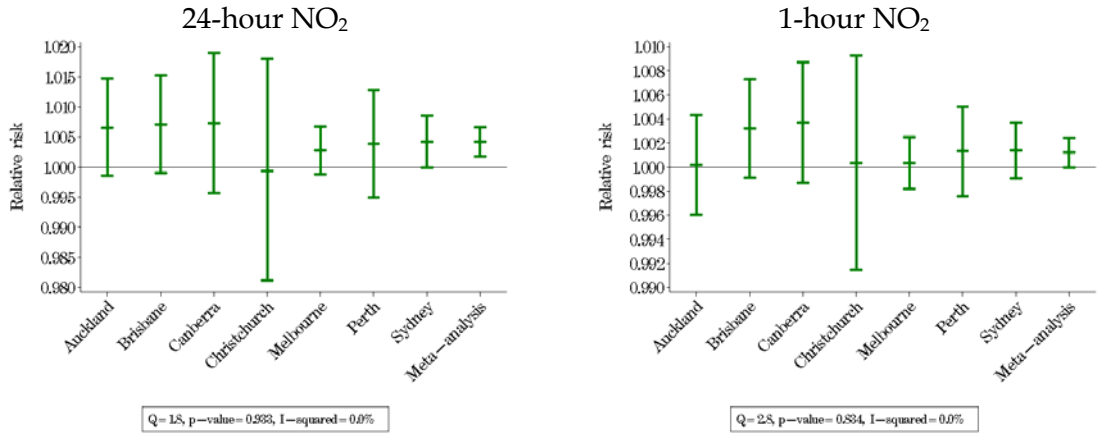
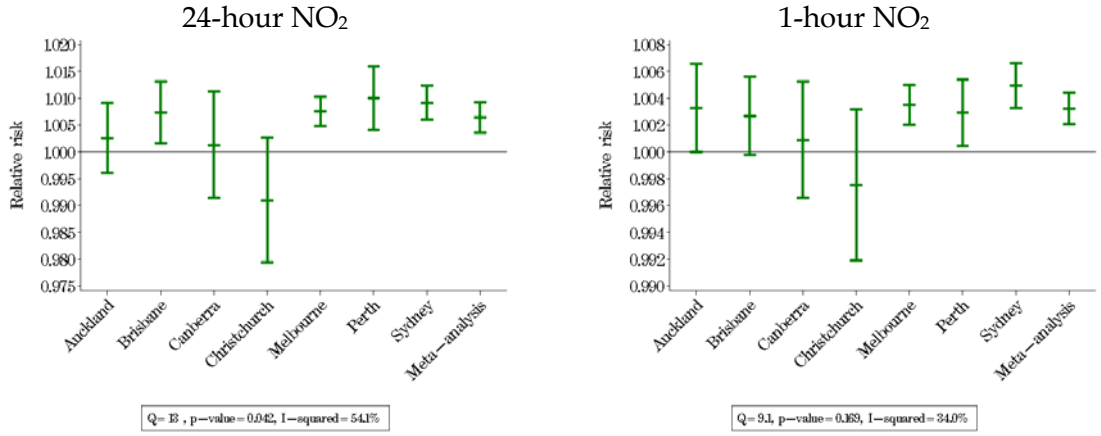


Figure 2.2.6: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Table 2.2.3 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis for the elderly group. It is clear the associations are statistically significant and positive only for the Australian cities as a group (this occurs for maximum 1-hour NO_2 and 24-hour average NO_2). The Christchurch results in particular appear to differ from the others, and this accounts for the heterogeneity found for 24-hour average NO_2 which also is apparent in Figure 2.2.6.

Leaving Christchurch out of the multi-city analysis still leads to a pooled estimate showing a significant increase (with no significant heterogeneity).

Table 2.2.3: Differences between cities using a leave-one-city-out sensitivity analysis for cardiac admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2			24-hour NO_2		
City left out	Inc	95%CI	I^2	Inc	95%CI	I^2
Auckland	2.9	1.6, 4.1	44.9	3.6	2.1, 5.2	54.1*
Brisbane	3.0	1.7, 4.2	42.9	2.8	0.5, 5.1	61.7*
Canberra	3.1	2.0, 4.2	34.8	3.5	2.0, 5.1	56.8*
Christchurch	3.3	2.5, 4.2	0.0	4.0	3.0, 5.0	7.4
Melbourne	2.7	1.2, 4.2	45.0	2.6	0.2, 5.2	61.6*
Perth	2.9	1.6, 4.2	43.5	3.0	1.3, 4.7	59.2*
Sydney	2.6	1.7, 3.6	1.6	2.8	1.0, 4.7	55.0*
Australian only	3.3	2.4, 4.3	13.3	4.2	3.3, 5.2	0.0
NZ only	0.9	-3.7, 5.6	66.7*	-1.1	-6.6, 4.8	65.7*
Overall	3.0	1.9, 4.0	34.0	3.4	1.9, 4.9	54.1*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.1.4 Ischemic heart disease

The associations with hospital admissions due to ischemic heart disease are shown in Figure 2.2.7 and Figure 2.2.8. There are significant associations for the older age group and there is no evidence for heterogeneity between the results for the cities (confirmed by the statistical tests).

Figure 2.2.7: City specific and meta-analysis relative risks (and 95% confidence intervals) for IHD admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

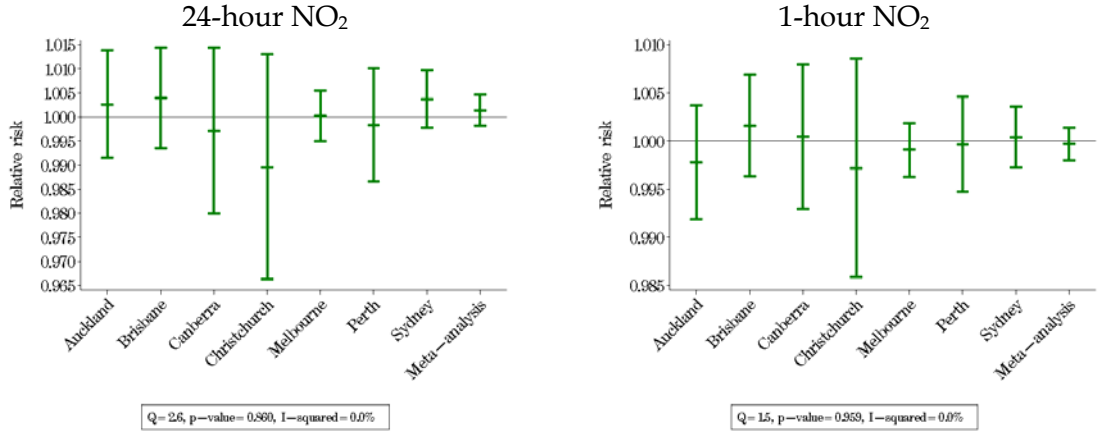
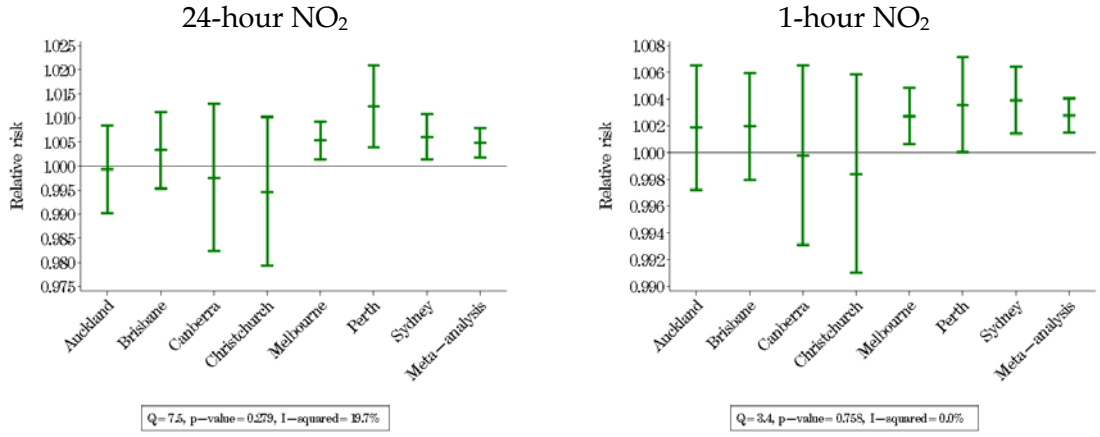


Figure 2.2.8: City specific and meta-analysis relative risks (and 95% confidence intervals) for IHD admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Table 2.2.4 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis for the elderly group.

Table 2.2.4: Differences between cities using a leave-one-city-out sensitivity analysis for IHD admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	2.6	1.4,	3.8	0.0	2.8	1.3,	4.4	14.2
Brisbane	2.6	1.4,	3.9	0.0	2.6	0.8,	4.4	31.1
Canberra	2.6	1.4,	3.8	0.0	2.6	1.0,	4.3	23.5
Christchurch	2.6	1.5,	3.8	0.0	2.7	1.3,	4.2	12.3
Melbourne	2.5	1.1,	4.0	0.0	2.2	0.0,	4.4	32.7
Perth	2.4	1.2,	3.7	0.0	2.2	0.9,	3.6	0.0
Sydney	2.2	0.8,	3.5	0.0	2.2	0.1,	4.2	30.7
Australian only	2.7	1.5,	4.0	0.0	3.0	1.6,	4.4	0.0
NZ only	0.8	-2.7,	4.4	0.0	-1.0	-4.9,	3.1	0.0
Overall	2.5	1.4,	3.7	0.0	2.5	1.0,	4.1	19.7

2.2.1.5 Myocardial infarction

The associations with hospital admissions due to myocardial infarction are shown in Figure 2.2.9 and Figure 2.2.10. For the older age group, the overall impact is positive (and statistically significant) and there is no evidence for heterogeneity between the results for all the cities.

Figure 2.2.9: City specific and meta-analysis relative risks (and 95% confidence intervals) for myocardial infarction admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

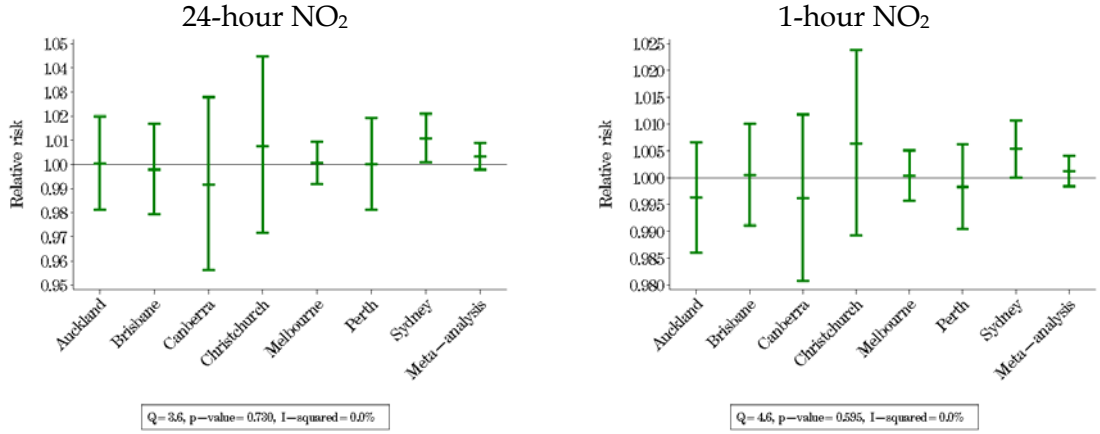
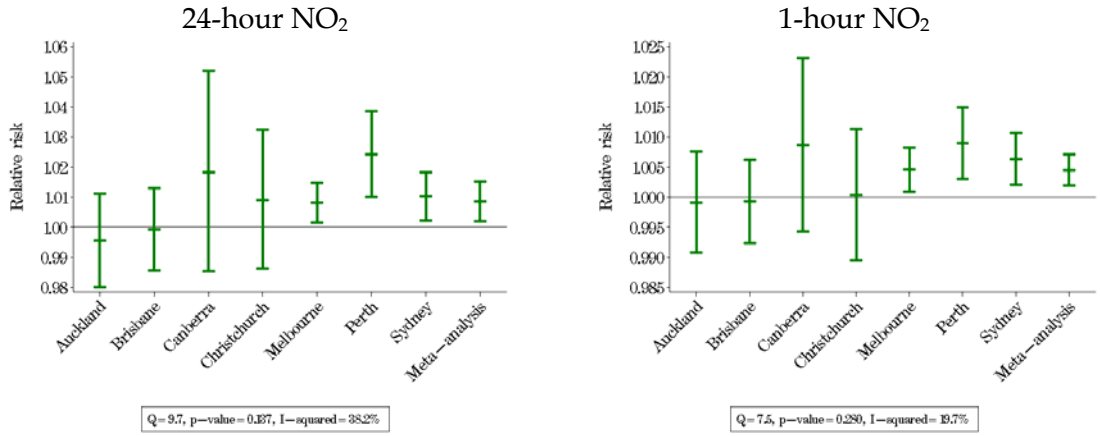


Figure 2.2.10: City specific and meta-analysis relative risks (and 95% confidence intervals) for myocardial infarction admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Table 2.2.5 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis for the older age group. The associations in Australia are positive (and statistically significant).

Table 2.2.5: Differences between cities using a leave-one-city-out sensitivity analysis for myocardial infarction admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2			24-hour NO_2		
City left out	Inc	95%CI	I^2	Inc	95%CI	I^2
Auckland	4.6	2.3, 7.0	11.9	5.2	2.3, 8.3	26.5
Brisbane	4.8	2.6, 7.0	0.0	5.2	1.6, 9.0	35.2
Canberra	3.9	1.3, 6.5	30.3	4.2	0.4, 8.2	46.7
Christchurch	4.3	1.7, 6.9	27.0	4.4	0.4, 8.6	48.5
Melbourne	3.8	0.6, 7.2	33.0	4.5	0.1, 9.0	48.3
Perth	3.6	1.3, 5.8	2.2	3.7	1.3, 6.1	0.0
Sydney	3.5	0.6, 6.5	25.2	4.2	-0.3, 8.8	47.3
Australian only	4.8	2.3, 7.4	18.8	5.4	1.6, 9.4	41.2
NZ only	-0.4	-6.2, 5.7	0.0	-0.1	-6.5, 6.7	0.0
Overall	4.1	1.7, 6.5	19.7	4.4	1.0, 8.0	38.2

2.2.1.6 Cardiac failure

The associations with hospital admissions due to cardiac failure are shown in Figure 2.2.11 and Figure 2.2.12. There are significant associations for both age groups. There is no evidence for heterogeneity between the results for both maximum 1-hour NO_2 and for 24-hour NO_2 for the younger age group. For the older age group there is evidence for heterogeneity between the results for 24-hour average NO_2 due to the results for Christchurch.

Figure 2.2.11: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac failure admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

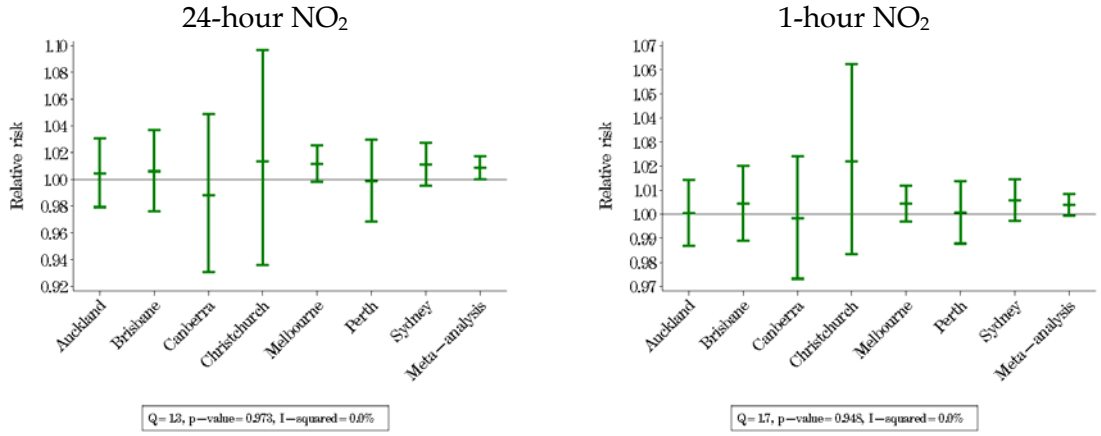
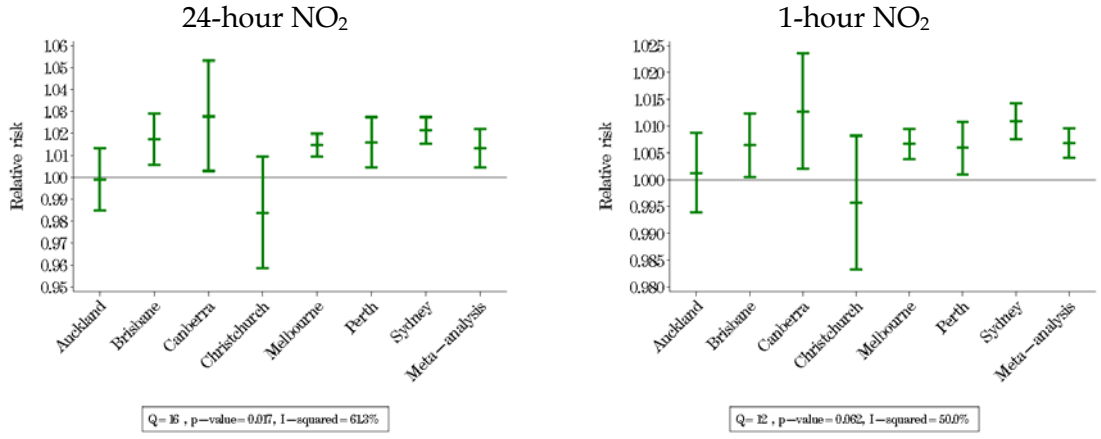


Figure 2.2.12: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac failure admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



Even in the largest city, Sydney, the mortality due to cardiac failure in the 15 to 64 year age group is only about 1.6 deaths per day. Interpretation of differing effects must consider that early mortality from cardiac failure may have a much different etiology from cardiac failure in later life.

The results in Table 2.2.6 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis. The associations for the older age group are positive (and statistically significant) for the Australian cities. Table 2.2.6 indicate that leaving out Christchurch shows no evidence for heterogeneity in the results for the remaining cities.

Table 2.2.6: Differences between cities using a leave-one-city-out sensitivity analysis for cardiac failure admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	7.0	4.4,	9.6	45.8	8.3	3.8,	13.1	48.9
Brisbane	6.3	3.3,	9.4	57.9*	6.3	0.7,	12.3	67.6*
Canberra	6.0	3.3,	8.7	54.7*	6.2	1.3,	11.4	65.8*
Christchurch	6.9	4.5,	9.3	41.9	8.5	5.6,	11.4	47.5
Melbourne	6.1	2.6,	9.7	56.8*	6.5	0.5,	12.8	66.9*
Perth	6.4	3.3,	9.5	56.9*	6.5	0.7,	12.5	67.8*
Sydney	5.4	3.2,	7.7	14.8	5.8	0.5,	11.4	54.7*
Australian only	7.5	5.3,	9.7	28.6	9.3	7.4,	11.3	0.0
NZ only	-0.1	-5.6,	5.7	0.0	-2.3	-8.5,	4.3	3.5
Overall	6.3	3.7,	9.0	50.0	6.9	2.2,	11.8	61.3*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.1.7 Arrhythmia

The associations with hospital admissions due to arrhythmia are shown in Figure 2.2.13 and Figure 2.2.14. There are significant associations that are similar for all cities (confirmed by the statistical tests) only for the 15-64 years age group.

Figure 2.2.13: City specific and meta-analysis relative risks (and 95% confidence intervals) for arrhythmia admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

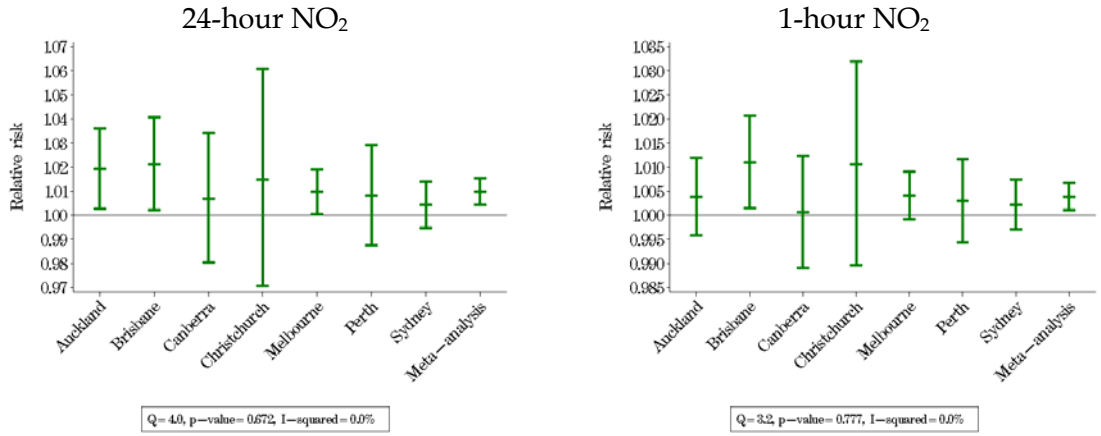
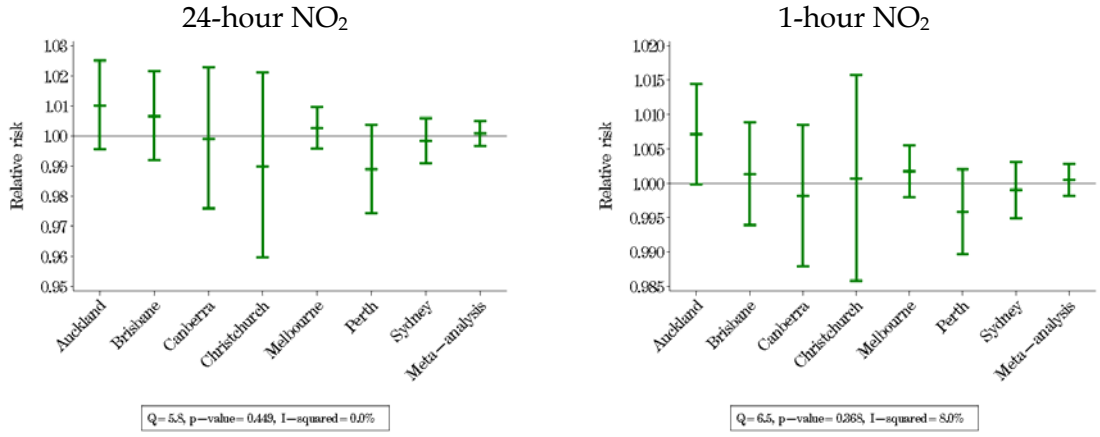


Figure 2.2.14: City specific and meta-analysis relative risks (and 95% confidence intervals) for arrhythmia admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Table 2.2.7 (all the results per inter-quartile range) show the effects of leaving cities out of the analysis on the 15-64 years age group.

Table 2.2.7: Differences between cities using a leave-one-city-out sensitivity analysis for arrhythmia admissions (15-64 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	I	95%	CI	I2	I	95%	CI	I2
Auckland	3.5	0.8,	6.4	0.0	4.5	1.4,	7.7	0.0
Brisbane	2.9	0.2,	5.7	0.0	4.6	1.6,	7.7	0.0
Canberra	3.7	1.1,	6.4	0.0	5.2	2.2,	8.3	0.0
Christchurch	3.4	0.8,	6.1	0.0	5.1	2.2,	8.1	0.0
Melbourne	3.5	0.4,	6.7	0.0	5.2	1.5,	8.9	0.0
Perth	3.6	0.9,	6.4	0.0	5.2	2.2,	8.3	0.0
Sydney	4.2	1.1,	7.4	0.0	6.6	3.0,	10.3	0.0
Australian only	3.4	0.7,	6.3	0.0	4.4	1.3,	7.6	0.0
NZ only	4.3	-2.5,	11.6	0.0	10.0	1.7,	19.0	0.0

2.2.1.8 Seasonal differences

Separate analyses were carried out for the warm (November–April) and cool (May–October) periods to identify if there are different effects in each period (due to different emission patterns, atmospheric chemistry). Individual city seasonal results for cardiovascular admissions are shown in *Volume 3: Appendix A3.4*, Tables WC.1 to WC.14. Meta-analysis and results from the leave-one-city-out analysis for cardiovascular admissions are shown in *Volume 3: Appendix A3.5*, Tables WCL.1 to WCL.14.

Significant seasonal associations found between cardiovascular admissions and NO_2 for both cool and warm seasons are shown in Table 2.2.8.

There was generally no evidence for heterogeneity between results for the cities when the pooled estimate was significant. The only exception was for cardiac admissions in the elderly, and when the Christchurch results (much lower estimate)

were removed, there was no evidence for heterogeneity between the results for the remaining cities.

The differences found between the cool and warm periods can be summarised as follows:

- the increases in hospital admissions due to all cardiovascular disease, cardiac disease, arrhythmia and cardiac failure, were only significant in the cool periods for adults aged between 15 and 64 years

- the increases in hospital admissions due to cardiac failure for adults aged 65 years or greater were only significant for the warm period.

Table 2.2.8: Significant seasonal increases in cardiovascular admissions associated with an IQR increase in NO_2 (average lag 0-1) (case-crossover meta-analysis)

Admissions (age group)		1-hour NO_2			24-hour NO_2		
		Inc	95%CI	I2	Inc	95%CI	I2
Cardiac (15-64 yrs)	cool	2.1	0.3, 3.8	0.0	3.2	1.5, 5.0	0.0
	warm	0.3	-1.4, 2.0	0.0	0.9	-1.3, 3.1	0.0
Cardiac (65+ yrs)	cool	3.6	1.5, 5.7	57.6*	4.0	1.3, 6.7	59.1*
	warm	2.5	1.3, 3.8	0.0	2.8	1.2, 4.5	0.0
IHD (65+ yrs)	cool	3.3	1.5, 5.2	0.0	3.3	1.4, 5.1	0.0
	warm	2.7	0.9, 4.6	0.0	2.4	-0.4, 5.2	24.2
Arrhythmia (15-64 yrs)	cool	4.7	0.7, 8.8	0.0	5.5	1.5, 9.6	0.0
	warm	1.6	-2.2, 5.6	0.0	4.5	-2.7, 12.3	35.1
Cardiac failure (15-64 yrs)	cool	5.2	-1.0, 11.9	0.0	6.8	0.6, 13.4	0.0
	warm	2.4	-3.8, 9.0	0.0	2.6	-5.3, 11.1	0.0
Cardiac failure (65+ yrs)	cool	5.6	-1.3, 13.1	72.7*	6.8	-0.8, 14.9	69.7*
	warm	4.8	2.1, 7.6	0.0	6.2	2.8, 9.8	0.0
Myocardial infarction (65+ yrs)	cool	3.8	0.7, 7.0	0.0	3.9	0.7, 7.1	0.0
	warm	4.0	0.6, 7.4	0.0	5.4	1.2, 9.8	0.0
Total cardiovascular (15-64 yrs)	cool	1.6	-0.5, 3.8	43.9	2.5	0.6, 4.5	29.9
	warm	1.2	-0.3, 2.7	0.0	1.2	-0.6, 3.1	0.0
Total cardiovascular (65+ yrs)	cool	3.3	1.7, 4.9	50.0	3.8	2.4, 5.3	37.3
	warm	2.0	0.9, 3.1	0.9	2.0	0.5, 3.5	12.1

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 95% level*

2.2.2 Hospital admissions due to respiratory disease

The respiratory disease groups considered included (see Table 1.2 for the ICD codes):

- total respiratory disease
- asthma
- chronic obstructive pulmonary disease (COPD)
- pneumonia and acute bronchitis.

Analyses for hospital admissions were stratified using the following age groups as appropriate for the disease:

- children
  - less than one year of age
  - 1–4 years
  - 5–14 years
- adults
  - 15–64 years
  - 65 years and greater.

In the following section, the impacts from exposure to air pollutant in the short-term (average of the same day and day before — lag [0,1]) are presented for these health outcomes.

2.2.2.1 Total respiratory disease

The associations with hospital admissions for all respiratory disease for all age groups are shown in Figures 2.2.15 to 2.2.19. The significant results for pooled estimates can be summarised as follows:

- there are overall positive associations between increases (statistically significant) in admissions for children in the age group 1–4 years and increases in maximum 1–hour NO_2. There is no evidence of heterogeneity between results for all the cities (as shown by the statistical tests)

- there are overall positive associations between increases (statistically significant) in admissions for children in the age group 5–14 years and increases in maximum 1–hour NO_2 and 24–hour NO_2. However, there is evidence of heterogeneity between results for all the cities for 24–hour NO_2 (as shown by the statistical tests) due to high results for Auckland (when Auckland is removed, the pooled estimates for the increases in the remaining cities are still positive and statistically significant)

- there are overall positive increases (statistically significant for maximum 1–hour NO_2) for the 15-64 years age group. There is no evidence of heterogeneity between results for all the cities (as shown by the statistical tests).

Figure 2.2.15: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (less than 1 year) associated with a one-unit increase in NO_2 (average lag 0-1)

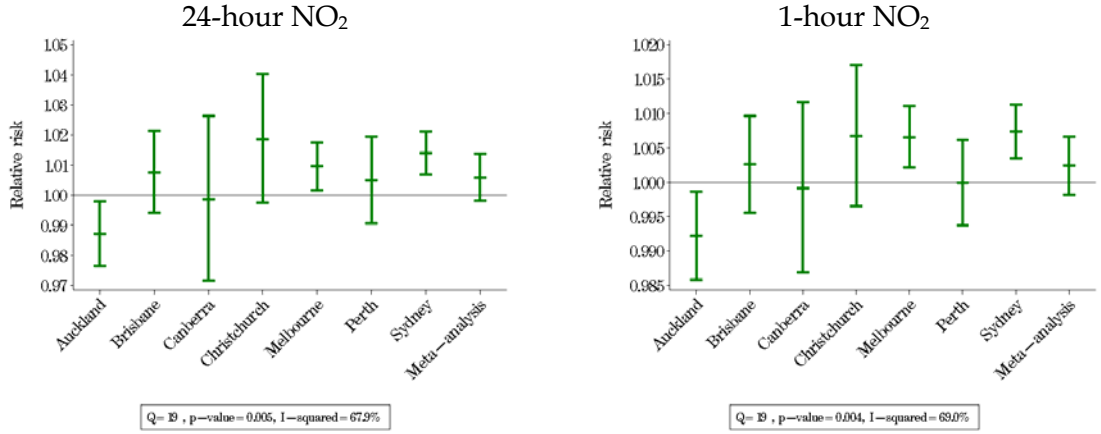


Figure 2.2.16: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (1-4 years) associated with a one-unit increase in NO_2 (average lag 0-1)

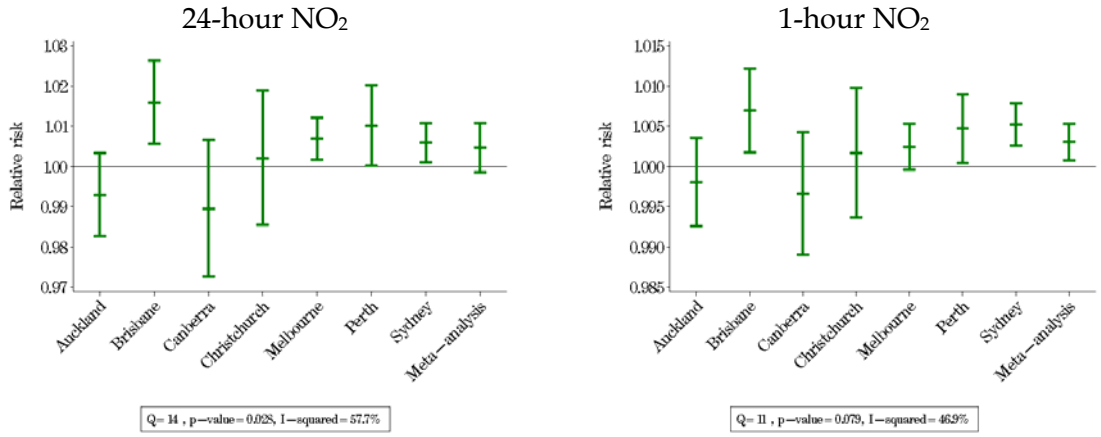


Figure 2.2.17: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (5-14 years) associated with a one-unit increase in NO_2 (average lag 0-1)

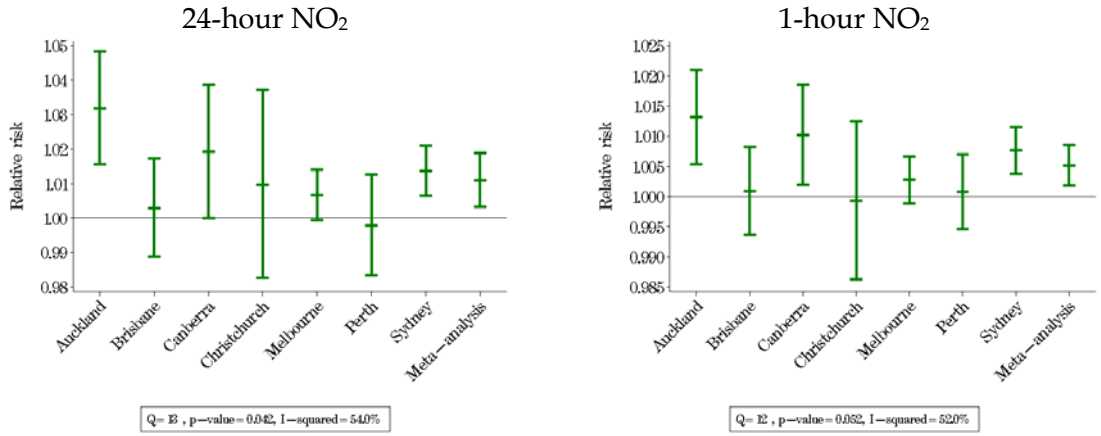


Figure 2.2.18: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

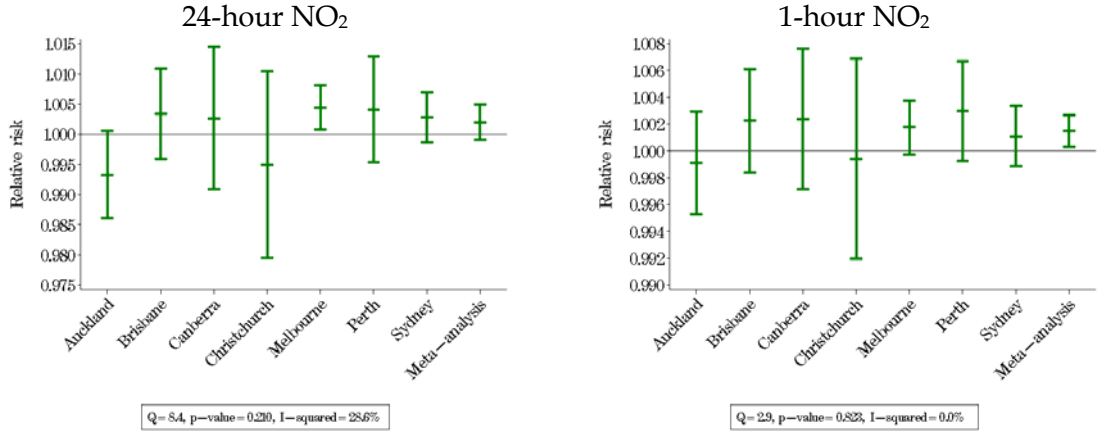
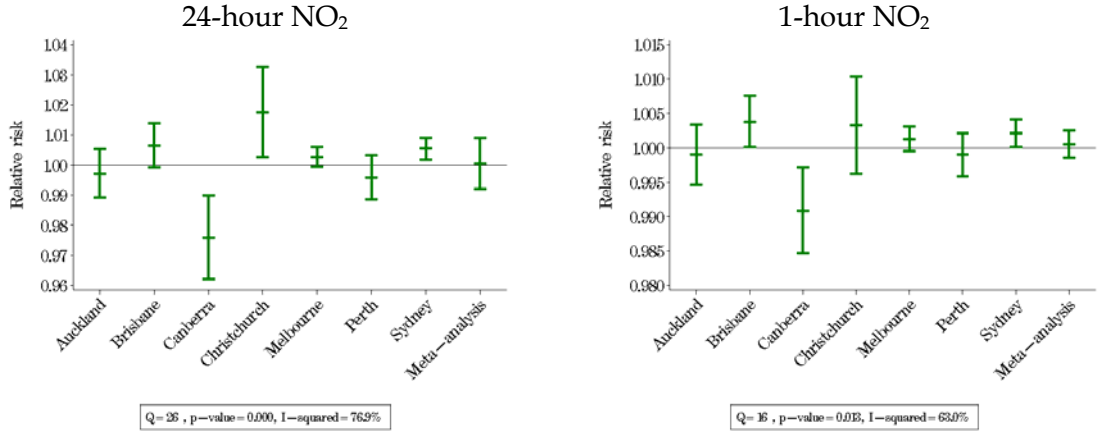


Figure 2.2.19: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Tables 2.2.9 to 2.2.13 (all the results per inter-quartile range) show the effects of leaving cities out of the analyses. The results can be summarised as follows:

- there are positive overall (and statistically significant) increases in admissions for children in the age groups less than 1 year and 1–4 years for Australian cities for 24–hour NO_2 and maximum 1–hour NO_2, and there is no evidence of heterogeneity between results for the cities

- for 24-hour NO_2 and the admissions for age groups less than 1 year, 1-4 years and 5-14 years, removing the results for Auckland removes the heterogeneity in the results (with the increase still positive and significant)

- as well as contributing to the heterogeneity within infancy there is notable heterogeneity in Auckland across age groups, with results ranging from significantly negative to significantly positive.

Table 2.2.9: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory admissions (0 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	4.6	2.1,	7.1	13.5	5.7	3.3,	8.1	0.0
Brisbane	2.1	-2.3,	6.7	74.0*	2.9	-1.8,	7.8	73.3*
Canberra	2.4	-1.7,	6.7	73.4*	3.3	-1.0,	7.9	72.7*
Christchurch	1.7	-2.4,	6.1	73.7*	2.4	-1.9,	6.9	71.6*
Melbourne	1.3	-3.2,	6.0	70.9*	2.6	-2.1,	7.6	72.7*
Perth	2.6	-1.7,	7.1	71.9*	3.1	-1.5,	8.0	73.1*
Sydney	1.1	-3.1,	5.5	64.8*	2.0	-2.6,	6.8	63.0*
Australian only	4.2	1.5,	7.1	29.7	5.5	3.1,	8.0	0.0
NZ only	-1.0	-12.8,	12.4	81.9*	0.7	-13.9,	17.9	85.2*
Overall	2.2	-1.6,	6.1	69.0*	3.1	-1.0,	7.3	67.9*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.10: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory admissions (1-4 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	3.5	1.6,	5.4	30.9	3.6	1.4,	5.9	36.0
Brisbane	2.3	0.1,	4.5	47.1	1.6	-1.3,	4.7	50.3
Canberra	3.3	1.4,	5.2	37.9	3.2	0.2,	6.2	52.9*
Christchurch	2.8	0.6,	5.1	55.0*	2.5	-1.1,	6.2	64.2*
Melbourne	2.8	0.2,	5.5	52.8*	2.0	-2.0,	6.1	64.1*
Perth	2.4	0.0,	4.9	54.3*	1.8	-1.8,	5.6	62.6*
Sydney	2.1	-0.3,	4.6	42.3	2.1	-1.9,	6.3	64.7*
Australian only	3.6	1.5,	5.7	42.2	3.8	0.7,	6.9	46.5
NZ only	-0.7	-4.7,	3.4	0.0	-2.3	-6.7,	2.2	0.0
Overall	2.8	0.7,	4.9	46.9	2.4	-0.8,	5.7	57.7*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.11: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory admissions (5-14 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	3.8	1.0,	6.7	38.0	4.5	1.9,	7.3	15.8
Brisbane	5.3	1.9,	8.8	54.9*	6.6	1.9,	11.5	57.9*
Canberra	4.2	0.9,	7.5	54.5*	5.3	0.7,	10.1	59.1*
Christchurch	5.0	1.8,	8.4	57.3*	5.9	1.2,	10.8	61.7*
Melbourne	5.3	1.6,	9.2	52.4*	6.5	1.4,	11.8	56.4*
Perth	5.5	2.1,	8.9	51.7*	6.9	2.7,	11.2	50.0
Sydney	4.1	0.5,	7.8	50.2	5.5	0.4,	10.9	58.0*
Australian only	4.0	1.1,	7.1	46.4	4.4	1.4,	7.5	32.6
NZ only	6.7	-5.3,	20.3	68.3*	12.7	1.4,	25.3	46.1
Overall	4.7	1.6,	7.9	52.0*	5.8	1.7,	10.1	54.0*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.12: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory admissions (15-64 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	1.6	0.4,	2.7	0.0	1.8	0.5,	3.0	0.0
Brisbane	1.2	0.1,	2.4	0.0	0.8	-0.9,	2.6	40.1
Canberra	1.3	0.2,	2.4	0.0	0.9	-0.7,	2.6	40.5
Christchurch	1.4	0.3,	2.5	0.0	1.1	-0.4,	2.7	33.1
Melbourne	1.2	-0.1,	2.5	0.0	0.5	-1.3,	2.3	24.8
Perth	1.2	0.0,	2.3	0.0	0.8	-0.9,	2.6	39.5
Sydney	1.5	0.2,	2.7	0.0	0.7	-1.3,	2.8	40.2
Australian only	1.6	0.5,	2.8	0.0	1.9	0.6,	3.2	0.0
NZ only	-0.8	-3.7,	2.3	0.0	-3.3	-6.5,	0.0	0.0
Overall	1.3	0.3,	2.4	0.0	1.0	-0.5,	2.5	28.6

Table 2.2.13: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	0.6	-1.3,	2.7	67.4*	0.5	-4.6,	5.9	79.2*
Brisbane	0.0	-1.9,	2.0	64.2*	-0.3	-5.3,	4.9	79.9*
Canberra	1.2	0.1,	2.4	12.8	1.6	-0.7,	4.0	57.0*
Christchurch	0.3	-1.6,	2.2	68.4*	-0.8	-4.8,	3.5	77.3*
Melbourne	0.1	-2.3,	2.6	69.0*	-0.0	-5.3,	5.5	80.7*
Perth	0.7	-1.3,	2.8	65.2*	0.6	-4.5,	6.0	77.7*
Sydney	0.0	-2.2,	2.3	66.0*	-0.3	-5.4,	5.1	78.0*
Australian only	0.4	-1.7,	2.6	73.3*	-0.8	-5.8,	4.5	80.3*
NZ only	0.2	-3.1,	3.6	1.6	3.3	-6.6,	14.3	81.9*
Overall	0.5	-1.3,	2.3	63.0*	0.2	-0.4,	4.7	76.9*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.2.2 Asthma

The associations with hospital admissions for asthma for selected age groups (excluding the youngest, less than 1 year, where it is difficult to diagnose asthma) are shown in Figures 2.2.20 to 2.2.23. There are significant associations between increases in 24-hour NO_2 and increases in admissions for children in the age groups 5-14 years. However, there is evidence of heterogeneity between the results for the cities (as shown by the statistical tests).

