

**EXPOSURE ASSESSMENT AND RISK  
CHARACTERISATION FOR THE DEVELOPMENT OF  
A PM<sub>2.5</sub> STANDARD**

**FINAL REPORT**

Prepared for

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Service Corporation**

by

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## 1. INTRODUCTION

This report describes the outcomes from the consultancy “Exposure Assessment and Risk Characterisation for the Development of a PM<sub>2.5</sub> Standard” undertaken for the National Environment Protection Council Service Corporation (NEPCSC).

The consultancy consisted of three main tasks:

### I. Assessment of Population Exposure

Estimates of population exposure to PM<sub>2.5</sub> have been prepared for Sydney, Melbourne, Brisbane and Perth using daily 24-hour average and annual average PM<sub>2.5</sub> concentrations for three consecutive years.

### II. Identification of Health Effects at Current Levels of Exposure

The health risks associated with current levels of PM<sub>2.5</sub> in Sydney, Melbourne, Brisbane and Perth have been estimated using the exposure data derived in I above and dose-response relationships provided by the NEPC Project Team. Table 1 lists the health endpoints considered in this study.

Table 1. Health endpoints included in the health risk characterisation.

<i>Endpoint</i>	<i>Description</i>	<i>Age group</i>
	<b>Short Term Mortality</b>	
S1	• All cause	All ages
S2	• Respiratory	All ages
S3	• Cardiovascular	All ages
	<b>Hospital Admissions</b>	
S4	• Asthma	All ages
S5	• Cardiovascular disease	Elderly
S6	• Chronic obstructive pulmonary disease	Elderly
	<b>Long Term Mortality</b>	
L1	• All cause	All ages
L2	• Lung cancer	All ages
L3	• Cardiopulmonary disease	All ages

Baseline health incidence data for each health endpoint and for each city (Sydney, Melbourne, Brisbane and Perth) have also been provided by the NEPC Project Team.

### III. Scenario Evaluation

To guide the development of PM<sub>2.5</sub> standards in Australia, the NEPC Project Team proposed a range of PM<sub>2.5</sub> concentrations that could be considered as possible standards. In this report, these concentrations are referred to as 'scenario levels'. The scenario levels considered are 35, 30, 25 and 20 µg/m<sup>3</sup> for 24-hour PM<sub>2.5</sub>, and 10, 8 and 5 µg/m<sup>3</sup> for annual average PM<sub>2.5</sub>.

To model each scenario, the current PM<sub>2.5</sub> concentrations in each city were adjusted so that they were at or below the scenario level. The health outcomes associated with these adjusted concentrations were then calculated. These results were compared with the health outcomes associated with current PM<sub>2.5</sub> levels, to estimate the health outcomes that would be avoided in each scenario.

## 2. METHODOLOGY

The methodology for this project is that described in the USEPA publication "*Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas*" (USEPA, 2002). In order to adapt the USEPA methodology to the requirements stated in the Consultancy Brief, some minor modifications were made. The methodology used for each main task is described below, with any changes from the USEPA methodology indicated in the text.

### 2.1. Assessment of Population Exposure

Exposure data have been derived for each city by averaging the reported PM<sub>2.5</sub> concentrations across the city's monitoring network. The averaged concentration data are assumed to be representative of the ambient PM<sub>2.5</sub> concentrations to which the city's population is exposed. This approach is consistent with what has been done in epidemiological studies estimating PM<sub>2.5</sub> concentration-response functions, used in the subsequent health risk characterisation.

#### 2.1.1 COMPOSITE CONCENTRATIONS

A spatially-averaged composite data set has been created for Sydney, Melbourne, Brisbane and Perth by averaging data across suitable population-oriented monitoring sites. The composite daily 24-hour data consist of the average of the daily 24-hour PM<sub>2.5</sub> concentrations at all sites for which data were available on each day. Table M1 displays some sample 24-hour PM<sub>2.5</sub> data for July 1998 in Melbourne, with the annual maximum concentrations for that year shown in the last row. Note that the annual maximum composite (bottom right entry in Table M1) is the maximum of the daily composite values (not the average of the station annual maximum values).

Table M1. Sample 24-hour PM<sub>2.5</sub> concentrations (in µg/m<sup>3</sup>) for the Alphington, Brighton and Footscray monitoring stations and composite data in Melbourne.

