

# **Vegetation fire smoke, indigenous status and cardio-respiratory hospital admissions in Darwin, Australia, 1996-2005.**

Ivan C. Hanigan<sup>1§</sup>, Fay H. Johnston<sup>1,2\*</sup>, Geoffrey G. Morgan<sup>3\*</sup>.

<sup>1</sup>School for Environmental Research, Charles Darwin University, Darwin, Northern Territory, Australia.

<sup>2</sup>Menzies School of Health Research, Charles Darwin University, Darwin, Northern Territory, Australia.

<sup>3</sup>North Coast Area Health Service and Department of Rural Health (Northern Rivers) University of Sydney, Lismore, New South Wales, Australia.

\*These authors contributed equally to this work

§Corresponding author

Email addresses:

ICH: [ivan.hanigan@anu.edu.au](mailto:ivan.hanigan@anu.edu.au)

FHJ: [fayj@utas.edu.au](mailto:fayj@utas.edu.au)

GGM: [GMORG@nrahs.nsw.gov.au](mailto:GMORG@nrahs.nsw.gov.au)

# **Abstract**

## **Background**

Air pollution in Darwin, Northern Australia, is dominated by smoke from seasonal fires in the surrounding savanna that burn during the dry season from April to November. Our aim was to study the association between particulate matter less than 10 microns diameter ( $PM_{10}$ ) and daily emergency hospital admissions for cardio-respiratory diseases for each fire season from 1996 to 2005. We also investigated whether the relationship differed in indigenous Australians; a disadvantaged population sub-group.

## **Methods**

Daily  $PM_{10}$  exposure levels were estimated for the population of the city from existing visibility data using a previously validated model. We used over-dispersed Poisson generalized linear models with parametric smoothing functions for time and meteorology to examine the association between admissions and  $PM_{10}$  at lags of 0 to 3 days. An interaction between indigenous status and  $PM_{10}$  was included to examine differences in the impact on indigenous people.

## **Results**

We found both positive and negative associations and our estimates had wide confidence intervals. There were generally positive associations between respiratory disease and  $PM_{10}$  but not with cardiovascular disease. There were greater effects in indigenous people than

non-indigenous people. A positive effect estimate was found with same-day asthma admissions; which was greater for indigenous people.

## **Conclusions**

The positive associations between vegetation fire smoke and daily hospital admissions for respiratory diseases was disproportionately higher in indigenous people. Our statistical power was limited by the low number of daily admissions; however these results do contribute to the currently small evidence base for health effects of vegetation fire smoke pollution.

## Background

Associations between daily hospital admissions for cardio-respiratory diseases and particulate matter less than 10 microns in aerodynamic diameter (PM<sub>10</sub>) have been described in many settings worldwide including North America, Europe, Asia and Australia.[1] In large cities, where the vast majority of research has been conducted, fossil fuel combustion in industry and transport are major sources of PM<sub>10</sub>. However, depending on the setting, there are potential contributions from a range of other sources including crustal particles and biomass combustion such as forest fires and wood fuels.[2] The relative effects of different sources of particulate pollution on adverse health outcomes, and differences in these effects across population sub-groups, remain major gaps in the currently available evidence.[1] Studies examining a single source of ambient PM<sub>10</sub> are infrequent because of the difficulty finding a site without a mixture of various pollutants, and the complexity of apportioning contributions from different sources.[2]

Particulate matter derived from biomass combustion has been identified as an increasing and unregulated source of outdoor air pollution. The use of wood burning for domestic heating is increasing in several countries,[3, 4] while the frequency and severity of uncontrolled vegetation fires is increasing the world over.[5] Vegetation fires generate pollution episodes across wide geographic areas, and major population centers are frequently affected.[6]

In Australia, the increasing use of deliberate fuel reduction burns as hazard reduction activities to avert major fire disasters is becoming more controversial in the light of the evidence of adverse health impacts of particulate air pollution.[7]

The tropical city of Darwin (Latitude: -12.462, Longitude: 130.842) provides an opportunity to specifically examine the health associations of vegetation fire smoke. Here 50-70% of the surrounding savanna burns annually during the 8 month dry season between April and November.[8] The months from December until March are referred to as the wet season when approximately 80% of Darwin's average annual total rain falls. Due to the rain, fires only occur during the dry season and the smoke from these fires are the source of 95% of measured PM<sub>10</sub> in the city.[9] There are no other important sources of air pollution such as traffic or industry and so PM is negligible during the wet season.[10] A comprehensive air quality study was conducted in the year 2000.[9] and the average concentrations of other pollutants including ozone, sulphur dioxide and nitrogen dioxide are negligible.

Darwin has a population of approximately 110,000 people and also provides an opportunity to examine the relative impact on indigenous Australians, a high-risk population subgroup comprising 11% of the population of Darwin.[11] Socio-economic disadvantage, chronic cardio-respiratory diseases and diabetes have all been shown to modify the effect of particulate air pollution on health outcomes.[12] Indigenous Australians have a high prevalence of all these health risks and have been recognized as being likely to be at much greater risk from poor air quality than other Australians.[13] This has been stated as a priority for Australian public health research.[14]

A previous case-crossover study of the hospital admissions and observed PM<sub>10</sub> in Darwin showed a positive association with respiratory diseases, and disproportionately higher effect estimates in indigenous people.[15] That study had limited statistical power as Darwin's population is relatively small and only three years of air quality data were

available for analysis. Here we attempt to address these limitations by using PM<sub>10</sub> estimations over a 10-season period using a previously validated predictive model based on visibility records;[16] and using the alternative method of time series modeling.[17]

## **Methods**

### **Outcome measures**

De-identified unit record data were obtained for emergency admissions to the Royal Darwin Hospital. This is the only hospital in Darwin and services the entire population of the city and surrounding areas.