Figure 2.2.20: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (1-4 years) associated with a one-unit increase in NO_2 (average lag 0-1)

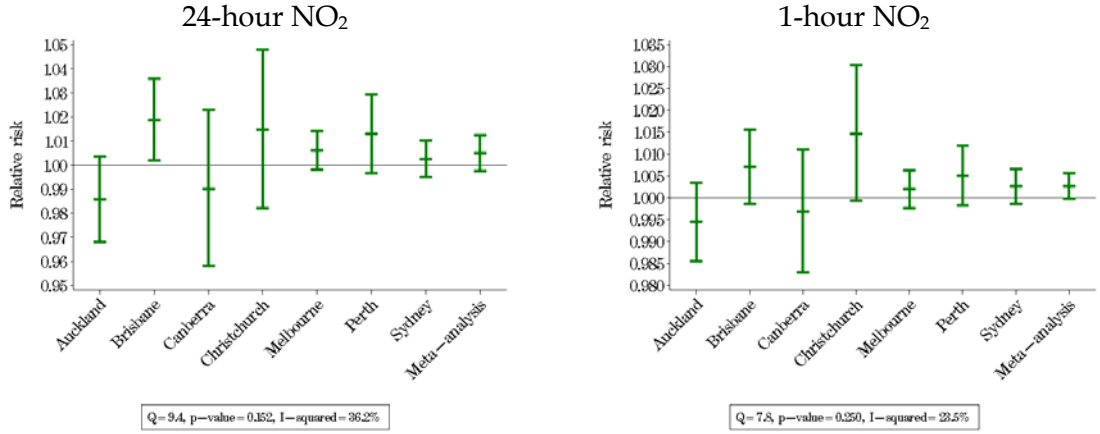


Figure 2.2.21: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (5-14 years) associated with a one-unit increase in NO_2 (average lag 0-1)

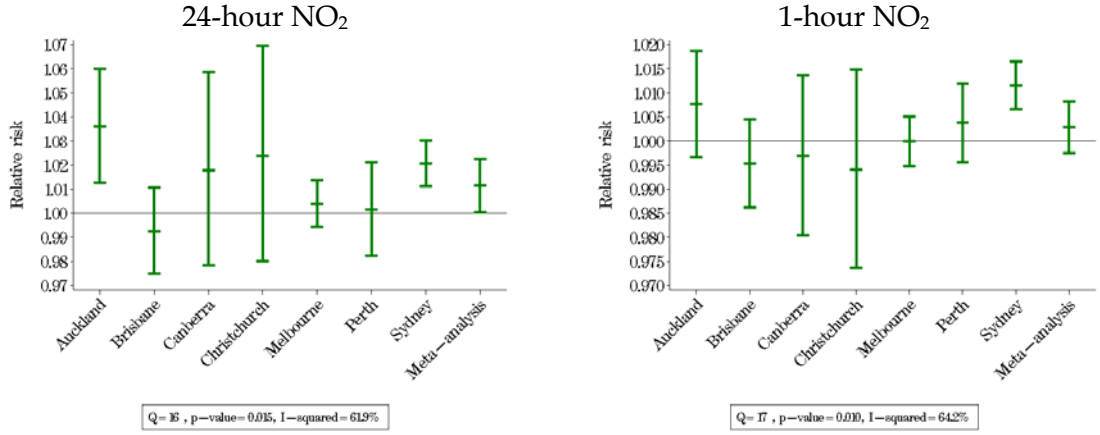


Figure 2.2.22: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

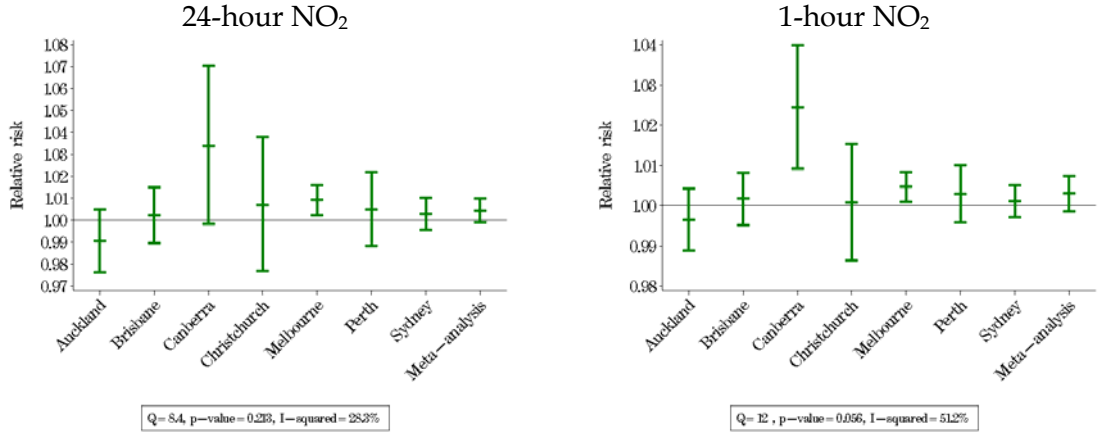
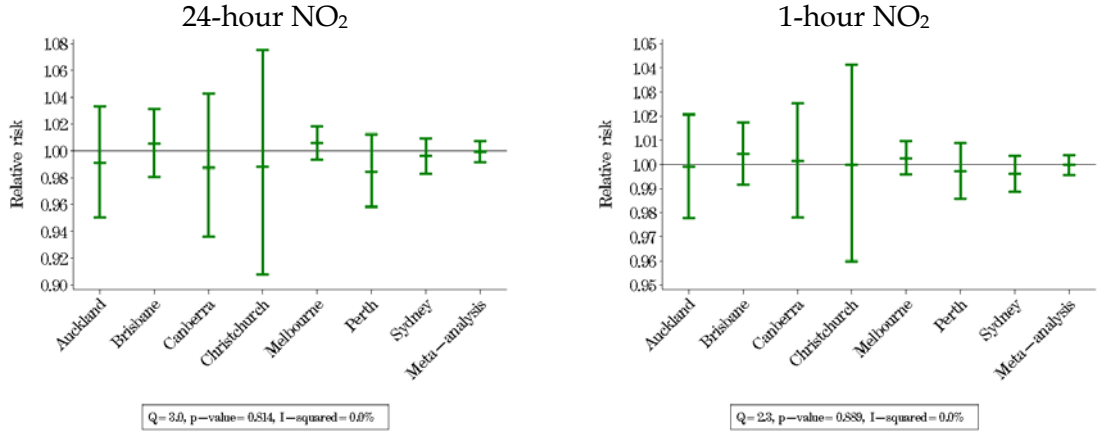


Figure 2.2.23: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Tables 2.2.14 to 2.2.17 (all the results per inter-quartile range) show the effects of leaving cities out of the analyses. The results can be summarised as follows:

- there are positive overall (and statistically significant) increases in admissions for children in the age group 1–4 years for Australian cities for both maximum 1–hour NO_2 and 24–hour average NO_2 and there are no significant differences between Australian cities
- there are positive overall increases in admissions for children in the 5–14 years age group in New Zealand cities (and statistically significant)
- there are positive overall increases in admissions for the age group 15-64 years for Australian cities (statistically significant for 24-hour NO_2).

Table 2.2.14: Differences between cities using a leave-one-city-out sensitivity analysis for asthma admissions (1-4 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	3.0	0.7,	5.3	0.0	3.2	0.6,	5.8	0.0
Brisbane	2.0	-0.8,	4.9	25.4	1.7	-1.4,	4.9	23.8
Canberra	2.7	-0.1,	5.6	30.4	3.0	-1.2,	7.2	41.9
Christchurch	2.1	-0.2,	4.5	8.2	2.3	-2.0,	6.7	44.8
Melbourne	2.7	-0.8,	6.4	35.3	2.4	-2.8,	7.8	46.0
Perth	2.1	-1.0,	5.2	31.2	1.9	-2.4,	6.4	40.2
Sydney	2.5	-1.1,	6.3	36.3	2.9	-2.2,	8.3	43.9
Australian only	2.7	0.4,	5.1	0.0	3.2	0.4,	6.1	12.3
NZ only	3.2	-13.5,	23.1	79.8*	-1.7	-14.6,	13.2	57.0*
Overall	2.5	-0.2,	5.2	23.5	2.6	-1.3,	6.6	36.2

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.15: Differences between cities using a leave-one-city-out sensitivity analysis for asthma admissions (5-14 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	2.1	-2.6,	7.1	69.5*	4.3	-0.4,	9.3	55.3*
Brisbane	3.9	-1.0,	9.1	60.7*	7.7	2.2,	13.5	55.3*
Canberra	3.0	-2.1,	8.4	68.7*	5.9	-0.6,	12.8	68.0*
Christchurch	3.0	-2.0,	8.3	68.4*	5.7	-0.6,	12.4	67.6*
Melbourne	3.3	-2.3,	9.1	61.0*	7.2	0.3,	14.6	59.3*
Perth	2.3	-3.4,	8.2	70.2*	7.0	0.4,	14.0	66.0*
Sydney	0.4	-2.7,	3.7	0.0	4.6	-2.0,	11.6	48.9
Australian only	2.3	-3.3,	8.3	74.2*	3.8	-1.3,	9.3	62.9*
NZ only	3.7	-6.3,	14.7	22.2	18.4	6.7,	31.4	0.0
Overall	2.6	-2.2,	7.6	64.2*	6.0	0.2,	12.1	61.9*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.16: Differences between cities using a leave-one-city-out sensitivity analysis for asthma admissions (15-64 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	3.3	0.1,	6.6	47.5	3.2	0.9,	5.5	0.0
Brisbane	3.3	-2.2,	9.2	58.9*	2.4	-1.4,	6.2	38.9
Canberra	2.0	0.0,	4.1	0.0	2.1	-0.4,	4.6	13.5
Christchurch	3.2	-2.0,	8.7	59.1*	2.1	-1.4,	5.7	40.1
Melbourne	2.8	-2.9,	8.8	52.7*	1.1	-2.0,	4.3	13.7
Perth	3.1	-2.4,	9.0	59.3*	2.1	-1.5,	5.9	40.2
Sydney	3.4	-1.8,	8.8	55.9*	2.4	-1.7,	6.6	37.0
Australian only	4.4	-1.2,	10.3	57.5*	3.1	0.7,	5.6	6.1
NZ only	-2.3	-8.0,	3.8	0.0	-3.3	-9.6,	3.4	0.0
Overall	2.7	-1.2,	6.8	51.2*	2.2	-0.6,	5.1	28.3

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.17: Differences between cities using a leave-one-city-out sensitivity analysis for asthma admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	-0.1	-3.9,	3.7	0.0	-0.1	-4.1,	4.2	0.0
Brisbane	-0.6	-4.5,	3.4	0.0	-0.5	-4.7,	3.8	0.0
Canberra	-0.2	-3.9,	3.6	0.0	-0.1	-4.1,	4.1	0.0
Christchurch	-0.2	-3.8,	3.7	0.0	-0.2	-4.1,	4.0	0.0
Melbourne	-1.6	-6.1,	3.1	0.0	-2.3	-7.3,	2.9	0.0
Perth	0.2	-3.7,	4.3	0.0	0.5	-3.7,	4.8	0.0
Sydney	1.4	-3.1,	6.1	0.0	0.7	-4.2,	5.9	0.0
Australian only	-0.1	-3.9,	3.8	0.0	0.0	-4.1,	4.2	0.0
NZ only	-0.8	-16.3,	17.7	0.0	-4.9	-21.5,	15.3	0.0
Overall	-0.2	-3.8,	3.6	0.0	-0.2	-4.2,	3.9	0.0

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.2.3 Chronic obstructive pulmonary disease (COPD)

The associations with hospital admissions for COPD for adults (15-64 years age group, and 65 years and greater age group) are shown in Figure 2.2.24 and 2.2.25 (there are few events of COPD for children). There were no statistically significant results for the pooled estimates.

Figure 2.2.24: City specific and meta-analysis relative risks (and 95% confidence intervals) for COPD admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

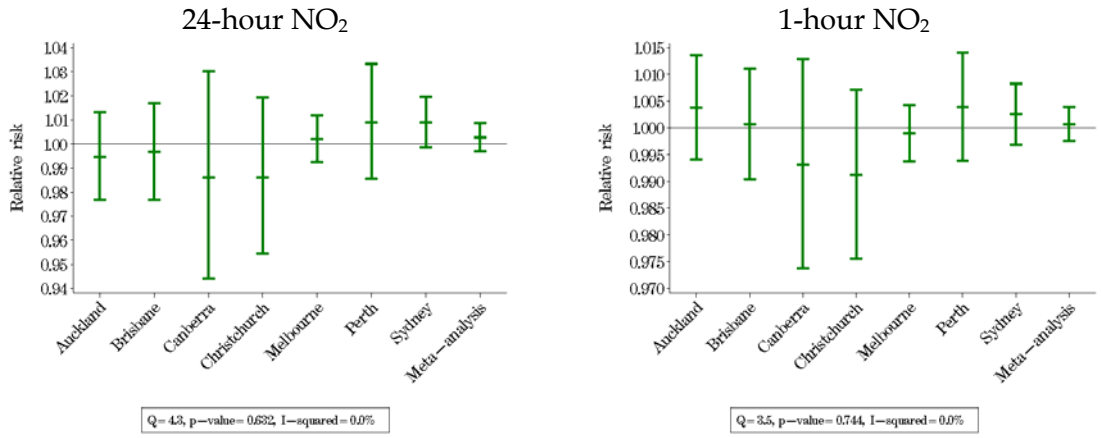
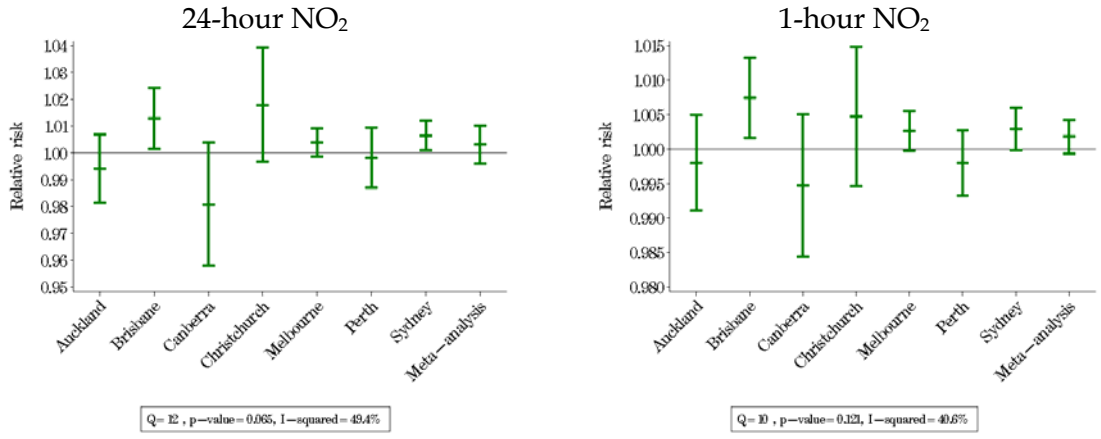


Figure 2.2.25: City specific and meta-analysis relative risks (and 95% confidence intervals) for COPD admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Tables 2.2.18 to 2.2.19 (all the results per inter-quartile range) show the effects of leaving cities out of the analyses. There were no statistically significant results in leaving out any cities.

Table 2.2.18: Differences between cities using a leave-one-city-out sensitivity analysis for COPD admissions (15-64 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	0.4	-2.6,	3.4	0.0	1.9	-1.3,	5.2	0.0
Brisbane	0.7	-2.2,	3.7	0.0	1.7	-1.4,	5.0	0.0
Canberra	0.9	-2.0,	3.7	0.0	1.6	-1.5,	4.8	0.0
Christchurch	1.0	-1.8,	3.9	0.0	1.7	-1.4,	4.9	0.0
Melbourne	1.5	-1.9,	5.1	0.0	1.6	-2.2,	5.6	0.0
Perth	0.4	-2.5,	3.4	0.0	1.2	-1.9,	4.4	0.0
Sydney	-0.0	-3.3,	3.4	0.0	-0.1	-3.7,	3.6	0.0
Australian only	0.7	-2.3,	3.8	0.0	2.3	-1.0,	5.6	0.0
NZ only	-0.6	-10.1,	9.9	42.6	-3.7	-11.3,	4.6	0.0
Overall	0.7	-2.1,	3.5	0.0	1.4	-1.6,	4.6	0.0

Table 2.2.19: Differences between cities using a leave-one-city-out sensitivity analysis for COPD admissions (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	2.0	-0.3,	4.4	42.4	2.4	-1.4,	6.3	45.8
Brisbane	1.1	-0.9,	3.2	23.1	0.8	-2.9,	4.7	47.4
Canberra	2.0	-0.2,	4.2	38.0	2.4	0.1,	4.8	36.2
Christchurch	1.4	-0.9,	3.9	49.1	1.2	-2.1,	4.6	51.1*
Melbourne	1.3	-1.7,	4.3	49.4	1.4	-3.3,	6.3	57.7*
Perth	2.4	0.2,	4.6	26.5	2.0	-2.3,	6.5	52.9*
Sydney	1.2	-1.6,	4.2	48.5	1.1	-3.4,	5.8	54.4*
Australian only	1.8	-0.7,	4.5	52.7*	1.8	-2.1,	5.8	48.5
NZ only	0.3	-5.2,	6.0	13.6	2.3	-9.1,	15.1	72.0*
Overall	1.6	-0.6,	3.9	40.6	1.6	-2.0,	5.4	49.4

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.2.4 Pneumonia and acute bronchitis

The associations with hospital admissions for pneumonia and acute bronchitis for all age groups (except the 5-14 years group — data very small) are shown in Figures 2.2.26 to 2.2.29. There are no statistically significant results for any pooled estimates.

Figure 2.2.26: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (0 years) associated with a one-unit increase in NO_2 (average lag 0-1)

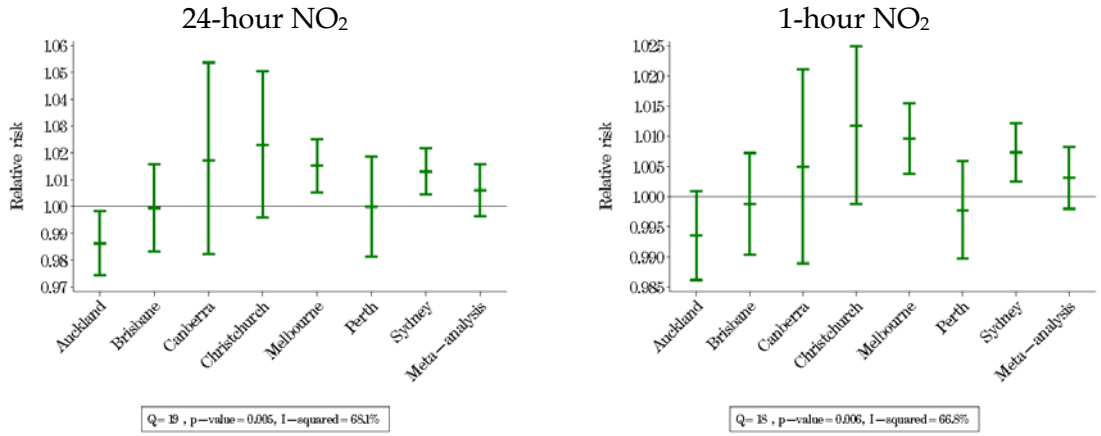


Figure 2.2.27: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (1-4 years) associated with a one-unit increase in NO_2 (average lag 0-1)

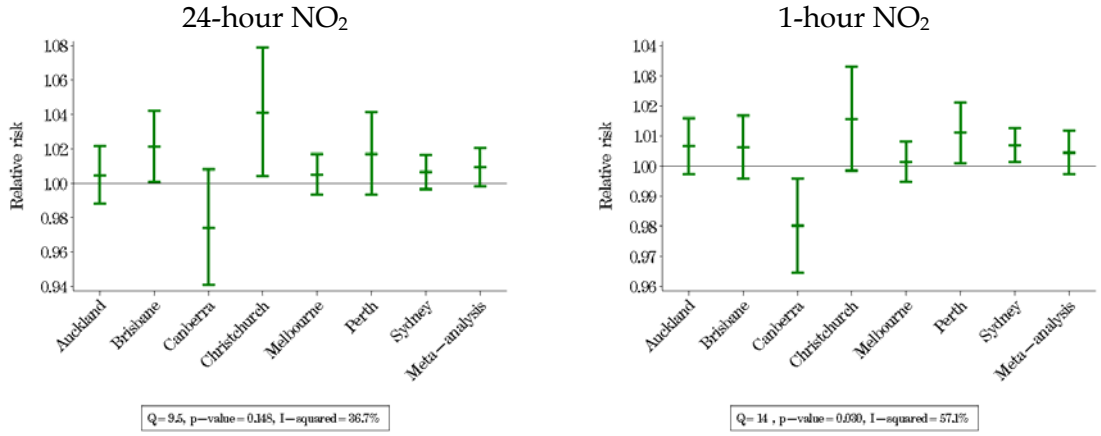


Figure 2.2.28: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (15-64 years) associated with a one-unit increase in NO_2 (average lag 0-1)

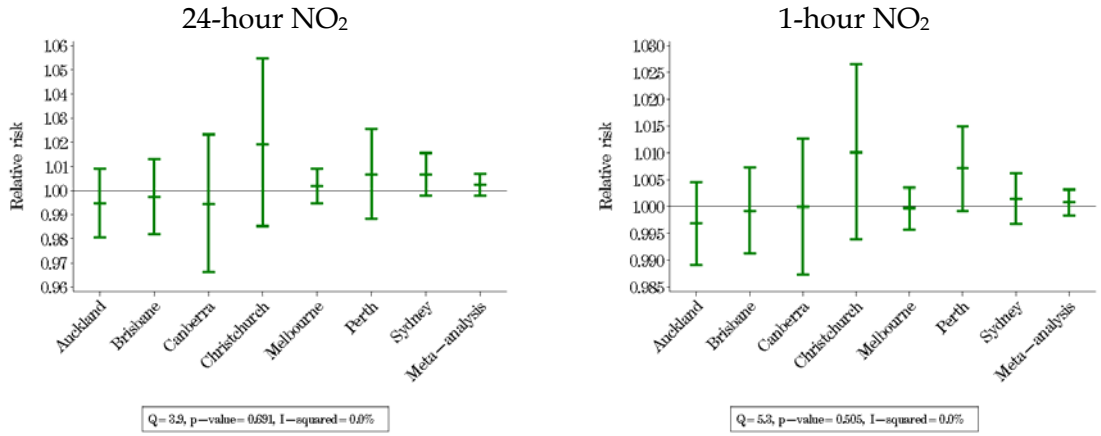
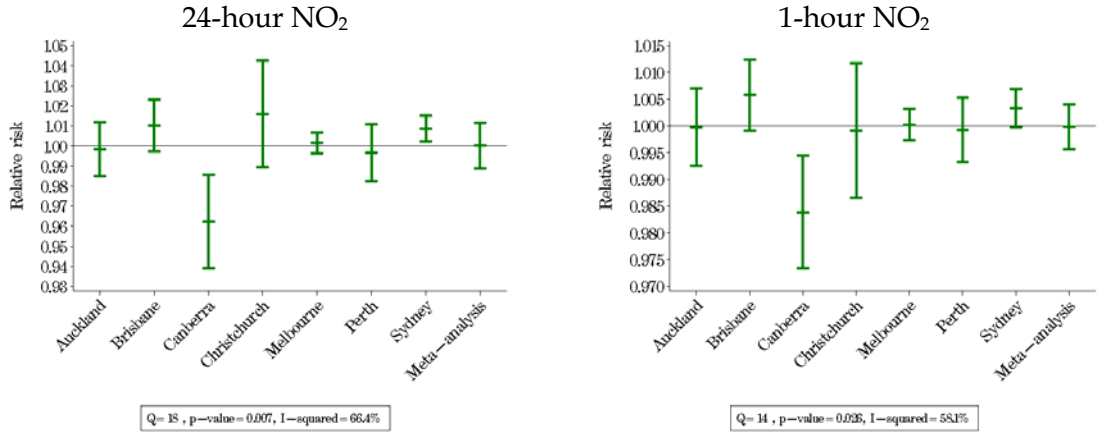


Figure 2.2.29: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (65+ years) associated with a one-unit increase in NO_2 (average lag 0-1)



The results in Tables 2.2.20 to 2.2.23 (all the results per inter-quartile range) show the effects of leaving cities out of the analyses. The results can be summarised as follows:

- there are statistically significant positive increases in admissions for children in the age group less than 1 year for Australian cities associated with 24-hour NO_2, and there is no evidence for heterogeneity between the results.

Table 2.2.20: Differences between cities using a leave-one-city-out sensitivity analysis for pneumonia and acute bronchitis (0 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	4.7	0.6,	8.8	45.9	6.1	3.1,	9.1	0.2
Brisbane	3.5	-1.6,	8.9	69.6*	3.8	-1.8,	9.8	71.9*
Canberra	2.7	-2.3,	7.9	72.3*	2.8	-2.5,	8.4	73.0*
Christchurch	2.0	-2.9,	7.1	70.0*	2.4	-2.6,	7.6	71.4*
Melbourne	1.5	-3.4,	6.6	62.8*	2.0	-3.4,	7.7	67.8*
Perth	3.7	-1.4,	9.0	67.8*	3.7	-1.9,	9.6	72.5*
Sydney	1.9	-3.6,	7.8	67.6*	2.2	-3.4,	8.1	68.8*
Australian only	4.0	-0.3,	8.5	52.2*	5.7	2.5,	9.0	7.2
NZ only	1.6	-13.3,	19.2	82.5*	1.2	-15.6,	21.4	83.2*
Overall	2.8	-1.8,	7.7	66.8*	3.2	-1.8,	8.4	68.1*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.21: Differences between cities using a leave-one-city-out sensitivity analysis for pneumonia and acute bronchitis (1-4 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	3.6	-4.2,	12.1	63.9*	5.4	-1.9,	13.3	46.4
Brisbane	3.7	-4.1,	12.2	64.1*	3.8	-2.8,	10.8	34.9
Canberra	5.7	2.5,	9.0	0.0	4.9	1.4,	8.7	10.5
Christchurch	3.0	-3.8,	10.2	59.9*	3.7	-0.5,	8.1	20.8
Melbourne	4.6	-3.4,	13.2	59.8*	5.5	-2.0,	13.5	45.6
Perth	3.0	-4.4,	10.9	60.1*	4.3	-2.6,	11.7	43.5
Sydney	3.5	-4.5,	12.1	62.5*	5.3	-2.3,	13.5	46.6
Australian only	2.1	-6.2,	11.2	67.4*	3.9	-2.3,	10.5	35.9
NZ only	8.0	0.4,	16.2	0.0	10.1	-7.5,	31.0	67.0*
Overall	4.1	-2.4,	11.0	57.1*	4.8	-1.0,	11.0	36.7

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 2.2.22: Differences between cities using a leave-one-city-out sensitivity analysis for pneumonia and acute bronchitis (15-64 years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	1.1	-1.2,	3.5	0.0	1.7	-0.8,	4.4	0.0
Brisbane	0.9	-1.5,	3.3	2.7	1.5	-1.0,	4.1	0.0
Canberra	0.8	-1.6,	3.1	5.4	1.4	-1.1,	3.9	0.0
Christchurch	0.5	-1.7,	2.8	0.0	1.1	-1.3,	3.6	0.0
Melbourne	1.3	-1.5,	4.2	0.0	1.5	-1.6,	4.7	0.0
Perth	0.1	-2.2,	2.4	0.0	1.1	-1.4,	3.6	0.0
Sydney	0.5	-2.1,	3.2	3.8	0.4	-2.4,	3.3	0.0
Australian only	0.9	-1.5,	3.3	0.0	1.6	-1.0,	4.2	0.0
NZ only	1.3	-9.2,	13.0	52.2	0.9	-9.7,	12.7	40.6
Overall	0.7	-1.5,	2.9	0.0	1.3	-1.1,	3.7	0.0

Table 2.2.23: Differences between cities using a leave-one-city-out sensitivity analysis for pneumonia and acute bronchitis (65+ years) associated with an IQR increase in NO_2 (average lag 0-1)

	1-hour NO_2				24-hour NO_2			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	-0.3	-4.7,	4.3	64.8*	0.1	-6.8,	7.5	71.1*
Brisbane	-1.0	-4.9,	3.0	58.5*	-0.9	-7.4,	6.0	69.9*
Canberra	1.3	-0.5,	3.0	0.0	2.2	-0.0,	4.4	18.8
Christchurch	-0.2	-4.4,	4.1	64.9*	-0.8	-6.9,	5.7	70.5*
Melbourne	-0.5	-5.3,	4.5	64.3*	-0.2	-7.2,	7.2	70.8*
Perth	-0.2	-4.7,	4.4	64.3*	0.2	-6.6,	7.6	70.6*
Sydney	-1.0	-5.3,	3.5	57.9*	-0.9	-7.6,	6.2	64.1*
Australian only	-0.6	-6.3,	5.5	71.7*	-1.1	-8.8,	7.2	75.8*
NZ only	-0.4	-5.9,	5.4	0.0	1.4	-5.6,	9.0	22.8
Overall	-0.2	-3.8,	3.6	58.1*	0.1	-5.6,	6.1	66.4*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.2.2.5 Seasonal differences

Separate analyses were carried out for the warm (November– April) and cool (May–October) periods to identify if there are different effects in each period (due to different emission patterns, atmospheric chemistry). Individual city seasonal results for respiratory admissions are shown in *Volume 3: Appendix A3.4*, Tables WC.15 to WC.29. Meta-analysis and results from the leave-one-city-out analysis for respiratory admissions are shown in *Volume 3: Appendix A3.5*, Tables WCL.15 to WCL.29.

Significant seasonal associations for respiratory admissions and NO_2 are shown in Table 2.2.24, and there was no evidence of heterogeneity between the results for the cities. The differences found between the cool and warm periods can be summarised as follows:

- the increases in hospital admissions due to pneumonia and acute bronchitis for children in the age group less than 1 year were significant in the warm season for both maximum 1-hour NO_2 and 24-hour average NO_2, and in the age group 1 to 4 years, for the warm period (maximum 1-hour NO_2) and the cool period (average 24-hour average NO_2)

- the increases in hospital admissions due to all respiratory disease for children aged between 1 and 4 years were significant for the cool period for both maximum 1-hour NO_2 and 24-hour average NO_2

- the increases in hospital admissions due to all respiratory disease and asthma for children aged between 5 and 14 years were significant for the warm period for both maximum 1-hour NO_2 and 24-hour average NO_2.

Table 2.2.24: Significant seasonal increases in respiratory admissions associated with an IQR increase in NO_2 (average lag 0-1) (case-crossover meta-analysis)

Admissions (age group)		1-hour NO_2			24-hour NO_2		
		Inc	95%CI		Inc	95%CI	
Total respiratory (1-4 years)	cool	2.7	0.7,	4.8	2.2	0.2,	4.3
	warm	3.4	-1.6,	68.3*	2.4	-5.8,	11.4
Total respiratory (5-14 years)	cool	5.7	-1.9,	14.0	6.0	-0.7,	13.0
	warm	8.6	4.0,	13.3	9.6	3.3,	16.3
Asthma (5-14 years)	cool	3.4	-5.1,	12.8	7.0	-2.4,	17.3
	warm	7.5	1.6,	13.8	10.2	2.6,	18.4
Pneumonia + acute bronchitis (0 years)	cool	0.7	-4.5,	6.1	1.9	-3.1,	7.0
	warm	9.7	4.0,	15.7	9.9	2.7,	17.7
Pneumonia + acute bronchitis (1-4 years)	cool	5.2	-0.9,	11.8	4.5	0.6,	8.6
	warm	5.9	0.1,	12.0	5.1	-2.2,	12.9

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

2.3 Single city results

There are often differences between the results for the cities, so combining the estimates for all the cities would not be appropriate in all instances, whether in combining the results for all cities or just combining results for all Australian cities. Additional significant associations were found with short-term exposure to NO_2 in some cities for the following categories of hospital admissions:

- stroke in adults aged 65 years or greater in Auckland
- stroke in adults aged between 15 and 64 years in Brisbane and Canberra
- myocardial infarction in adults aged between 15 and 64 years in Sydney
- asthma in children aged between 5 and 14 years in Auckland and Sydney
- COPD in adults aged 65 years or greater in Brisbane and Sydney
- pneumonia and acute bronchitis in children aged between 1 and 4 years in Brisbane, Christchurch, Perth and Sydney
- pneumonia and acute bronchitis in adults aged 65 years or greater in Sydney
- all respiratory disease in adults aged 65 years or greater in Brisbane, Christchurch and Sydney.

All individual city estimates for short-term associations are shown in *Volume 3: Appendix A3.1.*

Chapter 3 Carbon monoxide

Carbon monoxide (CO) is one of the air pollutants monitored on a daily basis in all the cities under study here, but the number of air pollution monitors measuring ambient outdoor air pollutant concentrations is different in each city. In Auckland there are 3 monitors, Brisbane 1 monitor, Canberra 1 monitor, Christchurch 2 monitors, Melbourne 3 monitors, Perth 3 monitors and Sydney 4 monitors. As the number of monitors used in each city ranges from 1 to 4, the representativeness of the network varies. The project team used the data supplied by the government agencies in each city. Detailed summary statistics for air pollutant data are supplied in *Volume 3: Appendix 1*.

The results are reported as the percentage increase in the daily health outcome data for 1998 to 2001 due to an increase in the daily maximum 8-hour concentrations of CO.

Summarised in Table 3.0.1 are all the significant meta-analysis estimates for health impacts associated with CO. Given are the associations for the short-term exposure (an average of the CO concentration on the same day as the health effect, and the day before) to CO on morbidity and mortality health outcomes. Table 3.0.2 summarises the associations for long-term exposure (an average of the CO concentration on the same day as the health effect, and all forty-days before).

Table 3.0.1: Significant increases in morbidity and mortality (and 95% confidence intervals) associated with a one-unit (ppm) increase in maximum 8-hour CO (lags 0-1) for all seven cities (case-crossover meta-analysis)

Health outcome	Age group (years)	% increase (95% CI)	I2 statistic
Hospital admissions			
Cardiovascular	15-64	1.3 (0.3,2.4)	6.6
	65+	2.5 (1.0,4.0)	69.5*
Cardiac	15-64	2.0 (0.6,3.3)	24.7
	65+	3.3 (1.5,5.1)	73.5*
Ischemic heart disease	65+	2.7 (1.1,4.4)	35.9
Myocardial infarction	65+	3.3 (0.9,5.8)	21.3
Cardiac failure	15-64	4.9 (0.7,9.1)	0.0
	65+	7.0 (4.1,10.1)	61.6*
Arrhythmia	15-64	2.9 (0.1,5.7)	5.6

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

It is clear from the analysis that the results for Sydney are different to other cities, the increases being higher, as shown in Table 3.2.1 for cardiovascular admissions, Table

3.2.3 for cardiac admissions, and Table 3.2.6 for cardiac failure (for example, Figure 3.2.6 shows this result quite clearly). However, even without Sydney in the analysis, the increases are still significant for all the other cities combined with no evidence for heterogeneity (see Table 3.2.1 for cardiovascular admissions, Table 3.2.3 for cardiac admissions; Table 3.2.6 for cardiac failure).

The APHEA2 project used the parameter — number of monitors — as an effect modifier, and tests on the results for CO using this parameter (see Table 6.1.9) found evidence to suggest there were differences in associations between cities that could be linked to the different number of monitors used in each city, as the city with the highest number of monitors, Sydney (with 4), shows the highest effect and is different to the other cities. It could be expected that cities with more monitors would likely have given a better estimate of the population exposure, and therefore this effect modification due to the number of monitors would most likely be due to an occurrence known as regression dilution (MacMahon 1990). In regression dilution a true association appears stronger when the exposure is measured with greater accuracy. It would be expected that, as the number of monitors increases, the exposure estimate based on the number of monitors used should improve. It is always a concern that cities with a small number of monitors may show unexpectedly high (or low) results. The increases for the city with the largest number of monitors (Sydney) is also notably the largest which, following MacMahon (1990), may well reinforce the conclusion that this is a true association.

Similar tests for other different city characteristics (such as climate, elderly population, and different average pollution levels) did not identify any additional city characteristics that could explain differences between the results for different cities.

Table 3.0.2: Comparison of significant forty-day increases in mortality (and 95% confidence intervals) with short-term increases (average lags 0-1) associated with a one-unit increase in maximum 8-hour CO

Health outcome	Age group (years)	% increase (95% CI) 40-day	% increase (95% CI) Av 0-1
Cardiovascular	All ages	9.3 (3.0,16.1)	0.4 (-0.7, 1.5)
Respiratory	All ages	32.4 (16.9,50.0)	1.4 (-1.3, 4.2)
Total all cause	All ages	11.0 (5.6,16.7)	0.6 (-0.1, 1.3)

Results for only Australian cities

When only the Australian cities were examined (Brisbane, Canberra, Melbourne, Perth and Sydney), there were significant increases in all hospital admissions due to myocardial infarction and ischemic heart disease in the adults aged between 15 and 64 years (see Table 3.0.3). There were also significant increases in hospital admissions due to respiratory disease for children aged 1 to 4 years, and significant increases in respiratory mortality for all ages.

Table 3.0.3: Significant increases in morbidity and mortality (and 95% confidence intervals) associated with maximum 8-hour CO (lags 0-1) for all (5) Australian cities – results only presented for those not identified in Table 3.0.1

Health outcome	Age group (years)	IQR* % increase (95% CI)	I2 statistic
Hospital admissions			
IHD	15-64	2.1 (0.3,3.9)	24.2
Myocardial infarction	15-64	3.0 (0.4,5.7)	0.0
Respiratory	1-4	1.5 (0.1,2.9)	0.0
Mortality			
Respiratory	All ages	2.7 (0.3,5.2)	0.0

* 0.9 ppm

However, it is not possible in statistical analyses like these to identify any causes for the increases, only significant associations can be noted. It is possible that the increases identified here with exposure to CO may not be due to CO, but to other pollutants which are correlated with CO as they arise from the same emissions source, such as motor vehicle exhausts.

3.1 Mortality

3.1.1 Short-term averages for air pollutants

The mortality impacts of CO for the short term (average of same day as, and day before death) are shown in Figure 3.1.1 for total deaths (non-accidental, all causes), deaths due to cardiovascular disease and deaths due to respiratory disease. The results are shown for two age groups: all age groups and the oldest (75 years and greater). The pooled estimates generally show positive increases in mortality associated with increases in CO, but they are not significant.

Figure 3.1.1: City specific and meta-analysis relative risks (and 95% confidence intervals) for mortality associated with a one-unit increase in 8-hour CO (average lag 0-1)

Figure 3.1.1.A: Total all cause mortality

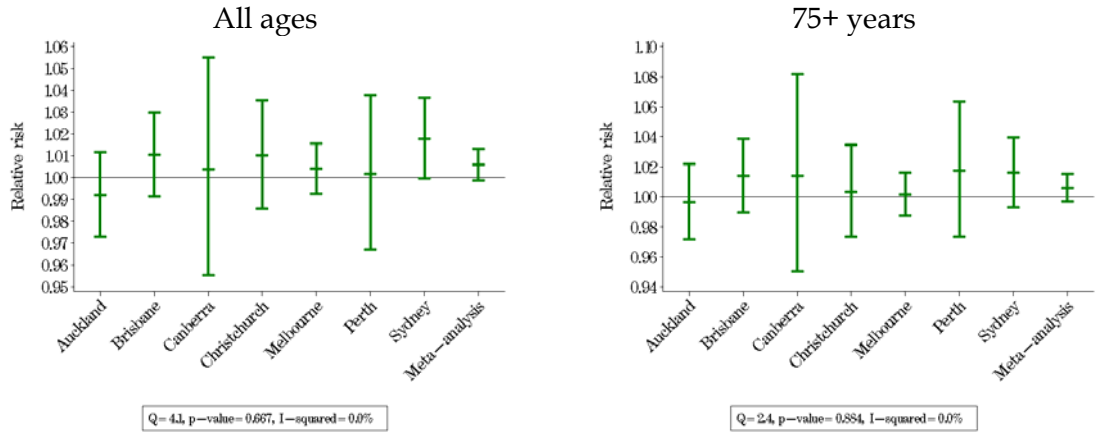


Figure 3.1.1.B: Respiratory mortality

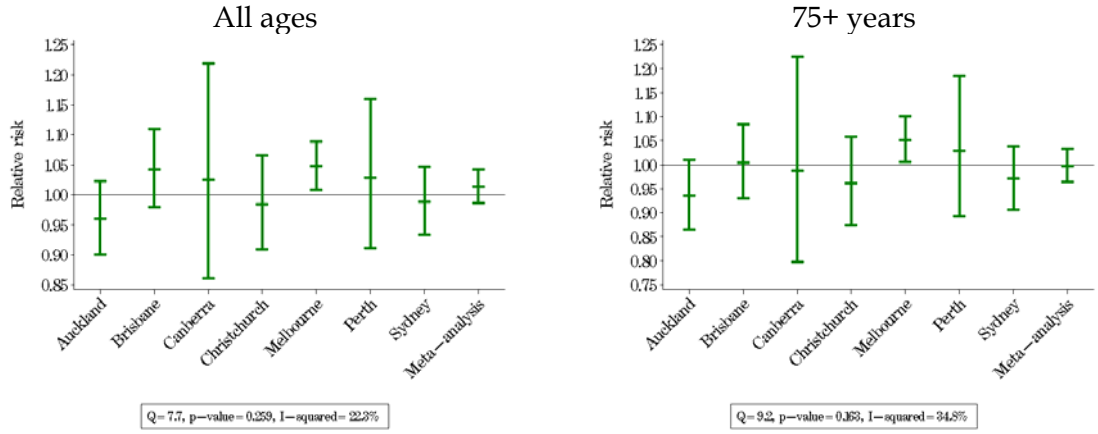
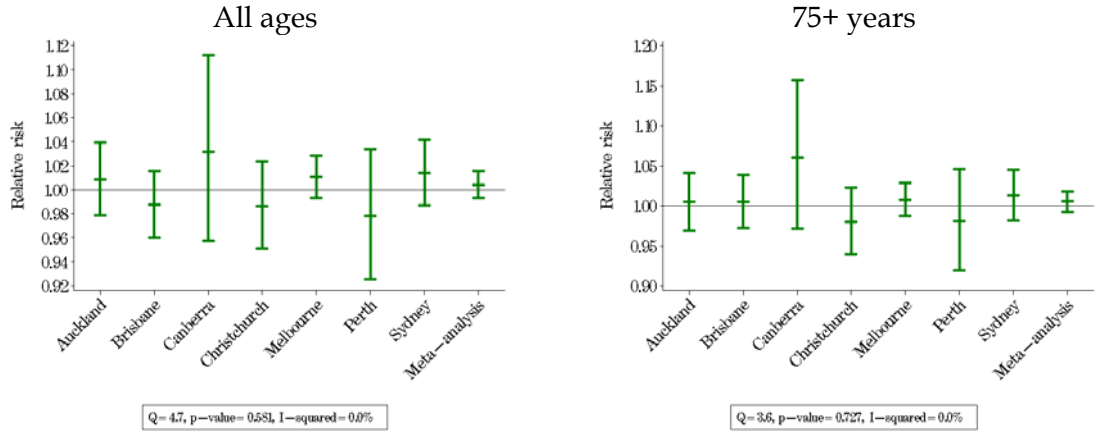


Figure 3.1.1.C: Cardiovascular mortality



Tables 3.1.1 to 3.1.3 show the impact of leaving out each city in turn from the analysis for each health category for all ages. The results for significant pooled estimates may be summarised as follows:

- the pooled estimates for Australian cities for respiratory mortality are significant and positive, and there is no evidence of heterogeneity between the results for the cities

- after removing Auckland, the pooled estimates for the remaining cities for all cause mortality and respiratory mortality are significant and positive, and there is no evidence of heterogeneity between the results for the cities.