Date	Alphington	Brighton	Footscray	Composite
...	...	...	...	...
20-Jul-98	13.0	11.0	12.4	12.1
21-Jul-98	19.9	15.2	14.5	16.5
22-Jul-98	12.2	7.0	6.3	8.5
23-Jul-98	15.4	16.0	12.0	14.5
24-Jul-98	22.4	18.0	20.1	20.1
25-Jul-98	34.0	24.8	24.6	27.8
26-Jul-98	25.0	24.9	22.0	24.0
...	...	...	...	...
Max. 1998	34.0	31.0	35.7	33.2

The composite annual average PM<sub>2.5</sub> concentrations for each city were calculated similarly by taking the mean of the monitor-specific annual

average concentrations. All calculations were performed for each year of data provided.

In the USEPA methodology, the annual average PM<sub>2.5</sub> concentrations at the individual monitoring sites are derived by calculating the mean of the average concentrations for the four quarters of the year. The average quarterly concentrations are based on 24-hour PM<sub>2.5</sub> concentrations. In this project, the annual average concentrations at the individual monitoring sites have been supplied directly by the NEPC Project Team.

### **2.1.2 FREQUENCY DISTRIBUTIONS**

For each city and for each year of data, a frequency distribution was calculated based on the composite daily 24-hour PM<sub>2.5</sub> data. This was done by allocating each daily concentration value  $x$  to a PM<sub>2.5</sub> concentration 'bin', representing a  $2 \mu\text{g}/\text{m}^3$  range of concentrations starting at  $0 < x \leq 2 \mu\text{g}/\text{m}^3$ , up to  $34 < x \leq 36 \mu\text{g}/\text{m}^3$ . All concentrations above  $36 \mu\text{g}/\text{m}^3$  were placed in a separate bin. The frequency of occurrence of each concentration range is expressed as a percentage of the total number of occurrences, ie. as a percentage of the total number of days for which data were available. The results are presented as bar graphs in Section 4 of this report.

For each frequency distribution an inverse cumulative frequency distribution was constructed linking PM<sub>2.5</sub> concentration  $x$  with the number of days on which the concentration was higher than  $x$ . The inverse cumulative frequency distribution is a useful tool for describing air pollution data as it indicates the frequency of events where levels are greater than  $x$ , in other words, the frequency of extreme events. It is expressed as a percentage of the total number of occurrences in the same way as for the main frequency distribution (see above), and results are presented as line graphs in Section 4.

The above calculations were repeated for each city using the combined data for the three-year period.

### **2.1.3 POPULATION**

Since city-wide average PM<sub>2.5</sub> concentrations are used, the number of people exposed in each city is assumed to be the population of that city. The population of each city has been estimated using the 2001 Census data for each city's Statistical Division from the Australian Bureau of Statistics.

## **2.2 Identification of Health Effects at Current Levels of Exposure**

The health effects at current levels of exposure to PM<sub>2.5</sub> have been estimated by calculating the *change* in the number of cases for each health endpoint corresponding to a change in PM<sub>2.5</sub> concentration from current levels down to background. Therefore, for a given health endpoint, the estimate of the number of cases associated with current levels of exposure is actually an estimate of the *difference* between the number of cases at current levels and the number of cases at background level. Note that the health outcomes associated with PM<sub>2.5</sub> levels below background are not necessarily zero.

For the short-term health endpoints S1 to S6 in Table 1, the current PM<sub>2.5</sub> concentrations are the composite daily concentrations for each city. For the long-term endpoints L1 to L3, the current PM<sub>2.5</sub> concentrations are the composite annual average concentrations for each city.

### **2.2.1 BACKGROUND LEVELS OF PM<sub>2.5</sub>**

Since the health outcomes are calculated only for PM<sub>2.5</sub> concentrations above background, estimates of background PM<sub>2.5</sub> concentrations are needed for each city.

The background PM<sub>2.5</sub> concentration is defined as the 'natural' background, excluding all anthropogenic contributions. Note that the concentration and composition of background PM<sub>2.5</sub> can vary with geographic location, from monitoring site to monitoring site; with season of the year; and with meteorological conditions which affect the emissions and secondary production of biogenic or geogenic species to the background (USEPA, 1996b).

Following the advice of the NEPC Project Team, the background concentration has been estimated by taking the average of the 5<sup>th</sup> percentile 24-hour PM<sub>2.5</sub> concentrations for the four cities. This results in a value of 4.81 µg/m<sup>3</sup>. The same background level has been used for all four cities.