Principal diagnosis, indigenous status and primary residence were recorded on discharge from the hospital. Patients whose primary residence was not in Darwin were excluded.

Data were extracted by their assigned principal diagnosis codes classified according to the International Classification of Diseases (ICD) codes. In 1999 there was a change in the coding system used to assign diagnoses from the ICD edition 9 to edition 10. A concordance list produced by the New Zealand Health Information Service was used to marry the diagnosis codes across these two classification systems.

Time series of daily admissions were constructed for each 8-month fire season between 1996 and 2005 for the following diagnosis groups: Total Cardiovascular (ICD9=390-459; ICD10=I00-I99), Ischemic Heart Disease - IHD (ICD9=410-414; ICD10=I20-I25), Total Respiratory (ICD9=460-519; ICD10=J00-J99), Asthma (ICD9=493;ICD10=J45-J46), Chronic Obstructive Pulmonary Disease - COPD (ICD9=490-492, 494-496; ICD10=J40-J44, J47, J67) and Respiratory Infections (ICD9=461-466, 480-487, 514; ICD10=J00-J22).

Ethical approval was gained from the Human Research Ethics Committees of the Northern Territory Government Department of Health and Community Services; the Menzies School of Health Research; and the Charles Darwin University.

### **Exposure measures**

During the dry season, prevailing south-easterly winds bring vegetation fire smoke over Darwin from a large region of savanna. The lower atmosphere of the airshed is characteristically stable during dry seasons, with little convective mixing, and there is a persistent inversion at about 3000 meters.[16] These conditions produce homogenous concentrations of ambient  $PM_{10}$  across the city. This was validated in 2005 when  $PM_{10}$  measurements at two monitors located 25 km to the north-west and south were highly correlated with the primary monitor.[16]

The pattern of vegetation burning has remained consistent throughout our study period. This has been validated by analysis of satellite data which have confirmed the ongoing regional and seasonal nature of annual landscape fires;[18] and by an air quality monitoring campaign conducted 25 km north-west of Darwin in the mid 1990s.[10]

We focus on  $PM_{10}$  in this study as this was the only size fraction with monitoring data available to develop a predictive model using the relationship between air quality observations with meteorological observations.[16] The estimated ambient  $PM_{10}$  levels from this predictive model are shown in Figure 1.

In development of the predictive model for  $PM_{10}$  a range of candidate models were assessed using the possible independent predictor variables: daily average visibility, precipitation, average relative humidity, total cloud cover, maximum temperature and

average wind speed.[16] The Akaike Information Criterion (AIC) was used for model selection and the final model included only visibility, rainfall and relative humidity.

The model was constructed using meteorological observations for dry seasons from the years 2000 and 2004 (observations were not available for the intervening years), and the predicted estimates were validated against observations from 2005. PM<sub>10</sub> data from the monitoring site for a 91 day period (April – June inclusive) in 2005 were used to independently assess the predictive power of the selected model using the corresponding meteorological and visibility data for that period. The model was not used to predict PM<sub>10</sub> during the wet season for the following reasons: firstly meteorological conditions are markedly different with 80% of Darwin's average annual rainfall of approximately 1700 mm falling. This monsoonal rainfall strongly affects visibility and suppresses all fire activity. Secondly, insufficient PM<sub>10</sub> data were available to build a valid model for these periods.

Daily visibility and meteorological data for the centrally located Darwin Airport were obtained from the Australian Bureau of Meteorology. Visibility measurements have been made at this location since the 1950s, following the international standard practice of determining whether or not reference objects at known distances from the site were visible to the human observer.

The predicted ambient PM<sub>10</sub> values correlated well with the observations during 2005 with an  $r^2$  of 0.68. The relationship between the predicted and measured ambient PM<sub>10</sub> from 2005 is shown in Figure 2, with a slope of 0.90. The mean deviation between the predicted

and measured values was  $-2 \mu\text{g}/\text{m}^3$  with a standard deviation of 3.6. No adjustment was made for this small bias.

Daily humidity, temperature, holidays and weekdays were included in our models. Three days missing temperatures were imputed with the prior and subsequent days.

Population data was obtained for each year of the study period from the Australian Bureau of Statistics.[19] The population of Darwin was separated into indigenous and non-indigenous groups; the proportion of indigenous people in the population of Darwin in the 2001 census was used to estimate the indigenous population for the other years.[11]

The relative percentages of persons stratified in three age groups and by indigenous status are given in Table 1.[11, 20]

Weekly influenza data (as a rate per 1000 consultations) were provided by the tropical influenza surveillance system network of sentinel General Practitioners (Northern Territory Department of Health and Community Services, Darwin).