Table 3.1.1: Differences between cities using a leave-one-city-out sensitivity analysis for total mortality (all ages) associated with an IQR increase in CO (average lag 0-1)

	8-hour CO			
City left out	Inc	95%CI		I2
Auckland	0.7	0.0,	1.4	0.0
Brisbane	0.4	-0.2,	1.1	0.0
Canberra	0.5	-0.1,	1.2	0.0
Christchurch	0.5	-0.2,	1.1	0.0
Melbourne	0.6	-0.2,	1.4	0.0
Perth	0.5	-0.1,	1.2	0.0
Sydney	0.3	-0.4,	1.0	0.0
Australian only	0.7	-0.0,	1.4	0.0
NZ only	-0.0	-1.5,	1.5	23.4
Overall	0.5	-0.1,	1.1	0.0

Table 3.1.2: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory mortality (all ages) associated with an IQR increase in CO (average lag 0-1)

	8-hour CO			
City left out	Inc	95%CI		I2
Auckland	2.3	0.0,	4.6	0.0
Brisbane	0.7	-1.9,	3.4	28.4
Canberra	1.0	-1.6,	3.7	35.2
Christchurch	1.5	-1.2,	4.1	28.7
Melbourne	-0.2	-2.8,	2.5	0.0
Perth	1.0	-1.6,	3.7	35.0
Sydney	1.6	-1.0,	4.4	24.5
Australian only	2.7	0.3,	5.2	0.0
NZ only	-2.7	-6.7,	1.6	0.0
Overall	1.2	-1.2,	3.6	22.3

Table 3.1.3: Differences between cities using a leave-one-city-out sensitivity analysis for cardiovascular mortality (all ages) associated with an IQR increase in CO (average lag 0-1)

	8-hour CO			
City left out	Inc	95%CI		I2
Auckland	0.3	-0.7,	1.3	0.0
Brisbane	0.6	-0.4,	1.7	0.0
Canberra	0.3	-0.6,	1.3	0.0
Christchurch	0.5	-0.5,	1.5	0.0
Melbourne	0.0	-1.2,	1.2	0.0
Perth	0.5	-0.5,	1.4	0.0
Sydney	0.2	-0.8,	1.3	0.0
Australian only	0.5	-0.6,	1.6	0.0
NZ only	-0.0	-2.0,	2.0	0.0
Overall	0.4	-0.6,	1.3	0.0

3.1.2 'Harvesting'?

It is clear from Table 3.0.2 that the associations for the cumulative impacts of outdoor CO concentrations **forty days** prior to the health outcome are much higher than those for short-term exposures. For example, for all ages, the results show a *one-ppm* increase in 8-hour CO is associated with significant mean increases in daily mortality of:

- 11% for all causes (compared to an insignificant increase of 0.6% for short-term), with a 95% confidence interval of 5.6% to 16.7%

- 9.3% for cardiovascular disease (an insignificant increase of 0.4% for short-term), with a 95% confidence interval of 3.0% to 16.1%

- 32.4% for respiratory disease (an insignificant increase of 1.4% for short-term), with a 95% confidence interval of 16.9% to 50%.

The uncertainty intervals are large, but the increases for the forty-day exposures are significantly greater than those for the short-term exposures. These results do not support the 'harvesting' hypothesis.

Although the I-squared statistic indicates that the harvesting results are the same for all cities, the results for the individual cities are shown in Tables 3.1.4 to 3.1.6, and these indicate that Brisbane and Sydney data are dominating the estimates for Australian cities, and Christchurch for New Zealand cities. It is also noticeable that the confidence intervals are very large.

Table 3.1.4: Per cent changes (and 95% confidence interval) in total mortality (all ages) over 40-day periods* associated with 8-hour CO

	8-hour CO		
City	Inc	95%CI	
Auckland	4.7	-4.4,	14.6
Brisbane	13.5	2.4,	25.8
Canberra	-5.0	-25.9,	21.8
Christchurch	14.3	5.3,	24.0
Melbourne	5.4	0.2,	10.8
Perth	15.4	-2.2,	36.3
Sydney	21.6	12.4,	31.6
I-squared (%)	54.0	-	-
Meta-analysis	11.0	5.6,	16.7
Meta-analysis IQR	9.4	4.8,	14.3

Table 3.1.5: Per cent changes (and 95% confidence interval) in respiratory mortality (all ages) over 40-day periods* associated with 8-hour CO

	8-hour CO		
City	Inc	95%CI	
Auckland	5.1	-19.5,	37.4
Brisbane	67.1	21.4,	130.1
Canberra	10.9	-45.4,	125.3
Christchurch	26.9	-1.9,	64.1
Melbourne	25.2	6.5,	47.1
Perth	52.8	-10.5,	160.7
Sydney	59.7	23.0,	107.4
I-squared (%)	24.4	-	-
Meta-analysis	32.4	16.9,	50.0
Meta-analysis IQR	27.9	14.6,	43.0

Table 3.1.6: Per cent changes (and 95% confidence interval) in cardiovascular mortality (all ages) over 40-day periods* associated with 8-hour CO

	8-hour CO		
City	Inc	95%CI	
Auckland	8.2	-4.7,	22.8
Brisbane	8.8	-6.5,	26.6
Canberra	12.5	-17.8,	53.9
Christchurch	6.6	-4.8,	19.4
Melbourne	1.4	-6.2,	9.7
Perth	12.2	-13.8,	46.1
Sydney	26.6	12.6,	42.4
I-squared (%)	38.5	-	-
Meta-analysis	9.3	3.0,	16.1
Meta-analysis IQR	8.0	2.6,	13.8

* Estimates from a random effects meta-analysis of cities using distributed lag models, individual city results are shown for a one-unit increase in pollutant and meta-analysis results are shown for an inter-quartile range (IQR) increase where indicated.

3.1.3 Seasonal differences

Separate analyses were carried out for the warm (November–April) and cool (May–October) periods to identify if there are different effects in each period (due to different emission patterns, atmospheric chemistry). Individual city seasonal results for mortality are shown in *Volume 3: Appendix A3.4*, Tables WC.30 to WC.38. Meta-analysis and results from the leave-one-city-out analysis for mortality are shown in *Volume 3: Appendix A3.5*, Tables WCL.30 to WCL.38.

In the seasonal analysis, no significant differences were found between the cities, and there were no significant differences found between the cool and warm periods.

3.2 Hospital admissions

3.2.1 Hospital admissions due to cardiovascular disease

The following categories for cardiovascular admissions were examined in this study (see Table 1.2 for the ICD codes):

- total cardiovascular disease
- stroke
- cardiac disease
- ischemic heart disease
- myocardial infarction
- cardiac failure
- arrhythmia.

In the following section, the associations between cardiac admissions for two age groups (15-64 years, 65 years and greater) and ambient concentrations of daily maximum 8-hour CO in the short term (average of the same day and day before – lag [0,1]) are presented.

3.2.1.1 Cardiovascular disease

The associations with total cardiovascular admissions are shown in Figures 3.2.1 and 3.2.2. There are statistically significant positive increases for the pooled estimates for both the 15-64 years age group, and for the older age group. There was no evidence for heterogeneity between the results for the cities for the 15-64 years age group, but there was for the older age group.

Figure 3.2.1: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiovascular admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

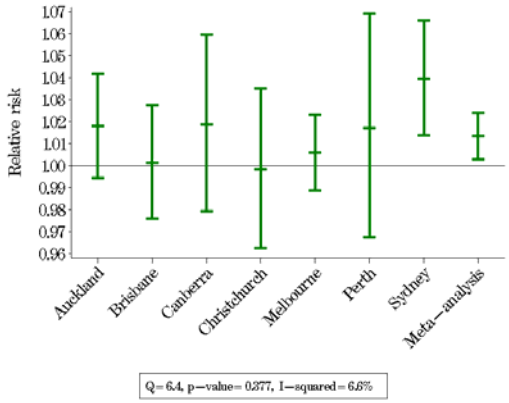


Figure 3.2.2: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiovascular admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

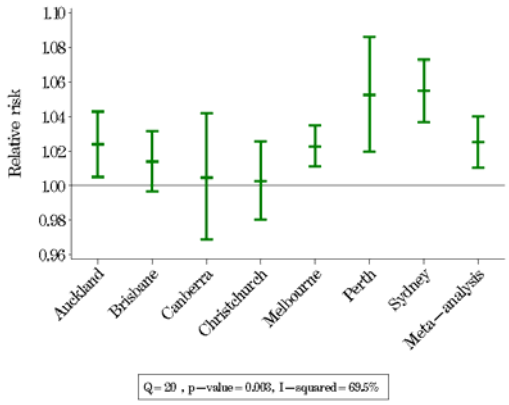


Table 3.2.1 shows the effects on the results of leaving cities out of the analysis, and the significant estimates for the pooled estimates can be summarised as follows:

- the increases are statistically significant and positive for the Australian cities as a group, and for both age groups, with the increases for Sydney being significantly larger

- leaving Sydney out of the analysis removes any significant heterogeneity in the results for the older group, with the pooled estimate for the increase in the remaining cities both positive and significant.

Table 3.2.1: Differences between cities using a leave-one-city-out sensitivity analysis for cardiovascular admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	1.1	-0.0,	2.2	19.9	2.2	0.7,	3.7	74.6*
Brisbane	1.3	0.3,	2.3	8.9	2.3	0.9,	3.8	71.9*
Canberra	1.1	0.1,	2.2	21.2	2.3	1.0,	3.7	72.9*
Christchurch	1.3	0.3,	2.3	12.7	2.5	1.2,	3.8	67.6*
Melbourne	1.5	0.4,	2.6	6.4	2.2	0.7,	3.8	74.2*
Perth	1.2	0.1,	2.2	21.9	1.9	0.6,	3.2	70.6*
Sydney	0.7	-0.2,	1.7	0.0	1.7	0.7,	2.7	33.4
Australian only	1.3	0.1,	2.5	28.8	2.6	1.0,	4.2	73.7*
NZ only	1.0	-0.6,	2.8	0.0	1.2	-0.5,	3.0	50.1
Overall	1.2	0.3,	2.1	6.6	2.2	0.9,	3.4	69.5*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

3.2.1.2 Stroke

The associations with hospital admissions due to stroke are shown in Figures 3.2.3 and 3.2.4. There are no significant impacts.

Figure 3.2.3: City specific and meta-analysis relative risks (and 95% confidence intervals) for stroke admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

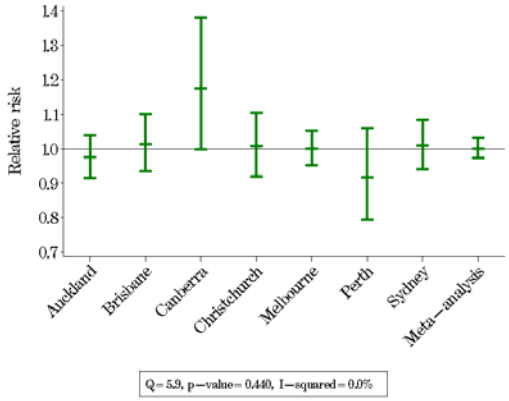


Figure 3.2.4: City specific and meta-analysis relative risks (and 95% confidence intervals) for stroke admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

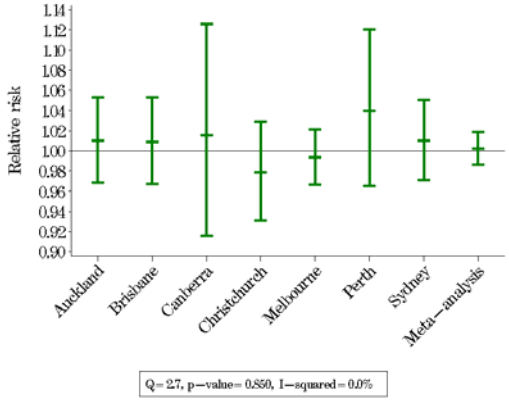


Table 3.2.2 shows the effects on the results of leaving cities out of the analysis. No significant pooled estimates were identified in this analysis.

Table 3.2.2: Differences between cities using a leave-one-city-out sensitivity analysis for stroke admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	0.7	-2.2,	3.6	1.3	0.1	-1.4,	1.6	0.0
Brisbane	0.0	-3.3,	3.4	12.9	0.1	-1.4,	1.6	0.0
Canberra	-0.4	-2.8,	2.2	0.0	0.2	-1.3,	1.6	0.0
Christchurch	0.1	-3.2,	3.6	14.3	0.4	-1.0,	2.0	0.0
Melbourne	0.2	-3.4,	4.0	14.6	0.6	-1.1,	2.4	0.0
Perth	0.4	-2.1,	2.9	0.0	0.0	-1.4,	1.5	0.0
Sydney	0.1	-3.3,	3.6	13.6	0.1	-1.5,	1.6	0.0
Australian only	0.9	-3.6,	5.7	21.1	0.4	-1.3,	2.0	0.0
NZ only	-1.2	-5.5,	3.3	0.0	-0.3	-3.0,	2.5	0.0
Overall	0.1	-2.4,	2.6	0.0	0.2	-1.2,	1.6	0.0

3.2.1.3 Cardiac admissions

The associations with cardiac admissions are shown in Figures 3.2.5 and 3.2.6. There are statistically significant positive increases for the pooled estimates for both the 15-64 years age group, and for the older age group. There was no evidence for heterogeneity between the results for the cities for the 15-64 years age group, but there was for the older age group.

Figure 3.2.5: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

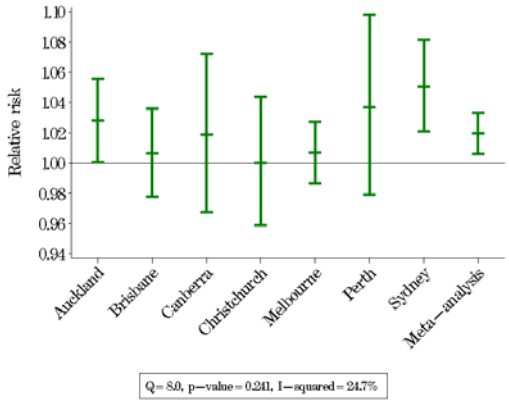


Figure 3.2.6: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

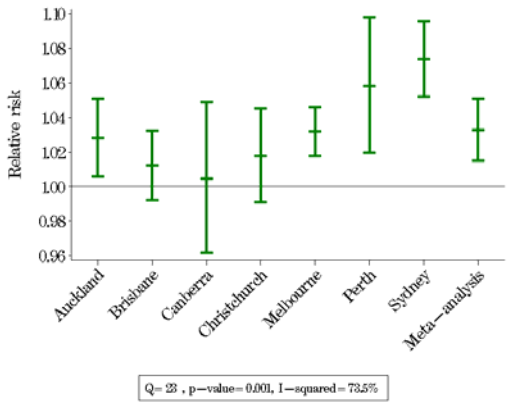


Table 3.2.3 shows the effects on the results of leaving cities out of the analysis, and the significant estimates for the pooled estimates can be summarised as follows:

- the increases are statistically significant and positive for the Australian cities as a group, and for both age groups
- leaving Sydney out of the analysis removes the heterogeneity in the results for the older group, with the pooled estimate for the increase in the remaining cities both positive and significant.

Table 3.2.3: Differences between cities using a leave-one-city-out sensitivity analysis for cardiac admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	1.5	0.2,	2.9	33.0	2.9	1.1,	4.7	77.7*
Brisbane	1.9	0.6,	3.2	29.7	3.2	1.5,	4.9	71.5*
Canberra	1.7	0.4,	3.1	37.2	3.1	1.5,	4.7	76.1*
Christchurch	1.8	0.6,	3.1	30.5	3.0	1.3,	4.8	76.5*
Melbourne	2.1	0.8,	3.5	15.6	2.8	1.0,	4.7	77.8*
Perth	1.6	0.3,	2.9	34.2	2.6	0.9,	4.2	76.3*
Sydney	1.1	0.0,	2.2	0.0	2.2	1.2,	3.1	21.7
Australian only	1.8	0.2,	3.3	41.5	3.2	1.0,	5.3	80.8*
NZ only	1.7	-0.4,	3.8	10.0	2.0	0.6,	3.5	0.0
Overall	1.7	0.5,	2.9	24.7	2.8	1.3,	4.4	73.5*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

3.2.1.4 Ischemic heart disease

The associations with hospital admissions due to ischemic heart disease are shown in Figures 3.2.7 and 3.2.8. There are statistically significant positive increases for the pooled estimates for the older age group, and there was no evidence for heterogeneity between the results for the cities.

Figure 3.2.7: City specific and meta-analysis relative risks (and 95% confidence intervals) for IHD admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

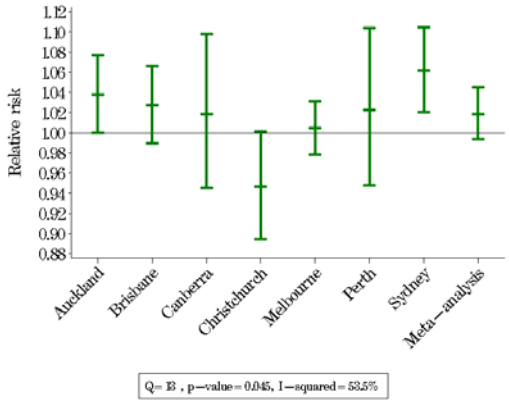


Figure 3.2.8: City specific and meta-analysis relative risks (and 95% confidence intervals) for IHD admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

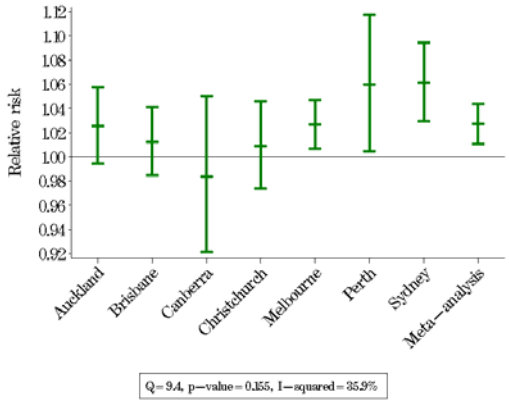


Table 3.2.4 shows the effects on the results of leaving cities out of the analysis. It is clear the associations are significant for the pooled estimate for the Australian cities for both age groups, with the Sydney results causing the evidence found for heterogeneity in the older group.

Table 3.2.4: Differences between cities using a leave-one-city-out sensitivity analysis for IHD admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	1.3	-1.3,	3.9	57.7*	2.3	0.6,	4.1	46.5
Brisbane	1.4	-1.2,	4.2	60.7*	2.6	0.9,	4.3	38.1
Canberra	1.6	-1.0,	4.2	61.3*	2.5	1.1,	3.9	34.5
Christchurch	2.3	0.8,	3.8	13.2	2.6	1.0,	4.2	39.7
Melbourne	1.9	-0.8,	4.7	54.7*	2.3	0.5,	4.2	46.5
Perth	1.6	-1.0,	4.2	61.2*	2.1	0.7,	3.6	37.4
Sydney	1.0	-1.2,	3.1	38.6	1.9	0.8,	3.0	0.0
Australian only	2.1	0.3,	3.9	24.2	2.7	0.6,	4.8	51.4*
NZ only	-0.6	-8.0,	7.5	85.9*	1.6	-0.4,	3.7	0.0
Overall	1.6	-0.6,	3.9	53.5*	2.3	0.9,	3.8	35.9

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

3.2.1.5 Myocardial infarction

The associations with hospital admissions due to myocardial infarction are shown in Figures 3.2.9 and 3.2.10. There are statistically significant positive increases for the pooled estimates for the older age group, and there was no evidence for heterogeneity between the results for the cities.

Figure 3.2.9: City specific and meta-analysis relative risks (and 95% confidence intervals) for myocardial admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

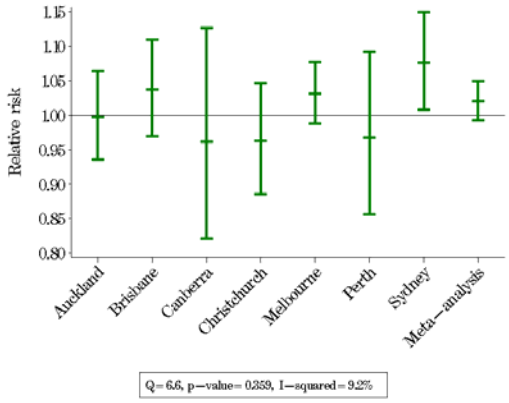


Figure 3.2.10: City specific and meta-analysis relative risks (and 95% confidence intervals) for myocardial admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

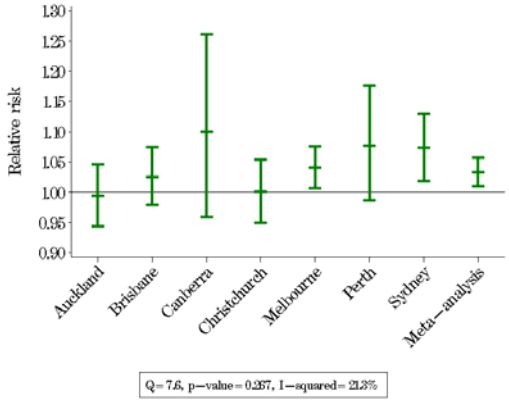


Table 3.2.5 shows the effects on the results of leaving cities out of the analysis. It is clear the associations are significant for the pooled estimate for the Australian cities for both age groups, with no evidence found for heterogeneity in either age group.

Table 3.2.5: Differences between cities using a leave-one-city-out sensitivity analysis for myocardial infarction admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	2.1	-0.8,	5.0	16.2	3.4	1.5,	5.3	2.4
Brisbane	1.3	-1.6,	4.3	21.7	3.0	0.5,	5.6	33.5
Canberra	1.9	-0.8,	4.6	17.1	2.7	0.6,	4.9	26.5
Christchurch	2.4	0.0,	4.9	0.0	3.3	1.2,	5.5	15.9
Melbourne	1.2	-2.0,	4.5	20.8	2.7	0.1,	5.4	31.9
Perth	2.0	-0.6,	4.6	13.6	2.6	0.5,	4.8	25.3
Sydney	1.0	-1.4,	3.5	0.0	2.3	0.4,	4.2	4.3
Australian only	3.0	0.4,	5.7	0.0	4.1	2.0,	6.1	0.0
NZ only	-1.3	-5.6,	3.1	0.0	-0.2	-3.4,	3.0	0.0
Overall	1.8	-0.7,	4.3	9.2	2.9	0.8,	4.9	21.3

3.2.1.6 Cardiac failure

The associations with hospital admissions due to cardiac failure are shown in Figures 3.2.11 and 3.2.12. There are statistically significant positive increases for the pooled estimates for both the age groups, but there was evidence for heterogeneity between the results in the older age group.

Figure 3.2.11: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac failure admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

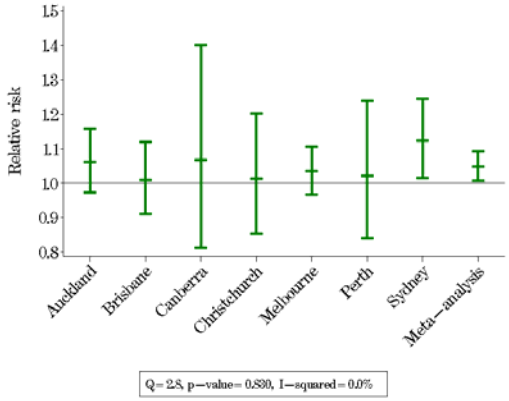


Figure 3.2.12: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac failure admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

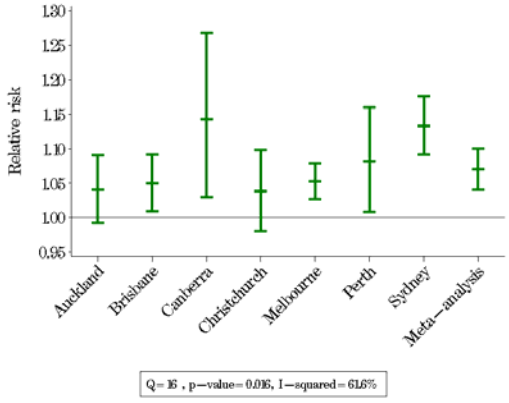


Table 3.2.6 shows the effects on the results of leaving cities out of the analysis. It is clear the associations are significant for the pooled estimate for the Australian cities for the older age group, with the Sydney results causing significant heterogeneity.

Table 3.2.6: Differences between cities using a leave-one-city-out sensitivity analysis for cardiac failure admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	3.9	-0.1,	8.0	0.0	6.5	3.7,	9.3	65.3*
Brisbane	4.8	0.9,	8.8	0.0	6.4	3.5,	9.4	66.3*
Canberra	4.1	0.6,	7.8	0.0	5.6	3.1,	8.2	64.1*
Christchurch	4.3	0.7,	8.1	0.0	6.4	3.6,	9.3	65.9*
Melbourne	4.9	0.5,	9.5	0.0	6.4	3.4,	9.5	63.6*
Perth	4.3	0.7,	8.0	0.0	5.9	3.0,	8.9	67.7*
Sydney	3.0	-0.8,	7.0	0.0	4.5	3.0,	6.1	0.0
Australian only	4.1	-0.0,	8.4	0.0	7.0	3.9,	10.2	69.7*
NZ only	4.4	-2.3,	11.6	0.0	3.4	0.2,	6.6	0.0
Overall	4.2	0.6,	7.8	0.0	6.0	3.5,	8.5	61.6*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

3.2.1.7 Arrhythmia

The associations with hospital admissions due to arrhythmia are shown in Figures 3.2.13 and 3.2.14. There are statistically significant positive increases for the pooled estimates for the age group 15-64 years, and no evidence for heterogeneity between the results for the cities.

Figure 3.2.13: City specific and meta-analysis relative risks (and 95% confidence intervals) for arrhythmia admissions (15-64 years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

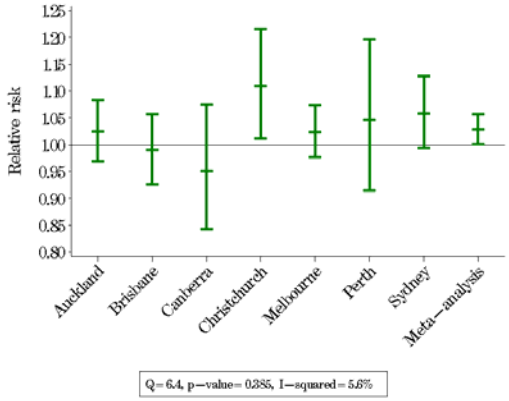


Figure 3.2.14: City specific and meta-analysis relative risks (and 95% confidence intervals) for arrhythmia admissions (65+ years) associated with a one-unit increase in 8-hour CO (average lag 0-1)

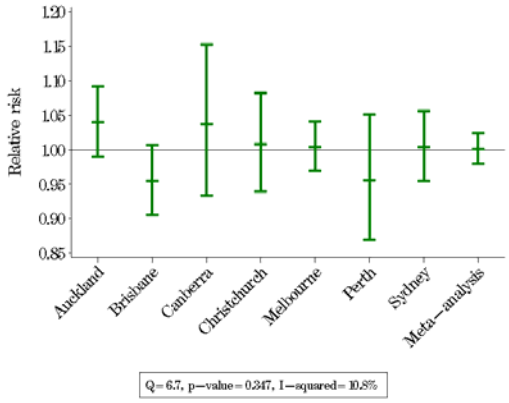


Table 3.2.7 shows the effects on the results of leaving cities out of the analysis. No new significant associations were found.

Table 3.2.7: Differences between cities using a leave-one-city-out sensitivity analysis for arrhythmia admissions associated with an IQR increase in CO (average lag 0-1)

	15-64 years				65+ years			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Auckland	2.6	-0.5,	5.8	21.1	-0.5	-2.5,	1.4	0.0
Brisbane	3.1	0.6,	5.6	0.0	0.9	-1.0,	2.8	0.0
Canberra	2.8	0.5,	5.2	0.0	-0.1	-2.1,	2.1	20.5
Christchurch	1.9	-0.5,	4.3	0.0	0.0	-2.2,	2.3	25.3
Melbourne	2.6	-0.6,	6.0	20.7	-0.0	-2.5,	2.5	25.4
Perth	2.4	-0.4,	5.3	20.5	0.3	-1.6,	2.3	12.9
Sydney	2.0	-0.7,	4.7	7.4	0.0	-2.3,	2.4	25.6
Australian only	1.8	-0.9,	4.5	0.0	-0.7	-2.7,	1.4	0.0
NZ only	4.9	-1.7,	11.9	51.7*	2.5	-1.0,	6.1	0.0
Overall	2.5	0.1,	4.9	5.6	0.1	-1.8,	2.1	10.8

3.2.1.8 Seasonal differences

It might be expected that some pollutant effects may be different in different seasons. For example, ozone peaks during the summer smog episodes in general (although Brisbane may also have winter smog events), but there is no evidence to show that this significantly affects CO concentrations. CO ambient concentrations should rise during winter due to higher emissions (home heating, vehicle emissions). Shown in Table 3.2.8 are the significant seasonal associations found between cardiovascular admissions and maximum 8-hour CO. The differences found between pooled estimates for the cool and warm periods can be summarised as follows:

- the increases in hospital admissions due to all cardiovascular disease, cardiac disease, IHD and cardiac failure, were only significant in the cool periods for adults aged 65 years or greater (and those aged between 15 and 64 years for cardiac disease)

- the increases in hospital admissions due to myocardial infarction for adults aged 65 years or greater were significant for both the warm and cool periods

- there was evidence for heterogeneity between the cities arising from the results for Sydney (as before).

Table 3.2.8: Significant seasonal increases in cardiovascular admissions associated with an IQR increase in CO (average lag 0-1) (case-crossover meta-analysis)

Admissions (age group)		8-hour CO			
		Inc	95%CI		I2
Cardiac (15-64 yrs)	cool	1.9	0.1,	3.7	52.5*
	warm	2.2	-0.3,	4.7	0.3
Cardiac (65+ yrs)	cool	3.3	1.9,	4.8	59.9*
	warm	1.2	-2.1,	4.6	53.2*
IHD (65+ yrs)	cool	2.2	0.9,	3.6	17.4
	warm	3.2	-0.5,	7.1	31.7
Cardiac failure (65+ yrs)	cool	6.3	4.5,	8.2	18.3
	warm	2.2	-4.1,	8.8	49.9
Myocardial infarction (65+ yrs)	cool	2.3	0.3,	4.4	0.0
	warm	7.3	1.2,	13.8	0.0
Total cardiovascular (65+ yrs)	cool	2.6	1.4,	3.7	56.9*
	warm	1.0	-0.6,	2.7	0.0

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

3.2.2 Hospital admissions due to respiratory disease

The respiratory disease groups considered included (see Table 1.2 for the ICD codes):

- total respiratory disease
- asthma
- chronic obstructive pulmonary disease (COPD)
- pneumonia and acute bronchitis.

Analyses for hospital admissions were stratified using the following age groups:

- children
  - less than one year of age
  - 1-4 years
  - 5-14 years
- adults
  - 15-64 years
  - 65 years and greater.

The associations between these health outcomes for these age groups and maximum 8-hour CO ambient concentrations in the short term (average of the same day and day before – lag (0,1)) are presented in *Volume 3: Appendix A3.1*, Tables CM.15 to CM.29. There were no significant associations for the pooled estimates for any age groups.

3.3 Single city results

There are often differences between the results for the cities, so combining the estimates for all the cities would not be appropriate in all instances, whether in combining the results for all cities or just combining results for all Australian cities. Additional significant associations were found with short-term exposure to CO in some cities for the following categories of hospital admissions:

- myocardial infarction in adults aged between 15 and 64 years in Sydney

- asthma in children aged between 1 and 4 years in Christchurch
- asthma in children aged between 5 and 14 years in Auckland
- all respiratory disease (especially pneumonia and acute bronchitis) in children aged less than 1 year in Sydney
- all respiratory disease (especially pneumonia and acute bronchitis) in adults aged 65 years and greater in Sydney
- all respiratory disease in children aged between 5 and 14 years in Auckland.

All individual city estimates for short-term associations are shown in *Volume 3: Appendix A3.1*.

Chapter 4 Particles

Particle concentrations are monitored in all the cities under study here, but not all data sets can be used in the analyses carried out, and the concentrations need to be monitored on a daily basis, something that does not happen in every city. Also, there are three different measures of particle pollution used in Australian and New Zealand cities:

- Nephelometry (light scattering by particles, bsp) - a measure of particles less than 1-2 microns in diameter
- $PM_{2.5}$ - measure of particles with diameters less than 2.5 microns using TEOMS
- PM_{10} - measure of particles with diameters less than 10 microns using TEOMS.

This study concentrates on $PM_{2.5}$ and PM_{10}, as bsp has previously only been used as a surrogate for such measures for which there are NEPC standards (NEPC 1998). These measures were not available on a daily basis in all the cities under study.
There are daily data sets for PM_{10} in five cities:

- Brisbane (4 monitors), Christchurch (2 monitors), Melbourne (4 monitors), Perth (1 monitors) and Sydney (11 monitors).

There are daily data sets for $PM_{2.5}$ in four cities:

- Brisbane (1 monitor), Melbourne (2 monitors), Perth (2 monitors) and Sydney (4 monitors).

The project team used the data supplied by the government agencies in each city. Detailed summary statistics for air pollutant data are supplied in *Volume 3: Appendix 1*. Essentially, the analyses for $PM_{2.5}$ are Australian studies. Christchurch has the highest PM_{10} concentrations in all the cities monitoring this pollutant.

The results are reported as the percentage increase in the daily health outcome data for 1998 to 2001 either due to an increase in the 24-hour average concentrations of $PM_{2.5}$, or due to an increase in the 24-hour average concentrations of PM_{10}.

The results in Table 4.0.1 show all the significant meta-analysis estimates for increases in mortality and hospital admissions associated with increases in short-term exposure to ambient particle concentrations (an average of the particle concentration on the same day as the health effect, and the day before), for those cases when the estimates for all cities in Australia and New Zealand can be reasonably combined to derive such estimates.

It is clear from Table 4.0.1 that the short-term averages of outdoor concentrations of particles are associated with significant increases in mortality for all causes, and for cardiovascular and respiratory disease. There are also significant associations for

both cardiovascular and respiratory hospital admissions. However, tests on the results found no evidence to suggest there was heterogeneity in the associations for the cities.

Table 4.0.1: Significant increases in mortality and morbidity (and 95% confidence intervals) associated with a one-unit ($\mu g.m^{-3}$) increase in particles (lags 0-1) for meta-analysis*

Health outcome	Age group (years)	Particle type**	Per Unit % Increase (95% CI)	I2 statistic
Mortality				
Cardiovascular	All ages	PM_{10}	0.2 (0.0,0.3)	27.1
		$PM_{2.5}$	0.4 (0.2,0.6)	0.0
	75+	PM_{10}	0.2 (0.0,0.4)	27.4
		$PM_{2.5}$	0.5 (0.2,0.7)	0.0
Respiratory	75+	$PM_{2.5}$	0.6 (0.0,1.1)	40.8
Total all cause	All ages	$PM_{2.5}$	0.2 (0.1,0.4)	40.8
	75+	$PM_{2.5}$	0.3 (0.1,0.5)	0.0
Hospital admissions				
Total cardiovascular	65+	$PM_{2.5}$	0.3 (0.1,0.5)	51.9
Cardiac	65+	PM_{10}	0.1 (0.0,0.3)	32.9
		$PM_{2.5}$	0.5 (0.3,0.7)	55.0
Ischemic heart disease	65+	$PM_{2.5}$	0.4 (0.2,0.6)	3.6
Myocardial infarction	65+	$PM_{2.5}$	0.7 (0.3,1.1)	0.0
Cardiac failure	65+	PM_{10}	0.4 (0.3,0.6)	0.0
		$PM_{2.5}$	0.9 (0.5,1.4)	58.6
Total respiratory	0	$PM_{2.5}$	0.6 (0.3,1.0)	0.0
	1-4	PM_{10}	0.2 (0.1,0.4)	0.0
		$PM_{2.5}$	0.4 (0.2,0.7)	0.0
	5-14	PM_{10}	0.3 (0.0,0.5)	0.0
	15-64	$PM_{2.5}$	0.3 (0.0,0.6)	40.7
	65+	$PM_{2.5}$	0.4 (0.2,0.6)	0.0
Asthma	15-64	$PM_{2.5}$	0.6 (0.2,0.9)	0.0
COPD	65+	$PM_{2.5}$	0.4 (0.2,0.7)	0.0
Pneumonia & acute bronchitis	0	$PM_{2.5}$	0.4 (0.0,0.9)	0.0
	1-4	$PM_{2.5}$	0.6 (0.0,1.2)	16.3
	65+	PM_{10}	0.2 (0.0,0.4)	0.0
		$PM_{2.5}$	0.5 (0.2,0.8)	0.0

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

*** PM_{10} - Brisbane, Christchurch, Melbourne, Perth, Sydney; $PM_{2.5}$ – Brisbane, Melbourne, Perth, Sydney*

Table 4.0.2 summarises the associations for long-term exposure (an average of the particle concentration on the same day as the health effect, and all forty days before); the uncertainty confidence intervals are large. The effect estimates for the forty-day exposures are significantly lower than those for the short-term exposures for PM_{10} and cardiovascular mortality. For the other outcomes, there is no statistical difference between the estimates for the two time periods.

Table 4.0.2: Significant forty-day increases in mortality (and 95% confidence intervals) associated with a one-unit ($\mu g.m^{-3}$) increase in 24-hour $PM_{2.5}$ and PM_{10}

Health outcome	Age group (years)	Particle type*	Average lags 0-1 Per unit % increase (95% CI)	40 days Per unit % increase (95% CI)
Cardiovascular	All ages	PM_{10}	0.2 (0.0,0.3)	-0.9 (-1.7, -0.1)
		$PM_{2.5}$	0.4 (0.2,0.6)	-0.3 (-1.5, 0.9)
	75+	PM_{10}	0.2 (0.0,0.4)	-1.0 (-1.9, -0.1)
		$PM_{2.5}$	0.5 (0.2,0.7)	-0.1 (-1.5, 1.3)
Respiratory	75+	$PM_{2.5}$	0.6 (0.0,1.1)	3.0 (-4.6, 11.2)
Total all cause	All ages	$PM_{2.5}$	0.2 (0.1,0.4)	0.3 (-0.3, 0.9)
	75+	$PM_{2.5}$	0.3 (0.1,0.5)	0.4 (-1.2, 2.1)

* *PM_{10} - Brisbane, Christchurch, Melbourne, Perth, Sydney; $PM_{2.5}$ - Brisbane, Melbourne, Perth, Sydney*

4.1 Mortality

4.1.1 Short-term averages for air pollutants

Three different age groups were examined: all ages, 0-74 years, and 75 years and greater. It is clear from Table 4.0.1 that:

- the 24-hour average concentrations of $PM_{2.5}$ are associated with significant increases in mortality for the 75+ years age group due to all causes, cardiovascular disease and respiratory disease, and there is no evidence of heterogeneity between the results for the cities

- the 24-hour average $PM_{2.5}$ concentrations are associated with significant increases in mortality for the all ages group for total mortality and cardiovascular mortality, and there is no evidence of heterogeneity between the results for the cities

- there are also significant increases in mortality due to cardiovascular disease (all ages, and 75+ years) associated with increases in 24-hour average PM_{10}, and there is no evidence of heterogeneity between the results for the cities.

The mortality impacts of particles ($PM_{2.5}$ and PM_{10}) for the short term (average of same day as, and day before death) are shown in Figure 4.1.1 for total deaths (non-accidental, all causes), deaths due to cardiovascular disease and deaths due to

respiratory disease. The results are shown for two age groups: all ages and the oldest (75 years and greater).

Figure 4.1.1: City specific and meta-analysis relative risks (and 95% confidence intervals) for mortality associated with a one-unit increase in particles (average lag 0-1)

Figure 4.1.1.A: Total all cause mortality (all ages)

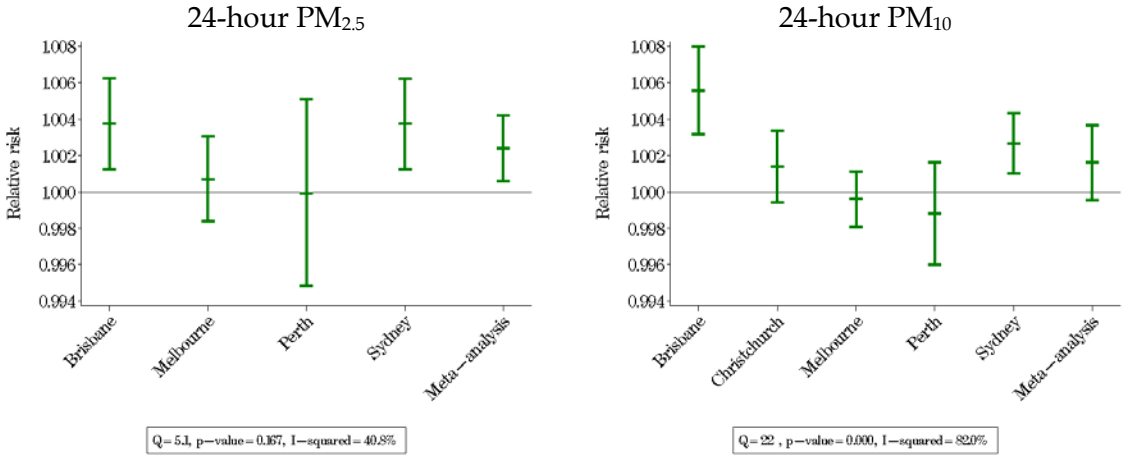


Figure 4.1.1.B: Total all cause mortality (75+ years)

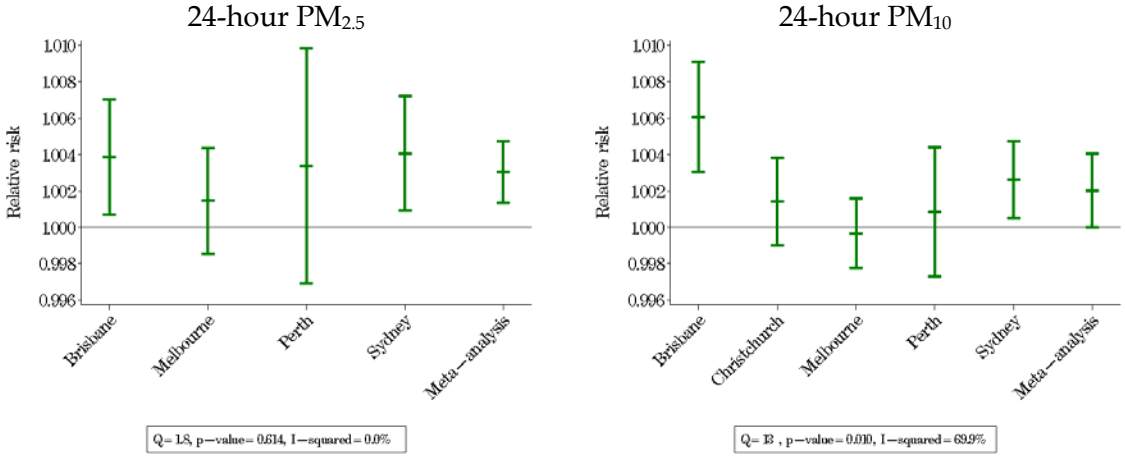


Figure 4.1.1.C: Respiratory mortality (all ages)

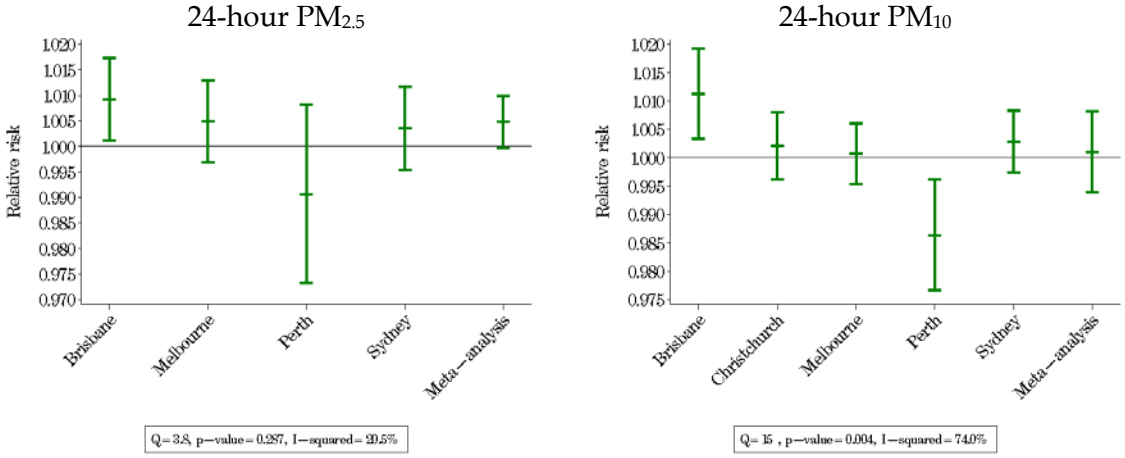


Figure 4.1.1.D: Respiratory mortality (75+ years)

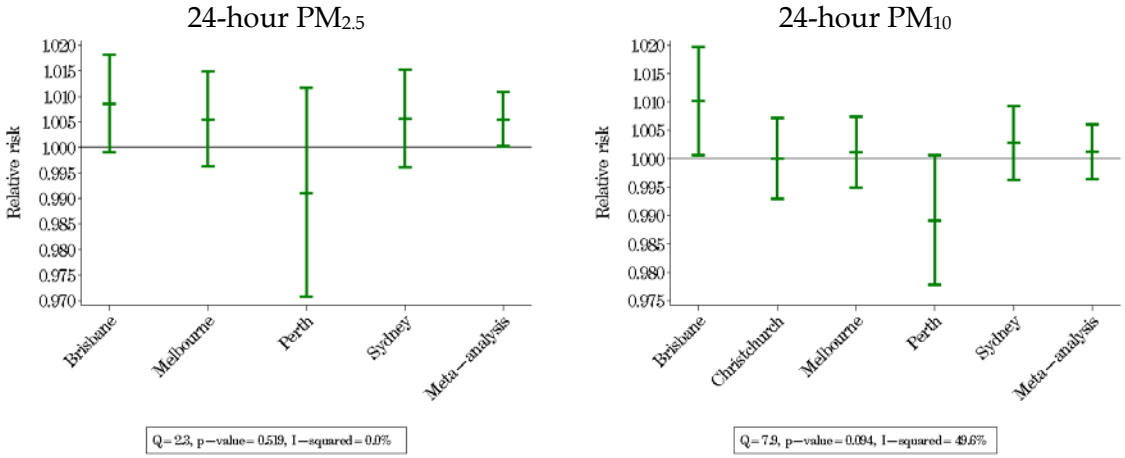


Figure 4.1.1.E: Cardiovascular mortality (all ages)

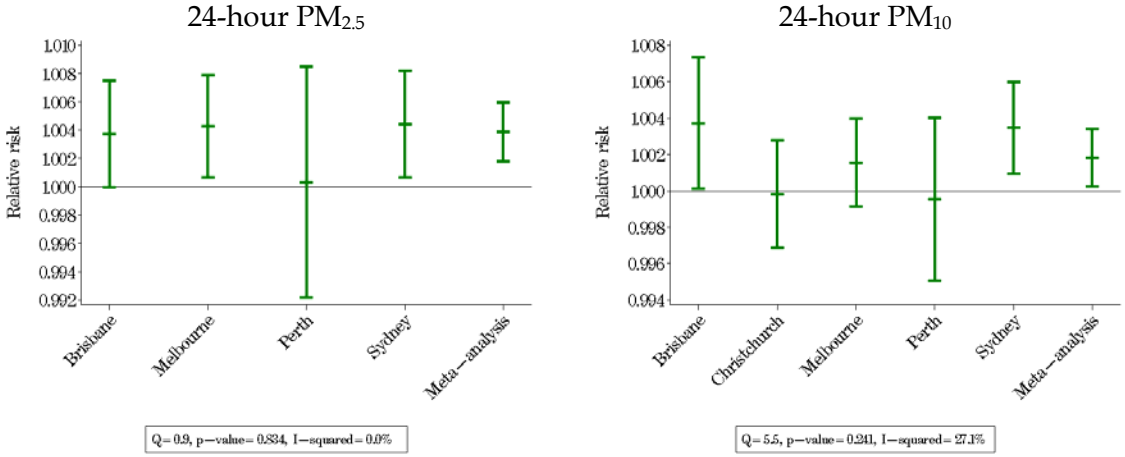
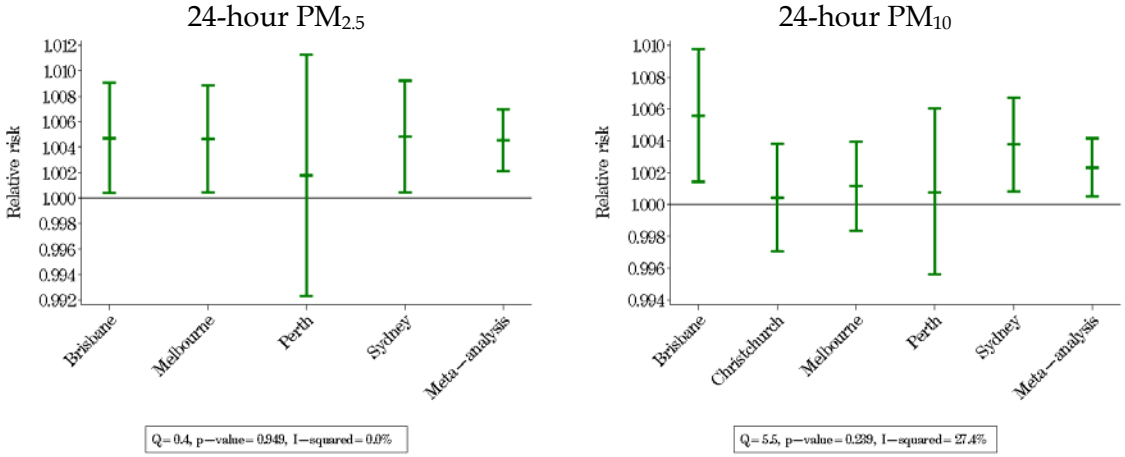


Figure 4.1.1.F: Cardiovascular mortality (75+ years)



The impacts on the overall average increase when each city is removed in turn are shown in Tables 4.1.1 to 4.1.6 for both particle measures for deaths due to cardiovascular disease, deaths due to respiratory disease, and total deaths (non-accidental, all causes), respectively.