### **Statistical modeling**

Statistical approaches for analyzing time series data in air pollution studies continue to be refined.[21-24] Here we have followed the methods of the American Medicare Air Pollution Study (MCAPS).[24] and the National Morbidity, Mortality and Air Pollution Study (NMMAPS).[25] by using over-dispersed Poisson generalized linear models with natural cubic splines for smoothed functions of time and meteorological variables. These authors suggested other studies reproduce their analyses using the same methods to increase the comparability of results of air pollution studies; and have made their computer code available on the web for adaptation. We adapted Peng's MCAPS code[26] to suit our data

as follows: we did not stratify by age; we included variables for indigenous status, influenza epidemics, holidays, and the change between ICD editions. We did not stratify by age because of the extremely low numbers of daily admissions this would create for some diagnosis groups.

Our regression models separately analyzed the association of same day estimated ambient PM<sub>10</sub> and lags up to three days with daily admission counts for each diagnostic group. Potential confounding or modifying explanatory variables were included in all analyses using previously established protocols for air pollution health studies.[27] We included additional parameters to control for time varying factors including influenza epidemics and school holidays. Annual estimates of the populations of indigenous and non-indigenous Darwin residents were included as an offset in the model as the total population of Darwin grew by 10% during the study period.

We used an over-dispersed Poisson model of daily hospital admissions as follows:

$$\log[E(Y_t)] = \beta_1 \text{ Lagged PM}_{10} + \beta_2 \text{ Indigenous} + \text{ns}(\text{Time}) + \text{ns}(\text{AvDailyTemp}) + \text{ns}(\text{AvDailyTempLag1-3}) + \text{ns}(\text{RHumAv}) + \text{ns}(\text{RHumAvLag1-3}) + \text{DOW} + \text{FluEpidemic} + \text{ICD10change} + \text{Holidays} + \text{offset}(\log(\text{Population}))$$

Where  $E(Y_t)$  is the expected admission count on day  $t$  and 'ns' represents natural cubic splines. These variables, and the degrees of freedom (df) used in splines that represent them, are summarized in Table 2.

Because school holidays are likely to be related to rates of hospital admissions in children [27] these were included as a dummy variable for total respiratory admissions, asthma and respiratory infections as these conditions had a high proportion of children aged less than 15 years.

In the second stage we used a sensitivity analysis similar to the method described by Dominici and colleagues [22] to select the optimal degrees of freedom for the smoothed function of time; to minimize bias in the estimates of the pollution coefficients. This sensitivity analysis was applied to the model for the estimated ambient PM<sub>10</sub> lag with the greatest absolute t-value. We adjusted the degree of smoothing on the time variable by applying different values of a multiplier ( $\alpha$ ) that ranged from 0.2 to 3 times the degrees of freedom which had been chosen a priori.[24, 26] The influence that this had on the effect estimate was assessed using the change in the mean squared error. Theoretically there is lower bias in the estimate caused by smoothing at higher values of  $\alpha$ , but there is larger statistical uncertainty. We conservatively selected the optimal smoothing function for minimizing bias in the point estimate.

Finally, an interaction term between indigenous status and estimated ambient PM<sub>10</sub> was added to the model to investigate the difference in the magnitude of the association in the two population sub-groups.

All analyses were conducted using the statistical software package R version 2.3.1 (<http://www.R-project.org>).

## Results

There were 2,410 days in the 10 dry seasons of our study period. There were 8,279 admissions during this period. The total numbers of hospital admissions (and proportion of patients under 15 years old) are given in Table 3; stratified by clinical grouping and indigenous status. Despite indigenous people representing 11% of the population of Darwin, they comprised 23% of these admissions.

Descriptive statistics for daily admissions in each disease category, estimated daily ambient PM<sub>10</sub> and meteorological parameters are summarized in Table 4.

The point estimates and 95% confidence intervals for the association between hospital admissions with estimated ambient PM<sub>10</sub> are reported here as the exponent value of the model coefficient (and standard errors) multiplied by a nominal change of 10 µg/m<sup>3</sup> of PM<sub>10</sub>. Subtracting one and multiplying by one hundred converts this to a percentage change in the relative risk per 10µg/m<sup>3</sup> change in exposure.

There was a positive association for total respiratory admissions with same day estimated ambient PM<sub>10</sub> (4.81%, 95%CI: -1.04%, 11.01%). The subgroups of respiratory infections, asthma and COPD all had positive associations with same day estimated ambient PM<sub>10</sub>. The small associations for all cardiovascular diseases and IHD were all negative or zero and not statistically significant. Due to small numbers in these groups the confidence intervals are wide.

Figure 3 shows the point estimates and 95% confidence intervals for the association between hospital admissions with estimated ambient PM<sub>10</sub> when an interaction term with indigenous status is included. The point estimate for the effect of same-day ambient PM<sub>10</sub> on total respiratory admissions in indigenous residents was much higher than the estimate for non-indigenous residents, 9.40% (95%CI: 1.04%, 18.46%) compared with 3.14% (95%CI: -2.99%, 9.66%) respectively. For asthma admissions and estimated ambient PM<sub>10</sub> there was a non-significant estimated increase at a lag of 1 day: 16.27% (95%CI: -3.55%, 40.17%) for indigenous compared with 8.54% (95%CI: -5.60%, 24.80%) for non-indigenous people.