The results for $PM_{2.5}$ are generally similar, although Perth is clearly lower for respiratory mortality (see also Figure 4.1.1).

For PM_{10} the pooled estimates for the increases are only significant for cardiovascular mortality. Brisbane and Sydney results appear to dominate in all categories. Perth PM_{10} results appear to be much lower and are negative (protective) for respiratory mortality (see Figures 4.1.1C and 4.1.1D).

Table 4.1.1: Differences between cities using a leave-one-city-out sensitivity analysis for total mortality (75+ years) associated with an IQR increase in particles (average lag 0-1)

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	1.0	0.3,	1.8	0.0	0.8	-0.2,	1.9	28.8
Christchurch	-	-	-	-	1.7	-0.3,	3.7	77.3*
Melbourne	1.5	0.7,	2.3	0.0	2.1	0.5,	3.6	56.6*
Perth	1.1	0.5,	1.8	0.0	1.7	-0.1,	3.6	77.0*
Sydney	1.0	0.2,	1.8	0.0	1.4	-0.5,	3.4	75.7*
Overall	1.2	0.5,	1.8	0.0	1.5	-0.0,	3.1	69.9*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 4.1.2: Differences between cities using a leave-one-city-out sensitivity analysis for total mortality (all ages) associated with an IQR increase in particles (average lag 0-1)

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	0.7	-0.2,	1.6	44.8	0.6	-0.7,	1.8	68.0*
Christchurch	-	-	-	-	1.3	-0.7,	3.3	86.5*
Melbourne	1.3	0.6,	1.9	0.0	1.6	-0.1,	3.4	78.5*
Perth	1.0	0.2,	1.8	51.1*	1.7	-0.1,	3.4	83.7*
Sydney	0.7	-0.2,	1.6	44.5	1.0	-1.0,	3.0	84.6*
Overall	0.9	0.2,	1.6	40.8	1.2	-0.3,	2.8	82.0*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 4.1.3: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory mortality (75+ years) associated with an IQR increase in particles (average lag 0-1)

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	1.6	-0.8,	4.1	0.0	-0.1	-3.4,	3.3	30.4
Christchurch	-	-	-	-	1.0	-4.6,	6.9	61.3*
Melbourne	2.0	-0.6,	4.7	11.7	0.7	-5.0,	6.8	62.1*
Perth	2.5	0.4,	4.6	0.0	2.0	-0.7,	4.8	2.0
Sydney	2.0	-0.6,	4.7	11.6	0.4	-5.2,	6.3	61.0*
Overall	2.1	0.1,	4.2	0.0	0.9	-2.7,	4.7	49.6

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 4.1.4: Differences between cities using a leave-one-city-out sensitivity analysis for respiratory mortality (all ages) associated with an IQR increase in particles (average lag 0-1)

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	1.1	-1.0,	3.3	7.5	-0.9	-5.8,	4.3	66.5*
Christchurch	-	-	-	-	0.5	-6.5,	7.9	80.5*
Melbourne	1.4	-2.0,	4.8	47.0	0.7	-6.3,	8.3	80.2*
Perth	2.3	0.5,	4.1	0.0	2.7	-0.3,	5.7	40.4
Sydney	1.6	-1.7,	5.0	44.6	0.3	-6.6,	7.8	80.3*
Overall	1.8	-0.1,	3.8	20.5	0.8	-4.5,	6.4	74.0*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

Table 4.1.5: Differences between cities using a leave-one-city-out sensitivity analysis for cardiovascular mortality (75+ years) associated with an IQR increase in particles (average lag 0-1)

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	1.7	0.6,	2.8	0.0	1.3	0.1,	2.6	0.0
Christchurch	-	-	-	-	2.1	0.6,	3.7	26.0
Melbourne	1.7	0.6,	2.8	0.0	2.1	0.3,	3.9	34.8
Perth	1.8	0.9,	2.8	0.0	1.9	0.3,	3.6	41.9
Sydney	1.7	0.6,	2.8	0.0	1.4	-0.3,	3.0	27.9
Overall	1.7	0.8,	2.7	0.0	1.8	0.4,	3.2	27.4

Table 4.1.6: Differences between cities using a leave-one-city-out sensitivity analysis for cardiovascular mortality (all ages) associated with an IQR increase in particles (average lag 0-1)

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	1.5	0.6,	2.5	0.0	1.1	-0.2,	2.4	30.3
Christchurch	-	-	-	-	1.8	0.6,	3.0	6.7
Melbourne	1.4	0.4,	2.4	0.0	1.4	-0.3,	3.1	44.5
Perth	1.6	0.8,	2.4	0.0	1.6	0.3,	2.9	31.4
Sydney	1.4	0.4,	2.4	0.0	0.9	-0.3,	2.2	9.5
Overall	1.5	0.7,	2.3	0.0	1.4	0.2,	2.6	27.1

4.1.2 'Harvesting'?

The concept of harvesting pertains to the phenomenon whereby an increase in air pollution on a particular day brings forward the death of frail individuals expected to die in the short term. Evidence of this can be found by examining whether there is a short-term (few days) increase in deaths, followed by a compensating reduction in mortality within the frail population over the succeeding period. This has been proposed to occur particularly with particulate matter.

If harvesting occurs, therefore, we expect it to be most evident in the elderly in the population, the 75 years and older age group. We thus compare the effect estimates for the short-term to those derived from a distributed lag model, taken over 40 days.

The purpose of the latter is to identify a negative or compensating effect of positive effects of the former.

Figures 4.1.1.B, 4.1.1.D, and 4.1.1.F show that there are significant increases in all cause mortality, respiratory mortality, and cardiovascular mortality in 75+ year olds of around 0.3-0.5% per $\mu g.m^{-3}$ of $PM_{2.5}$. However, the 40-day distributed lag model shows no evidence of a decline in mortality, all cause or specific, suggesting that harvesting is not occurring for $PM_{2.5}$.

This contrasts with the results for PM_{10}, for which we see short-term effects of around 0.2% % per $\mu g.m^{-3}$ of PM_{10} for all cause and cardiovascular, with no effect seen for respiratory deaths. The 40-day model for total mortality shows a small negative effect (-0.3%, 95% CI (-4.7%, 4.3%)) with a wide confidence interval covering zero effect (Table 4.1.7). This suggests the short-term increase might be compensated for by a longer-term decrease. The wide confidence limits (due to the large number of parameters incorporated in the 40-day model) do, however, make interpretation difficult. For respiratory mortality and PM_{10}, the question of harvesting does not arise since the effect estimate for the short-term effect is virtually zero: no short-term increase in deaths occurs. For cardiovascular mortality, the data are somewhat more convincing. The 4-day model shows a decline of 1.0%, 95% CI (-1.9%, -0.1%), which falls just short of significance, the confidence interval just excluding zero effect. It is noteworthy that effect estimates are consistently negative across all cities, except Christchurch. This suggests that the 0.5% short-term mortality increase may be counterbalanced by a longer-term decrease.

Thus, it is concluded that there is some evidence for the 'harvesting' hypothesis in the results for PM_{10} and cardiovascular mortality. This may merit further detailed analyses, although conclusions will inevitably be limited by the relatively small numbers.

Although the I-squared statistic indicates that the harvesting results are the same for all cities, the results for the individual cities are shown in Tables 4.1.7 to 4.1.9 for the greater than 75 years age group for total mortality (all causes), respiratory mortality and cardiovascular mortality, respectively.

Even though the estimate for Christchurch is significant and positive for total mortality, the wide confidence intervals do not allow a differentiation of this estimate from the short-term average.

Table 4.1.7: Per cent changes (and 95% confidence interval) in total mortality (75+ years) over 40-day periods* associated with particles

	24-hour $PM_{2.5}$			24-hour PM_{10}		
City	Inc	95%CI		Inc	95%CI	
Brisbane	-0.9	-2.2,	0.5	-0.5	-1.7,	0.6
Christchurch	-	-	-	20.8	0.6,	44.9
Melbourne	3.1	-2.7,	9.3	-4.1	-9.6,	1.6
Perth	2.6	-0.5,	5.8	-0.0	-1.6,	1.6
Sydney	0.3	-1.4,	2.1	-1.1	-2.0,	-0.1
I-squared (%)	45.9	-	-	44.0	-	-
Meta-analysis	0.4	-1.2,	2.1	-0.3	-4.7,	4.3
Meta-analysis IQR	1.7	-4.4,	7.8	-2.0	-35.0,	32.6

Table 4.1.8: Per cent changes (and 95% confidence interval) in respiratory mortality (75+ years) over 40-day periods* associated with particles

	24-hour $PM_{2.5}$			24-hour PM_{10}		
City	Inc	95%CI		Inc	95%CI	
Brisbane	-5.9	-9.7,	-2.0	-2.3	-5.8,	1.3
Christchurch	-	-	-	35.4	-20.7,	131.4
Melbourne	13.4	-5.6,	36.2	-11.1	-26.0,	6.7
Perth	7.9	-2.2,	19.1	2.1	-3.1,	7.5
Sydney	5.7	-0.1,	11.8	-0.9	-3.9,	2.2
I-squared (%)	81.2	-	-	11.4	-	-
Meta-analysis	3.0	-4.6,	11.2	-0.9	-3.5,	1.7
Meta-analysis IQR	11.2	-17.5,	42.2	-6.9	-26.1,	12.9

Table 4.1.9: Per cent changes (and 95% confidence interval) in cardiovascular mortality (75+ years) over 40-day periods* associated with particles

	24-hour $PM_{2.5}$			24-hour PM_{10}		
City	Inc	95%CI		Inc	95%CI	
Brisbane	-0.3	-2.2,	1.6	-0.7	-2.4,	0.9
Christchurch	-	-	-	11.0	-12.7,	41.1
Melbourne	0.8	-7.2,	9.5	-4.4	-12.0,	4.0
Perth	1.8	-2.7,	6.4	-0.2	-2.5,	2.1
Sydney	-0.5	-2.9,	2.0	-1.4	-2.7,	-0.0
I-squared (%)	0.0	-	-	0.0	-	-
Meta-analysis	-0.1	-1.5,	1.3	-1.0	-1.9,	-0.1
Meta-analysis IQR	-0.5	-5.7,	4.8	-7.5	-14.5,	-0.4

* Estimates from a random effects meta-analysis of cities using distributed lag models, individual city results are shown for a one-unit increase in pollutant and meta-analysis results are shown for an inter-quartile range (IQR) increase where indicated.

The results in Table 4.1.10 show results for the total mortality outcome when each city is left out in turn. As noted before, the PM_{10} results for Christchurch dominate the results for all cause mortality.

Table 4.1.10: Differences between cities using a leave-one-city-out sensitivity analysis for 40-day changes in total mortality (75+ years) associated with an IQR increase in particles

	24-hour $PM_{2.5}$				24-hour PM_{10}			
City left out	Inc	95%CI		I2	Inc	95%CI		I2
Brisbane	4.0	-1.7,	10.1	1.7	4.7	-40.4,	84.0	57.1*
Christchurch	-	-	-	-	-5.6	-10.2,	-0.7	0.0
Melbourne	1.1	-5.4,	8.1	54.9*	7.7	-31.2,	68.6	48.0
Perth	-0.9	-5.4,	3.8	19.9	3.6	-41.2,	82.5	51.9*
Sydney	3.1	-6.2,	13.3	62.4*	5.8	-39.4,	84.7	51.6*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

4.1.3 Seasonal differences

Individual city seasonal results for mortality are shown in *Volume 3: Appendix A3.4*, Tables WC.30 to WC.38. Meta-analysis and results from the leave-one-city-out analysis for mortality are shown in *Volume 3: Appendix A3.5*, Tables WCL.30 to WCL.38.

It might be expected that some pollutant effects may be different in different seasons, For example, ozone peaks during the summer smog episodes in general (although Brisbane may also have winter smog events) and this will lead to an increase in the photochemical aerosol load. Ambient particle concentrations also rise during smog events but there are many sources (home heating, vehicle emissions) that may also peak during winter.

Table 4.1.11 shows the results in different warm and cool periods for the associations of particle concentrations with cardiovascular, respiratory and total mortality:

- for both $PM_{2.5}$ and PM_{10}, there are only significant pooled estimates for the cool periods
- for $PM_{2.5}$ in the cool period there are significant pooled estimates for the associations with increases in respiratory mortality (75+ years), total mortality (all ages, 75+ years), and cardiovascular mortality (all ages, 75+ years)
- for PM_{10} in the cool period there are significant pooled estimates for the associations with increases in total mortality (all ages, 75+ years) and cardiovascular mortality (all ages, 75+ years).

Table 4.1.11: Significant seasonal increases in mortality associated with an IQR increase in particles (average lag 0-1) (case-crossover meta-analysis)

		24-hour $PM_{2.5}$			24-hour PM_{10}		
		Inc	95%CI		Inc	95%CI	
Total all cause (75+ yrs)	cool	1.3	0.6,	2.1	2.0	0.9,	3.1
	warm	0.2	-2.1,	2.5	0.1	-2.3,	2.7
Total all cause (all ages)	cool	1.0	0.4,	1.7	1.6	0.3,	2.8
	warm	0.1	-2.0,	2.2	-0.0	-2.3,	2.4
Respiratory (75+ yrs)	cool	2.4	0.0,	4.8	1.4	-1.8,	4.7
	warm	-0.3	-4.8,	4.3	-7.6	-24.6,	13.3
Cardiovascular (75+ yrs)	cool	1.6	0.5,	2.7	2.2	0.6,	3.9
	warm	0.7	-1.3,	2.7	-0.3	-2.3,	1.8
Cardiovascular (all ages)	cool	1.4	0.4,	2.3	1.8	0.1,	3.6
	warm	0.5	-1.2,	2.3	-0.5	-2.3,	1.3

4.2 Hospital admissions

4.2.1 Hospital admissions due to cardiovascular disease

The following categories for cardiovascular admissions were examined in this study (see Table 1.2 for the ICD codes):

- total cardiovascular disease
- stroke
- cardiac disease
- ischemic heart disease
- myocardial infarction
- cardiac failure
- arrhythmia.

In the following sections, the associations between admissions for two age groups (15-64 years, 65 years and greater) and ambient concentrations of 24-hour average $PM_{2.5}$ and PM_{10} in the short term (average of the same day and day before – lag [0,1]) are presented for these health outcomes.

4.2.1.1 Cardiovascular disease

The associations with total cardiovascular admissions are shown in Figures 4.2.1 and 4.2.2. There are only significant estimates for the pooled estimates for $PM_{2.5}$, and only then for the older age group with no evidence for heterogeneity between the city results (confirmed by the statistical tests).

Figure 4.2.1: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiovascular admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

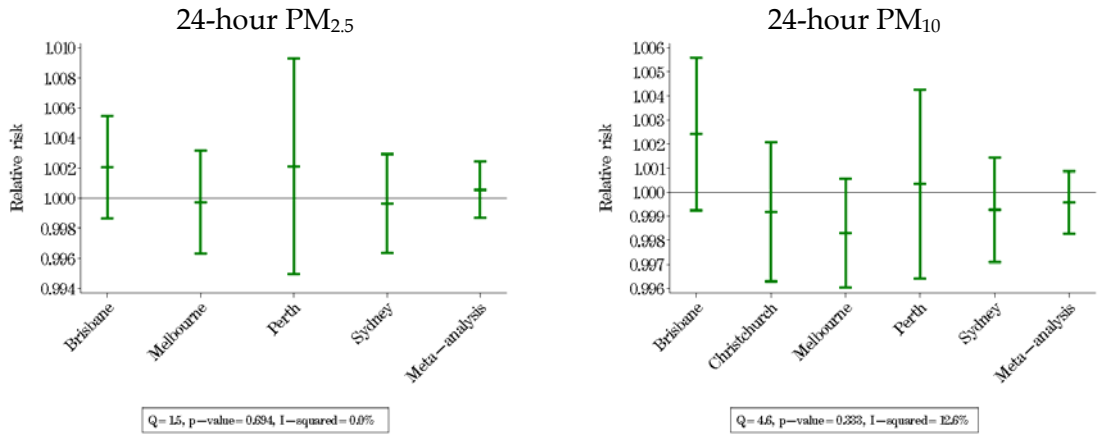
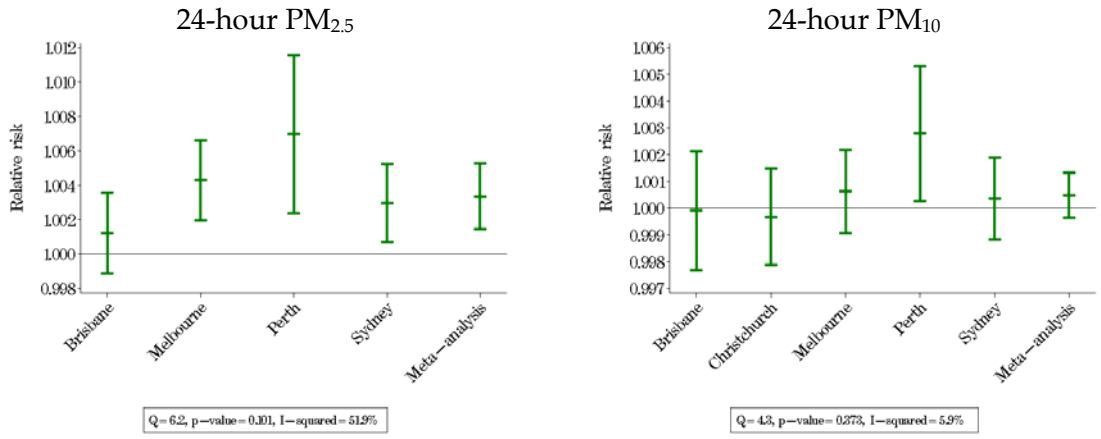


Figure 4.2.2: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiovascular admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.1.2 Stroke

The associations with hospital admissions due to stroke are shown in *Volume 3: Appendix A3.1*, Tables CM.5 to CM.6. There were no significant associations.

4.2.1.3 Cardiac admissions

The associations with cardiac admissions are shown in Figures 4.2.3 and 4.2.4, respectively. There are significant estimates for the pooled estimates for both $PM_{2.5}$ and PM_{10} only for the older age group, with no evidence for heterogeneity between the city results (confirmed by the statistical tests).

Figure 4.2.3: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

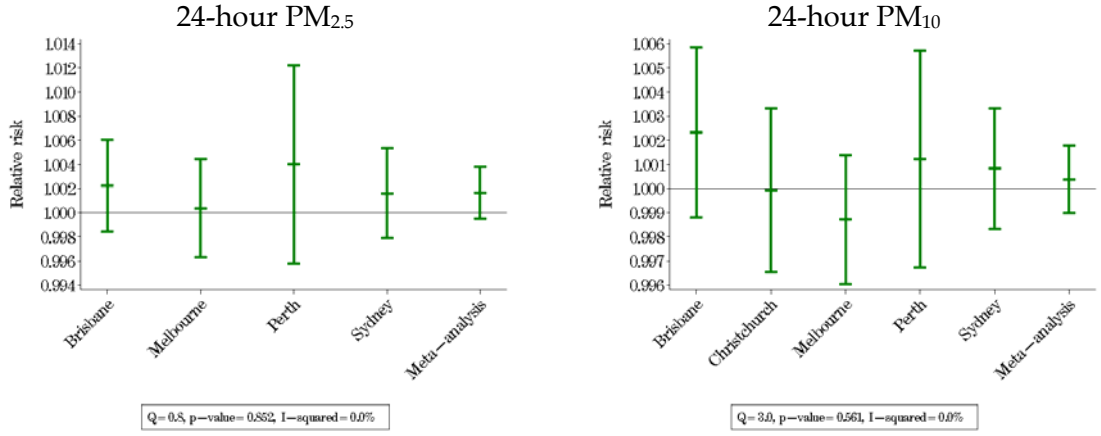
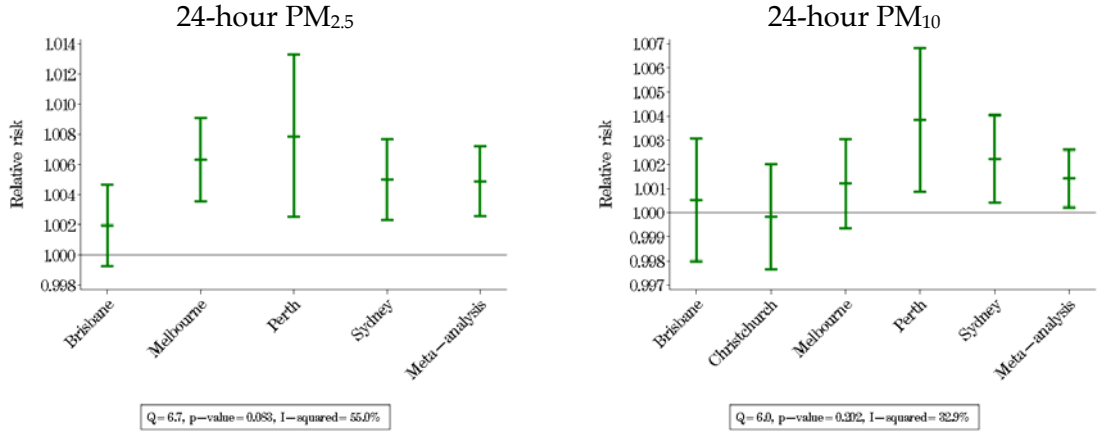


Figure 4.2.4: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.1.4 Ischemic heart disease

The associations with hospital admissions due to ischemic heart disease are shown in Figures 4.2.5 and 4.2.6. There are significant estimates for the pooled estimates only for $PM_{2.5}$ and then only for the older age group, with no evidence for heterogeneity between the city results (confirmed by the statistical tests).

Figure 4.2.5: City specific and meta-analysis relative risks (and 95% confidence intervals) for IHD admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

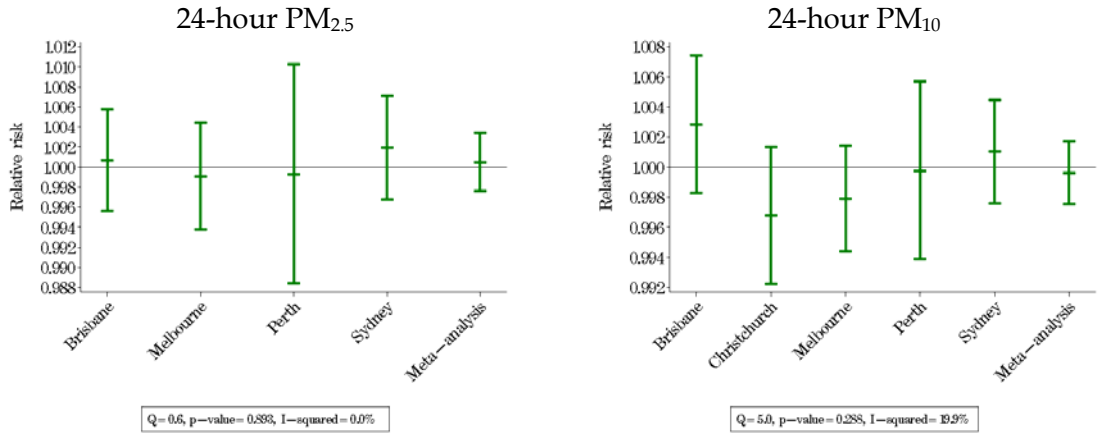
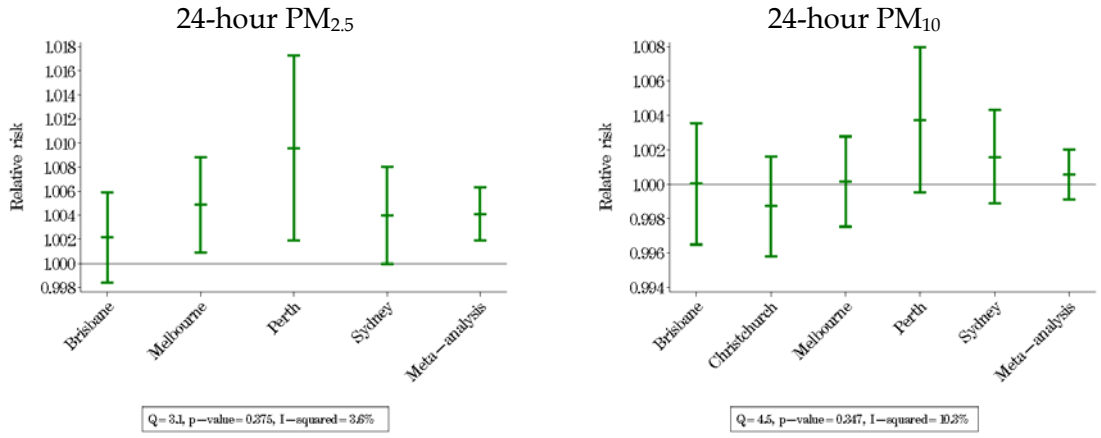


Figure 4.2.6: City specific and meta-analysis relative risks (and 95% confidence intervals) for IHD admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.1.5 Myocardial infarction

The associations with hospital admissions due to myocardial infarction are shown in Figures 4.2.7 and 4.2.8. There are significant estimates for the pooled estimates only for $PM_{2.5}$ and then only for the older age group, with no evidence for heterogeneity between the city results (confirmed by the statistical tests).

Figure 4.2.7: City specific and meta-analysis relative risks (and 95% confidence intervals) for myocardial infarction admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

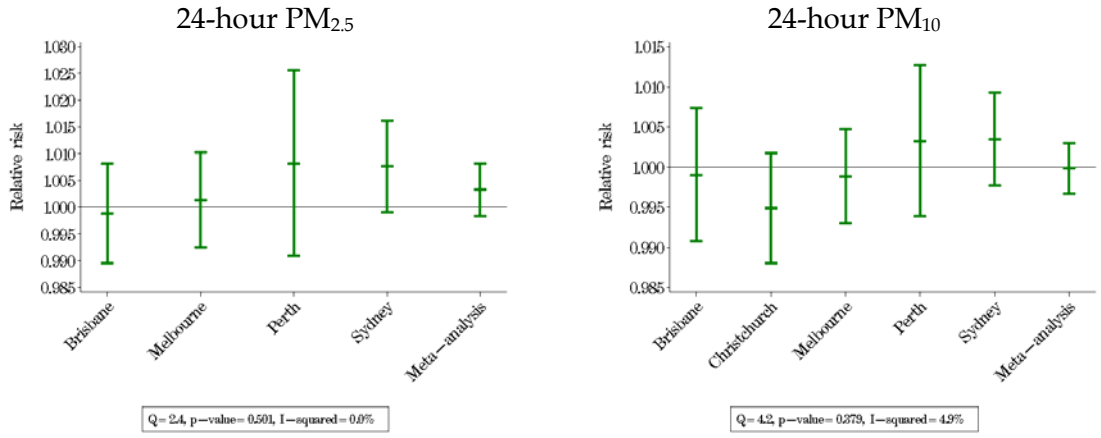
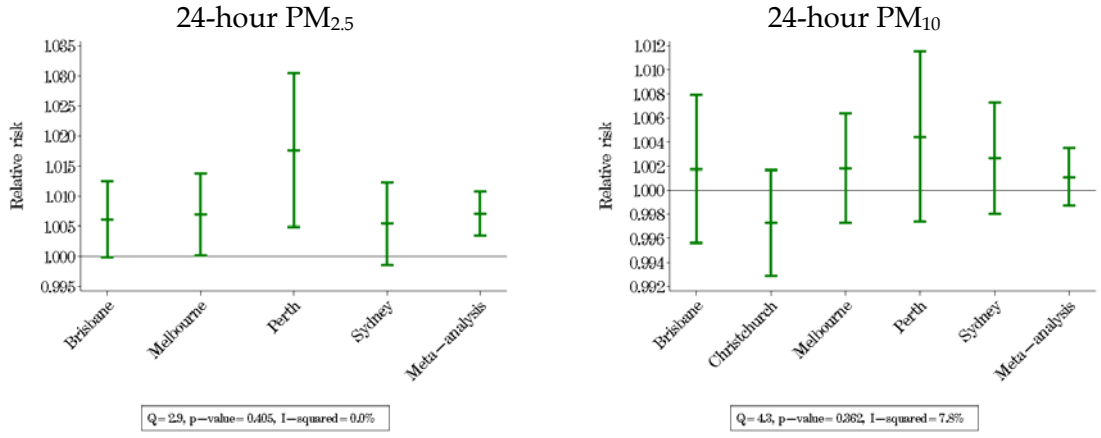


Figure 4.2.8: City specific and meta-analysis relative risks (and 95% confidence intervals) for myocardial infarction admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.1.6 Cardiac failure

The impact of particles on hospital admissions due to cardiac failure for two age groups (15-64 years, 65 years and greater) for the short term (lag 0-1) are shown in Figures 4.2.9 and 4.2.10. There are significant estimates for the pooled estimates for both $PM_{2.5}$ and PM_{10} only for the older age group, with no evidence for heterogeneity between the city results (confirmed by the statistical tests).

Figure 4.2.9: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac failure admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

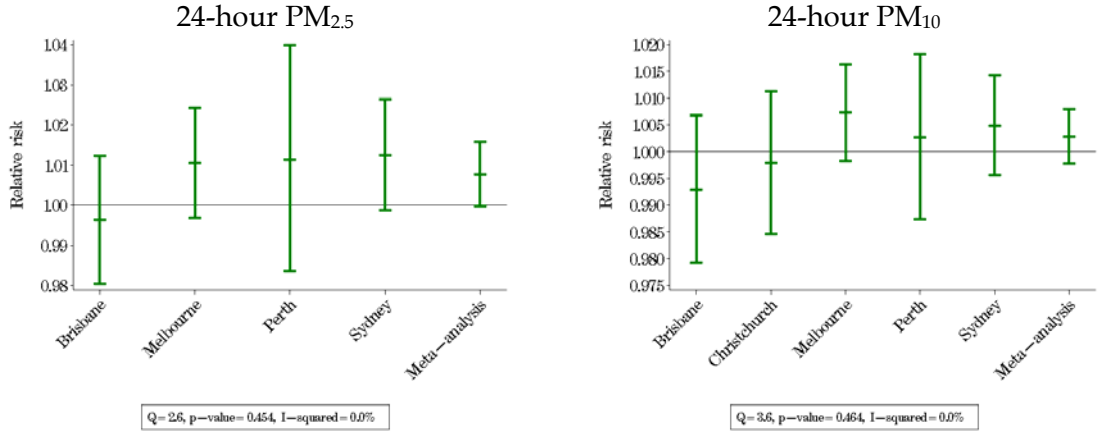
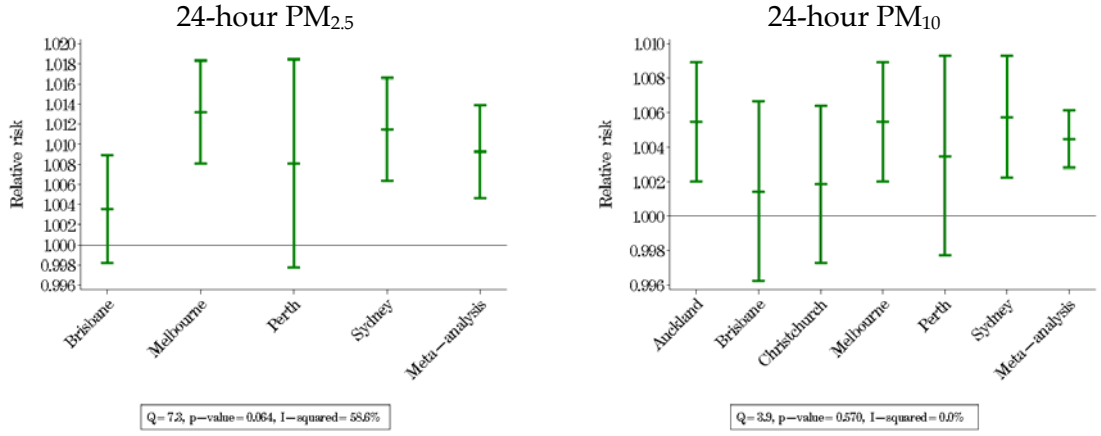


Figure 4.2.10: City specific and meta-analysis relative risks (and 95% confidence intervals) for cardiac failure admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.1.7 Arrhythmia

The associations with hospital admissions due to arrhythmia are shown in Figures 4.2.11 and 4.2.12. There are no significant associations.

Figure 4.2.11: City specific and meta-analysis relative risks (and 95% confidence intervals) for arrhythmia admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

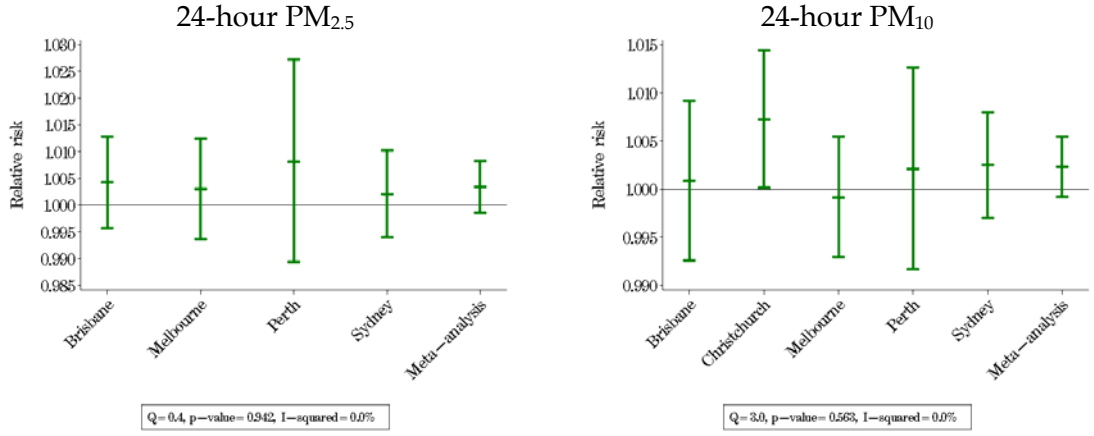
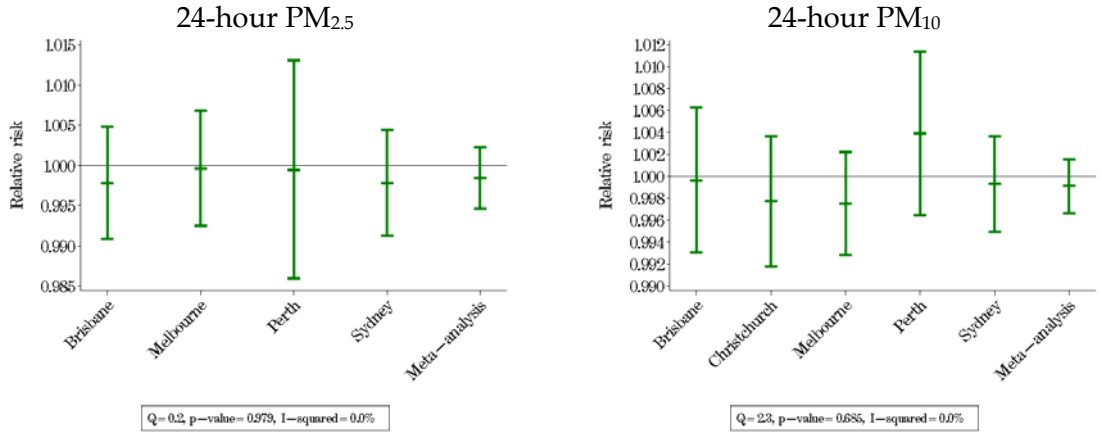


Figure 4.2.12: City specific and meta-analysis relative risks (and 95% confidence intervals) for arrhythmia admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.1.8 Seasonal differences

Separate analyses were carried out for the warm (November–April) and cool (May–October) periods to identify if there are different effects in each period (due to different emission patterns, atmospheric chemistry). A complete set of individual city seasonal results for cardiovascular admissions is shown in *Volume 3: Appendix A3.4*, Tables WC.1 to WC.14. A complete set of meta-analysis results and results from the leave-one-city-out analysis for cardiovascular admissions are shown in *Volume 3: Appendix A3.5*, Tables WCL.1 to WCL.14.

Table 4.2.1 shows the significant meta-analysis results for cool and warm periods, and there are clearly significant cool period associations (especially for $PM_{2.5}$), and often evidence for heterogeneity in the results. This heterogeneity is arising because the Brisbane results are often showing a significant warm period association only.

Table 4.2.1: Significant seasonal increases in cardiovascular admissions associated with an IQR increase in particles (average lag 0-1) (case-crossover meta-analysis)

Admissions (age group)		24-hour $PM_{2.5}$				24-hour PM_{10}			
		Inc	95%CI		I2	Inc	95%CI		I2
Cardiac (65+ yrs)	cool	2.3	0.6,	4.0	81.5*	1.6	-0.3,	3.4	71.7*
	warm	1.9	-0.4,	4.3	71.8*	1.1	-1.1,	3.4	64.3*
IHD (65+ yrs)	cool	1.8	0.4,	3.2	40.7	0.8	-1.4,	3.0	58.5
	warm	1.7	-0.6,	4.2	47.3	0.5	-1.9,	3.0	42.3
Cardiac failure (65+ yrs)	cool	4.0	1.0,	7.0	78.3*	4.5	1.8,	7.2	47.6
	warm	2.2	-0.3,	4.7	0.0	0.3	-2.3,	3.0	0.0
Myocardial infarction (65+ yrs)	cool	2.3	0.6,	4.1	0.0	0.1	-2.5,	2.8	25.4
	warm	2.2	-0.7,	5.2	0.0	0.5	-2.5,	3.7	0.0
Total cardiovascular (65+ yrs)	cool	1.8	0.3,	3.4	83.6*	1.2	-0.3,	2.7	69.0*
	warm	1.5	-1.7,	4.8	86.7*	-0.2	-2.3,	2.0	72.7*

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

4.2.2 Hospital admissions due to respiratory disease

The respiratory disease groups considered included (see Table 1.2 for the ICD codes):

- total respiratory disease
- asthma
- chronic obstructive pulmonary disease (COPD)
- pneumonia and acute bronchitis.

The hospital admissions analyses were stratified by age groups as appropriate for the diseases:

- children age groups
  - less than one year of age
  - 1-4 years
  - 5-14 years
- 15-64 years
- greater or equal to 65 years.

The following sections present the associations between these health outcomes and short-term (lag 0-1) ambient concentrations of average 24-hour $PM_{2.5}$ and PM_{10}.

4.2.2.1 Total respiratory disease

The associations with hospital admissions for all respiratory disease for all age groups are shown in Figures 4.2.13 to 4.2.17. These results can be summarised as follows:

- there are overall (statistically significant) increases in admissions for children in the age groups less than 1 year, and 1-4 years, for both $PM_{2.5}$ and PM_{10}, with no evidence for heterogeneity between results for the cities (as shown by the statistical tests)

- there are overall (statistically significant) increases in admissions for children in the age groups 5-14 years for PM_{10}, with no evidence for heterogeneity between results for the cities

- there are overall (statistically significant) increases for the adult age groups (15-64 years, and 65 years and greater) for $PM_{2.5}$, with no evidence for heterogeneity between results for the cities.