A strong positive association was observed for respiratory infections in indigenous people of 15.02% (95%CI: 3.73%, 27.54%) at a lag of 3 days while no association was evident for this condition in non-indigenous people at this lag (0.67%, 95%CI: -7.55%, 9.61%).

There were positive non-significant estimates for same day estimated ambient PM<sub>10</sub> with COPD admissions in both groups. This is in contrast to negative associations with COPD admissions and lagged estimated ambient PM<sub>10</sub> in both groups.

There were no clear associations with estimated ambient PM<sub>10</sub> and total cardiovascular admissions or IHD. In non-indigenous people there were negative non-significant estimates for these conditions at all lags, while indigenous people had some positive non-significant estimates at lags 2 and 3.

## **Discussion**

We found generally positive associations between PM<sub>10</sub> with total respiratory admissions, asthma and respiratory infections that were statistically significant among indigenous people for total respiratory admissions (at lag 0) and respiratory infections (at lag 3). There were generally negative non-significant associations for cardiovascular outcomes in both population groups. These results may be due to chance; reflecting the low precision of our estimates due to the relatively small numbers of daily admissions. However our findings are consistent with other studies of ambient biomass smoke and contribute to the limited evidence concerning the health effects of vegetation fire PM<sub>10</sub>.

A previous case-crossover study in Darwin had similar findings to this study with positive associations reported between observed ambient PM<sub>10</sub> and respiratory admissions (OR 1.077 95%CI 0.98, 1.18) and a tendency towards negative associations with cardiovascular

admissions (OR 0.91 95%CI 0.81,1.02).[15] Similarly, the estimates from that analysis for total respiratory admissions were also approximately double for indigenous rather than non-indigenous people.

A study in Christchurch, New Zealand, where ambient PM<sub>10</sub> predominantly arises from the combustion of wood for domestic heating, found a 3.37% (95%CI: 2.34%, 4.40%) increase in total respiratory admissions per interquartile rise in ambient PM<sub>10</sub> (IQR = 14.8 µg/m<sup>3</sup>) at a lag of 2 days.[28] That study found an association with admissions for heart failure but not other cardiac diagnoses, while a later study in Christchurch found no association with cardiovascular admissions.[29]

In a study of the South East Asian forest fires of 1997 Mott et al found large fire-period related increases in respiratory hospitalizations for asthma and COPD, ranging from 40-80% in adults but no association with cardiovascular admissions although people with pre-existing cardio-respiratory diagnoses were at greatest risk.[30]

A recent study from Brisbane, Australia, directly compared the association between bushfire and non-bushfire derived particulates on total respiratory hospital admissions excluding influenza.[31] That study analyzed the PM<sub>10</sub> distribution as a three-level factor with levels defined as low (<15 µg/m<sup>3</sup>), medium (15-20 µg/m<sup>3</sup>) and high (>20 µg/m<sup>3</sup>).

They found that for an increase in same-day PM<sub>10</sub> from low to high there was an increase in the relative risk for total respiratory hospital admissions of 19% (95%CI: 9%, 30%) whereas on non-bushfire days the associated increase was 13% (95%CI: 6%, 23%).

A similar study from Sydney, Australia, directly compared associations between cardio-respiratory hospitalizations and ambient PM<sub>10</sub> derived from vegetation fire smoke with associations between these outcomes and ambient PM<sub>10</sub> derived from other sources

(Morgan, submitted for publication). They apportioned ambient PM<sub>10</sub> on vegetation fire days into particulate matter derived from burning biomass and particulates due to other sources. They found a 1.24% (95%CI: 0.22%, 2.27%) increase in relative risk for all respiratory admissions per 10 µg/m<sup>3</sup> increase in vegetation fire derived ambient PM<sub>10</sub> at lag 0. Ambient PM<sub>10</sub> due to other sources at lag 0 was associated with an increase in all respiratory admissions of 1.04% (95%CI: 0.02%, 2.07%) per 10 µg/m<sup>3</sup> increase. They also failed to find an association between cardiovascular outcomes and vegetation fire smoke in contrast to findings of a positive association between cardiovascular admissions and ambient PM<sub>10</sub> from all other (non bushfire) sources.

Our positive association with asthma was of similar magnitude to previous biomass smoke studies but did not achieve statistical significance. The lack of a clear association with COPD was unexpected as previous biomass studies have generally found strong positive associations.[28, 30, 32, 33] This finding may have been influenced by the smaller numbers in these sub-groups leading to unstable effect estimates and wide confidence intervals.

The magnitude of the point estimates from our study, the studies discussed above and several other studies of outpatient attendances for respiratory conditions in association with vegetation fires,[28, 30, 34-37] are much greater than multi-city studies of associations between admissions for respiratory diseases (including asthma, COPD, and total respiratory admissions) with positive associations for a 10 µg/m<sup>3</sup> change in ambient PM<sub>10</sub> of the order of just 1-1.5%.[38, 39] Dominici et al 2006 found similar associations of around 1% increase in respiratory admissions per 10 µg/m<sup>3</sup> change in PM<sub>2.5</sub>. [24] The greater

magnitude of adverse respiratory effects reported in studies specifically examining biomass smoke might reflect a true difference in the adverse outcomes associated with this source of PM. However, studies of biomass smoke are usually conducted in cities and towns with small populations, or around short episodes of extreme exposures, and their results inevitably are less precise than those from multi-city studies making direct comparisons difficult to interpret. Similarly, the absent or negative associations between biomass smoke and cardiovascular disease outcomes in our study and in three previous studies of vegetation fire smoke,[15, 30] (and Morgan *et al*, submitted for publication) might also reflect a different pattern of adverse health outcomes from biomass smoke. However these findings require replication as cardiovascular admissions have been clearly associated with ambient PM<sub>10</sub> in many large studies, usually conducted in urban settings where fossil fuel combustion is a major source of PM.[1]

The primary strengths of this study are the spatially homogenous population exposure to particulates across Darwin;[16] the specific source from vegetation fires;[9] the hospital data collection which represents the admissions patterns for the entire population of the city; and the inclusion of details of indigenous status in the health records. These factors all minimized the problems of exposure and outcome misclassification inherent in population-level studies. Additionally due to Darwin's tropical climate, there was minimal variation of daily temperature and humidity; minimizing confounding by meteorological changes.

An important limitation of this study is the small population of Darwin and hence the low numbers of daily admissions for cardio-respiratory diseases in spite of our relatively long 10-season period of data for analysis. This inevitably limited the statistical power and

reduced the precision of our point estimates. However, population-level studies of the health effects of ambient biomass smoke have inherent limitations. Vegetation fire events affecting large populations are rare, unpredictable and often of short duration. In addition settings where biomass is the predominant source of ambient particulate matter tend to have smaller populations as larger cities will have a more complex mix of pollutants often dominated by fossil fuel combustion by industry and transport. For this reason the results from studies specifically examining vegetation fire smoke pollution will almost inevitably have lower precision than studies examining ambient PM regardless of source.

This study also compared rates of admissions between indigenous and non-indigenous subpopulations finding the suggestion of disproportionate burdens of health effects due to the seasonal fire smoke pollution. This is consistent with the only previous study examining this issue in Australia.[15] Many factors could contribute to this including the socio-economic disadvantage, chronic cardio-respiratory diseases and diabetes which all modify the effects of ambient PM<sub>10</sub> on cardio-respiratory admissions.[12] Other factors could include reduced access to health services and therefore early management of chronic conditions,[40] and different patterns of smoking, physical activity or diet among this population sub-group.[13] Residential segregation is less likely to explain the difference in this setting as exposure is relatively uniform across the city.[16]

## **Conclusions**

Our results suggest an association between vegetation fire smoke and daily hospital admissions for respiratory diseases that was disproportionately higher in indigenous people. The analysis found approximately three-fold higher associations between same-day

estimated ambient PM<sub>10</sub> and total respiratory admissions in indigenous people than non-indigenous people. This has implications for local public health policy and practice, such as the identification of sensitive sub-groups, the setting of air quality guidelines, targeting of public health messages in relation to air pollution and the regulation of deliberate burning practices.[14]

This is an important research area to pursue. With global change bringing changes in vegetation burning regimes and increasing population exposures to pollution from vegetation fires, understanding and managing the health impacts of biomass combustion smoke will become an increasingly important public health activity.

## **Competing interests**

The authors declare that they have no competing interests.

## **Authors' contributions**

ICH carried out the analysis and drafted the manuscript. FHJ conceived the study and helped to draft the manuscript. GGM provided theoretical and conceptual guidance and helped to draft the manuscript.

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## Figure legends

Figure 1 - Time series of daily estimated ambient  $PM_{10}$  ( $\mu\text{g}/\text{m}^3$ ) from visibility and weather data for Darwin during each 8-month dry seasons (no estimates were made for the 4-month wet seasons), 1996-2005.

Figure 2 - Validation of the model daily predictions of ambient  $PM_{10}$  ( $\mu\text{g}/\text{m}^3$ ) in 2005 against measured data for Darwin (reproduced from Bowman et al 2006 with permission).

Figure 3 - Point estimates and 95% confidence intervals for the association between hospital admissions for non-indigenous and indigenous people with estimated ambient  $PM_{10}$  in Darwin 1996-2005, as the percentage change in relative risk per  $10\mu\text{g}/\text{m}^3$  rise in  $PM_{10}$ .  $\alpha$  represents the optimal level of a multiplication factor for the smooth function of time, selected using sensitivity analysis.

## Tables

**Table 1 – Estimated resident population of Darwin in each age and indigenous status group in 2004.**

Age Group	Indigenous		Non-indigenous		Total Population	
	Population	Proportion	Population	Proportion	Population	Proportion
0-14	4,295	37%	20,742	21%	25,037	23%
15-64	6,992	60%	72,259	74%	79,251	72%
65plus	303	3%	4,887	5%	5,190	5%
Total	11,591	100%	97,887	100%	109,478	100%