Figure 4.2.13: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (0 years) associated with a one-unit increase in particles (average lag 0-1)

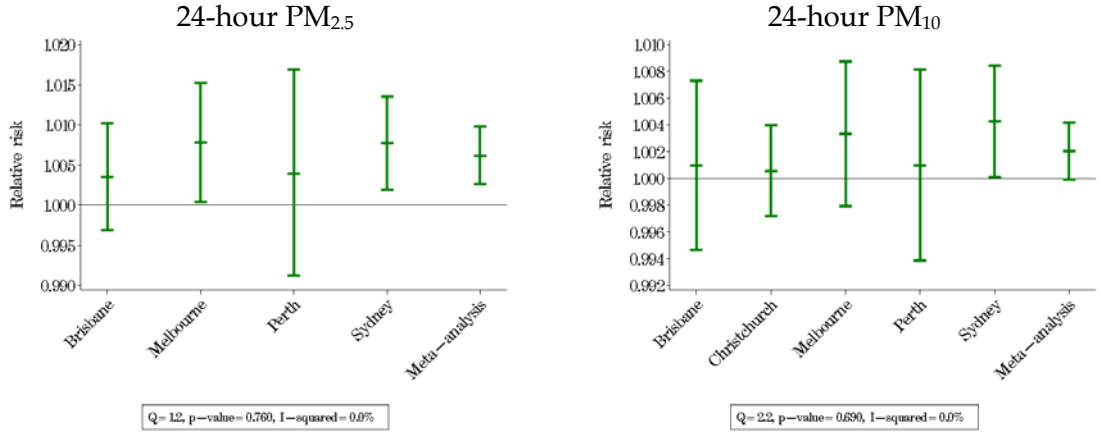


Figure 4.2.14: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (1-4 years) associated with a one-unit increase in particles (average lag 0-1)

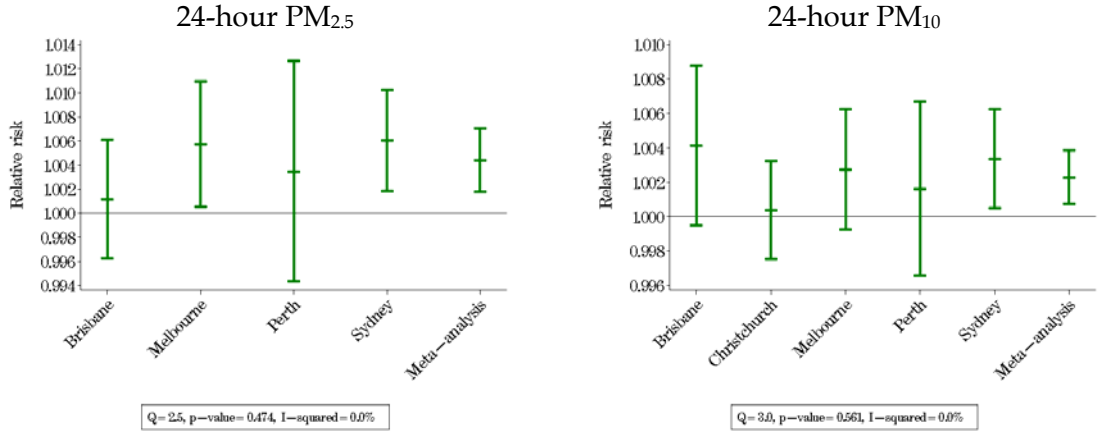


Figure 4.2.15: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (5-14 years) associated with a one-unit increase in particles (average lag 0-1)

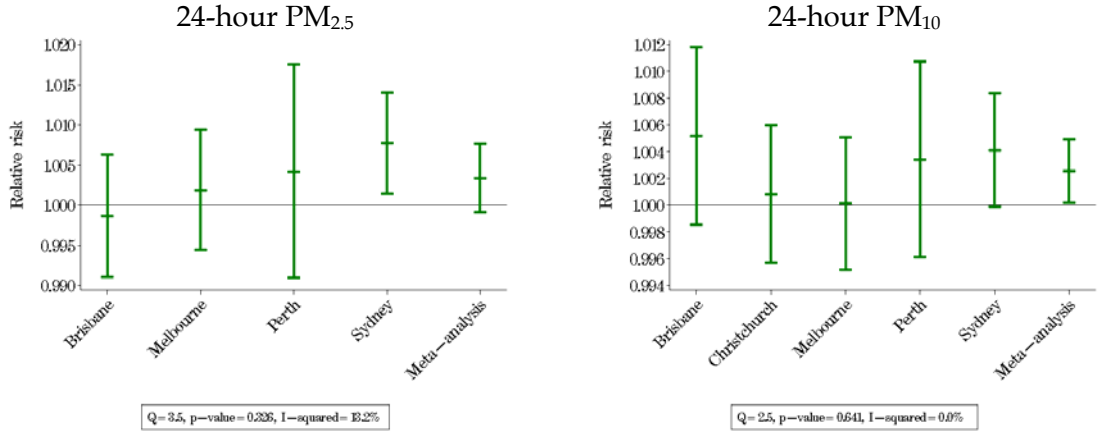


Figure 4.2.16: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

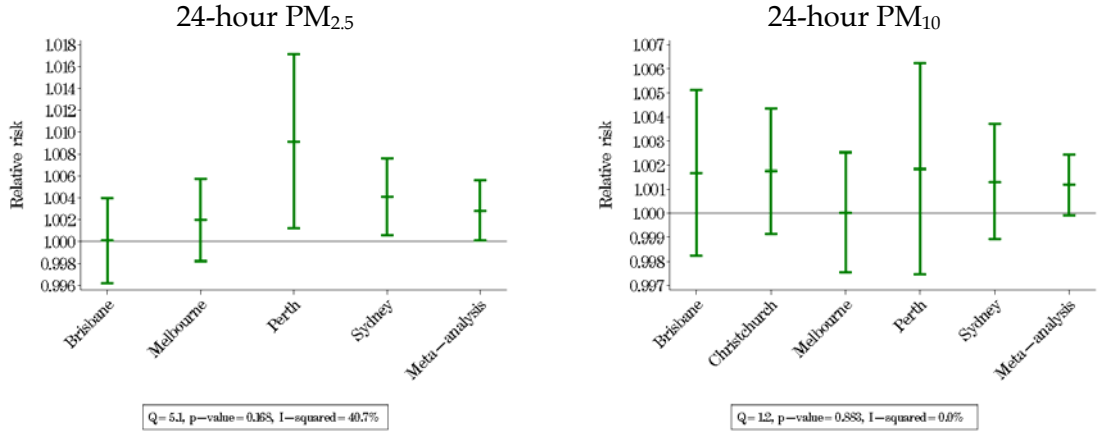
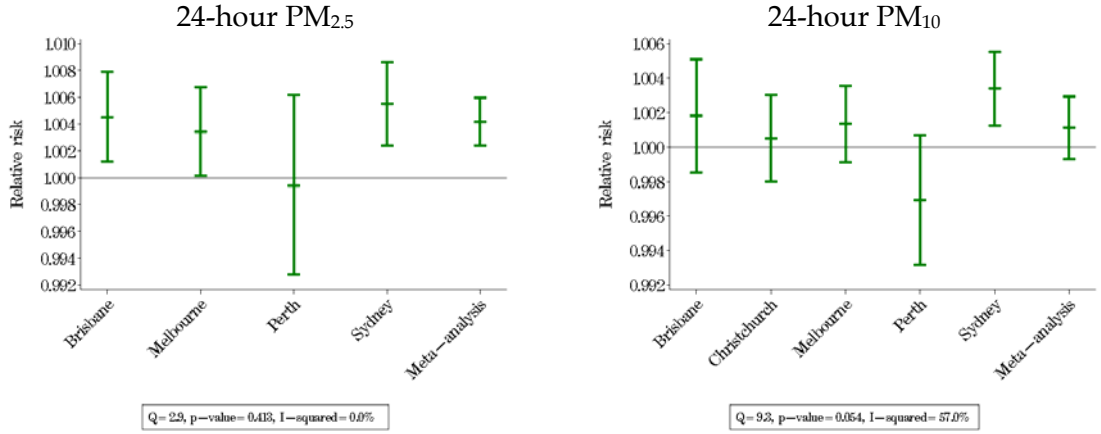


Figure 4.2.17: City specific and meta-analysis relative risks (and 95% confidence intervals) for respiratory admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.2.2 Asthma

The associations with hospital admissions for asthma for all age groups except the youngest (less than 1 year, where it is difficult to diagnose asthma) are shown in Figures 4.2.18 to 4.2.21. There are significant results found for the pooled estimates only for $PM_{2.5}$ and only for the 15-64 years age group, with no evidence for heterogeneity between results for the cities.

Figure 4.2.18: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (1-4 years) associated with a one-unit increase in particles (average lag 0-1)

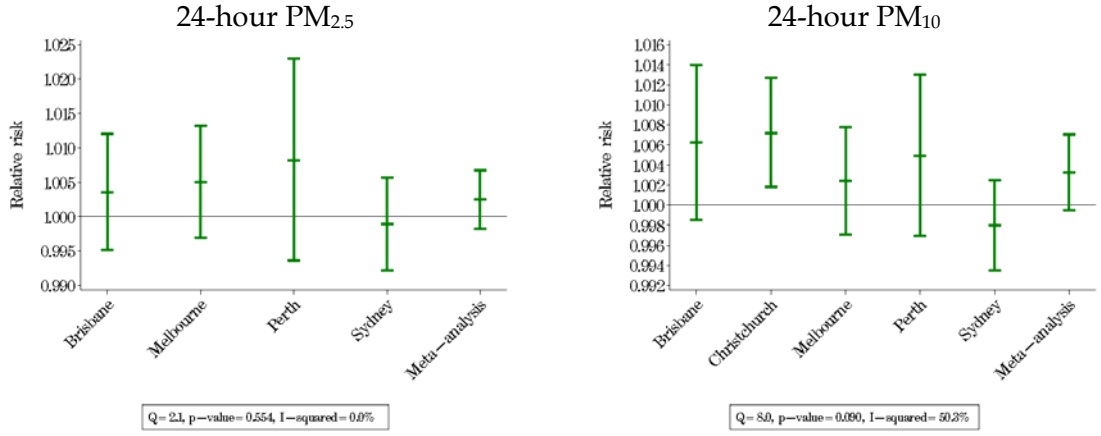


Figure 4.2.19: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (5-14 years) associated with a one-unit increase in particles (average lag 0-1)

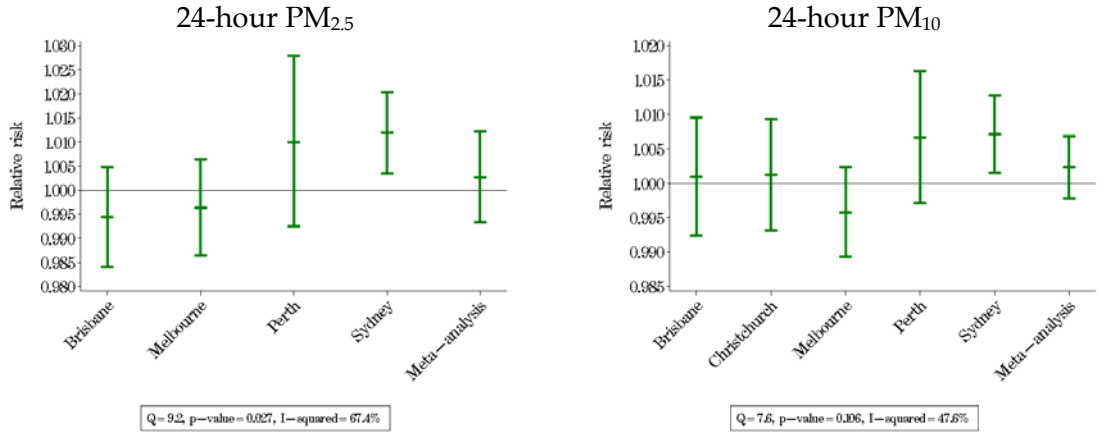


Figure 4.2.20: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

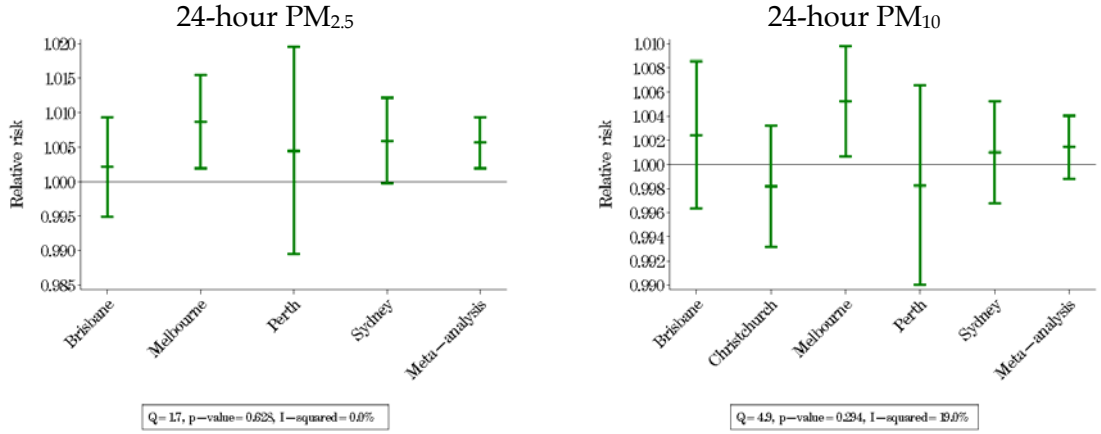
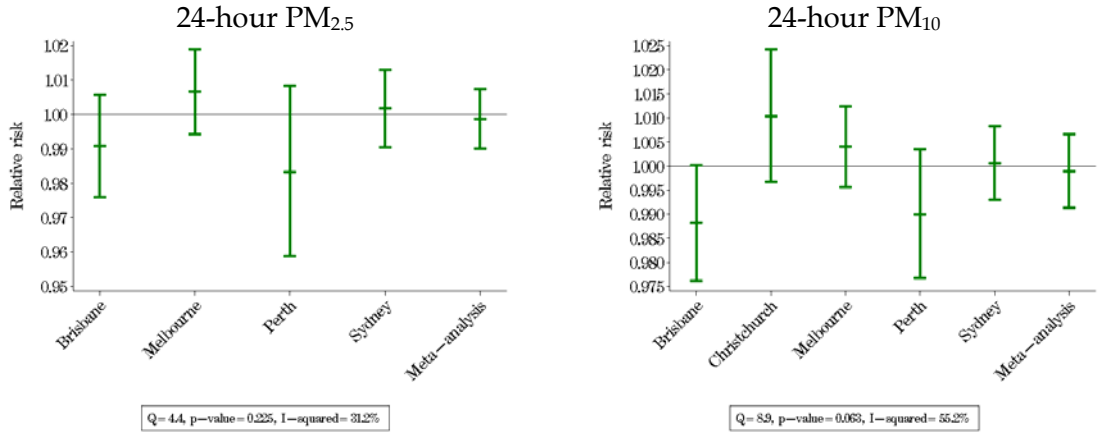


Figure 4.2.21: City specific and meta-analysis relative risks (and 95% confidence intervals) for asthma admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.2.3 Chronic obstructive pulmonary disease (COPD)

The associations with hospital admissions for COPD for adults:15-64 years age group, and 65 years and greater age group, are shown in Figure 4.2.22 and 4.2.23 (there is little COPD in children). For $PM_{2.5}$ exposure, there were overall increases for COPD admissions for the 65 years and greater age group (statistically significant).

Figure 4.2.22: City specific and meta-analysis relative risks (and 95% confidence intervals) for COPD admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

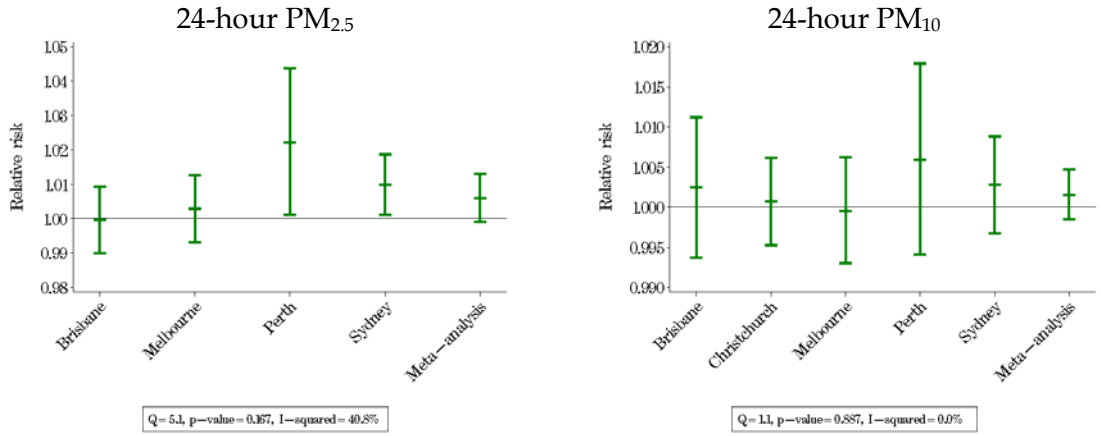
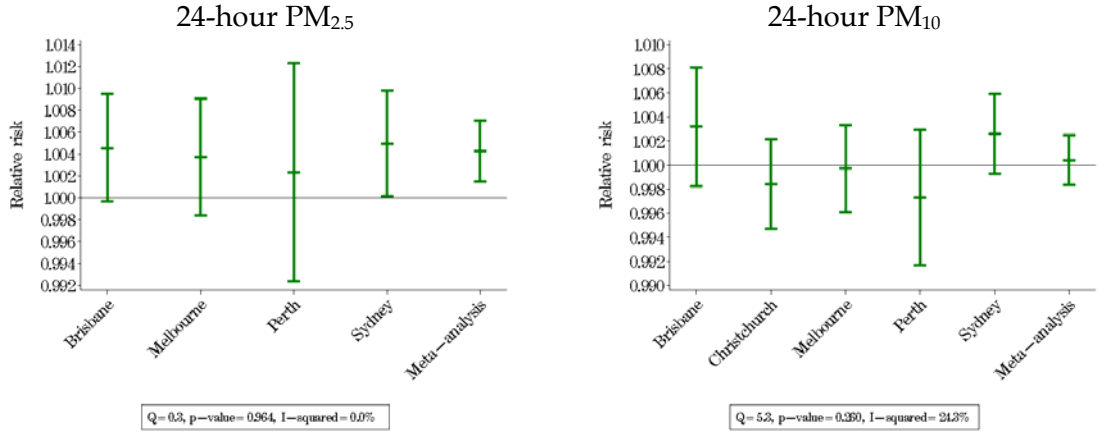


Figure 4.2.23: City specific and meta-analysis relative risks (and 95% confidence intervals) for COPD admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.2.4 Pneumonia and acute bronchitis

The associations with hospital admissions for pneumonia and acute bronchitis for all age groups except the 5-14 years group (events are very few) are shown in Figures 4.2.24 to 4.2.27. These results can be summarised as follows:

- there are overall (statistically significant) increases in admissions for pre-school children associated with $PM_{2.5}$, with no evidence for heterogeneity between results for the cities (as shown by the statistical tests)

- there are overall positive (and statistically significant) increases on hospital admissions for the elderly (65 years and greater age group) for exposure to $PM_{2.5}$ and PM_{10}, with no evidence for heterogeneity between results for the cities.

Figure 4.2.24: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (0 years) associated with a one-unit increase in particles (average lag 0-1)

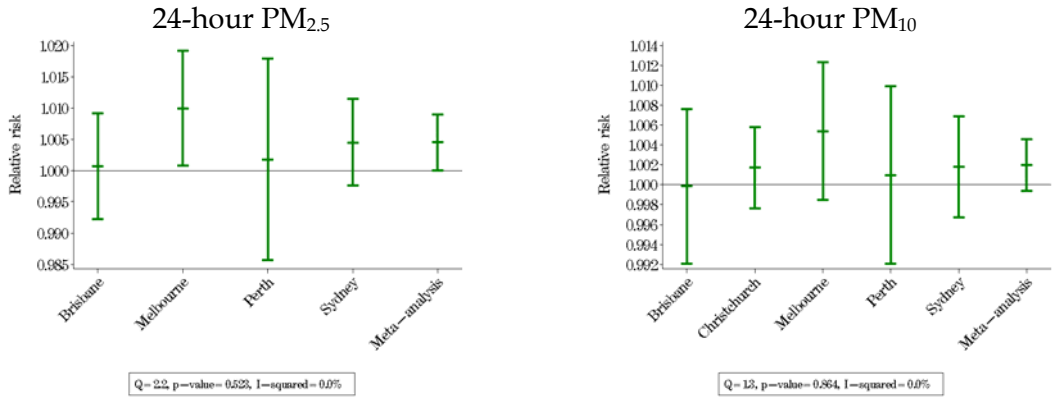


Figure 4.2.25: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (1-4 years) associated with a one-unit increase in particles (average lag 0-1)

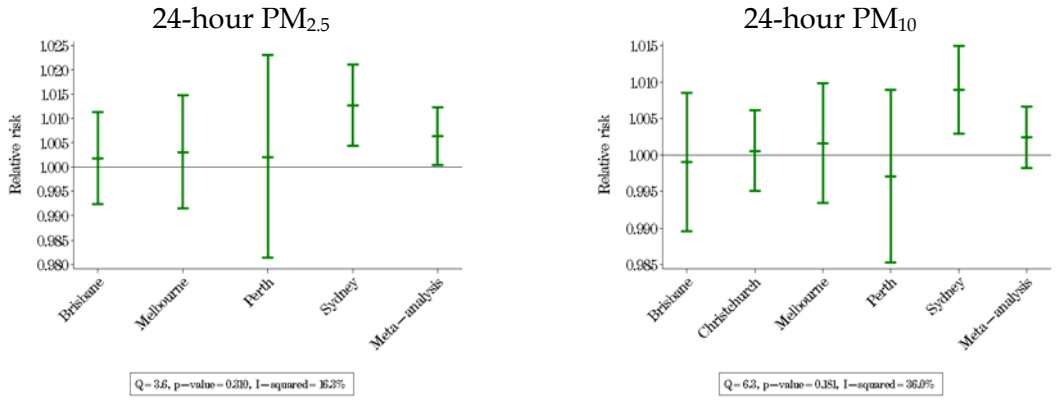


Figure 4.2.26: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (15-64 years) associated with a one-unit increase in particles (average lag 0-1)

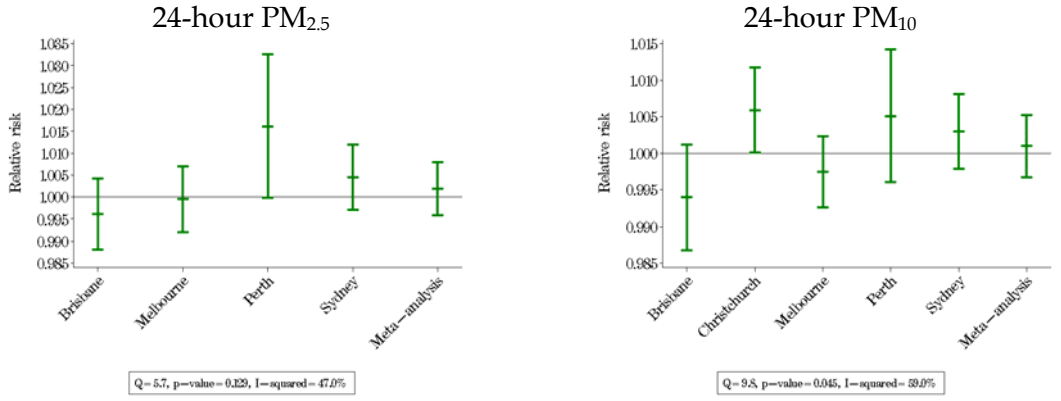
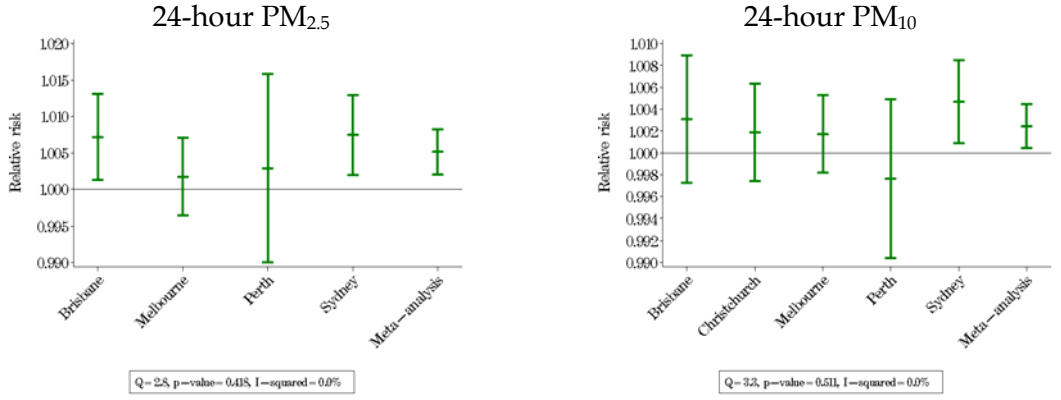


Figure 4.2.27: City specific and meta-analysis relative risks (and 95% confidence intervals) for pneumonia and acute bronchitis admissions (65+ years) associated with a one-unit increase in particles (average lag 0-1)



4.2.2.5 Seasonal differences

Separate analyses were carried out for the warm (November–April) and cool (May–October) periods to identify if there are different effects in each period (due to different emission patterns, atmospheric chemistry). A complete set of individual city seasonal results for respiratory admissions are shown in *Volume 3: Appendix A3.4*, Tables WC.15 to WC.29. A complete set of meta-analysis results and results from the leave-one-city-out analysis for respiratory admissions are shown in *Volume 3: Appendix A3.5*, Tables WCL.15 to WCL.29.

Table 4.2.2 shows the meta-analysis results for cool and warm periods. There are clearly significant cool period associations for $PM_{2.5}$ for asthma (15-64 years), COPD (65+ years), pneumonia and acute bronchitis (65+ years), total respiratory (less than 1 year, 1-4 years, 15-64 years (also for PM_{10}), 65+ years). Also, there are significant warm period increases for asthma (1-4 years) and total respiratory (1-4 years) (also for PM_{10}).

Table 4.2.2: Significant seasonal increases in respiratory admissions associated with an IQR increase in particles (average lag 0-1) (case-crossover meta-analysis)

		24-hour $PM_{2.5}$				24-hour PM_{10}			
		Inc	95%CI		I2	Inc	95%CI		I2
Asthma (1-4 yrs)	cool	-0.6	-2.6,	1.4	0.0	0.9	-3.6,	5.6	66.5
	warm	3.5	0.0,	7.1	12.2	2.8	-0.7,	6.3	0.0
Asthma (15-64 yrs)	cool	2.4	0.7,	4.1	0.0	1.0	-1.8,	3.9	30.9
	warm	1.9	-1.1,	5.0	0.0	0.3	-4.9,	5.7	54.8
COPD (65+ yrs)	cool	1.4	0.1,	2.6	0.0	-0.1	-1.8,	1.6	0.0
	warm	0.7	-1.6,	3.2	0.0	-0.3	-4.7,	4.3	42.9
Pneumonia + acute bronchitis (65+ yrs)	cool	2.2	0.6,	3.9	20.8	2.4	-0.5,	5.4	52.6
	warm	-0.4	-2.9,	2.3	0.0	-1.4	-4.2,	1.4	0.0
Total respiratory (0 yrs)	cool	1.8	0.2,	3.4	0.0	0.8	-1.0,	2.7	0.0
	warm	2.8	-0.9,	6.7	0.0	0.8	-3.2,	5.1	0.0
Total respiratory (1-4 yrs)	cool	1.1	-0.1,	2.3	0.0	0.6	-0.8,	2.1	0.0
	warm	3.0	0.8,	5.2	0.0	3.1	0.2,	6.0	27.5
Total respiratory (15-64 yrs)	cool	1.3	0.3,	2.2	0.0	1.5	0.3,	2.8	0.0
	warm	-0.2	-3.8,	3.6	69.5*	0.0	-2.7,	2.8	51.0
Total respiratory (65+ yrs)	cool	1.4	0.4,	2.5	33.6	0.9	-1.0,	2.8	63.1
	warm	0.7	-0.8,	2.3	0.0	-0.2	-2.0,	1.6	11.7

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

4.3 Single city results

There are often differences between the results for the cities so combining the estimates for all the cities would not be appropriate in all instances, whether in combining the results for all cities or just combining results for all Australian cities.

Additional significant associations were found with short-term exposure to particles in different cities for the following categories of **mortality**:

- respiratory mortality for all ages ($PM_{2.5}$ and PM_{10}) and adults aged 75 years and greater (PM_{10}) in Brisbane

- all cause mortality for both age groups in Brisbane and Sydney (PM_{10}).

Additional significant associations were found with short-term exposure to particles in some cities for the following categories of **hospital admissions**:

- arrhythmia in adults aged between 15 and 64 years in Christchurch (PM_{10})
- cardiovascular disease in adults aged 65 years and greater in Perth (PM_{10})
- asthma in children aged between 1 and 4 years in Christchurch (PM_{10})
- asthma in children aged between 5 and 14 years in Sydney ($PM_{2.5}$ and PM_{10})
- asthma in adults aged between 15 and 64 years in Melbourne (PM_{10})

- chronic obstructive pulmonary disease in adults aged between 15 and 64 years in Sydney ($PM_{2.5}$) and Perth ($PM_{2.5}$)
- pneumonia and acute bronchitis in children aged between 1 and 4 years in Sydney (PM_{10})
- pneumonia and acute bronchitis in adults aged between 15 and 64 years in Christchurch (PM_{10})
- all respiratory disease in children aged less than 1 year in Sydney (PM_{10})
- all respiratory disease in children aged between 5 and 14 years in Sydney ($PM_{2.5}$).

All individual city estimates for short-term associations are shown in *Volume 3: Appendix A3.1*.

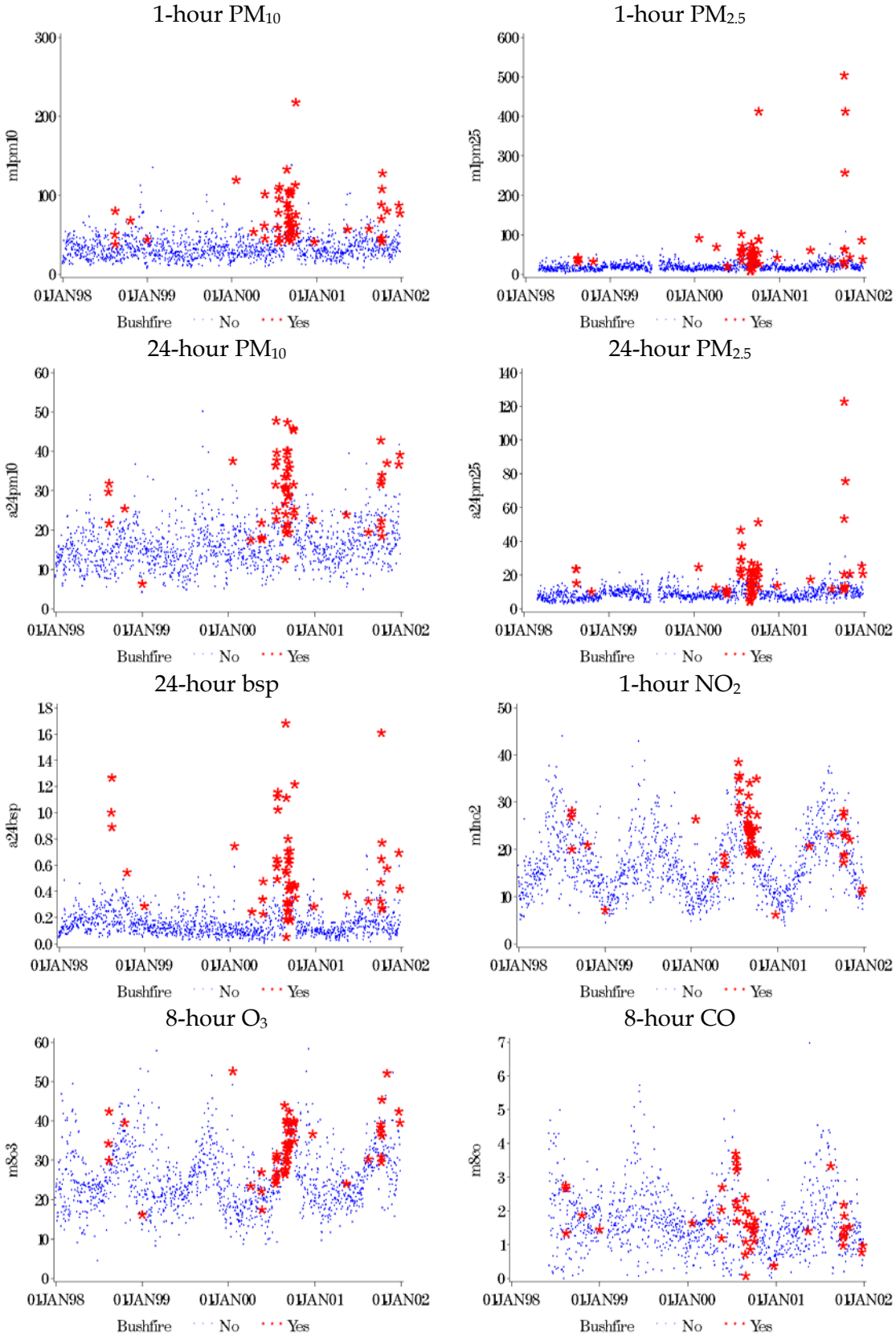
4.4 Bushfires

Bushfire information was not generally available for this study. However, it is possible from published reports for Brisbane to identify days when bushfires were reported during 1998 to 2001. Comparison between air pollutant levels for these days and other days are shown in Figure 4.4.1 for bsp, $PM_{2.5}$, PM_{10}, NO_2, CO and ozone.

These figures show how bushfires seriously affect the higher records for daily pollutant readings of PM and bsp. For PM_{10} there is some overlap between results with and without bushfire, but for bsp and $PM_{2.5}$ the highest values are all bushfires, and some values are extreme. This means the extra effect of bushfires can possibly be unravelled from the usual pollutant effect for PM_{10}, (because there are high pollutant days with and without bushfires), but for $PM_{2.5}$ the highest values are completely confounded with bushfires.

By contrast, the bushfire events do not necessarily coincide with peak values of NO_2, CO and ozone.

Figure 4.4.1: Plots of selected Brisbane daily pollutants over time by bushfire



The effect on the parameter estimates is shown in Tables 4.4.1 to 4.4.3, which show three different models:

- the first model ignores the effect of bushfires (standard practice thus far);
- the second model includes an interaction between the pollutant and bushfires
- the third model includes a binary indicator term for bushfires.

Model 1 assumes there is a pollutant effect only, and that the extremes on bushfires days are captured by this effect. Model 2 assumes that the effect of the pollutant is different on bushfire days. Model 3 assumes an independent bushfire effect.

Table 4.4.1: Estimating the effect of different techniques for dealing with bushfires using the case-crossover method and total mortality in Brisbane. Cells show per cent mortality increase (and 95% confidence interval)

	1. Ignore	2. Interaction		3. Indicator	
Pollutant	Pollutant	Pollutant	Interaction	Pollutant	Bushfire
1-hour $PM_{2.5}$†	0.4 (-0.0, 0.9)	0.2 (-1.0, 1.5)	0.2 (-1.0, 1.4)	0.2 (-0.3, 0.7)	8.7 (1.7, 16.1)
24-hour $PM_{2.5}$†	2.5 (0.3, 4.7)	1.0 (-2.2, 4.2)	1.6 (-1.8, 5.2)	1.4 (-1.0, 3.8)	8.1 (1.1, 15.5)
1-hour PM_{10}†	1.6 (0.9, 2.3)	1.6 (0.7, 2.4)	0.1 (-0.7, 1.0)	1.5 (0.7, 2.2)	3.7 (-2.9, 10.7)

†10 unit increase

Table 4.4.2: Estimating the effect of different techniques for dealing with bushfires using the case-crossover method and total respiratory mortality in Brisbane. Cells show per cent mortality increase (and 95% confidence interval)

	1. Ignore	2. Interaction		3. Indicator	
Pollutant	Pollutant	Pollutant	Interaction	Pollutant	Bushfire
1-hour $PM_{2.5}$†	1.1 (-0.3, 2.5)	-1.0 (-4.9, 3.1)	2.2 (-1.7, 6.2)	0.4 (-1.2, 1.9)	30.9 (7.2, 59.9)
1-hour PM_{10}†	1.8 (-0.4, 4.2)	0.4 (-2.3, 3.2)	2.4 (-0.3, 5.2)	0.6 (-1.9, 3.1)	29.8 (5.9, 58.9)

†10 unit increase

Table 4.4.3: Estimating the effect of different techniques for dealing with bushfires using the case-crossover method and total cardiovascular mortality in Brisbane. Cells show per cent mortality increase (and 95% confidence interval)

	1. Ignore	2. Interaction		3. Indicator	
Pollutant	Pollutant	Pollutant	Interaction	Pollutant	Bushfire
1-hour $PM_{2.5}$†	0.5 (-0.2, 1.2)	-0.5 (-2.3, 1.4)	1.0 (-0.8, 2.8)	0.4 (-0.4, 1.1)	4.2 (-5.7, 15.1)
1-hour PM_{10}†	1.4 (0.3, 2.4)	1.1 (-0.2, 2.3)	0.5 (-0.8, 1.8)	1.3 (0.2, 2.4)	1.5 (-8.1, 12.0)

†10 unit increase

Using bushfire only (that is, no pollutants) results in an 8.7% increase in total mortality on bushfire days, 95% CI: 1.7, 16.1%.

There seems to be an independent bushfire effect when considering $PM_{2.5}$ but not PM_{10} for all cause mortality and cardiovascular mortality. For any PM_{10} model, adding an interaction or bushfire indicator does not change the pollutant effect by

much (when compared to model 1). However, this is not the case for the results for respiratory mortality. For $PM_{2.5}$ the pollutant effect decreases in models 2 and 3, and the independent bushfire effect is, in some cases, very large.

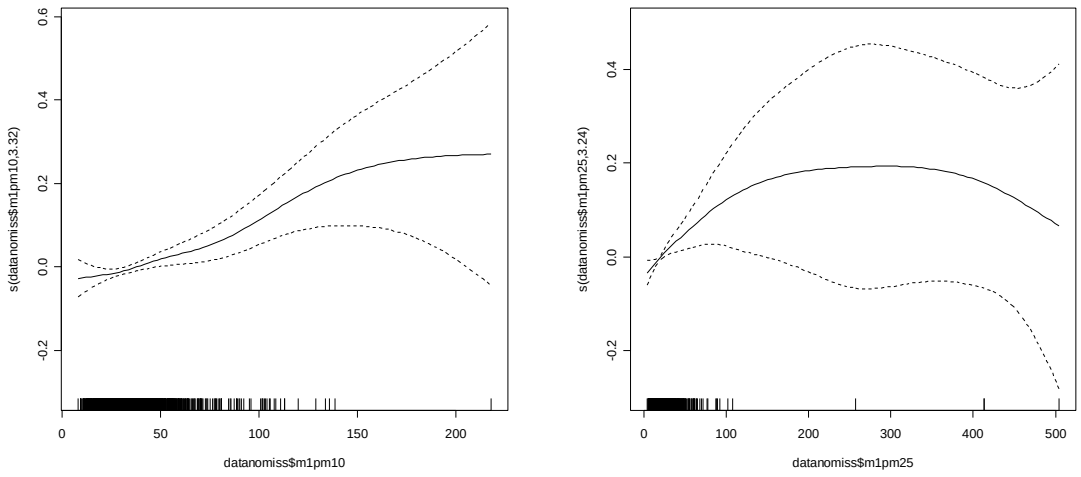
To investigate the effects further an analysis was run with both pollutants and the bushfire indicator. The results below show the per cent increase in mortality and 95% confidence interval (in brackets), and use 10-unit increase in 1-hour PM:

Bushfire 5.9 (-1.1, 13.4); $PM_{2.5}$ -0.2 (-0.8, 0.4); PM_{10} 1.4 (0.5, 2.3).

Figure 4.4.2 shows how non-linear the outcomes will be for PM in Brisbane, especially for $PM_{2.5}$.

Figure 4.4.2: Non-linearity of PM effect on total mortality in Brisbane using GAMs (without bushfire indicator)

1-hour PM_{10} 1-hour $PM_{2.5}$



The horizontal axis of Figure 4.4.2 covers the entire observed range of PM. It is important to note that most data occur at the lower end of the range (<150 for PM_{10} and <125 for $PM_{2.5}$) — see the 'rug' plot just above the horizontal axis, which indicates the density of observations — and this is responsible for the extremely wide confidence limits in the right-hand part of the plots. The y-axis indexes the log relative-risks centred on the mean exposure concentration.

Chapter 5 Ozone

Ozone exposure analysis has been included in this study since it has significant health effects and is well recognised as an issue for many of the cities included. However, ozone needs to be treated cautiously in the meta-analysis due to its complex chemistry and the difficultly of obtaining representative exposure estimates from the monitoring networks. In contrast to the other pollutants examined, ozone is not emitted directly, but forms in the atmosphere from emitted precursors (oxides of nitrogen and hydrocarbons play a major role). These react in the presence of light to form ozone over a period of several hours. During this time, the peaks of ozone concentration can move around the city and form in locations well away from the emissions sources. This process and its complexities are very specific to the city.

Ozone is monitored in all the cities under study here except Christchurch: in Auckland (with 2 monitors), in Brisbane (7 monitors), Canberra (1 monitor), Melbourne (8 monitors), Perth (3 monitors) and Sydney (12 monitors). Therefore, the representativeness of the network in some cities varies. For cities that have an extensive and well-designed ozone monitoring system, the exposure results are probably reasonably representative of the target population exposure (Sydney, Melbourne and Brisbane).

However, for the other cities — particularly Auckland — the ozone network is sparse and does not represent population exposure very well. Indeed, the Auckland monitor can show low concentrations at times when ozone levels are high in other parts of the city — and vice versa. The monitoring network in Auckland (and possibly Canberra also) gives indicative concentrations rather than fully represent population exposure. Thus a full meta-analysis of ozone across all of the cities may be problematical.

The project team reviewed the data supplied by the government agencies in each city and it was decided to only use the data for some Australian cities — Brisbane, Melbourne, Perth, and Sydney.

Detailed summaries for ozone data in each city are shown in *Volume 3: Appendix 1*. Results were derived for daily maximum 1-hour, 4-hour and 8-hour concentrations for ozone (maxima derived for daytime period).

Given the most serious impacts of ozone occur during photochemical smog episodes, usually in summer, we will concentrate on seasonal analyses. It is apparent that the summer periods are important for cities such as Melbourne and Perth. Brisbane usually has wet summers and warm dry winters, so seasonal variation in ozone is not marked there. Sydney ozone patterns will probably have characteristics between those of Brisbane and Melbourne/Perth.

Summarised in Table 5.0.1 are all the significant pooled estimates for health impacts associated with maximum 1-hour, 4-hour and 8-hour ozone concentrations. The associations for the short-term exposure (an average of the ozone concentration on the same day as the health effect, and the day before) with morbidity and mortality health outcomes are presented for Brisbane, Melbourne, Perth and Sydney. As any

significant associations for ozone are seasonal, with only warm periods contributing, the methodology adopted here for estimating long-term associations (40 days previous) cannot be used effectively, so long term estimates for ozone have not been calculated here.

Table 5.0.1 also shows the results for pooling the estimates for mortality and morbidity. It was found that there were significant pooled estimates for the associations between increases in ozone concentrations and increases in:

- total all cause mortality, cardiovascular mortality and respiratory mortality for all ages for all year, and there is no evidence of heterogeneity between results for all cities

- total all cause mortality and cardiovascular mortality for all ages for the warm season, and there is no evidence of heterogeneity between results for all cities

- cardiovascular mortality for 75+ years age group for the warm season, and there is no evidence of heterogeneity between results for all cities

- cardiovascular and respiratory mortality for 75+ years age group for all year, and there is no evidence of heterogeneity between results for all cities

- all respiratory hospital admissions and asthma hospital admissions in the child age group 1 to 4 years, for the warm period, and there is no evidence of heterogeneity between results for all cities

- all respiratory admissions in the child age groups, 1 to 4 years for all year, and there is no evidence of heterogeneity between results for all cities.