**Table 2 – explanatory variables used in all models.**

Variable	Description
Lagged PM <sub>10</sub>	Estimated ambient PM <sub>10</sub> for lags 0-3 in (µg/m <sup>3</sup> )
Indigenous	An index of counts for indigenous status where indigenous = 1 and non-indigenous = 0
Time	Time in days, represented by a natural cubic spline with 40 df (4 df per season)
AvDailyTemp	Average daily temperature (calculated by averaging the max and min temperatures), in Degrees Celsius (°C), with 6 df
AvDailyTempLag1-3	Rolling averages of average daily temperatures at lags 1, 2 and 3, with 6 df
RHumAv	Average daily relative humidity in percent (%) with 3df
RHumAvLag1-3	Rolling averages of relative humidity at lags 1, 2 and 3 days, with 3 df
DOW	Day of the week. Factor with 7 levels
FluEpidemic	Influenza epidemics. Dummy for days above the 90th centile
ICD10change	The change between ICD editions. Dummy variable indicating the changeover
Holidays	Dummy variable for public holidays
Population	The estimated yearly population for indigenous or non-indigenous residents included as an offset

**Table 3 – Emergency hospitalizations to the Royal Darwin Hospital (with proportion of patients aged under 15 years) for the dry seasons 1996-2005.**

Diagnosis	ICD9	ICD10	Total population		Non-Indigenous admissions		Indigenous admissions	
			Counts	<15 yr	Counts	<15 yr	Counts	<15 yr
<b>Cardiovascular</b>								
Total	390-459	I00-I99	3443	1%	2854	1%	589	2%
IHD	410-414	I20-I25	1533	0%	1287	0%	246	0%
Other	-	-	1910	2%	1567	1%	343	4%
<b>Respiratory</b>								
Total	460-519	J00-J99	4836	40%	3551	40%	1285	40%
Asthma	493	J45-J46	1008	58%	776	59%	232	56%
COPD	490-492, 494-496	J40-J44, J47, J67	995	1%	753	1%	242	1%
Infections	461-466, 480-487, 514	J00-J22	2409	53%	1681	55%	728	50%
Other	-	-	424	16%	341	16%	83	17%

**Table 4 – Summary statistics for daily hospital admissions, estimated ambient PM<sub>10</sub> and meteorological parameters in Darwin during the dry seasons 1996-2005.**

Diagnosis		Mean	Standard Deviation	Range
Cardiovascular	Total	1.4	1.2	6.0
	Indigenous	0.2	0.5	4.0
	Non-Indigenous	1.2	1.1	6.0
IHD	Total	0.6	0.8	5.0
	Indigenous	0.1	0.3	2.0
	Non-Indigenous	0.5	0.7	5.0
Respiratory	Total	2.0	1.5	10.0
	Indigenous	0.5	0.7	4.0
	Non-Indigenous	1.5	1.2	7.0
Asthma	Total	0.4	0.7	5.0
	Indigenous	0.1	0.3	2.0
	Non-Indigenous	0.3	0.6	3.0
COPD	Total	0.4	0.7	4.0
	Indigenous	0.1	0.3	2.0
	Non-Indigenous	0.3	0.6	4.0
Respiratory infections	Total	1.0	1.1	7.0
	Indigenous	0.3	0.5	3.0
	Non-Indigenous	0.7	0.9	5.0
Daily Estimated Ambient PM <sub>10</sub> (µg/m <sup>3</sup> )		21.2	8.2	55.2
Daily Average Temperature (°C)		27.4	2.2	13.1
Daily Average Relative Humidity (%)		65.0	11.1	70.4
Influenza rates (per 1000 consults)		13.2	12.3	82.4

Fig. 1

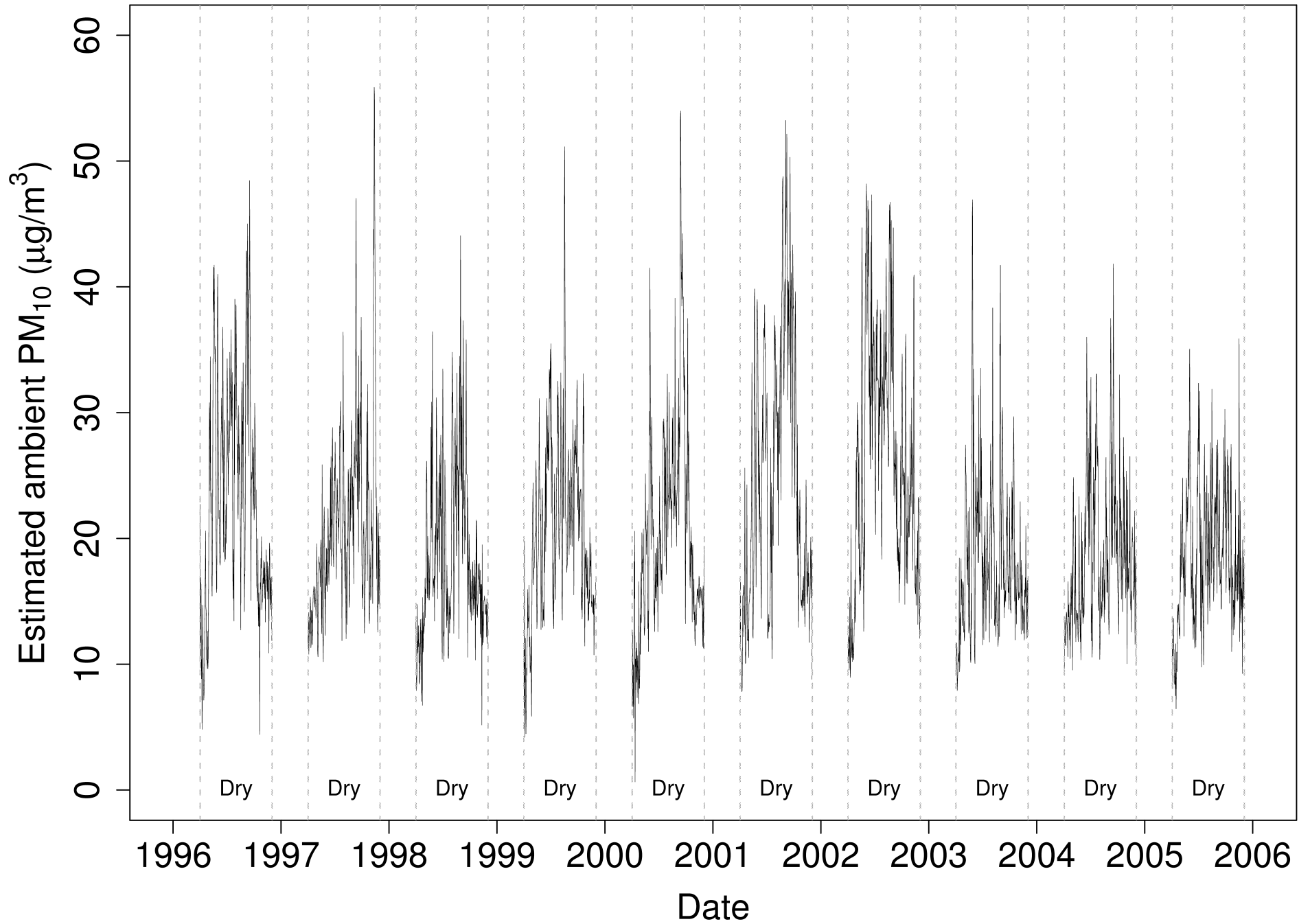


Figure 1

Fig. 2

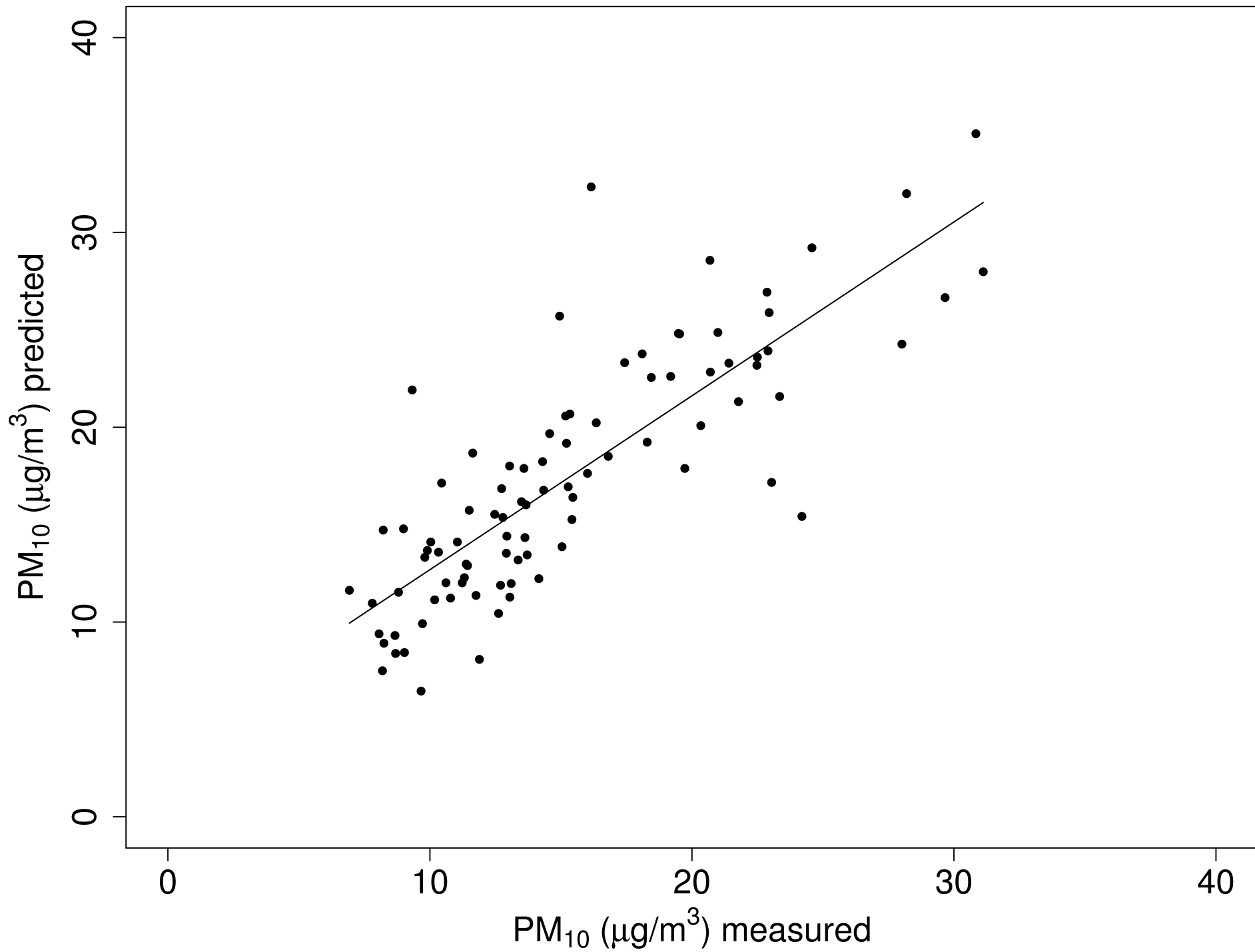


Figure 2

Fig. 3

Non-indigenous people

Indigenous people

All Respiratory  
 $\alpha = 1$

Asthma  
 $\alpha = 1.8$

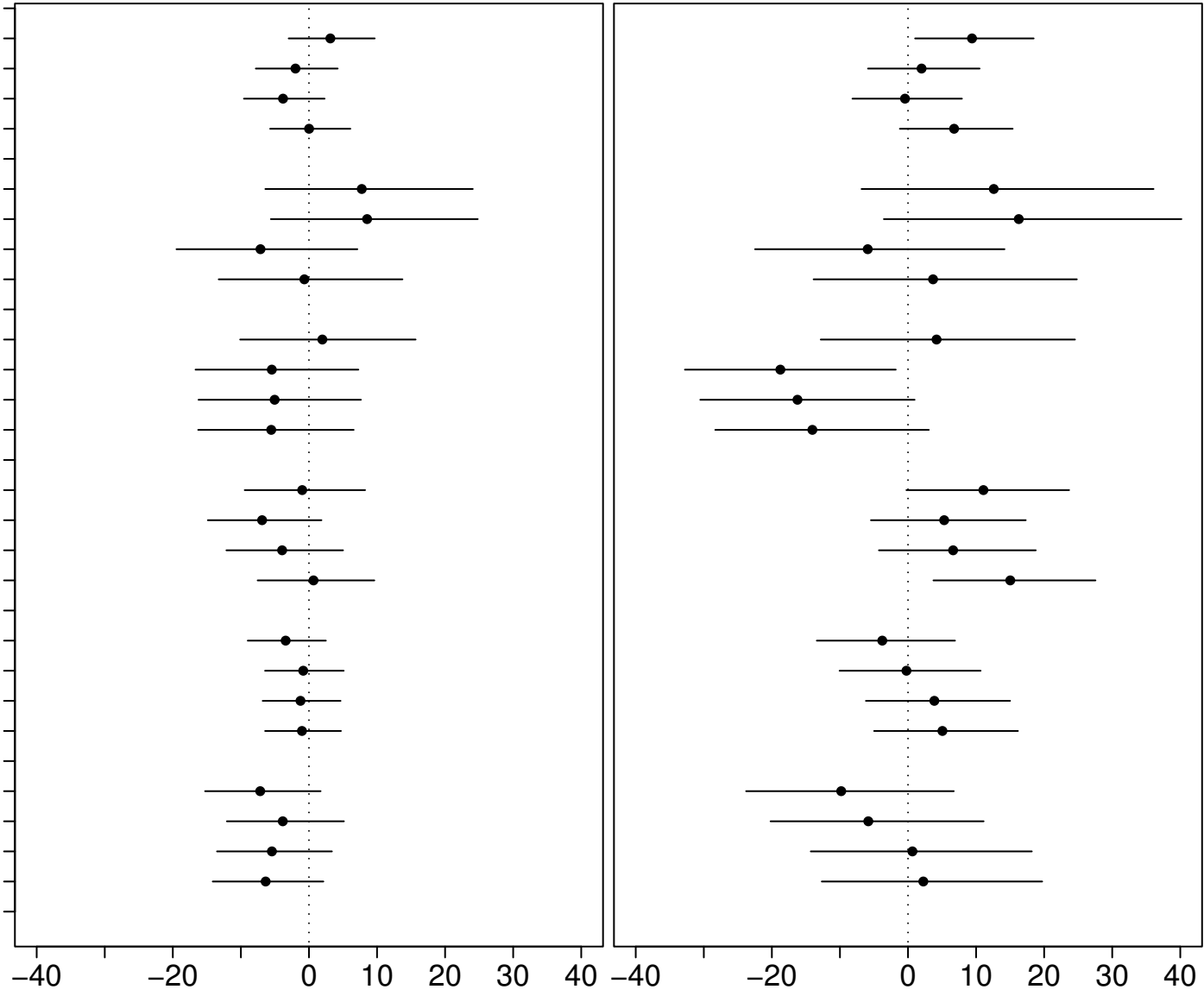
COPD  
 $\alpha = 0.6$

Respiratory Infections  
 $\alpha = 1$

All Circulatory  
 $\alpha = 0.4$

IHD  
 $\alpha = 0.4$

Lag0  
Lag1  
Lag2  
Lag3  
  
Lag0  
Lag1  
Lag2  
Lag3  
  
Lag0  
Lag1  
Lag2  
Lag3  
  
Lag0  
Lag1  
Lag2  
Lag3  
  
Lag0  
Lag1  
Lag2  
Lag3



% Change in Hospital Admissions per 10- $\mu\text{g}/\text{m}^3$  Increase in  $\text{PM}_{10}$