Table 5.0.1: Significant increases in morbidity and mortality (and 95% confidence intervals) associated with a one-ppb increase in ozone (lags 0-1) for meta-analyses of the cities: Brisbane, Melbourne, Perth and Sydney

			1-hour ozone				4-hour ozone				8-hour ozone			
			Inc	95% CI		I2	Inc	95% CI		I2	Inc	95% CI		I2
Mortality														
Total all cause	75+	warm	0.1	-0.0,	0.2	30.5	0.1	-0.0,	0.3	28.4	0.2	-0.0,	0.3	29.2
		all year	0.1	-0.0,	0.3	70.8*	0.1	-0.0,	0.3	71.8*	0.1	-0.0,	0.3	66.9*
Total all cause	all ages	warm	0.1	0.0,	0.2	0.0	0.1	0.0,	0.2	0.0	0.1	0.0,	0.2	0.0
		all year	0.1	0.0,	0.2	60.5	0.1	0.0,	0.3	61.0	0.2	0.0,	0.3	55.6
Cardiovascular	75+	warm	0.2	0.0,	0.3	0.0	0.2	0.0,	0.4	0.0	0.2	0.0,	0.4	0.0
		all year	0.2	0.0,	0.3	48.3	0.2	0.0,	0.4	48.3	0.3	0.0,	0.5	44.6
Cardiovascular	all ages	warm	0.1	-0.0,	0.2	0.0	0.1	-0.0,	0.3	0.0	0.2	0.0,	0.4	0.0
		all year	0.2	0.1,	0.3	0.0	0.2	0.1,	0.3	0.0	0.3	0.1,	0.4	0.0
Respiratory	75+	all year	0.3	0.0,	0.5	0.0	0.3	0.0,	0.6	0.0	0.3	0.0,	0.7	0.0
Respiratory	all ages	all year	0.2	0.0,	0.4	0.0	0.2	-0.0,	0.5	0.0	0.3	-0.0,	0.6	0.0
Admissions														
Respiratory-all	1-4	warm	0.4	0.2,	0.5	0.0	0.5	0.3,	0.6	0.0	0.6	0.4,	0.8	0.0
		all year	0.2	0.0,	0.3	18.2	0.2	0.0,	0.4	33.6	0.2	-0.0,	0.5	53.3
Asthma	1-4	warm	0.5	0.3,	0.7	0.0	0.6	0.3,	0.8	0.0	0.6	0.3,	0.9	0.0

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

5.1 Correlation between ozone and other pollutants

Correlation between ozone and other pollutants is discussed earlier in this report, while Table 1.13 shows how ozone concentrations are correlated with those for other pollutants and weather variables.

It is notable that the results for particles, NO_2 and ozone are similar for respiratory hospital admissions for children. An examination of the particle and NO_2 associations in the cool and warm periods did sometimes show significant differences between the two periods, with results often higher and significant for the warm period.

5.2 Mortality

Table 5.0.1 shows the results for all the meta-analyses. The associations for all cause (total) mortality outcomes for the short term (average of same day as, and day before death) are shown in Tables 5.2.1 to 5.2.6 for deaths due to cardiovascular disease, deaths due to respiratory disease, and for total deaths (non-accidental, all causes) for each of the cities (Brisbane, Melbourne, Perth, Sydney). The results are presented separately for the warm and cool periods. The statistical tests show there is no

evidence for heterogeneity between the cities for outcomes for which there were significant pooled estimates. (See *Volume 3, Appendix A3.5, A3.6* for complete results.)

It might be expected that some pollutant effects may be different in different seasons. For example, ozone peaks during the summer smog episodes in general (although Brisbane may also have winter smog events). These warm and cool period results are shown in Tables 5.2.

Table 5.2.1: City specific and meta-analysis percentage increases (and 95% confidence intervals) in total mortality (75+ years) associated with a one-ppb increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.1	-0.3,	0.5	0.1	-0.3,	0.5	0.1	-0.4,	0.6
	warm	0.2	-0.1,	0.4	0.2	-0.1,	0.5	0.3	-0.1,	0.6
Melbourne	cool	-0.4	-0.8,	0.0	-0.4	-0.7,	0.0	-0.2	-0.6,	0.1
	warm	-0.0	-0.2,	0.2	0.0	-0.2,	0.2	0.0	-0.2,	0.3
Perth	cool	-0.1	-0.7,	0.5	-0.2	-0.9,	0.5	-0.2	-0.9,	0.4
	warm	-0.0	-0.3,	0.2	-0.1	-0.5,	0.3	-0.1	-0.6,	0.3
Sydney	cool	0.1	-0.1,	0.4	0.1	-0.2,	0.4	0.1	-0.2,	0.4
	warm	0.2	0.1,	0.3	0.2	0.1,	0.4	0.3	0.1,	0.5
Overall	cool	-0.0	-0.3,	0.2	-0.1	-0.3,	0.2	-0.0	-0.2,	0.2
	warm	0.1	-0.0,	0.2	0.1	-0.0,	0.3	0.2	-0.0,	0.3
I-squared (%)	cool	43.1	-	-	32.3	-	-	0.0	-	-
	warm	30.5	-	-	28.4	-	-	29.2	-	-

Table 5.2.2: City specific and meta-analysis percentage increases (and 95% confidence intervals) in total mortality (all ages) associated with a one-ppb increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.2	-0.1,	0.5	0.2	-0.1,	0.6	0.2	-0.2,	0.7
	warm	0.2	-0.0,	0.3	0.2	-0.0,	0.4	0.2	-0.0,	0.5
Melbourne	cool	-0.1	-0.4,	0.2	-0.1	-0.4,	0.2	-0.1	-0.4,	0.3
	warm	-0.0	-0.2,	0.2	0.0	-0.2,	0.2	0.0	-0.2,	0.2
Perth	cool	-0.0	-0.5,	0.5	-0.0	-0.6,	0.5	-0.0	-0.6,	0.5
	warm	0.1	-0.2,	0.3	0.0	-0.2,	0.3	0.0	-0.3,	0.4
Sydney	cool	0.1	-0.1,	0.3	0.1	-0.1,	0.3	0.0	-0.2,	0.3
	warm	0.1	0.0,	0.2	0.1	0.0,	0.3	0.2	0.0,	0.3
Overall	cool	0.1	-0.1,	0.2	0.1	-0.1,	0.2	0.0	-0.1,	0.2
	warm	0.1	0.0,	0.2	0.1	0.0,	0.2	0.1	0.0,	0.2
I-squared (%)	cool	0.0	-	-	0.0	-	-	0.0	-	-
	warm	0.0	-	-	0.0	-	-	0.0	-	-

Table 5.2.3: City specific and meta-analysis percentage increases (and 95% confidence intervals) in respiratory mortality (75+ years) associated with a one-ppb increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	-0.2	-1.4,	1.0	-0.3	-1.7,	1.1	-0.4	-2.0,	1.3
	warm	0.8	-0.0,	1.6	0.9	-0.0,	1.9	1.3	0.1,	2.5
Melbourne	cool	0.3	-0.9,	1.6	0.2	-1.0,	1.4	0.0	-1.2,	1.2
	warm	-0.2	-0.8,	0.5	-0.2	-0.9,	0.6	-0.2	-1.1,	0.8
Perth	cool	-0.3	-2.2,	1.6	-0.5	-2.5,	1.6	-0.7	-2.7,	1.4
	warm	0.7	-0.4,	1.7	0.8	-0.5,	2.0	1.0	-0.5,	2.6
Sydney	cool	0.6	-0.2,	1.3	0.6	-0.2,	1.4	0.6	-0.2,	1.5
	warm	0.1	-0.4,	0.5	0.1	-0.5,	0.7	0.1	-0.6,	0.8
Overall	cool	0.3	-0.2,	0.9	0.3	-0.3,	0.8	0.2	-0.4,	0.8
	warm	0.2	-0.2,	0.6	0.3	-0.2,	0.7	0.4	-0.2,	1.0
I-squared (%)	cool	0.0	-	-	0.0	-	-	0.0	-	-
	warm	28.7	-	-	22.4	-	-	36.3	-	-

Table 5.2.4: City specific and meta-analysis percentage increases (and 95% confidence intervals) in respiratory mortality (all ages) associated with a one-ppb increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.1	-0.9,	1.1	0.0	-1.1,	1.2	0.0	-1.4,	1.4
	warm	0.3	-0.4,	1.0	0.3	-0.5,	1.1	0.5	-0.5,	1.5
Melbourne	cool	0.3	-0.7,	1.4	0.2	-0.8,	1.2	0.1	-0.9,	1.2
	warm	-0.2	-0.8,	0.4	-0.2	-0.9,	0.4	-0.2	-1.0,	0.5
Perth	cool	-0.4	-2.0,	1.3	-0.6	-2.3,	1.1	-0.8	-2.6,	1.0
	warm	0.2	-0.6,	1.1	0.3	-0.7,	1.4	0.4	-0.9,	1.7
Sydney	cool	0.7	0.0,	1.3	0.7	-0.0,	1.3	0.7	-0.0,	1.4
	warm	0.1	-0.3,	0.5	0.1	-0.4,	0.6	0.1	-0.5,	0.7
Overall	cool	0.4	-0.1,	0.9	0.3	-0.1,	0.8	0.3	-0.2,	0.8
	warm	0.1	-0.2,	0.3	0.1	-0.3,	0.4	0.1	-0.3,	0.5
I-squared (%)	cool	0.0	-	-	0.0	-	-	0.0	-	-
	warm	0.0	-	-	0.0	-	-	0.0	-	-

Table 5.2.5: City specific and meta-analysis percentage increases (and 95% confidence intervals) in cardiovascular mortality (75+ years) associated with a one-ppb increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.2	-0.3,	0.7	0.2	-0.4,	0.8	0.2	-0.5,	0.9
	warm	0.0	-0.3,	0.3	0.0	-0.4,	0.4	0.1	-0.4,	0.6
Melbourne	cool	-0.4	-0.9,	0.2	-0.3	-0.9,	0.2	-0.2	-0.8,	0.3
	warm	0.2	-0.1,	0.5	0.2	-0.1,	0.6	0.3	-0.1,	0.7
Perth	cool	-0.2	-1.1,	0.7	-0.2	-1.2,	0.8	-0.2	-1.1,	0.8
	warm	-0.1	-0.5,	0.4	-0.1	-0.6,	0.4	-0.2	-0.8,	0.5
Sydney	cool	0.3	-0.0,	0.7	0.3	-0.1,	0.7	0.3	-0.2,	0.7
	warm	0.2	0.0,	0.4	0.3	0.0,	0.5	0.3	0.0,	0.6
Overall	cool	0.1	-0.2,	0.4	0.1	-0.2,	0.4	0.1	-0.2,	0.4
	warm	0.2	0.0,	0.3	0.2	0.0,	0.4	0.2	0.0,	0.4
I-squared (%)	cool	34.3	-	-	23.3	-	-	0.0	-	-
	warm	0.0	-	-	0.0	-	-	0.0	-	-

Table 5.2.6: City specific and meta-analysis percentage increases (and 95% confidence intervals) in cardiovascular mortality (all ages) associated with a one-ppb increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.1	-0.4,	0.5	0.0	-0.5,	0.6	0.1	-0.6,	0.7
	warm	-0.0	-0.3,	0.3	0.0	-0.3,	0.4	0.1	-0.4,	0.5
Melbourne	cool	-0.2	-0.7,	0.2	-0.2	-0.7,	0.3	-0.1	-0.6,	0.4
	warm	0.2	-0.1,	0.4	0.2	-0.1,	0.5	0.3	-0.1,	0.6
Perth	cool	-0.0	-0.8,	0.8	0.1	-0.8,	0.9	0.2	-0.6,	1.1
	warm	0.1	-0.3,	0.5	0.1	-0.3,	0.6	0.1	-0.5,	0.7
Sydney	cool	0.2	-0.1,	0.5	0.2	-0.1,	0.5	0.2	-0.2,	0.5
	warm	0.2	-0.0,	0.3	0.2	-0.0,	0.4	0.2	-0.0,	0.5
Overall	cool	0.1	-0.1,	0.3	0.1	-0.2,	0.3	0.1	-0.2,	0.3
	warm	0.1	-0.0,	0.2	0.1	-0.0,	0.3	0.2	0.0,	0.4
I-squared (%)	cool	0.0	-	-	0.0	-	-	0.0	-	-
	warm	0.0	-	-	0.0	-	-	0.0	-	-

5.3 Hospital admissions

5.3.1 Cardiovascular disease

The health outcomes are as described earlier in this report. However, there were few significant associations found between cardiovascular hospital admissions and ozone in any single city results. There are no significant meta-analysis results.

5.3.2 Hospital admissions due to respiratory disease

The health outcomes are as described earlier in this report.

Tables 5.3.1 and 5.3.2 show that, in pooling the estimates for all four Australian cities, there are significant increases in all respiratory and asthma admissions for the 1-4 years age group during the warm period, with no evidence for heterogeneity between the cities. There are similar estimates for each ozone averaging period used.

Table 5.3.1: City specific and meta-analysis percentage increases (and 95% confidence intervals) in respiratory admissions (1-4 years) associated with a one-unit increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.2	-0.4,	0.8	0.3	-0.3,	1.0	0.5	-0.3,	1.3
	warm	0.4	-0.0,	0.7	0.5	0.0,	0.9	0.6	0.1,	1.2
Melbourne	cool	-1.2	-1.9,	-0.5	-1.2	-1.9,	-0.5	-1.1	-1.8,	-0.5
	warm	0.3	-0.1,	0.7	0.4	-0.0,	0.9	0.5	-0.0,	1.0
Perth	cool	-0.3	-1.2,	0.5	-0.5	-1.4,	0.4	-0.5	-1.4,	0.4
	warm	0.5	0.0,	1.0	0.6	0.0,	1.2	0.8	0.0,	1.5
Sydney	cool	-0.1	-0.4,	0.3	-0.1	-0.5,	0.3	-0.2	-0.6,	0.2
	warm	0.4	0.2,	0.6	0.4	0.2,	0.7	0.6	0.3,	0.8
Overall	cool	-0.3	-0.8,	0.2	-0.4	-0.9,	0.2	-0.4	-0.9,	0.2
	warm	0.4	0.2,	0.5	0.5	0.3,	0.6	0.6	0.4,	0.8
I-squared (%)	cool	70.7*	-	-	73.4*	-	-	69.7*	-	-
	warm	0.0	-	-	0.0	-	-	0.0	-	-

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 95% level*

Table 5.3.2: City specific and meta-analysis percentage increases (and 95% confidence intervals) in asthma admissions (1-4 years) associated with a one-unit increase in ozone (average lag 0-1)

		1-hour ozone			4-hour ozone			8-hour ozone		
		Inc	95%CI		Inc	95%CI		Inc	95%CI	
Brisbane	cool	0.6	-0.3,	1.6	0.7	-0.4,	1.8	0.7	-0.6,	2.1
	warm	0.6	0.1,	1.2	0.8	0.1,	1.4	0.8	-0.0,	1.7
Melbourne	cool	-1.5	-2.6,	-0.4	-1.5	-2.6,	-0.4	-1.3	-2.4,	-0.3
	warm	0.3	-0.3,	0.8	0.4	-0.2,	1.0	0.3	-0.5,	1.0
Perth	cool	-0.0	-1.3,	1.3	-0.2	-1.6,	1.2	-0.2	-1.7,	1.2
	warm	0.8	0.0,	1.5	0.9	0.0,	1.8	1.1	-0.0,	2.2
Sydney	cool	-0.4	-1.0,	0.2	-0.5	-1.1,	0.2	-0.6	-1.3,	0.2
	warm	0.5	0.2,	0.8	0.5	0.2,	0.8	0.6	0.2,	1.0
Overall	cool	-0.3	-1.2,	0.5	-0.4	-1.3,	0.5	-0.4	-1.2,	0.3
	warm	0.5	0.3,	0.7	0.6	0.3,	0.8	0.6	0.3,	0.9
I-squared (%)	cool	64.4*	-	-	60.9*	-	-	49.2	-	-
	warm	0.0	-	-	0.0	-	-	0.0	-	-

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 95% level*

Chapter 6 Summary and discussion of results

In this chapter, we summarise the results for the study and discuss the interpretation of such results for policy-makers. The detailed results for each pollutant and city are given in previous chapters and in *Volume 3* of the study report.

6.1 Pooled results for all the cities

The methodology used in this study required monitoring of the pollutant concentrations on a daily basis, and this only occurred for the air pollutants, NO_2 and CO, so it was only for these air pollutants that results could be pooled for all cities and an overall mean estimate could be calculated. However, results could be derived for smaller groupings of the cities for particles (as measured by $PM_{2.5}$ — Brisbane, Melbourne, Perth, Sydney; as measured by PM_{10} — these cities and Christchurch) and ozone (Brisbane, Melbourne, Perth, Sydney).

NO_2 is one of the air pollutants monitored on a daily basis in all the cities under study; however, the number of air pollution monitors measuring ambient outdoor air pollutant concentrations is different in each city. For NO_2 in Auckland there are 2 monitors, Brisbane 7 monitors, Canberra 1 monitor, Christchurch 1 monitor, Melbourne 8 monitors, Perth 5 monitors, and Sydney 13 monitors. CO is also one of the air pollutants monitored on a daily basis in all the cities under study, and the number of air pollution monitors measuring ambient outdoor air pollutant concentrations is different in each city. For CO in Auckland there are 3 monitors, Brisbane 1 monitor, Canberra 1 monitor, Christchurch 2 monitors, Melbourne 3 monitors, Perth 3 monitors and Sydney 4 monitors.

We tested as to whether the results for all cities showed any statistical differences, that is, whether all cities were showing increases or decreases, and whether these changes were similar in magnitude, given the statistical uncertainty in the analyses. If the results were not similar (that is, not homogeneous or heterogeneous), we needed to examine why that might be the case, as in such instances the pooled estimate would overestimate or underestimate the associations found.

6.1.1 Association with death counts

The significant meta-analysis results for the case-crossover analysis for mortality are shown in Table 6.1.1 for all deaths and for the age group 75 years and greater (75+), and for mortality categories of total all cause, deaths due to cardiovascular disease and deaths due to respiratory disease. The results refer to associations between mortality and pollutant concentrations averaged over the same day and the day before (lags 0 to 1) to the day of the health outcome.

There are significant associations between NO_2 (1-hour maximum) and all health outcomes for all cities in the study, and there is no evidence to suggest there is heterogeneity in the results for the cities.

For Brisbane, Christchurch, Melbourne, Perth, and Sydney, there are significant associations between PM_{10} and all health outcomes for cardiovascular mortality, and there is no evidence to suggest there is heterogeneity in the results for the cities.

For the four Australian cities (Brisbane, Melbourne, Perth, Sydney) it is clear that there are significant associations for both $PM_{2.5}$ and ozone (warm period) with all health outcomes, and there is no evidence to suggest there is heterogeneity in the results for the cities.

Table 6.1.1: Significant per cent changes in total deaths (and 95% confidence interval) by age group due to air pollutant concentrations averaged over same day and day before (lags 0 to 1) *

	Total (all ages)			75 + years	
Outcome (age group) and pollutant (units)	**Increase %* (95% CI)**	**I-squared %†**	**Cities missing**	**Increase %* (95% CI)**	**I-squared %†**
Total all cause					
24-hour average $PM_{2.5}$ ($\mu g.m^{-3}$)	0.2 (0.1, 0.4)	40.8	A, Ca, Ch	0.3 (0.1, 0.5)	0.0
Max 1-h average NO_2 (ppb)	0.2 (0.0, 0.3)	51.5	None	0.2 (0.0, 0.3)	46.5
Max 8-h av. O_3 (ppb) 2(warm)1	0.1 (0.0, 0.2)	0	A, Ca, Ch		
Cardiovascular mortality					
24-hour average $PM_{2.5}$ ($\mu g.m^{-3}$)	0.4 (0.2, 0.6)	0.0	A, Ca, Ch	0.5 (0.2, 0.7)	0.0
24-hour average PM_{10} ($\mu g.m^{-3}$)	0.2 (0.0, 0.3)	27.1	A, Ca	0.2 (0.0, 0.4)	27.4
Max 1-h average NO_2 (ppb)	0.2 (0.0, 0.3)	0.0	None	0.2 (0.0, 0.3)	14.3
Max 8-h av. O_3 (ppb)2 (warm)1	0.2 (0.0, 0.4)	0.0	A, Ca, Ch	0.2 (0.0, 0.4)	0.0
Respiratory mortality					
24-hour average $PM_{2.5}$ (μg.m-3)			A, Ca, Ch	0.6 (0.0, 1.1)	0.0
Max 1-h average NO_2 (ppb)	0.4 (0.1, 0.7)	0.0	None	0.4 (0.1, 0.8)	0.0

**Estimates from a random effects meta-analysis of cities shown per unit increase in pollutant*
† I-squared is the percentage of total variation in the estimated increase that is due to heterogeneity between cities
[1] Warm period only
[2]Results similar for 1-h and 4-h ozone (except for cardiovascular)
CI=confidence interval; ppb=parts per billion: A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney

The results for matching pollutants that have significant associations with mortality outcomes are shown in Tables 6.1.2, 6.1.3 and 6.1.4 for cardiovascular, respiratory and all cause mortality, respectively.

The results indicate that NO_2 confounds results for particles (both $PM_{2.5}$ and PM_{10}) and ozone, increases in mortality associated with increases in NO_2 cannot be added to the increases arising from particles or ozone, as they may refer to similar events and are therefore not additive. It is notable there is no confounding between particle and ozone associations, nor between $PM_{2.5}$ and PM_{10} associations.

Table 6.1.2: Multi-pollutant models: statistically significant increases* in deaths (all ages) for cardiovascular disease, and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %*	95% CI	Cities included
1-hour maximum O_3	24-hour average PM_{10}	2.9	0.9, 4.9	B, M, P, S
	24-hour average $PM_{2.5}$	3.5	2.2, 4.8	B, M, P, S
	1-hour maximum NO_2	2.0	0.2, 3.9	B, M, P, S
	Unmatched	2.1	1.1, 3.1	B, M, P, S
24-hour average PM_{10}	1-hour maximum NO_2	0.5	-1.6, 2.5	B, Ch, M, P, S
	Unmatched	1.4	0.2, 2.6	B, Ch, M, P, S
24-hour average $PM_{2.5}$	24-hour average PM_{10}	3.9	2.2, 5.7	B, M, P, S
	1-hour maximum NO_2	0.4	-2.3, 3.2	B, M, P, S
	1-hour maximum O_3	2.1	0.6, 3.7	B, M, P, S
	Unmatched	1.5	0.7, 2.3	B, M, P, S

**Estimates from a random effects meta-analysis of cities shown per IQR increase in pollutant.*
A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

Table 6.1.3: Multi-pollutant models: statistically significant increases* in deaths (75+ years age group) for respiratory disease, and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %*	95% CI	Cities included
24-hour average $PM_{2.5}$	Max 1-h NO_2	-1.0	-4.3, 2.5	B, M, P, S
	Unmatched	2.1	0.1, 4.2	B, M, P, S

**Estimates from a random effects meta-analysis of cities shown per IQR increase in pollutant.*
A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

Table 6.1.4: Multi-pollutant models: statistically significant increases* in deaths (75+ years) for all cause mortality, and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %*	95% CI	Cities included
24-hour average $PM_{2.5}$	Max 1-h NO_2	0.3	-2.3, 2.9	B, M, P, S
	Unmatched	1.2	0.5, 1.8	B, M, P, S

**Estimates from a random effects meta-analysis of cities shown per IQR increase in pollutant.*
A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

The examination for possible 'harvesting' effects by including distributed lags for pollutants up to 40 days prior to the death has found little evidence for such effects (see Tables 6.1.5 and 6.1.7) when examining the gases CO and NO_2.

Table 6.1.5: Per cent changes in total deaths (and 95% confidence interval) by age group over forty-day periods*

Outcome (age group) and pollutant (units)	40-day: Lags 0 to 40		
	Change	95% CI	I2
Total all cause (all ages)			
24-hour average $PM_{2.5}$ ($\mu g.m^{-3}$)	1.0	(-1.2, 3.3)	0.0
24-hour average PM_{10} ($\mu g.m^{-3}$)	-4.0	(-18.4, 10.6)	29.6
24-hour average NO_2 (ppb)	8.8	(4.6, 13.0)	0.0
8-hour average CO (ppm)	9.4	(4.8, 14.3)	54.0

** Estimates from a random effects meta-analysis of cities using distributed lag models, shown for an inter-quartile range increase in pollutant.*
CI=confidence interval, ppb=parts per billion, ppm=parts per million.

Table 6.1.6: Per cent changes in respiratory deaths (and 95% confidence interval) by age group over forty-day periods*

Outcome (age group) and pollutant (units)	40-day: Lags 0 to 40		
	Change	95% CI	I2
Respiratory deaths (all ages)			
24-hour average $PM_{2.5}$ ($\mu g.m^{-3}$)	9.5	(-13.3, 33.7)	80.3
24-hour average PM_{10} ($\mu g.m^{-3}$)	-4.0	(-50.3, 45.4)	35.6
24-hour average NO_2 (ppb)	26.3	(12.2, 40.7)	0.0
8-hour average CO (ppm)	27.9	(14.6, 43.0)	24.4

* *Estimates from a random effects meta-analysis of cities using distributed lag models, shown for an inter-quartile range increase in pollutant.*
CI=confidence interval, ppb=parts per billion, ppm=parts per million.

Table 6.1.7: Per cent changes in cardiovascular deaths (and 95% confidence interval) by age group over forty-day periods*

Outcome (age group) and pollutant (units)	40-day: Lags 0 to 40		
	Change	95% CI	I2
Cardiovascular deaths (all ages)			
24-hour average $PM_{2.5}$ (µg.m-3)	-1.1	(-5.6, 3.4)	0.0
24-hour average PM_{10} (µg.m-3)	-7.0	(-12.9, -0.9)	0.0
24-hour average NO_2 (ppb)	14.3	(6.9, 21.8)	0.0
8-hour average CO (ppm)	9.2	(3.5, 15.2)	19.0

* *Estimates from a random effects meta-analysis of cities using distributed lag models, shown for an inter-quartile range increase in pollutant.*
CI=confidence interval, ppb=parts per billion, ppm=parts per million.

6.1.1.1 Summary

In pooling the estimates for all seven cities, we found that increases in concentrations of NO_2 are significantly associated with increases in death counts due to all causes, cardiovascular disease and respiratory disease, often significantly, and there is no evidence to suggest there is heterogeneity between the results for the cities.

In pooling the estimates for the Australian cities — Brisbane, Melbourne, Perth, Sydney — we found that increases in particle concentrations (as measured by $PM_{2.5}$) and ozone concentrations (warm period) are associated with increases in death counts due to all causes, cardiovascular disease and respiratory disease, and most of these increases are statistically significant (more for $PM_{2.5}$ than ozone). There is no evidence to suggest there is heterogeneity in the results between the cities.

When NO_2 is matched on particles and with ozone, there is confounding, indicating that these results may be referring to common air pollution 'events', with the pollutants being surrogates either for another pollutant, or for a pollutant 'mix'.

Given there is no evidence to suggest there is heterogeneity in the results for the cities used to derive the significant pooled estimates shown in Table 6.1.1, then these estimates may be used to estimate the effect of pollution in each of the cities; that is:

- the significant pooled estimates in Table 6.1.1 for the associations between increases in maximum 1-hour NO_2 and increases in total all cause mortality, cardiovascular mortality and respiratory mortality can be applied to all the cities — Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney

- the significant pooled estimates in Table 6.1.1 for the associations between increases in 24-hour average PM_{10} and increases in cardiovascular mortality can be applied to the cities Brisbane, Christchurch, Melbourne, Perth and Sydney

- the significant pooled estimates in Table 6.1.1 for the associations between increases in 24-hour average $PM_{2.5}$ and ozone (warm period) and increases in total all cause mortality, cardiovascular mortality and respiratory mortality, can be applied to the cities Brisbane, Melbourne, Perth and Sydney.

6.1.2 Associations with hospital admissions

6.1.2.1 Cardiovascular admissions

The changes in daily cardiovascular hospital admissions are shown in Table 6.1.8 (the table also shows the estimated heterogeneity between cities):

- in the elderly (65 years and greater), stroke was the only disease category that did not show a statistically significant association with a pollutant

- significant associations were found for all pollutants but O_3

- the significant associations with arrhythmia were only found in the 15-64 years age group

- the other significant associations found in the 15-64 years group (cardiac and cardiac failure disease categories) were also found in the elderly group.

Table 6.1.8: Significant increases (per unit increase in pollutant concentration) in cardiovascular hospital admissions in adults and the elderly using a meta-analysis of case-crossover estimates (urban Australia and New Zealand, 1998-2001)

Disease category	Age group (years)	Pollutant (units)	Increase %1 (95% CI)	I2 %†	Missing cities
Arrhythmia	15-64	24-h NO_2 (ppb)	1.0 (0.4, 1.5)	0.0	None
		Max 1-h NO_2 (ppb)	0.4 (0.1, 0.7)	0.0	
		Max 8-h CO (ppm)	2.9 (0.1, 5.7)	5.6	None
Cardiac	65+	24-h $PM_{2.5}$ ($\mu g.m^{-3}$)	0.5 (0.3, 0.7)	55.0	A, Ca, Ch
		24-h PM_{10} ($\mu g.m^{-3}$)	0.1 (0.0, 0.3)	32.9	A, Ca
		24-h NO_2 (ppb)	0.6 (0.4, 0.9)	54.1*	None
		Max 1-h NO_2 (ppb)	0.3 (0.2, 0.4)	34.0	None
		8-hour CO (ppm)	3.3 (1.5, 5.1)	73.5*	None
	15-64	24-hour NO_2 (ppb)	0.4 (0.2, 0.7)	0.0	None
		Max 8-h CO (ppm)	2.0 (0.6, 3.3)	24.7	None
Cardiac failure	65+	24-h $PM_{2.5}$ ($\mu g.m^{-3}$)	0.9 (0.5, 1.4)	58.6	A, Ca, Ch
		24-h PM_{10} ($\mu g.m^{-3}$)	0.4 (0.3, 0.6)	0.0	A, Ca
		24-h NO_2 (ppb)	1.3 (0.4, 2.2)	61.3*	None
		Max 1-h NO_2 (ppb)	0.7 (0.4, 1.0)	50.0	None
		Max 8-h CO (ppm)	7.0 (4.1, 10.0)	61.6*	None
	15-64	24-h NO_2 (ppb)	0.9 (0.0, 1.8)	0.0	None
		Max 8-h CO (ppm)	4.9 (0.7, 9.1)	0.0	None
IHD	65+	24-h $PM_{2.5}$ ($\mu g.m^{-3}$)	0.4 (0.2, 0.6)	3.6	A, Ca, Ch
		24-h NO_2 (ppb)	0.5 (0.2, 0.8)	19.7	None
		Max 1-h NO_2 (ppb)	0.3 (0.2, 0.4)	0.0	None
		Max 8-h CO (ppm)	2.7 (1.1, 4.4)	35.9	None
Myocardial infarction	65+	24-h $PM_{2.5}$ ($\mu g.m^{-3}$)	0.7 (0.3, 1.1)	0.0	A, Ca, Ch
		24-h NO_2 (ppb)	0.8 (0.2, 1.5)	38.2	None
		Max 1-h NO_2 (ppb)	0.4 (0.2, 0.7)	19.7	None
		Max 8-h CO (ppm)	3.3 (0.9, 5.8)	21.3	None
Total cardiovascular	65+	24-h $PM_{2.5}$ ($\mu g.m^{-3}$)	0.3 (0.1, 0.5)	51.9	A, Ca, Ch
		24-h NO_2 (ppb)	0.6 (0.4, 0.8)	18.4	None
		Max 1-h NO_2 (ppb)	0.3 (0.2, 0.4)	3.5	None
		Max 8-h CO (ppm)	2.5 (1.0, 4.0)	69.5*	None
	15-64	24-h NO_2 (ppb)	0.3 (0.1, 0.5)	0.0	None
		Max 1-h NO_2 (ppb)	0.1 (0.0, 0.2)	0.0	None
		Max 8-h CO	1.3 (0.3, 2.4)	6.6	None

[1] Per cent increase in admissions per unit increase in pollutant concentration using the average over the current and previous day

† I-squared is the percentage of total variation in the estimated increase that is due to heterogeneity between cities

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 5% level*

CI=confidence interval; A=Auckland; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth

For those pooled estimates where we found evidence for heterogeneity between cities, we attempted to explain these differences by examining the association between the city-specific effect estimates and city-specific variables (effect modifiers) used in the European studies (APHEA2). The statistically significant effect modifiers found are shown in Table 6.1.9.

The APHEA2 project used the parameter — number of monitors — as an effect modifier, and tests on the results for CO using this parameter found evidence (see Table 6.1.9) to suggest there were differences in associations between cities that could

be linked to the different number of monitors used in each city (the city with the highest number of monitors, Sydney, shows the highest effect and is different to the other cities). This effect modification due to the number of monitors can be interpreted using the regression dilution concept (MacMahon 1990). In regression dilution a true association appears stronger when the exposure is measured with greater accuracy. It would be expected that, as the number of monitors increases, the exposure estimate based on the number of monitors used should improve. It is always a concern that the average exposure estimated in cities with a small number of monitors may lead to spurious results. It is reassuring that the increases for the city with the largest number of monitors (Sydney) is so notably the largest which, following MacMahon (1990), may well reinforce the conclusion that this is a true association.

The effect of 24-hour $PM_{2.5}$ showed a city-level association with rainfall for arrhythmia, and humidity for cardiac admissions. To investigate this modification further, the case-crossover models in each city with available $PM_{2.5}$ data (Brisbane, Melbourne, Perth and Sydney) were re-run including an interaction term for 24-hour $PM_{2.5}$ and rainfall. There was a negative interaction between rainfall and $PM_{2.5}$ in all four cities, and in Sydney the association was highly statistically significant.

Tests for other different city characteristics, such as proportion of elderly in the city population, and different average pollution levels, did identify city characteristics that could explain differences between the results for different cities (see Table 6.1.9). For cardiac failure, cities with a higher proportion of elderly in the city population did show significantly higher $PM_{2.5}$ associations, while for IHD, this occurred in cities with lower average $PM_{2.5}$ concentrations.

Table 6.1.9: City-level effect modifiers of the significant associations between cardiovascular admissions and pollutants in the elderly

Disease category	Pollutant	Effect modifier	# cities	p-value	Interpretation
Cardiac	8-hour CO	# CO monitors	7	0.013	Greater association with CO in cities with more monitors
	24-hour $PM_{2.5}$	Humidity	4	0.023	Greater association with $PM_{2.5}$ in cities with less humidity
Cardiac failure	24-hour $PM_{2.5}$	Barometric pressure	4	0.043	Greater association with $PM_{2.5}$ in cities with higher pressure
	24-hour $PM_{2.5}$	% over 65	4	0.008	Greater association with $PM_{2.5}$ in cities with an older population
IHD	8-hour CO	# CO monitors	7	0.024	Greater association with CO in cities with more monitors
	24-hour $PM_{2.5}$	24-hour $PM_{2.5}$	4	0.043	Greater association with $PM_{2.5}$ in cities with lower average $PM_{2.5}$ levels
Myocardial infarction	8-hour CO	Humidity	7	0.026	Greater association with CO in cities with less humidity

Typical examples of the effects of matching the pollutants with significant associations are shown in Tables 6.1.10 and 6.1.11.

The results indicate that inclusion of CO in the model estimating effects for all the cities confounds the significant associations for NO_2 (and the reverse, but not so much).

The results indicate the inclusion of NO_2 in the model estimating effects for the cities — Brisbane, Melbourne, Perth and Sydney — confounds the significant associations for $PM_{2.5}$, while the inclusion of CO also reduces the $PM_{2.5}$ estimate, but not as much as NO_2.

These results indicate it is highly probable that increases in cardiovascular hospital admissions due to increases to NO_2, CO and particles cannot be added together, as they may often refer to similar air pollution events.

Table 6.1.10: Multi-pollutant models — statistically significant increases in cardiovascular hospital admissions in the elderly (65 years or greater) and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %* (95% CI)	Cities included
Total cardiovascular			
24-hour average NO_2	8-hour average CO (max)	0.5 (-0.9, 1.9)	All
	Unmatched	3.0 (2.1, 3.9)	All
24-hour average $PM_{2.5}$	24-hour average NO_2	0.2 (-1.7, 2.2)	B, M, P, S
	8-h average CO (max)	1.0 (-0.2, 2.3)	B, M, P, S
	Unmatched	1.3 (0.6, 2.0)	B, M, P, S
8-hour average CO (max)	24-hour average NO_2	2.6 (-0.0, 5.3)	All
	Unmatched	2.2 (0.9, 3.4)	All
Cardiac			
24-hour average NO_2	8-haverage CO (max)	0.6 (-1.7, 3.0)	All
	Unmatched	3.4 (1.9, 4.9)	All
24-hour average $PM_{2.5}$	24-hour average NO_2	0.4 (-1.7, 2.5)	B, M, P, S
	8-haverage CO (max)	1.2 (0.0, 2.4)	B, M, P, S
	Unmatched	1.9 (1.0, 2.7)	B, M, P, S
8-hour average CO (max)	24-hour average NO_2	2.8 (0.2, 5.4)	All
	Unmatched	2.8 (1.3, 4.4)	All
Myocardial infarction			
24-h average $PM_{2.5}$	24-h average NO_2	1.5 (-1.3, 4.4)	B, M, P, S
	8-h average CO (max)	2.4 (-0.6, 5.4)	B, M, P, S
	Unmatched	2.7 (1.3, 4.2)	B, M, P, S
24-h average NO_2	8-hour average CO (max)	1.9 (-2.1, 6.1)	All
	Unmatched	4.4 (1.0, 8.0)	All
8-h average CO (max)	24-hour average NO_2	2.3 (-4.0, 9.1)	All
	Unmatched	2.9 (0.8, 4.9)	All

** Per cent increase in admissions for an inter-quartile range (IQR) increase in pollutant using the average over the current and previous day*
A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

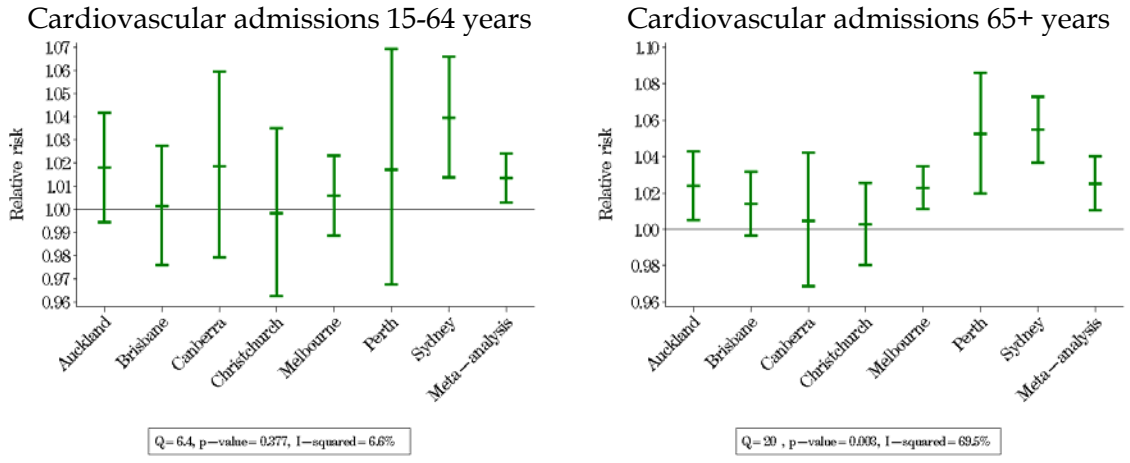
Table 6.1.11: Multi-pollutant models — statistically significant increases in cardiovascular hospital admissions in the 15-64 years age group and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %* (95% CI)	Cities included
Total cardiovascular			
24-h average NO_2	8-h average CO (max)	-0.1 (-1.6, 1.4)	All
	Unmatched	1.7 (0.6, 2.8)	All
8-h average CO (max)	24-h average NO_2	2.0 (-1.8, 5.8)	All
	Unmatched	1.2 (0.3, 2.1)	All
Cardiac failure			
24-h average NO_2	8-h average CO (max)	0.0 (-5.4, 5.8)	All
	Unmatched	4.6 (0.1, 9.3)	All
8-h average CO (max)	24-h average NO_2	2.6 (-7.3, 13.2)	All
	Unmatched	4.2 (0.6, 7.8)	All
Arrhythmia			
24-h average NO_2	8-h average CO (max)	3.8 (0.3, 7.5)	All
	Unmatched	5.1 (2.2, 8.1)	All
8-h average CO (max)	24-h average NO_2	2.5 (-1.6, 6.7)	All
	Unmatched	2.5 (0.1, 4.9)	All

* *Per cent increase in admissions for an inter-quartile range (IQR) increase in pollutant using the average over the current and previous day*
A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney

Figure 6.1.1 shows the estimated increase in cardiovascular admissions for adults and the elderly associated with increases in CO concentrations.

Figure 6.1.1: Case-crossover relative risks and 95% confidence intervals by age group for total cardiovascular admissions and CO (8h max, average lag 0-1)



6.1.2.1.1 Summary

In pooling the estimates for all the cities, we found there is no evidence to suggest there is heterogeneity between the results for the cities for the significant pooled estimates for the following categories of cardiovascular hospital admissions (so the pooled estimates can be applied to all the cities used in the analysis):

a) The associations between increases in NO_2 (maximum 1-hour) and, for 15-64 years age group, increases in total cardiovascular and arrhythmia admissions; for the 65+ years age group, increases in cardiac, cardiac failure, IHD, MI and total cardiovascular hospital admissions (these pooled estimates in Table 6.1.8 can be applied to the cities — Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney).

b) The associations between increases in NO_2 (24-hour average) and, for 15-64 years age group, increases in cardiac, total cardiovascular, cardiac failure, and arrhythmia admissions; for the 65+ years age group, increases in IHD, MI and total cardiovascular hospital admissions (these pooled estimates in Table 6.1.8 can be applied to the cities — Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney).

c) The associations between increases in CO (8-hour maximum) and increases in arrhythmia admissions for 15-64 years age group and, for the 65+ years age group, increases in IHD and MI hospital admissions (these pooled estimates in Table 6.1.8 can be applied to the cities — Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney).

d) The associations between increases in PM_{10} (24-hour average) and, for the 65+ years age group, increases in all cardiac and cardiac failure hospital admissions (these pooled estimates in Table 6.1.8 can be applied to the cities — Brisbane, Christchurch, Melbourne, Perth and Sydney).

e) The associations between increases in $PM_{2.5}$ (24-hour average) and, for the 65+ years age group, increases in IHD, MI and total cardiovascular hospital admissions (these pooled estimates in Table 6.1.8 can be applied to the cities — Brisbane, Melbourne, Perth and Sydney).

In pooling the estimates for the associations between increases in CO in all the cities and cardiac and total cardiovascular hospital admissions for the 65+ years age group, we found evidence to suggest there is heterogeneity in the results for the cities, due to the high associations for Sydney. However, if we accept the regression dilution concept, then the pooled estimates shown in Table 6.1.8 for associations between increases in CO and increases in these health outcomes could well be applied to all the cities — Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney.

For the cases where there is evidence of heterogeneity (as shown in Table 6.1.8) we suggest the following approach:

a) Associations between increases in CO and increases in cardiac, total cardiovascular, and cardiac failure hospital admissions for the 65+years age group be based on the pooled estimates for the set of cities excluding Sydney (per increase in IQR range of CO) in Tables 3.2.3, 3.2.1, and 3.2.6, respectively, and could be applied to the cities – Auckland, Brisbane, Canberra, Christchurch, Melbourne and Perth.

b) Associations between increases in 24-hour average NO_2 and increases in cardiac and cardiac failure hospital admissions for the 65+ years age group be based on the pooled estimates for the set of cities excluding Christchurch (per increase in IQR range of NO_2) in Tables 2.2.3, 2.2.1 and 2.2.6, respectively, and could be applied to the cities — Auckland, Brisbane, Canberra, Melbourne, Perth and Sydney.

6.1.2.2 Respiratory admissions

The changes in daily respiratory hospital admissions associated with increases in air pollution concentrations are shown in Table 6.1.12. No significant associations between CO and respiratory admissions were found, and ozone results for only the warm period are shown.

Table 6.1.12: Significant increases in respiratory hospital admissions using a meta-analysis of case-crossover estimates (urban Australia and New Zealand, 1998-2001)

Age group in years	Cities	Pollutant (units)	Increase %** (95% CI)	I2 %†
Pneumonia & acute bronchitis				
< 1	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.4 (0.0, 0.9	0
1-4	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.6 (0.0, 1.2)	16.3
65+	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.5 (0.2, 0.8)	0.0
	B, Ch, M, P, S	24hr PM_{10} ($\mu g.m^{-3}$)	0.2 (0.0, 0.4)	0.0
Respiratory admissions				
<1	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.6 (0.3, 1.0)	0
1-4	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.4 (0.2, 0.7)	0
	B, Ch, M, P, S	24hr PM_{10} ($\mu g.m^{-3}$)	0.2 (0.1, 0.4)	0
	All	Max 1h NO_2 (ppb)	0.3 (0.1, 0.5)	46.9
	B, M, P, S	Max 8-h O_3 (ppb) (warm)1	0.6 (0.4, 0.8)	0.0
5-14	B, Ch, M, P, S	24hr PM_{10} ($\mu g.m^{-3}$)	0.3 (0.0, 0.5)	0.0
	All	Max 1h NO_2 (ppb)	0.5 (0.2, 0.9)	52.0
	All	24hr NO_2 (ppb)	1.1 (0.3, 1.9)	54.0*
15-64	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.3 (0.0, 0.6)	40.7
	All	Max 1h NO_2 (ppb)	0.1 (0.0, 0.3)	0.0
65+	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.4 (0.2, 0.6)	0.0
Asthma admissions				
1-4	B, M, P, S	Max 8-h O_3 (ppb) (warm)1	0.6 (0.3, 0.9)	0.0
5-14	All	24hr NO_2 (ppb)	1.1 (0.0, 2.2)	61.9*
15-64	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.6 (0.2, 0.9)	0.0
COPD admissions				
65+ years	B, M, P, S	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	0.4 (0.2, 0.7)	0.0

*** Per cent increase in admissions per unit concentration increase in pollutant using the average over the current and previous day*

† I-squared is the percentage of total variation in the estimated increase that is due to heterogeneity between cities

** Statistical test shows there is evidence for heterogeneity (differences between cities) at 95% level*

1 results significant also for max 1h and max 4h ozone

CI=confidence interval; A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

Matched pollutant models were run where significant increases were found with more than one pollutant. Shown in Tables 6.1.13 and 6.1.14 are results from this matched analysis for the child (< 1, 1-4, 5-14 years) age groups and adult (15-64, 65+ years) age groups, respectively.

Table 6.1.13: Multi-pollutant models — statistically significant increases in respiratory hospital admissions in child (< 1, 1-4, 5-14 years) age groups and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %* (95% CI)	Cities included
Age group 1-4 years			
24-hour average PM_{10}	1-hour maximum NO_2	-0.0 (-2.1, 2.1)	B, Ch, M, P, S
	Unmatched	1.7 (0.5, 2.9)	
24-h average $PM_{2.5}$	24-hour average PM_{10}	2.9 (0.2, 5.6)	B, M, P, S
	1-hour maximum NO_2	-1.5 (-3.2, 0.2)	
	1-hour maximum O_3	2.0 (-0.6, 4.8)	
	Unmatched	1.7 (0.7, 2.7)	
1-hour maximum O_3	24-hour average PM_{10}	2.3 (-0.7, 5.3)	B, M, P, S
	24-h average $PM_{2.5}$	5.0 (2.7, 7.3)	
	1-hour maximum NO_2	-1.1 (-3.6, 1.4)	
	Unmatched	1.9 (0.5, 3.4)	
Age group 5-14 years			
24-hour average PM_{10}	24-hour average NO_2	1.2 (-1.8, 4.4)	B, Ch, M, P, S
	Unmatched	1.9 (0.1, 3.8)	

* *Per cent increase in admissions for an IQR increase in pollutant using the average over the current and previous day*

A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

Table 6.1.14: Multi-pollutant models - statistically significant increases in respiratory hospital admissions in adult (15-64, 65+ years) age groups and increases after matching for other exposures (increase in events and 95% confidence intervals)

Single pollutant	Matched exposure	Increase %* (95% CI)	Cities included
Age group 15-64 years			
Asthma			
24-hour average $PM_{2.5}$	24-hour average NO_2	3.2 (0.1, 6.3)	B, M, P, S
	Unmatched	2.2 (0.7, 3.6)	
Total respiratory			
24-hour average $PM_{2.5}$	1-hour maximum NO_2	-0.2 (-2.1, 1.8)	B, M, P, S
	Unmatched	1.1 (0.0, 2.1)	
Age group 65+ years			
COPD			
24-hour average $PM_{2.5}$	24-hour average NO_2	1.1 (-0.5, 2.7)	B, M, P, S
	Unmatched	1.6 (0.6, 2.7)	
Pneumonia & acute bronchitis			
24-hour average PM_{10}	24-hour average NO_2	0.6 (-2.1, 3.4)	B, Ch, M, P, S
	Unmatched	1.8 (0.3, 3.4)	
24-hour average $PM_{2.5}$	24-hour average NO_2	2.0 (0.1, 3.9)	B, M, P, S
	24-hour average PM_{10}	3.7 (1.0, 6.5)	
	Unmatched	2.0 (0.8, 3.2)	
Total respiratory			
24-hour average $PM_{2.5}$	24-hour average NO_2	1.5 (0.1, 2.9)	B, M, P, S
	Unmatched	1.6 (0.9, 2.3)	

* *Per cent increase in admissions for an IQR increase in pollutant using the average over the current and previous day*

A=Auckland; B=Brisbane; Ca=Canberra; Ch=Christchurch; M= Melbourne; P=Perth; S=Sydney.

The inclusion of NO_2 in the models estimating the associations between increases in respiratory hospital admissions and increases in particles (both $PM_{2.5}$ and PM_{10}) and

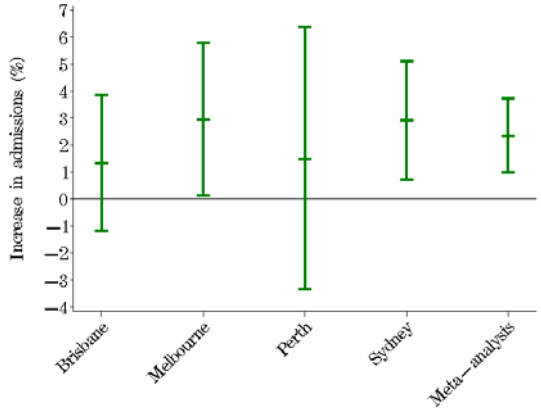
ozone, confounds the associations for both particles for all age groups (in both Tables 6.1.13 and 6.1.14) and ozone for children in the 5–14 years age group (in Table 6.1.13). Sometimes this confounding is a small reduction (see Table 6.1.14 for pneumonia and acute bronchitis), but it can be large (see results for age group 1–4 years in Table 6.1.13).

The results where ozone and particles (both $PM_{2.5}$ and PM_{10}) are matched show little confounding (Table 6.1.13). There is also little confounding between $PM_{2.5}$ and PM_{10} (see Tables 6.1.13 and 6.1.14).

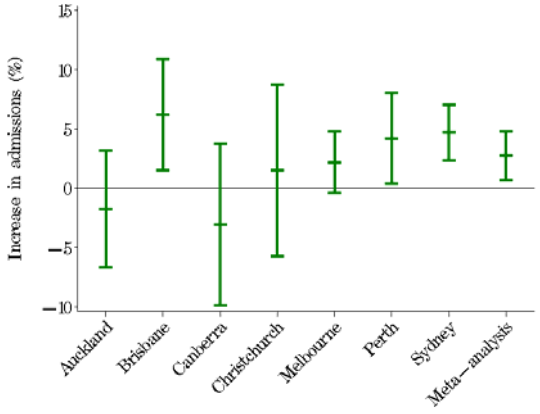
The contrasting increases between cities are apparent in Figure 6.1.2, which shows three of the significant meta-analysis increases for respiratory admissions (age groups 0, 1–4 and 5–14 years).

Figure 6.1.2: Selected statistically significant increases in hospital respiratory admissions in children, city-specific and meta-analysis estimates by age group:

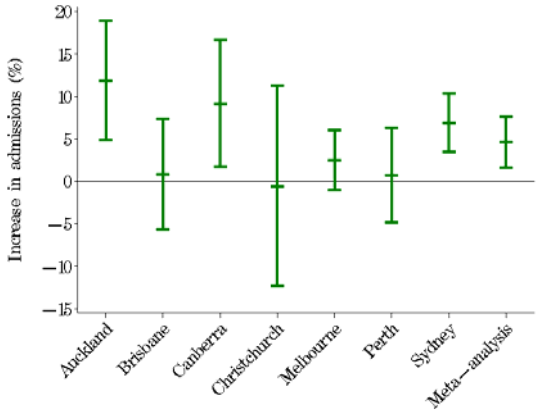
a) Age group <12 months; 24–hour average $PM_{2.5}$ (average lag 0-1)



b) Age group 1–4 years; 1–hour average NO_2 (average lag 0–1)



c) Age group 5-14 years; 1-hour average NO_2 (average lag 0-1)



The significant effects of pollutants were stratified by cool and warm seasons (Table 6.1.15), but only for child age groups where significant ozone associations were identified. Clearly, the ozone significant associations occur in the warm period. There is some evidence, shown in the results in Table 6.1.15, to indicate that there are distinct warm period associations for particles and NO_2 but it is not as obvious as for the ozone associations.

Table 6.1.15: Increases in hospital admissions in children stratified by cool and warm seasons using a meta-analysis of case-crossover estimates (urban Australia and New Zealand, 1998-2001)

		Cool*		Warm*	
Age group in years	**Pollutant (units)**	**Increase† (95% CI)**	**I-squared‡ %**	**Increase† (95% CI)**	**I-squared‡ %**
Pneumonia & acute bronchitis					
< 1	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	1.1 (-0.7, 3.0)	0	2.2 (-3.1, 7.8)	0
1-4	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	2.0 (-0.4, 4.5)	7.1	3.3 (-6.1, 13.6)	58.4
Respiratory admissions					
< 1	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	1.8 (0.2, 3.4)	0	2.8 (-0.9, 6.7)	0
1-4	24hr $PM_{2.5}$ ($\mu g.m^{-3}$)	1.1 (-0.1, 2.3)	0	3.0 (0.8, 5.2)	0
1-4	24hr PM_{10} ($\mu g.m^{-3}$)	0.6 (-0.8, 2.1)	0	3.1 (0.2, 6.0)	27.5
1-4	1hr NO_2 (ppb)	2.7 (0.7, 4.8)	0	3.4 (-1.6, 8.6)	68.3
1-4	1 hr O_3 (ppb)	-2.9 (-7.8, 2.3)	70.7	3.8 (2.3, 5.4)	0
1-4	4 hr O_3 (ppb)	-3.2 (-8.2, 2.0)	73.4	4.3 (2.7, 6.1)	0
1-4	8 hr O_3 (ppb)	-3.1 (-8.0, 2.0)	69.7	5.3 (3.3, 7.3)	0
5-14	24hr PM_{10} ($\mu g.m^{-3}$)	2.1 (-0.2, 4.6)	0	1.9 (-2.4, 6.4)	51.1
5-14	1hr NO_2 (ppb)	5.7 (-1.9, 14.0)	73.7	8.6 (4.0, 13.3)	39.8
5-14	24hr NO_2 (ppb)	6.0 (-0.7, 13.0)	66.0	9.6 (3.3, 16.3)	37.6
Asthma admissions					
1-4	1 hr O_3 (ppb)	-3.1 (-10.9, 5.3)	64.4	5.0 (2.8, 7.2)	0
1-4	4 hr O_3 (ppb)	-3.6 (–10.9, 4.2)	60.9	5.3 (2.9, 7.8)	0
1-4	8 hr O_3 (ppb)	-3.8 (-10.0, 2.9)	49.2	5.5 (2.7, 8.5)	0
5-14	24hr NO_2 (ppb)	7.0 (-2.4, 17.3)	66.8	10.2 (2.6, 18.4)	49.2

* *cool=May to October, warm=November to April*

† *Per cent increase in admissions for an IQR increase in pollutant using the average over the current and previous day*

‡ *I-squared is the percentage of total variation in the estimated increase that is due to heterogeneity between cities*

The only significant effect modification found was that cities with higher average temperatures had greater increases in hospital respiratory admissions in the 1-4 year age group.

6.1.2.2.1 Summary

In pooling the estimates for all the cities we found there is no evidence to suggest there is heterogeneity in the results for the cities for the significant pooled estimates for the following categories of respiratory hospital admissions (so the pooled estimates can be applied to all the cities used in the analysis):

a) The associations between increases in NO_2 (maximum 1-hour) and for the age groups — 1 to 4 years, 5 to 14 years and 15 to 64 years — increases in total respiratory admissions (these pooled estimates in Table 6.1.12 can be applied to the cities — Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney).

b) The associations between increases in $PM_{2.5}$ (24-hour average) and increases in total respiratory hospital admissions for the age groups — less than 1 year, 1 to 4 years, 15 to 64 years, and 65+ years; for increases in pneumonia and acute bronchitis hospital admissions for age groups — less than 1 year, 1 to 4 years, and 65+ years; for increases in asthma hospital admissions for the 15 to 64 years age group; and for increases in COPD hospital admissions in the 65+ years age group (these pooled estimates in Table 6.1.12 can be applied to the cities — Brisbane, Melbourne, Perth and Sydney).

c) The associations between increases in PM_{10} (24-hour average) and increases in total respiratory hospital admissions for the age groups 1 to 4 years and 5 to 14 years, and for increases in pneumonia and acute bronchitis hospital admissions for the 65+ years age group (these pooled estimates in Table 6.1.12 can be applied to the cities –Brisbane, Christchurch, Melbourne, Perth and Sydney).

d) The associations between increases in ozone (warm period) and increases in total respiratory hospital admissions and asthma hospital admissions for the 1 to 4 years age group (these pooled estimates in Table 6.1.12 can be applied to the cities — Brisbane, Melbourne, Perth and Sydney).

There is evidence of heterogeneity in the associations between increases in NO_2 (24-hour average) and respiratory hospital admissions for the 5 to 14 years age group, which does not occur for the Australian cities or when Auckland results are removed.

There is also evidence of heterogeneity in the associations between increases in NO_2 (24-hour average) and asthma hospital admissions for the 5 to 14 years age group, but it is not clear how this might be removed.

6.1.3 Comparisons with other multi-city and meta-analysis studies

The meta-analysis studies overseas for mortality have been for the US (NMMAPS: Samet et al. 2000, 2003; Dominici et al. 2006) and European (APHEA2: Katsouyanni et al. 2001, 2003). These results are compared with those found here in Table 6.1.16 for PM_{10}. Recently, three separate meta-analyses were carried out for ozone and mortality (Bell et al. 2005; Ito et al. 2005; Levy et al. 2005), and these results are also compared with those here (see Table 6.1.16).

Effect sizes for PM_{10} and ozone on all cause mortality found in the present study are larger than those observed in both Europe and the US. Possible explanations for these findings are considered under the usual epidemiological headings.

Chance: The overseas studies are large, providing estimates with narrow confidence intervals. We have pointed out that the confidence intervals of the EPHC estimates are much wider, and it is this imprecision that needs to be considered. If we assume that the overseas estimate of 0.6/0.7% for the increase in total mortality per $10\mu g/m^3$ of PM_{10} is sufficiently precise as to be able to neglect the measurement error, we see that this lies within the 95% confidence interval of (-0.5%, 3.7%) for the EPHC study (the point estimate being 1.6%), albeit towards the lower end (Table 6.1.16). Thus, the EPHC study cannot exclude a 'true' estimate of 1.6%, according to the usual interpretation of confidence intervals. Thus, the PM_{10} findings could be explained by the imprecision in the EPHC estimate. Similar considerations would apply to hospital admissions (Table 6.1.17). Allowing for imprecision in the international estimates reinforces this.

Similarly, the 95% confidence interval for the EPHC estimate of the ozone effect on all cause mortality is (0.0, 2.1), with a point estimate of 1.1%. This also overlaps the international estimates of 0.35%, 0.39% and 0.41%, and suggests the difference between the EPHC and overseas findings may be attributable to chance.

Despite the above observations, it is of some concern that the differences observed between EPHC estimates and those found overseas all favour a greater effect in the Australasian data. This leads to consideration of possible biases.

Bias: It appears unlikely that methodological approaches could be a source of bias. The EPHC study has closely followed the statistical techniques adopted by international studies, and the close collaboration with Professor Joel Schwartz, as well as the advice and access to programs used by other groups, has assured this. In addition, we find similar results within the EPHC study for different approaches, which, in any case, have been found to be robust. We also see consistent results between EPH and the earlier Australian SPIRT Study (Tables 6.1.20, 6.1.21).

Measurement error can act to produce different observed estimates of effects. Measurement error is the difference between the exposure as used in the analysis and the actual exposure distribution in the population. Observed effect estimates are attenuated if measurement error occurs and the amount of attenuation increases with increasing error. If exposure in Australasian populations were somewhat more homogeneous, and monitors carefully chosen to reflect usual exposure, then perhaps the observed exposures may be better measures of actual exposure. This would

apply if the population spent greater periods outdoors, where air pollution is measured. Further comparison of monitoring procedures in both international and the EPHC studies may be useful.

An additional consideration relates to dose response. If a plateau effect occurs, effects are greater at lower doses than higher doses. This would be consistent with the observation of lower concentrations of air pollutants in Australasian cities and the observation of larger effects. Further analyses of the shape of dose-response curves may elucidate this, but it may also be the case that the range of exposures is not sufficient.

Confounding and effect modification: We have observed some evidence of inter-city variability which may be attributed to different behavioural or other environmental factors. These same factors may contribute to differences between Australasian and overseas cities. Limited data are available to the present study to explore this.

Table 6.1.16: Comparison of EPHC results to results from overseas meta-analyses for short-term increases in total mortality (all ages) associated with a 10-unit increase in PM_{10} ($\mu g.m^{-3}$) and ozone (ppb)

Pollutant	Units	Studies	Age group	Increase %	95% CI
PM_{10}	Per 10 $\mu g.m^{-3}$	EPHC	All ages	1.6	(-0.5, 3.7)
		APHEA2 a ^		0.6	(0.4, 0.8)
		Schwartz 10 US cities b ^		0.7	(0.5, 0.8)
Ozone	Per 10 ppb	EPHC	All ages	1.1	(0.0, 2.1)
	Per 10 ppb	Bell et al. (2005)		0.35	(0.22, 0.47)
		Ito et al. (2005)		0.39	(0.26, 0.51)
	Per 10 $\mu g.m^{-3}$	Levy et al. (2005)		0.21*	(0.16, 0.26)

[a] *29 European cities, average lags 0-1*

[b] *10 US cities with daily monitoring for PM_{10}*

^ *Revised analyses – GAM with LOESS and stricter convergence criteria; HEI 2003*

* *equivalent to 0.41% increase per 10 ppb (Levy et al. 2005)*

The other major multi-city studies overseas for hospital admissions have been for US (NMMAPS) and European (APHEA2) cities (HEI 2003; Le Tertre et al. 2002; Atkinson et al. 2001). Most of these studies have published their results as increases in deaths or hospital counts for each 10 $\mu g.m^{-3}$ increase in PM_{10}. The results comparing EPHC and APHEA2 results are summarised in Table 6.1.17. The results here are showing estimates for increases in cardiovascular and respiratory hospital admissions with increases in PM_{10}. Again, confidence intervals for the EPHC estimates include those for APHEA2, despite the difference in point estimates, suggesting imprecision of the EPHC estimates may be the explanation for this difference. However, for cardiac admissions in the elderly, both EPHC and APHEA2 estimates are positive and significant, and there is no evidence to suggest that there is any difference between the results for this EPHC study and the APHEA2 studies.

Table 6.1.17: Comparison of EPHC results to results from overseas meta-analyses for hospital admissions. Increases in health outcomes for a 10 μg.m^{-3} increase in PM_{10} results are shown for the average of lags 0 and 1 unless indicated

	Outcome	Studies	Age group	Increase %	95% CI
CVD admissions	Cardiac	EPHC	65+	1.4	(0.2, 2.6)
		APHEA2 a ^	65+	0.7	(0.4, 0.9)
	IHD	EPHC	65+	0.6	(-0.9, 2.0)
		APHEA2 a ^	65+	0.7	(0.3, 1.2)
Respiratory admissions	Asthma	EPHC	15-64	1.4	(-1.2, 4.1)
		APHEA2 a ^	15-64	1.0	(0.3, 1.8)
	All resp.	EPHC	65+	1.1	(-0.7, 3.0)
		APHEA2 a ^	65+	1.0	(0.7, 1.3)

[a] *8 European cities - Le Tertre et al. 2002, Atkinson et al. 2001*
^ *Revised analyses – GAM with LOESS and stricter convergence criteria; HEI 2003*

In Table 6.1.18 we compare the case crossover results with the GAM models (also discussed in *Volume 1: Project description and methods used*). Given the estimates and the confidence intervals, there is no evidence to suggest that there is any difference between the results using the different methods.

Table 6.1.18: Comparison of case-crossover and GAM results for EPHC study for a one-unit increase in pollutant associated with selected health outcomes (average of lags 0 and 1)

Outcome	Pollutant	Analysis	Increase % (95% CI)
Cardiac admissions (65+ years)	24-hour PM_{10} (μg.m^{-3})	EPHC (case-crossover)	0.1 (0.0, 0.3)
		EPHC (GAM)	0.1 (0.0, 0.2)
IHD admissions (65+ years)	24-hour PM_{10} (μg.m^{-3})	EPHC (case-crossover)	0.1 (-0.1, 0.2)
		EPHC (GAM)	0.1 (-0.1, 0.3)
Respiratory mortality (all ages)	24-hour PM_{10} (μg.m^{-3})	EPHC (case-crossover)	0.1 (-0.6, 0.8)
		EPHC (GAM)	0.2 (-0.0, 0.4)
Cardiovascular mortality (all ages)	24-hour PM_{10} (μg.m^{-3})	EPHC (case-crossover)	0.2 (0.0, 0.3)
		EPHC (GAM)	0.2 (-0.5, 0.9)
Respiratory mortality (all ages)	8-hour O_3 (ppb)	EPHC (case-crossover)	0.3 (-0.0, 0.6)
		EPHC (GAM)	0.2 (-0.0, 0.5)
Cardiovascular mortality (all ages)	8-hour O_3 (ppb)	EPHC (case-crossover)	0.3 (0.1, 0.4)
		EPHC (GAM)	0.1 (-0.0, 0.3)

6.1.4 Comparison between SPIRT and EPHC results

The results for the previous multi-city study for Brisbane, Melbourne, Perth and Sydney (SPIRT study) for 1996 to 1999 (the SPIRT study) are compared with those here (the EPHC study) in Tables 6.1.19, 6.1.20, and 6.1.21 for NO_2, fine particles (as measured by bsp), and ozone, respectively, for (0-1) averages (Simpson et al. 2001; 2005a; 2005b). It was noted from the selected estimates in Table 6.1.18 and in *Volume 1: Project description and methods used*, that there is reasonable agreement between the results arising from case-crossover and GAM models; therefore, any differences between our results here and the SPIRT results may not be due to different modelling.

Given the confidence intervals, there is no evidence to suggest the estimates for the EPHC and SPIRT studies are different for total mortality and cardiac admissions, when the estimates are significant in both studies.

For respiratory admissions, there are clearly some differences between the SPIRT and EPHC case-crossover results for bsp and respiratory admissions that may be due to using case-crossover rather than GAM, especially for Melbourne and Perth. As mentioned in *Volume 1: Project description and methods used,*, the methodology using GAM in the SPIRT study used dummy variables for epidemics based on respiratory admissions data. For the GAM analysis in the EPHC study, smoothers or splines were used to represent epidemic periods, which could be several weeks, to avoid any numerical problems in using dummy variables for such periods. It is usually essential in GAM modelling, especially for respiratory health outcomes, to use such controls to remove the autocorrelation in the data, otherwise autoregressive models will be needed and much of any potential pollutant effect will be difficult to identify. However, one problem in using such controls for respiratory admission health outcomes in the GAM approach is that there will be over-correction, as the dummy variables (based on respiratory admissions) are closely related to the respiratory admissions data used as health outcomes. Such controls are not used for case-crossover as the design allows for control for changes over several weeks, so this problem should be avoided. If we had reliable data on actual epidemics, then these could easily be included in the case-crossover model.

Also, for particles, we did not correct for any bushfire or control burn effects in the EPHC study unlike the SPIRT study, and this may be a problem in Australian cities especially for Melbourne and Perth (this is discussed elsewhere in this document). The results obtained for adding corrections for bushfires in Brisbane (see Chapter 4) indicate that the particle associations may be significantly reduced when this is done. It is notable that there are fewer differences between the case-crossover and GAM approaches for ozone and NO_2 (where the levels should be less sensitive to bushfire or control burn effects — see the results in Chapter 4 for Brisbane).

Even with these differences in approach, the results for respiratory admissions, given the estimates and confidence intervals, indicate that there is no evidence to suggest the estimates for the EPHC and SPIRT studies are different, when both are statistically significant.

It is clear that the pooled estimates for all outcomes for both SPIRT and EPHC studies are more similar than for single cities.

Table 6.1.19: Comparison of results from the EPHC study with the SPIRT study for Brisbane, Melbourne, Perth and Sydney for a one-unit increase in NO_2 (average of lags 0-1)

	NO_2 (1-hour max.)		NO_2 (24-hour av.)	
	EPHC study Increase % (95% CI)	**SPIRT study Increase % (95% CI)**	**EPHC study Increase % (95% CI)**	**SPIRT study Increase % (95% CI)**
Total mortality all ages				
Brisbane	0.5 (0.3,0.8)*	0.2 (-0.1,0.4)	0.9 (0.3,1.4)*	0.1 (-0.3,0.5)
Melbourne	0.0 (-0.1,0.2)	0.1 (-0.0,0.2)	-0.0 (-0.3,0.2)	0.2 (-0.1,0.4)
Perth	0.1 (-0.1,0.4)	0.2 (-0.1,0.4)	0.2 (-0.4,0.7)	0.3 (-0.3,0.8)
Sydney	0.2 (0.1,0.4)*	0.1 (-0.0,0.2)	0.4 (0.1,0.7)*	0.3 (0.1,0.5)*
Meta-analysis†	0.2 (0.0,0.3)*	0.1 (0.0,0.2)*	0.2 (-0.0,0.5)	0.2 (0.1,0.3)*
Respiratory admissions 65+ yrs				
Brisbane	0.4 (0.0,0.8)*	0.3 (-0.1,0.7)	0.6 (-0.1,1.4)	0.1 (-0.6,0.8)
Melbourne	0.1 (-0.1,0.3)	0.2 (-0.0,0.4)	0.3 (-0.1,0.6)	0.2 (-0.1,0.6)
Perth	-0.1 (-0.4,0.2)	-0.4 (-0.7,-0.0)*	-0.4 (-1.2,0.3)	-1.3 (-2.0,-0.5)*
Sydney	0.2 (0.0,0.4)*	0.5 (0.3,0.7)*	0.5 (0.2,0.9)*	1.1 (0.8,1.5)*
Meta-analysis†	0.1 (-0.1,0.3)	0.2 (0.1,0.4)*	0.0 (-0.8,0.9)	0.5 (0.3,0.7)*
Cardiac admissions 65+ yrs				
Brisbane	0.3 (-0.0,0.6)	0.0 (-0.3,0.2)	0.7 (0.2,1.3)*	0.1 (-0.3,0.6)
Melbourne	0.4 (0.2,0.5)*	0.3 (0.2,0.4)*	0.8 (0.5,1.0)*	0.6 (0.4,0.8)*
Perth	0.3 (0.0,0.5)*	0.2 (-0.1,0.4)	1.0 (0.4,1.6)*	0.5 (-0.1,1.0)
Sydney	0.5 (0.3,0.7)*	0.5 (0.3,0.6)*	0.9 (0.6,1.2)*	0.9 (0.7,1.2)*
Meta-analysis†	0.3 (0.2,0.4)*	0.3 (0.2,0.4)*	0.6 (0.4,0.9)*	0.6 (0.5,0.8)*

** Statistically significant*

† EPHC meta-analysis for five cities – Brisbane, Canberra, Melbourne, Perth Sydney (1998-2001); SPIRT meta-analysis for four cities – Brisbane, Melbourne, Perth, Sydney (1996-1999)

Table 6.1.20: Comparison of results from the EPHC study with the SPIRT study for Brisbane, Melbourne, Perth and Sydney for a one-unit ($10^{-4}.m^{-1}$) increase in average 24-hour bsp (average of lags 0-1)

	EPHC study case-crossover	SPIRT study GAM
	% increase (95% CI)	% increase (95% CI)
Total mortality all ages		
Brisbane	16.2 (5.5,27.9)*	7.0 (-0.8,15.5)
Melbourne	0.8 (-2.7,4.4)	0.2 (-3.5,4.1)
Perth	-0.7 (-11.7,11.6)	4.8 (-4.5,15.1)
Sydney	6.7 (0.0,13.8)*	5.9 (0.9,11.1)*
Meta-analysis†	3.3 (-3.9,11.0)	3.1 (0.4,5.9)*
Respiratory admissions 65 + yrs		
Brisbane	17.1 (3.3,32.7)*	18.4 (4.6,34.0)*
Melbourne	5.5 (0.4,10.8)*	-0.7 (-7.5,6.6)
Perth	3.6 (-10.8,20.4)	-19.4 (-29.9,-7.4)*
Sydney	10.9 (2.5,20.0)*	20.8 (14.1,27.9)*
Meta-analysis†	6.5 (-0.8,14.4)	5.9 (1.0,11.0)*
Cardiac admissions 65+ yrs		
Brisbane	2.9 (-7.2,14.1)	3.1 (-5.2,12.3)
Melbourne	9.6 (5.2,14.2)*	10.4 (6.1,14.9)*
Perth	20.5 (6.9,35.9)*	6.6 (-3.1,17.2)
Sydney	12.3 (4.9,20.2)*	12.7 (6.5,19.3)*
Meta-analysis†	9.8 (5.2,14.7)*	9.8 (6.6,13.0)*

** Statistically significant*
† EPHC meta-analysis for five cities – Brisbane, Canberra, Melbourne, Perth Sydney (1998-2001); SPIRT meta-analysis for four cities – Brisbane, Melbourne, Perth, Sydney (1996-1999)

Table 6.1.21: Comparison of results from the EPHC study with the SPIRT study for Brisbane, Melbourne, Perth and Sydney for a one-unit (ppb) increase in maximum 1-hour O_3 (lag 0-1)

	EPHC study case-crossover	SPIRT study GAM
	% Increase (95% CI)	% Increase (95% CI)
Total mortality all ages		
Brisbane	0.3 (0.1, 0.4) *	0.1 (-0.1, 0.2)
Melbourne	0.0 (-0.1, 0.1)	0.0 (-0.1, 0.1)
Perth	0.1 (-0.1, 0.3)	0.1 (-0.1, 0.2)
Sydney	0.2 (0.1, 0.2) *	0.0 (-0.1, 0.1)
Meta-analysis†	0.1 (0.0, 0.2)*	0.0 (-0.0, 0.1)

** Statistically significant*
†EPHC meta-analysis for four cities – Brisbane, Melbourne, Perth Sydney (1998-2001); SPIRT meta-analysis for four cities – Brisbane, Melbourne, Perth, Sydney (1996-1999)

6.1.5 Pooled estimates for cities

Table 6.1.22 summarises the significant associations between increases in each mortality health outcome and increases in concentrations (lag 0-1) for each air pollutant.

Table 6.1.22: Mortality outcomes showing significant increases associated with increases in air pollutants — by age group — and the cities where there is no evidence for heterogeneity between results

Pollutant	All ages	75+ years	Cities
NO_2 (maximum 1-h average)	All cause mortality Total cardiovascular Total respiratory	All cause mortality Total cardiovascular Total respiratory	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
Carbon monoxide (maximum 8-h average)	- *	- *	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
PM_{10} (24-h average)	Total cardiovascular	Total cardiovascular	Brisbane, Canberra, Melbourne, Perth, Sydney
$PM_{2.5}$ (24-h average)	All cause mortality Total cardiovascular	All cause mortality Total cardiovascular Total respiratory	Brisbane, Melbourne, Perth, Sydney
Ozone1 (warm)2	All cause mortality Total cardiovascular	Total cardiovascular	Brisbane, Melbourne, Perth, Sydney

[1] *Same for maximum 1-h, 4-h, 8-h averages*
[2] *Ozone levels for warm period of year (November-April)*
* *Carbon monoxide generally was associated with increases, but they were not statistically significant*

Given there is no evidence for heterogeneity between the results for cities in each group of cities, the pooled estimates in Table 6.1.1 can be used for each city in the groups shown in Table 6.1.22.

Tables 6.1.23 and 6.1.24 summarise the significant associations between increases in the concentrations of each air pollutant and increases in hospital admissions due to cardiovascular disease for adults (15 years or older) and due to respiratory disease, respectively.

Table 6.1.25 summarises the significant associations between increases in the concentrations of each air pollutant and increases in hospital admissions due to respiratory disease for children (aged less than 15 years).

Table 6.1.23: Significant increases in hospital admissions due to cardiovascular disease for adults (15 years and older) associated with increases in air pollutant concentrations

Pollutant	15-64 years	65+ years	Cities
Nitrogen dioxide (max 1-h)	Total cardiovascular, arrhythmia	Total cardiovascular, all cardiac, cardiac failure, IHD*, MI*	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
Nitrogen dioxide (av. 24-h)	Total cardiovascular, all cardiac, cardiac failure, arrhythmia	Total cardiovascular[2], all cardiac[2], cardiac failure[2], IHD*, MI*	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
Carbon monoxide (max 8-h)	Total cardiovascular, all cardiac, cardiac failure, arrhythmia	Total cardiovascular[3], all cardiac3, cardiac failure[3], IHD*, MI*	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
PM_{10} (av. 24-h)	-	All cardiac, cardiac failure	Brisbane, Christchurch, Melbourne, Perth, Sydney
$PM_{2.5}$ (av. 24-h)	-	Total cardiovascular, all cardiac, cardiac failure, IHD*, MI*	Brisbane, Melbourne, Perth, Sydney

** IHD = ischemic heart disease; MI = myocardial infarction*

[2] Evidence for heterogeneity for these outcomes with Christchurch results different and showing no effect

[3] Evidence for heterogeneity for these outcomes with Sydney results higher than the other cities, but pooled estimates for the remaining cities still significant and positive

Apart from some results for carbon monoxide and 24-hour average nitrogen dioxide, the results in Table 6.1.8 indicate there is no evidence for heterogeneity between the results for cities in each group of cities, so the pooled estimates in Table 6.1.8 can be used for each city in each city group shown in Table 6.1.23.

There is also evidence for heterogeneity between cities for the nitrogen dioxide results, with the pooled estimates possibly overestimating the associations with increases in cardiovascular disease in the elderly (especially for cardiac failure) in Christchurch.

Table 6.1.24: Significant increases in hospital admissions due to respiratory disease for adults (15 years and older) associated with increases in air pollutant concentrations

Pollutant	15-64 years	65+ years	Cities
Nitrogen dioxide (max 1-h)	Total respiratory	-	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
Nitrogen dioxide (av. 24-h)	-	-	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
PM_{10} (av. 24-h)	-	Pneumonia & acute bronchitis	Brisbane, Christchurch, Melbourne, Perth, Sydney
$PM_{2.5}$ (av. 24-h)	Total respiratory, asthma	Total respiratory, COPD*, pneumonia & acute bronchitis	Brisbane, Melbourne, Perth, Sydney

** COPD = chronic obstructive pulmonary disease*

Noting the results in Table 6.1.12, it is clear that there is no evidence for heterogeneity between the results for cities in each group of cities, so the pooled estimates for elderly respiratory hospital admissions in Table 6.1.12 can be used for each city in each city group shown in Table 6.1.24.

Table 6.1.25: Significant increases in hospital admissions due to respiratory disease for children (aged less than 15 years) associated with increases in air pollutant concentrations

Pollutant	<1 year	1 -4 years	5-14 years	Cities
Nitrogen dioxide (max 1-h)	-	Total respiratory	Total respiratory	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
Nitrogen dioxide (av. 24-h)	-	-	Total respiratory[2], asthma[2]	Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth, Sydney
PM_{10} (av. 24-h)	-	Total respiratory	Total respiratory	Brisbane, Christchurch, Melbourne, Perth, Sydney
$PM_{2.5}$ (av. 24-h)	Total respiratory, pneumonia & acute bronchitis	Total respiratory, pneumonia & acute bronchitis	-	Brisbane, Melbourne, Perth, Sydney
Ozone (warm)1	-	Total respiratory, asthma	-	Brisbane, Melbourne, Perth, Sydney

[1] *Ozone levels for warm period of year (November-April)*
[2] *Evidence for heterogeneity for these outcomes with Auckland results higher than the other cities, but pooled estimates for the remaining cities still significant and positive*

Apart from results for 24-hour average nitrogen dioxide, the results in Table 6.1.12 indicate there is no evidence for heterogeneity between the results for cities in each group of cities, so the pooled estimates in Table 6.1.12 can be used for each city in each city group shown in Table 6.1.25.

6.2 Comparison with single city studies

In examining the results for single cities and comparing them to other single city studies, it is important to note the amount of data collected for each pollutant in each city. It is clear from Table 6.2.1 that PM data is restricted for Auckland, Canberra and Christchurch. It should also be noted that the PM_{10} data set in Christchurch has less than 3 years of data in it. All individual city estimates for short-term associations are shown in *Volume 3: Appendix A3.1.*

It is always difficult to obtain estimates for cities with small populations or with a limited air pollution monitoring network. One of the advantages of the multi-city approach is that we can pool data from a wide range of cities to obtain an average estimate, which can then be applied to all the cities. The best estimate for each single city in the EPHC study is the pooled estimate based on using the results for all the cities, unless there is evidence for heterogeneity in the results, and especially when the results for the city under study may be giving rise to that heterogeneity.

The previous sections indicated where this might be occurring. In this section, the pooled estimates will be compared with those from single city studies, and the latter will also be compared with the individual city estimates derived here. (see earlier in this document)

Table 6.2.1: Number of readings for daily pollutant concentrations for the different cities for January 1998 – December 2001

	Auckland	Brisbane	Canberra	Christchurch	Melbourne	Perth	Sydney
O_3 (ppb)							
8 hour	1439	1461	1197		1454	1461	1461
4 hour	1439	1461	1197		1454	1461	1461
1 hour	1439	1461	1197		1454	1461	1461
NO_2 (ppb)							
24 hour	1455	1461	1275	1328	1455	1461	1461
1 hour	1455	1461	1275	1328	1455	1461	1461
CO (ppm)							
8 hour	1249		1302	1461	1448	1461	1461
1 hour	1249		1302	1461	1448	1461	1461
bsp (10^{-4} .m^{-1})							
24 hour	517	1461	1087		1456	1360	1461
1 hour	517	1461	1087		1456	1360	1461
PM_{10} ($\mu g.m^{-3}$)							
24 hour	287	1461	212	1065	1457	1461	1461
1 hour		1461		1065	1457	1461	1461
$PM_{2.5}$ ($\mu g.m^{-3}$)							
24 hour	274	1337			1450	1461	1458
1 hour		1337			1450	1461	1458

6.2.1 Mortality counts

Brisbane

The pooled estimate results in Table 6.1.1 for mortality for all ages indicate that, for Brisbane:

- increases in (maximum 1-hour) NO_2, $PM_{2.5}$, and ozone (warm period) are significantly associated with increases in mortality for all causes and due to all cardiovascular disease
- increases in (maximum 1-hour) NO_2 are significantly associated with increases in mortality due to all respiratory disease
- increases in PM_{10} are significantly associated with increases in mortality due to all cardiovascular disease.

Tables 6.1.19 to 6.1.21 also compare the results for single city estimates derived in the SPIRT study with those in the EPHC study. For total all cause mortality (all ages), the EPHC estimates for the associations with NO_2 (24-hour and 1-hour) particles and ozone are higher (positive and statistically significant) than for the SPIRT results

(positive and insignificant) but, given the confidence intervals, there is no evidence to show the estimated increases are different. The pooled results in Table 6.1.1 for mortality indicate that increases in concentrations of ozone (warm period), $PM_{2.5}$, PM_{10} and (maximum 1-hour) NO_2 are significantly associated with increases in mortality for all cause, cardiovascular and respiratory disease, and these results are also applicable to Brisbane.

Table 6.2.2 compares the results here with a previous study for Brisbane. The analysis carried out by Simpson et al. (1997) used quite different time series modelling to the GAMS approach adopted in APHEA2 (Katsouyanni et al. 2001). The approach used harmonic analysis or trigonometric analysis adopted in APHEA1 (Katsouyanni et al. 1996) and controlled for weather using linear functions and at only one optimal lag. The APHEA2 approach is viewed as a superior to the APHEA1 approach in that general functions for the weather variables and in adjusting for time allows more options in controlling for temporal and weather effects. A comparison of these two techniques in the Melbourne study (EPA Victoria 2000) indicated that the GAM results showed similar estimates to the trigonometric approach but there were fewer significant results.

Table 6.2.2: Relative risk of mortality associated with air pollutants in Brisbane — comparison of results from the current EPHC study to a previous study in Brisbane* (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total mortality						
	1h O_3 (ppb)	All ages	0	EPHC	1.017	(1.004, 1.030)
	1h O_3 (ppb)	All ages	0	Brisbane*	1.016	(1.006,1.026)
	24h bsp (10^{-5}/m)	All ages	0	EPHC	1.014	(1.006, 1.023)
	24h bsp (10^{-5}/m)	All ages	0	Brisbane*	1.009	(1.003, 1.015)
	1h bsp (10^{-5}/m)	All ages	0	EPHC	1.004	(1.002, 1.007)
	1h bsp (10^{-5}/m)	All ages	0	Brisbane*	1.002	(1.000,1.004)
Cardiovascular mortality						
	1h bsp (10^{-5}/m)	All ages	0	EPHC	1.005	(1.001, 1.009)
	1h bsp (10^{-5}/m)	All ages	0	Brisbane*	1.004	(1.001, 1.008)

** Simpson et al. 1997. Results shown for 0_3 for a 10 ppb increase, and bsp per 1 unit (10^{-5}/m) increase*

Christchurch

Table 6.2.3 compares the results here with a previous study for Christchurch. The approach adopted by Hales et al. (2000) used a similar approach to APHEA1 (a harmonic analysis) so the time series approach is different to the GAMS approach which should yield more accurate results. However, Hales et al. (2000) used 5.5 years of mortality data compared to 4 years here, and there was also 5.5 years of PM_{10} data, compared to less than 3 years here. The significant EPHC pooled estimates applicable to Christchurch are shown in Table 6.1.1 (summarised in Table 6.1.22) and show also that increases in (maximum 1-hour) NO_2 are significantly

associated with increases in mortality for all cause, cardiovascular and respiratory disease.

Table 6.2.3: Relative risk of mortality associated with air pollutants in Christchurch - comparison of results from the current EPHC study to a previous study in Christchurch* (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	Increase %	95% CI
Total mortality						
	24h PM_{10} ($\mu g.m^{-3}$)	All ages	1	EPHC (single city)	1.1	(-0.5, 2.8)
			0-1	EPHC (pooled)	1.6	(-0.5, 3.7)
	24h PM_{10} ($\mu g.m^{-3}$)		1	Christchurch*	1.0	(0.5, 2.2)

**Hales et al. 2000. Results are shown for a 10-unit increase.*

Melbourne

Tables 6.1.19 to 6.1.21 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. For total all cause mortality (all ages), both the SPIRT and EPHC estimates for the associations with NO_2 (24-hour and 1-hour), particles and ozone, are positive and insignificant.

Table 6.2.4: Relative risk of mortality associated with air pollutants in Melbourne - comparison of results from the current EPHC study to a previous study in Melbourne* (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total mortality						
	1h O_3 (ppb)	All ages	0	EPHC	1.0012	(1.0003,1.0022)
	4h O_3 (ppb)	All ages	0	Melbourne*	1.0006	(1.0000,1.0012)
	24h NO_2 (ppb)	All ages	1	EPHC	1.0005	(0.9987,1.0024)
	24h NO_2 (ppb)	All ages	1	Melbourne*	1.0016	(1.004,1.0028)
	1h NO_2 (ppb)	All ages	1	EPHC	1.0006	(0.9997,1.0016)
	1h NO_2 (ppb)	All ages	1	Melbourne*	1.0006	(1.0000,1.0012)
Respiratory mortality						
	1h O_3 (ppb)	All ages	0	EPHC	1.0015	(0.9981,1.0049)
	1h O_3 (ppb)	All ages	0	Melbourne*	1.0023	(1.0001,1.0045)
Cardiovascular mortality						
	24h NO_2 (ppb)	All ages	1	EPHC	1.0025	(0.9995,1.0054)
	24h NO_2 (ppb)	All ages	1	Melbourne*	1.0021	(1.0003,1.0039)

** EPA Victoria 2000*

The EPHC pooled estimate results in Table 6.1.1 for mortality for all ages indicate that, for Melbourne:

- increases in (maximum 1-hour) NO_2, $PM_{2.5}$, and ozone (warm period) are significantly associated with increases in mortality for all causes and due to all cardiovascular disease

- increases in (maximum 1-hour) NO_2 are significantly associated with increases in mortality due to all respiratory disease

- increases in PM_{10} are significantly associated with increases in mortality due to all cardiovascular disease.

Perth

Tables 6.1.19 to 6.1.21 also compare the results for single city estimates derived in the SPIRT study with those in the EPHC study. For total all cause mortality (all ages), both the SPIRT and EPHC estimates for the associations with NO_2 (24-hour and 1-hour), particles and ozone, are positive and insignificant.

In the Perth study (DoE 2003), O_3 was found to be significantly associated with an increase in cardiovascular deaths for all ages for the period, 1992-1997. Table 6.2.5 indicates that the results derived here for the period 1998–2001 show a similar estimate but it is not statistically significant.

The pooled estimate results in Table 6.1.1 for mortality for all ages indicate that, for Perth:

- increases in (maximum 1-hour) NO_2, $PM_{2.5}$, and ozone (warm period) are significantly associated with increases in mortality for all causes and due to all cardiovascular disease

- increases in (maximum 1-hour) NO_2 are significantly associated with increases in mortality due to all respiratory disease

- increases in PM_{10} are significantly associated with increases in mortality due to all cardiovascular disease.

Table 6.2.5: Relative risk of mortality associated with air pollutants in Perth — comparison of results from the current EPHC study to a previous study in Perth* (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Cardiovascular mortality						
	8h O_3 (ppb)	All ages	0-1	EPHC	1.0027	0.9982,1.0072
	8h O_3 (ppb)	All ages	0-1	Perth*	1.0042	1.0006,1.0079

* *DoE 2003*

Sydney

The pooled estimate results in Table 6.1.1 for mortality for all ages indicate that, for Sydney:

- increases in (maximum 1-hour) NO_2, $PM_{2.5}$, and ozone (warm period) are significantly associated with increases in mortality for all causes and due to all cardiovascular disease
- increases in (maximum 1-hour) NO_2 are significantly associated with increases in mortality due to all respiratory disease
- increases in PM_{10} are significantly associated with increases in mortality due to all cardiovascular disease.

Tables 6.1.19 to 6.1.21 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. For total all cause mortality (all ages), both the SPIRT and EPHC estimates for the associations with NO_2 (24-hour), particles and ozone, are positive and significant, but only significant for 1-hour NO_2 and ozone in the EPHC study.

The Sydney study by Morgan et al. (1998a) for 1989-1993 used earlier filtering techniques than the trigonometric methods and found significant associations between particles (as measured by bsp), ozone, and NO_2 with all cause mortality, and between particles and cardiovascular mortality.

6.2.2 Cardiovascular hospital admissions

Brisbane

The pooled estimate results in Table 6.1.23 for cardiovascular hospital admissions in the elderly indicate that, for Brisbane:

- increases in (24-hour average and maximum 1-hour) NO_2, $PM_{2.5}$, and CO are significantly associated with increases in hospital admissions due to all cardiovascular disease, all cardiac disease, cardiac failure, IHD and MI

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all cardiac disease and cardiac failure.

Tables 6.1.19 to 6.1.20 compare the results for single city estimates derived in the SPIRT study with the EPHC study. Both the SPIRT and EPHC estimates for the associations with particles are positive and insignificant; the results for NO_2 (both 1-hour and 24-hour) are stronger for EPHC than SPIRT (but only significant for 24-hour NO_2). There were no significant cardiovascular admissions results in the previous study (Petroeschevsky et al 2001) for Brisbane.

Christchurch

The pooled estimate results in Table 6.1.23 for cardiovascular hospital admissions in the elderly indicate that, for Christchurch:

- increases in (maximum 1-hour) NO_2 and CO are significantly associated with increases in hospital admissions due to all cardiovascular disease, all cardiac disease, cardiac failure, IHD and MI

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all cardiac disease and cardiac failure.

The study by McGowan et al. (2002) showed significant associations between cardiac admissions and PM_{10} (lag 0) for the 65+ years age group for the period 1988-1998. The study used GAM methodology but not the same as that adopted in the APHEA and NMMAPS studies. However, the 10-year period should yield more accurate results than that using the 3 years here, and there are differences as shown in Table 6.2.6.

Table 6.2.6: Relative risk for cardiovascular hospital admissions associated with air pollutants in Christchurch - comparison of results from the current EPHC study to a previous study in Christchurch* (Only significant results shown)

Pollutant	Age group	Lag	Studies	Increase %	95% CI
Total cardiac admissions					
24h PM_{10} (µg.m-3) per 7.5 µg.m-3 increase	65+	(0-1)	EPHC (pooled)	1.1	(0.2,2.0)
24h PM_{10} (µg.m-3) per 10 µg.m^{-3} increase	65+	2	Christchurch*	1.22	(0.11,2.33)

**McGowan et al. 2002*

The pooled results in Table 6.1.9 for cardiovascular hospital admissions indicate that increases in CO, PM_{10} and (maximum 1-hour) NO_2 are significantly associated with increases in admissions for a range of cardiovascular disease categories (including all cardiac) and these results are also applicable to Christchurch**.**

Melbourne

The pooled estimate results in Table 6.1.23 for cardiovascular hospital admissions in the elderly indicate that, for Melbourne:

- increases in (24-hour average and maximum 1-hour) NO_2, $PM_{2.5}$, and CO are significantly associated with increases in hospital admissions due to all cardiovascular disease, all cardiac disease, cardiac failure, IHD and MI

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all cardiac disease and cardiac failure.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. For Melbourne, both the SPIRT and EPHC estimates for the associations with NO_2 and particles are similar in magnitude, positive and significant.

The GAM approach used in the Melbourne study (EPA Victoria 2001) for the period 1994-1997 is similar to that used here and the results in Table 6.2.7 show the results here are similar.

Table 6.2.7: Relative risk for cardiovascular hospital admissions associated with air pollutants in Melbourne - comparison of results from the current EPHC study to a previous study in Melbourne * (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total cardiovascular admissions						
	24h bsp ($10^{-4}.m^{-1}$)	65+	1	EPHC	1.0261	(0.9962,1.0569)
	24h bsp ($10^{-4}.m^{-1}$)	65+	1	Melbourne*	1.0560	(1.0208,1.0924)
	24h NO_2 (ppb)	65+	0-1	EPHC	1.0060	(1.0037,1.0083)
	24h NO_2 (ppb)	65+	3 day av	Melbourne*	1.0045	(1.0023,1.0067)
	8h CO (ppm)	65+	0-1	EPHC	1.0179	(1.0060,1.0299)
	8h CO (ppm)	65+	3 day av	Melbourne*	1.0329	(1.0185,1.0476)
IHD admissions						
	24h bsp ($10^{-4}.m^{-1}$)	65+	0	EPHC	1.0682	(1.0133,1.1260)
	24h bsp ($10^{-4}.m^{-1}$)	All ages	0	Melbourne*	1.0631	(1.0188,1.1094)
	1h bsp ($10^{-4}.m^{-1}$)	65+	0	EPHC	1.0217	(0.9972,1.0469)
	1h bsp ($10^{-4}.m^{-1}$)	All ages	0	Melbourne*	1.0297	(1.0090,1.0509)
	24h NO_2 (ppb)	65+	0	EPHC	1.0048	(1.0014,1.0081)
	24h NO_2 (ppb)	All ages	0	Melbourne*	1.0037	(1.0013,1.0061)
	8h CO (ppm)	65+	0-1	EPHC	1.0214	(1.0012,1.0421)
	8h CO (ppm)	All ages	3 day av	Melbourne*	1.0368	(1.0180,1.0558)

** EPA Victoria 2001*

Perth

The pooled estimate results in Table 6.1.23 for cardiovascular hospital admissions in the elderly indicate that, for Perth:

- increases in (24-hour average and maximum 1-hour) NO_2, $PM_{2.5}$, and CO are significantly associated with increases in hospital admissions due to all cardiovascular disease, all cardiac disease, cardiac failure, IHD and MI
- increases in PM_{10} are significantly associated with increases in hospital admissions due to all cardiac disease and cardiac failure.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. For Perth, the SPIRT and EPHC estimates for the associations with NO_2 and particles are positive, but larger for the EPHC study (and significant).

The previous Perth study (DoE 2003) also used a case-crossover approach but a smaller window than here, and Table 6.2.8 indicates similar results.

Table 6.2.8: Relative risk for cardiovascular hospital admissions associated with air pollutants in Perth - comparison of results from the current EPHC study to a previous study in Perth * (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total cardiovascular admissions						
	24h NO_2 (ppb)	65+	1	EPHC	1.0064	(1.0022,1.0106)
	24h NO_2 (ppb)	65+	1	Perth*	1.0047	(1.0013,1.0081)
	1h NO_2 (ppb)	65+	1	EPHC	1.0029	(1.0011,1.0046)
	1h NO_2 (ppb)	65+	1	Perth*	1.0016	(1.0001,1.0031)

** DoE 2003*

Sydney

The pooled estimate results in Table 6.1.23 for cardiovascular hospital admissions in the elderly indicate that, for Sydney:

- increases in (24-hour average and maximum 1-hour) NO_2, $PM_{2.5}$, and CO are significantly associated with increases in hospital admissions due to all cardiovascular disease, all cardiac disease, cardiac failure, IHD and MI

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all cardiac disease and cardiac failure.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. For Sydney, both the SPIRT and EPHC estimates for the associations with NO_2 and particles are similar in magnitude, positive and significant.

The Morgan et al. study (1998b) for the period 1990-1994 derived significant associations between counts for admissions due to heart disease (ICD-9 codes: 410, 413, 427, 428) and particles (as measured by bsp) and NO_2 (both at lag 0), especially for the 65+ years age group. The study concluded that NO_2 has a stronger association than particles.

The pooled results in Table 6.1.9 for cardiovascular hospital admissions indicate that increases in CO, PM_{10}, $PM_{2.5}$ and (maximum 1-hour) NO_2 are significantly associated with increases in admissions for a range of cardiovascular disease categories, and these results are also applicable to Sydney.

6.2.3 Respiratory hospital admissions

Significant associations were found for individual cities for respiratory hospital admissions from the analysis of the short-term (average of same and previous day pollution) effects of air pollution.

Brisbane

The pooled estimate results in Tables 6.1.24 and 6.1.25 for respiratory hospital admissions indicate that, for Brisbane:

- increases in $PM_{2.5}$ are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups less than 1 year, 1-4 years, 15-64 years and 65+ years; increases in asthma hospital admissions for 15-64 years age group; increases in COPD admissions for 65+years age group; and increases in hospital admissions due to pneumonia and acute bronchitis for the age groups less than 1 year, 1-4 years and 65+ years

- increases in NO_2 are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years (only max 1-hour NO_2), 5-14 years (only max 1-hour NO_2), and 15-64 years (only max 1-hour NO_2); increases in asthma hospital admissions for 5-14 years age group (only 24-hour NO_2)

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years and 5-14 years; and increases in hospital admissions due to pneumonia and acute bronchitis for the age group 65+ years

- increases in ozone (warm period) are significantly associated with increases in hospital admissions due to all respiratory disease and due to asthma in the age group 1-4 years.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. For Brisbane, both the SPIRT and EPHC estimates for the associations with particles are similar in magnitude, positive and significant. For NO_2, the EPHC estimates are larger (and significant for 1-hour NO_2, although there is little difference).

Petroeschevsky et al. (2001) carried out a study for the period 1987-1994 using the APHEA1 approach (trigonometric filtering), and found significant associations (see Table 6.2.9) between ozone concentrations and hospital admissions for respiratory disease (age groups 15-64 years at lag 2, 65 years and greater at lag 3), and for asthma (0-14 years at lag 1; 15-64 years at lag 2). There was also a significant association for particles (5 day average for 1 hour maximum; as measured by bsp) and respiratory admissions (15-64 years).

The pooled results in Tables 6.1.13 and 6.1.14 for respiratory hospital admissions indicate that increases in ozone (warm period), PM_{10}, $PM_{2.5}$ and (maximum 1-hour) NO_2 are significantly associated with increases in admissions for a range of respiratory disease categories (including all respiratory, asthma, COPD, pneumonia

and acute bronchitis) for both children and adult age groups, and these results are also applicable to Brisbane.

Table 6.2.9: Relative risk for respiratory hospital admissions associated with air pollutants in Brisbane - comparison of results from the current EPHC study to a previous study in Brisbane * (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total respiratory admissions						
	8h O_3 (pphm)	15-64	2	EPHC	1.044	(1.017,1.071)
	8h O_3 (pphm)	15-64	2	Brisbane*	1.045	(1.013,1.079)
	8h O_3 (pphm)	65+	3	EPHC	0.994	(0.969,1.020)
	8h O_3 (pphm)	65+	3	Brisbane*	1.054	(1.016,1.094)
Asthma admissions						
	8h O_3 (pphm)	15-64	2	EPHC	1.077	(1.028,1.128)
	8h O_3 (pphm)	15-64	2	Brisbane*	1.084	(1.037,1.133)

** Petroeschevsky et al. 2001*

Christchurch

The pooled estimate results in Tables 6.1.24 and 6.1.25 for respiratory hospital admissions indicate that, for Christchurch:

- increases in NO_2 are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years (only max 1-hour NO_2), 5-14 years (maximum 1-hour NO_2), and 15-64 years (only max 1-hour NO_2); increases in asthma hospital admissions for 5-14 years age group (only 24-hour NO_2)

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years and 5-14 years; and increases in hospital admissions due to pneumonia and acute bronchitis for the age group 65+ years

- increases in ozone (warm period) are significantly associated with increases in hospital admissions due to all respiratory disease and due to asthma in the age group 1-4 years.

The McGowan et al. study (2002) found significant associations between PM_{10} (lag 2) and hospital admissions for respiratory admissions (age groups 0-14 years, 15-64 years, 65+ years), and for asthma, chronic lung diseases (ICD codes: 490-2, 494-6), pneumonia/influenza (480-7), and acute respiratory infections (460-6). Generally, the EPHC results were lower than those for the McGowan et al. study.

Table 6.2.10: Relative risk for total respiratory hospital admissions associated with air pollutants in Christchurch - comparison of results from the current EPHC study to a previous study in Christchurch* (Only significant results shown)

Pollutant	Age group	Lag	Studies	Increase %	95% CI
24h PM_{10} ($\mu g.m^{-3}$) per 7.5 $\mu g.m^{-3}$ increase	1-4	0-1	EPHC (pooled)	1.7	(0.5, 2.9)
24h PM_{10} ($\mu g.m^{-3}$) per 7.5 $\mu g.m^{-3}$ increase	5-14	0-1	EPHC (pooled)	1.9	0.1, 3.8
24h PM_{10} ($\mu g.m^{-3}$) per 10 $\mu g.m^{-3}$ increase	0-14	2	Christchurch*	3.62	2.34,4.90
24h PM_{10} ($\mu g.m^{-3}$) per 7.5 $\mu g.m^{-3}$ increase	15-64	0-1	EPHC (pooled)	0.9	-0.1, 0.9
24h PM_{10} ($\mu g.m^{-3}$) per 10 $\mu g.m^{-3}$ increase	15-64	2	Christchurch*	3.39	1.85,4.93
24h PM_{10} ($\mu g.m^{-3}$) per 7.5 $\mu g.m^{-3}$ increase	65+	0-1	EPHC (pooled)	0.8	(-0.5, 2.2)
24h PM_{10} ($\mu g.m^{-3}$) per 10 $\mu g.m^{-3}$ increase	65+	2	Christchurch*	2.86	1.23,4.49

**McGowan et al. 2002*

Melbourne

The pooled estimate results in Tables 6.1.24 and 6.1.25 for respiratory hospital admissions indicate that, for Melbourne:

- increases in $PM_{2.5}$ are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups less than 1 year, 1-4 years, 15-64 years and 65+ years; increases in asthma hospital admissions for 15-64 years age group; increases in COPD admissions for 65+years age group; and increases in hospital admissions due to pneumonia and acute bronchitis for the age groups less than 1 year, 1-4 years and 65+ years

- increases in NO_2 are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years (only max 1-hour NO_2), 5-14 years (only max 1-hour NO_2), and 15-64 years (only max 1-hour NO_2); increases in asthma hospital admissions for 5-14 years age group (only 24-hour NO_2)

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years and 5-14 years; and increases in hospital admissions due to pneumonia and acute bronchitis for the age group 65+ years

- increases in ozone (warm period) are significantly associated with increases in hospital admissions due to all respiratory disease and due to asthma in the age group 1-4 years.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. Both the SPIRT and EPHC estimates for the associations with NO_2 are similar in magnitude, positive but insignificant. For particles, the EPHC estimates are much larger (perhaps due to bushfire and control burn effects).

The GAM approach used in the Melbourne study (EPA Victoria 2001) for the period 1994-1997 is similar to that used here and the results in Table 6.2.11 show the results here are generally similar to the SPIRT study (see Tables 6.1.20 to 6.1.22), but less likely to be significant.

Table 6.2.11: Relative risk for respiratory hospital admissions associated with air pollutants in Melbourne — comparison of results from the current EPHC study to a previous study in Melbourne * (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total respiratory admissions						
	1h O_3 (ppb)	65+	2	EPHC	0.9995	(0.9982,1.0008)
	1h O_3 (ppb)	65+	2	Melbourne*	1.0015	(1.0003,1.0027)
	24h bsp ($10^{-4}.m^{-1}$)	15-64	0-1	EPHC	1.0303	(0.9728,1.0912)
	24h bsp ($10^{-4}.m^{-1}$)	15-64	3 day av	Melbourne*	1.0784	(1.0121,1.1491)
	24h bsp ($10^{-4}.m^{-1}$)	65+	0-1	EPHC	1.0580	(1.0060,1.1128)
	24h bsp ($10^{-4}.m^{-1}$)	65+	5 day av	Melbourne*	1.0745	(1.0041,1.1499)
	24h NO_2 (ppb)	15-64	0-1	EPHC	1.0049	(1.0011,1.0086)
	24h NO_2 (ppb)	15-64	5 day av	Melbourne*	1.0084	(1.0043,1.0126)
	24h NO_2 (ppb)	65+	0-1	EPHC	1.0033	(1.0000,1.0066)
	24h NO_2 (ppb)	65+	5 day av	Melbourne*	1.0110	(1.0070,1.0149)
	8h CO (ppm)	15-64	0-1	EPHC	0.9903	(0.9722,1.0087)
	8h CO (ppm)	15-64	3 day av	Melbourne*	1.0328	(1.0098,1.0564)
	8h CO (ppm)	65+	0-1	EPHC	1.0064	(0.9902,1.0228)
	8h CO (ppm)	65+	5 day av	Melbourne*	1.0305	(1.0069,1.0546)
Asthma admissions						
	24h bsp ($10^{-4}.m^{-1}$)	1-4	0	EPHC	1.1738	(1.0525,1.3091)
	24h bsp ($10^{-4}.m^{-1}$)	5-14	0	EPHC	1.0574	(0.9020,1.2395)
	24h bsp ($10^{-4}.m^{-1}$)	0-14	0	Melbourne*	1.1481	(1.0628,1.2403)
	24h NO2 (ppb)	1-4	0-1	EPHC	1.0087	(1.0007,1.0169)
	24h NO2 (ppb)	5-14	0-1	EPHC	1.0070	(0.9972,1.0168)
	24h NO2 (ppb)	0-14	5 day av	Melbourne*	1.0118	(1.0058,1.0177)
	8h CO (ppm)	1-4	0-1	EPHC	1.0065	(0.9654,1.0493)
	8h CO (ppm)	5-14	0-1	EPHC	1.0048	(0.9563,1.0557)
	8h CO (ppm)	0-14	3 day av	Melbourne*	1.0606	(1.0274,1.0948)

**EPA Victoria 2001*

Perth

The pooled estimate results in Tables 6.1.24 and 6.1.25 for respiratory hospital admissions indicate that, for Perth:

- increases in $PM_{2.5}$ are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups less than 1 year, 1-4 years, 15-64 years and 65+ years; increases in asthma hospital admissions for 15-64 years age group; increases in COPD admissions for 65+years age group; and increases in hospital admissions due to pneumonia and acute bronchitis for the age groups less than 1 year, 1-4 years and 65+ years

- increases in NO_2 are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years (only maximum 1-hour NO_2), 5-14 years (only maximum 1-hour NO_2), and 15-64 years (only maximum 1-hour NO_2); increases in asthma hospital admissions for 5-14 years age group (only average 24-hour NO_2)

- increases in PM_{10} are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years and 5-14 years; and increases in hospital admissions due to pneumonia and acute bronchitis for the age group 65+ years

- increases in ozone (warm period) are significantly associated with increases in hospital admissions due to all respiratory disease and due to asthma in the age group 1-4 years.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. The SPIRT estimates for the associations with NO_2 and particles are negative and significant. The EPHC estimates are insignificant (positive for particles, negative for NO_2)

The previous Perth study (DoE 2003) also used a case-crossover approach but a smaller window than here (and therefore fewer data points and less power), but Table 6.2.12 indicates similar results, but less significant for this study.

Table 6.2.12: Relative risk for respiratory hospital admissions associated with air pollutants in Perth — comparison of results from the current EPHC study to a previous study in Perth * (Only significant results shown)

Outcome	Pollutant	Age group	Lag	Studies	RR	95% CI
Total respiratory admissions						
	1h bsp ($10^{-4}.m^{-1}$)	65+	2	EPHC	1.0227	0.9938,1.0523
	1h bsp ($10^{-4}.m^{-1}$)	65+	2	Perth*	1.0196	1.0048,1.0347
	24h NO_2 (ppb)	65+	1	EPHC	0.9974	0.9912,1.0036
	24h NO_2 (ppb)	65+	1	Perth*	1.0058	1.0003,1.0113
Asthma admissions						
	24h $PM_{2.5}$ ($\mu g.m^{-3}$)	1-4	2	EPHC	1.0092	0.9975,1.0210
	24h $PM_{2.5}$ ($\mu g.m^{-3}$)	5-14	2	EPHC	1.0125	0.9984,1.0268
	24h $PM_{2.5}$ ($\mu g.m^{-3}$)	0-14	2	Perth*	1.0034	1.0008,1.0060
	1h O_3 (ppb)	1-4	0	EPHC	1.0029	0.9982,1.0076
	1h O_3 (ppb)	5-14	0	EPHC	1.0011	0.9953,1.0069
	1h O_3 (ppb)	0-14	0	Perth*	1.0031	1.0003,1.0058
COPD admissions						
	1h bsp ($10^{-4}.m^{-1}$)	65+	2	EPHC	1.0428	1.0006,1.0868
	1h bsp ($10^{-4}.m^{-1}$)	65+	2	Perth*	1.0492	1.0239,1.0752
	24h bsp ($10^{-4}.m^{-1}$)	65+	2	EPHC	1.1324	0.9468,1.3544
	24h bsp ($10^{-4}.m^{-1}$)	65+	2	Perth*	1.2431	1.0377,1.4892
Pneumonia admissions						
	24h $PM_{2.5}$ ($\mu g.m^{-3}$)	65+	3	EPHC#	1.0009	0.9904,1.0114
	24h $PM_{2.5}$ ($\mu g.m^{-3}$)	65+	3	Perth*	1.0048	1.0001,1.0097

 # Pneumonia and acute bronchitis hospital admissions
** DoE 2003*

Sydney

The pooled estimate results in Tables 6.1.24 and 6.1.25 for respiratory hospital admissions indicate that, for Sydney:

- increases in $PM_{2.5}$ are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups less than 1 year, 1-4 years, 15-64 years and 65+ years; increases in asthma hospital admissions for 15-64 years age group; increases in COPD admissions for 65+years age group; and increases in hospital admissions due to pneumonia and acute bronchitis for the age groups less than 1 year, 1-4 years and 65+ years

- increases in NO_2 are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years (only maximum 1-hour NO_2), 5-14 years (only maximum 1-hour NO_2), and 15-64 years (only maximum 1-hour NO_2); increases in asthma hospital admissions for 5-14 years age group (only average 24-hour NO_2)

- increases PM_{10} are significantly associated with increases in hospital admissions due to all respiratory disease in the age groups 1-4 years and 5-14 years; and increases in hospital admissions due to pneumonia and acute bronchitis for the age group 65+ years

- increases in ozone (warm period) are significantly associated with increases in hospital admissions due to all respiratory disease and due to asthma in the age group 1-4 years.

Tables 6.1.19 to 6.1.20 also compare the results for single city estimates derived in the SPIRT study with the EPHC study. The SPIRT and EPHC estimates for the associations with NO_2 and particles are positive and significant, with the SPIRT estimate larger in magnitude (overlap in confidence intervals).

Morgan et al. (1998b) only found significant associations between NO_2 (lag 0) and asthma admissions (1-4 years).

6.3 Interpretation

One of the most difficult problems in interpreting results such as those in Section 6.1 and 6.2 is that the increases identified cannot be proven here to be causal; that is, the studies reveal that there are increases in some air pollutant concentrations that are statistically significantly associated with increases in death counts or hospital admissions, but they cannot prove that air pollutants cause the increases in deaths and admissions. There are many mechanisms that have been identified in toxicological studies (Pope 2000) that show that the associations may be causal but epidemiological studies cannot do this.

Another problem is that the increases in death counts or hospital admissions associated with increases in concentrations of NO_2, CO and particles may be referring to the same effect, as these pollutants arise from similar sources (such as motor vehicle exhausts). Therefore, the air pollutants often increase and decrease in concentration on the same days; that is, they are correlated, and these correlations are often statistically significant. These patterns of increase and decrease in concentrations are compared with the increases and decreases in death counts or hospital admissions in the statistical models used here to identify whether the patterns are similar (that is, deaths or hospitalisations increase and decrease when air pollutant concentrations increase or decrease), and whether these similarities are statistically significant after controlling for other factors that might also be influencing the increases and decreases in health outcomes (such as hot, cold and wet weather).

Therefore, the results are mainly showing the associations between increases in deaths or hospitalisations and the increases in air pollutant emissions from sources such as motor vehicle exhausts.

6.3.1 Pollutant results from similar sources

Table 6.3.1 shows the relationship between CO and NO_2 all year round in all the cities. Considering these pollutants have similar emission sources, such as motor vehicle exhausts, the associations between these two gaseous pollutants and deaths or hospitalisations would be expected to be similar. Section 6.2 clearly shows this is the case for increases in death counts and hospital admissions due to cardiovascular disease, and when we matched these pollutants together in subsequent analyses we showed the estimates for CO and NO_2 were referring to similar air pollutant events.

Table 6.3.1: Correlation matrix of air pollutants by city and season (maximum 8-hour CO and average 24-hour NO_2)

City	All year	Cool period	Warm period
Auckland	0.53	0.47	0.39
Brisbane	0.64	0.64	0.54
Canberra	0.55	0.52	0.52
Christchurch	0.69	0.65	0.57
Melbourne	0.68	0.65	0.71
Perth	0.73	0.67	0.73
Sydney	0.70	0.65	0.73

There are similar sources for particles and the gases CO and NO_2, such as motor vehicle exhausts (especially for fine particles, $PM_{2.5}$), and these are shown in correlations given in Table 6.3.2 between particles and the gases. It is clear that the correlations are strongest in winter, indicating that the emission sources are most similar then (motor vehicle exhausts, home fires). It is notable that the results for death counts and hospital admissions due to cardiovascular admissions are also similar for particles, NO and CO, although only NO_2 and particles show similar results for respiratory hospital admissions (especially in the younger age groups).

The associations between increases in respiratory hospital admissions for specific conditions such as childhood asthma, COPD in the elderly, and pneumonia and acute bronchitis in general is significant only for particles (usually $PM_{2.5}$). When matched analyses were carried out including particles and NO_2 together, and particles and CO together, the results showed the impacts were arising from the same pattern of air pollutant concentration (probably arising from common sources, such as motor vehicle exhaust emissions) so it was not possible to identify whether one pollutant or the other was the primary cause of the associations found.

Table 6.3.2: Correlation matrix of air pollutants by city and season (maximum 8-hour CO and average 24-hour NO_2 with average 24-hour $PM_{2.5}$ and PM_{10})

City	All year NO_2	Cool period NO_2	Warm period NO_2	All year CO	Cool period CO	Warm period CO
Auckland						
PM_{10}	0.25	0.37	0.04	0.38	0.49	0.05
Brisbane						
$PM_{2.5}$	0.34	0.44	0.18	0.20	0.24	0.09
PM_{10}	0.36	0.33	0.23	0.21	0.17	0.16
Canberra						
PM_{10}	0.59	0.62	0.47	0.76	0.82	0.32
Christchurch						
PM_{10}	0.57	0.57	0.20	0.88	0.89	0.39
Melbourne						
$PM_{2.5}$	0.68	0.77	0.62	0.61	0.71	0.47
PM_{10}	0.38	0.66	0.43	0.27	0.55	0.25
Perth						
$PM_{2.5}$	0.51	0.65	0.46	0.54	0.66	0.47
PM_{10}	0.21	0.36	0.30	0.30	0.52	0.30
Sydney						
$PM_{2.5}$	0.45	0.73	0.35	0.40	0.62	0.32
PM_{10}	0.29	0.62	0.30	0.22	0.48	0.26

Table 6.3.3 shows how ozone concentrations are correlated with NO_2 in the warm period but not in the cool period. During photochemical smog events, the concentrations of ozone, particles (especially $PM_{2.5}$) and NO_2 increase (at different places and different times), so the fixed point monitors record increase for all these pollutants during these episodes. As smog events usually occur during the warm periods there is usually only significant correlation then between the air pollutants as shown in Table 6.3.3. The exception is Brisbane with its wet summers and high sunshine winters when 'cool' period smog events are possible. Perth and Melbourne, with their hot dry summers and cold wet winters, show the cool and warm period differences most strongly (as shown also by the higher correlations between temperature and ozone in Table 2.3.3; results for Auckland and Brisbane show the influence of wet summers).

CO is not influenced in the same way so the correlations between CO and ozone are usually different. It is notable that the results in Section 2.2 for particles, NO_2 and ozone are similar for respiratory hospital admissions for children. An examination of the particle and NO_2 associations in the cool and warm periods did often show significant differences between the two periods, with results often higher and significant for the warm period. This is particularly the case for Melbourne and Perth, the two cities where the relationship between the particles and gases in summer would be expected to be the most prominent. Therefore, the results here do indicate that the summer smog events are having a significant impact on respiratory disease in children as shown by increases in hospitalisations.

However, in cities such as Christchurch and Auckland, there are also examples showing respiratory disease being significant in the cool period, probably due to the extensive use of home fires. The results for Melbourne also sometimes show this impact.

Table 6.3.3: Correlation matrix of air pollutants by city and season (maximum 8-hour CO, average 24-hour NO_2, average 24-hour $PM_{2.5}$ with maximum 8-hour O_3, temperature also included)

City	All year O_3	Cool period O_3	Warm period O_3
Brisbane			
$PM_{2.5}$	0.32	0.42	0.39
PM_{10}	0.40	0.28	0.48
NO_2	0.11	-0.11	0.26
CO	-0.10	-0.20	-0.05
Temperature	0.12	0.41	0.23
Melbourne			
$PM_{2.5}$	-0.02	-0.51	0.43
PM_{10}	0.27	-0.30	0.39
NO_2	-0.15	-0.62	0.52
CO	-0.36	-0.56	0.29
Temperature	0.68	0.46	0.73
Perth			
$PM_{2.5}$	0.25	-0.07	0.48
PM_{10}	0.32	0.14	0.39
NO_2	0.18	-0.19	0.52
CO	0.02	-0.19	0.29
Temperature	0.37	0.17	0.54
Sydney			
$PM_{2.5}$	0.30	-0.16	0.54
PM_{10}	0.45	0.04	0.59
NO_2	-0.08	-0.30	0.33
CO	-0.33	-0.57	0.13
Temperature	0.56	0.51	0.57

6.3.2 Emission source studies

A recent study funded by the National Heritage Trust has indicated that the sources of particle emissions are complex and varied (L Denison 2005, pers. comm.). The study examined the chemical composition of airborne particles in Adelaide, Brisbane, Melbourne and Sydney and identified potential sources of these particles. The results for the particles in the Melbourne, Brisbane and Sydney samples are, in general, comparable to those observed in previous studies. The results show that the contribution of the fine particle fraction ($PM_{2.5}$) to the total PM_{10} fraction is lower in Australian cities to those overseas, it being approximately 40% in Australia compared to 60-80% found in US cities.

The results indicate that although there is variability between cities, the main components are crustal matter and sea-salt from natural sources, particles and soot from human sources, and an organic component from both. The organic component comprises approximately 30-40% in both the fine fraction ($PM_{2.5}$) and the coarse fraction (the fraction between 2.5 microns and 10 microns in size, $PM_{2.5\text{-}10}$). Particles from human sources comprise 24-29%, and soot 10-20%, of the finer $PM_{2.5}$ fraction, while particles from human sources comprise approximately 5% and soot 0.2% of the coarse fraction. It is clear, therefore, that emissions from human combustion sources (motor vehicle exhausts, home fires) and smog production mainly influence the measures of $PM_{2.5}$, and not PM_{10}. Sea salt and crustal matter comprise about 23% to 40% of $PM_{2.5}$ and 57-70% of the coarse fraction. The significant contribution of

crustal matter to the $PM_{2.5}$ indicates that windblown dust in Australian cities comprises a significant proportion of fine particles. It has usually been assumed that the crustal components are primarily in the coarse fraction and are not significant in the fine fraction of PM_{10}.

The results also show that within each city there is significant seasonal variability in the composition of particles in both the fine and coarse fractions. Also, the results indicate that the composition of $PM_{2.5}$ varies from city to city. The composition of $PM_{.2.5}$ in Sydney and Brisbane is very similar and has a much higher contribution of soot (an indicator of combustion processes) than Melbourne. The contribution from estimated organic components is higher in Melbourne than in the other cities as is the contribution from secondary particles during the summer, showing that smog production has a strong influence on particle production in summer in Melbourne.

6.3.3 Bushfires

The bushfire study for Brisbane (Chen et al. 2006) indicates that bushfires can dominate the associations found for $PM_{2.5}$ and, to a lesser extent, PM_{10}. Therefore, such data sets may not be a good surrogate for emission from sources such as traffic.

6.3.4 Air pollution exposure and interpretation of results

Only data sets for air pollutants that are monitored on a daily basis at outdoor fixed-point sites in the cities under study are used here in the analyses. In each city, all the data from all the air pollution monitors is collated and an average air pollution concentration is derived which is assumed to be representative of the average outdoor exposure of the population under study to the air pollutant.

However, the number of air pollution monitors measuring ambient outdoor air pollutant concentrations is different in each city. The project team have used the data supplied by the government agencies in each city and have assumed the data supplied is representative.

The air pollution concentrations measures at fixed outdoor locations are referred to as 'ambient' concentrations. These readings are used here as measures for actual exposure by the population, but clearly they are not equivalent, as people spend most of their time in an indoor or enclosed (motor vehicle) environment. This is especially the case in winter, with houses sealed for heating, and even in summer if there is extensive air conditioning (especially for adults). There are also indoor sources of air pollutants such as NO_2 (gas heaters, stoves), CO (home heating, smoking) and particles (home heating, stoves, smoking), so it is highly unlikely that the outdoor ambient air pollution concentrations are true measures for actual exposure. However, they may serve as good surrogates. For example, here we are examining the associations between day-to-day change of deaths or hospitalisations and air pollution, and sources such as smoking, home heating, and gas cooking probably do not vary much from day to day, while the sources of outdoor pollution, such as motor vehicle exhausts, do. Therefore, the variability of the actual air pollution exposure on a day-to-day basis may be reflected in the day-to-day variability of the ambient air pollution levels, and it is this variability we are

examining when we consider the associations between increases or decreases in deaths or hospitalisations and increases or decreases in air pollution levels. In using ambient air pollution data, we are assuming that the relative increase or decrease in actual air pollution exposure on a day-to-day basis is effectively given by the relative increase or decrease in ambient air pollution concentrations, but this may well not be the case for gases such as NO_2, CO or ozone which may not penetrate sealed buildings easily.

There have been numerous studies on the relationship between actual air pollution exposure and outdoor air pollution concentrations, such as a study in Boston (Sarnat et al. 2005). This study generally found little relationship between outdoor ambient air pollution concentrations and actual exposures, particularly in winter. The exceptions were fine particle (good representation in summer especially). The Boston study showed that outdoor concentrations of NO_2, ozone and CO in summer are good surrogates for changes in actual exposure to fine particles in that there are strong correlations between actual exposure to particles (as measured by $PM_{2.5}$) and outdoor ambient concentrations for ozone, NO_2 and CO. However, there are poor correlations between the actual exposures to these gases and their outdoor ambient concentrations.

By contrast, the NSW Indoor Air Survey found strong correlations between indoor and outdoor nitrogen dioxide concentrations, especially in homes with no significant use of gas appliances (Sheppeard 2002). Similarly, there were strong correlations between indoor PM_{10} and PM_{10} monitored at ambient stations, particularly in homes without smokers.

It is important to note that the results for the air pollutants are not additive. For example, the results for short-term exposure to pollutants for the estimated increases in the number of hospital admissions due to all cardiovascular disease in the elderly age group per unit of air pollutant exposure is 0.3% for $PM_{2.5}$, 0.6% for NO_2 and 2.5% for CO. The results for allowing air pollution levels to rise from their concentration at the 25^{th} percentile to that at the 75^{th} percentile (the middle 50% of the data) are increases in admission counts of 1.3% for $PM_{2.5}$, 3.0% for NO_2, 2.2% for CO. The total impact is not obtained by adding these estimates for any given air pollution event, as they are referring potentially to the same increase in cardiovascular admissions, given each pollutant is a marker for the same human combustion sources. The correct use of these data, therefore, is to indicate that in raising air pollution from the 25^{th} percentile levels of the data to the 75^{th} percentile levels (the middle 50% of the data), the resulting estimate for the increase in hospital admissions due to all cardiovascular disease is in the range 1.35 to 3.0%.

6.3.5 'Harvesting'

The results presented focus on the influence on a health outcome (death count or hospital admission count) of exposure to the average concentrations of air pollutants on the same day or the day previous to the event, or *short-term impacts*. However, identifying associations between increases in short-term exposures and increases in deaths can be misleading. The 'harvesting hypothesis' suggests that air pollutants may be causing sick people to die a few days or weeks earlier than they would have otherwise, but the total number of deaths averaged over a year is effectively the

same, the time of death has been displaced (mortality displacement) or the sick people 'harvested' earlier.

We have used the statistical approach adopted by the Harvard School of Public Health to test this hypothesis. Overseas studies suggest that when the influence of the previous 30 to 40 days of air pollution exposure is estimated, then the increase in deaths is much higher, indicating that the short-term calculations underestimate the impact of air pollution and that 'harvesting' does not occur. Most overseas studies have concentrated on the impact of $PM_{2.5}$ or PM_{10}. Here, there are limited data sets for $PM_{2.5}$ and PM_{10} and the use of the overseas approach for these pollutants indicates that most of the effects reduce when considered over the longer term. However, the use of this statistical approach for the more complete data sets for NO2 and CO shows significant increases in impact over 40 days, compared to the short-term impacts.

It should be noted that mortality displacement or 'harvesting' of a frail population is not incompatible with the advancement of mortality by much greater time, such as weeks or even years. The extreme 'harvesting hypothesis' was that air pollution might only effect a small susceptible pool of individuals, that their day of death would be advanced by only a few days, that a period of increased mortality would be followed by a corresponding period of reduced mortality due to the removal of these frailest individuals from the population, and that this mortality displacement was not of public health concern. This extreme 'harvesting hypothesis' has not been supported by any epidemiologic study of short-term exposures, and has been disproved by epidemiologic studies of survival cohorts that show considerable advancement of mortality in larger populations. Nevertheless, the general population is composed of individuals with varying susceptibility to the adverse effects of particulate matter either due to chronic conditions, such as diabetes and pre-existing cardiopulmonary diseases, or to transient conditions, such as a respiratory infection or a recent cardiovascular event.

6.4. Conclusions

In all the Australian and New Zealand cities studied here, the results indicate that increases in concentrations of this mixture of air pollutants in urban airsheds are significantly associated with:

- increases in mortality for total all cause, cardiovascular and respiratory disease categories (and the impact on the elderly is the strongest)

- increases in cardiovascular hospital admissions for a range of disease categories including all cardiac, IHD, MI and cardiac failure (and the impact on the elderly is the strongest)

- increases in respiratory hospital admissions for a range of disease categories including all respiratory, asthma, COPD, pneumonia and acute bronchitis (and the impact on the child age groups is the strongest, except for COPD).

Statistical tests generally indicated that there was no evidence for heterogeneity between the results for cities sharing common data sets, so the pooled estimates derived were applicable to all the cities considered in each analysis. This was found

for cities sharing data sets for (maximum 1-hour) NO_2, 24-hour average PM_{10}, 24-hour average $PM_{2.5}$, and ozone (warm period). For the associations between CO and cardiovascular admissions, pooled estimates can also be used although generally Sydney yielded higher results.

These air pollutants are usually significantly correlated with each other (probably due to similar emissions sources, such as motor exhausts), so the results for each pollutant may be a surrogate for another, and may be referring to the same impacts. Therefore, the increases are not additive; that is, it is inappropriate to add all the increases in each health outcome arising from all pollutant increases. Throughout the analysis, we found NO_2 and particle associations confounded each other, as did NO_2 and CO where CO associations became significant. Some of the summer ozone associations also appeared to be confounded by the NO_2 associations.

The results here indicate the impacts of air pollution 'mixtures' of gases and particles, including all or part of the concentrations for CO, NO_2, $PM_{2.5}$, and PM_{10} in winter, and NO_2, $PM_{2.5}$, and ozone in summer. All the components of such a mixture probably share similar emission sources (such as motor vehicle exhausts) and the most notable surrogate for this mixture found here is NO_2. It is suggested that the NO_2 associations are the most consistent in this report as NO_2 is measured best, and it seems that NO_2 is a valid indicator for combustion related pollution in urban areas in Australia and New Zealand.

Most of the analyses here examined the short-term effects, that is, the acute health effects arising from exposure to pollutants on the same day or the day before. It has been suggested that the resulting mortality results may exaggerate the effects of air pollutants as people who are already very ill may simply die a few days or weeks earlier because of enhanced air pollution exposures — the 'harvesting' or mortality displacement hypothesis. The results here show no evidence for that effect if we use NO_2 as the surrogate for the effects.

This study indicates that other Australian and New Zealand studies need to be carried out to investigate this air pollution mix more closely. For example, the following questions need to be addressed:

- What is the relationship between the air pollutant concentrations monitored at fixed outdoor sites (such as the NEPM network) and the actual exposure to air pollution?

- What are the constituents of particles that contribute to the health associations found here?

The first question acknowledges that any one of the pollutants here may be a surrogate for another, or even for the effects of a mixture of air pollutants, and not necessarily just the air pollutants considered here. That is, are the effects found for NO_2 and CO arising only because these gases are 'markers' for others (such as particles, or a mixture of gases and particles)?

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