### **2.2.2 DOSE-RESPONSE FUNCTIONS AND ESTIMATING HEALTH OUTCOME CHANGES**

For a given health endpoint, the change in the number of cases,  $\Delta y = y_0 - y$ , corresponding to a change in concentration  $\Delta x = x_0 - x$ , is given by:

$$\Delta y = y(e^{\beta \Delta x} - 1) \quad [1]$$

where  $y_0$  is the number of cases of that endpoint associated with a specified ambient PM<sub>2.5</sub> concentration  $x_0$ ,  $y$  is the baseline incidence rate,

and  $x$  is the measure of ambient "as is"  $PM_{2.5}$  concentration (USEPA, 2002).

### *Calculation of the coefficient $\beta$*

The dose-response functions provided by the NEPC Project Team are expressed as a percentage increase in the number of cases of a given health endpoint associated with a  $10 \mu\text{g}/\text{m}^3$  increase in  $PM_{2.5}$  concentration. For example, a  $10 \mu\text{g}/\text{m}^3$  change in peak 24-hour  $PM_{2.5}$  is associated with a 1.7% increase in hospital admissions for cardiovascular disease in the elderly (endpoint S5 in Table 1). In order to apply this dose-response function to other concentration changes according to Equation 1 above, it was necessary to calculate the value of the coefficient  $\beta$  for each health endpoint.

The percentage increase in the number of cases of a given health endpoint,  $z$ , corresponding to a given change in concentration,  $\Delta x$ , is given by

$$z = \Delta y/y * 100 \quad [2]$$

Using Equation 1,

$$e^{\beta \Delta x} = 1 + z/100 \quad [3]$$

The term  $e^{\beta \Delta x}$  is also known as the relative risk associated with the concentration change  $\Delta x$ . Rearranging Equation 3 gives:

$$\beta = [ \ln(1 + z/100) ] / \Delta x \quad [4]$$

Equation 4 was used to estimate values of the coefficient  $\beta$  for each health endpoint using the dose-response data provided by the NEPC Project Team (see Section 3.3) and a value of  $10 \mu\text{g}/\text{m}^3$  for  $\Delta x$ .

### *Baseline health effects incidence data*

Calculating the change in the number of cases  $\Delta y$  for a given health endpoint corresponding to a given change in concentration according to Equation 1 also requires knowledge of  $y$ , the baseline health effect incidence rate. Baseline health incidence rates express the occurrence of a disease or event (e.g., hospital admission for asthma, death) in a specific period of time, usually per year or per day. Typically the rates are expressed as a value per population group (e.g. the number of cases in Melbourne). Incidence rates vary among geographic areas due to differences in population characteristics (e.g., age distribution) and factors promoting illness (e.g., smoking, air pollution levels). For a given geographic area, incidence rates also vary over time.

Baseline health effect incidence data have been provided for Sydney, Melbourne, Brisbane and Perth by the NEPC Project Team (see Section 3.2).

The baseline health incidence rates were converted to per-capita rates so that they could be applied to the appropriate population size. Any incidence rates provided as annual values were also converted to average daily rates by dividing by 365.

#### *Short-term health effects*

The health outcomes for the short-term endpoints S1 to S6 in Table 1 were estimated for each city on an annual basis by summing the daily changes in the number of cases for each health endpoint, calculated using Equation 1, for each year of data provided. The daily changes in the number of cases (cases at current levels – cases at background level) for each endpoint were calculated using 24-hour  $PM_{2.5}$  concentrations, a constant background concentration, the  $\beta$  coefficient derived from the endpoint's dose-response function, and the daily baseline incidence rate for the endpoint specific to the city.

The annual health outcomes for each endpoint were adjusted for any missing 24-hour  $PM_{2.5}$  data to take into account the full year. This was done by multiplying the annual estimates by the ratio of the total number of days in the year (365 or 366) to the number of days for which 24-hour  $PM_{2.5}$  data were available. For example, data were missing at all Melbourne monitoring sites on 6 March 1998. As a result the health outcomes for Melbourne in 1998 were initially calculated for 364 days. The health outcomes were then adjusted by multiplying with the factor 365/364.

Whilst the USEPA risk analysis is based on a single year, the current analysis is based on a three-year period. The annual health outcomes for the three years of  $PM_{2.5}$  data for each city were averaged to provide estimates of the average annual number of cases of each health endpoint attributable to  $PM_{2.5}$ .

#### *Long-term health effects*

The health outcomes for the long-term endpoints L1 to L3 in Table 1 were estimated for each city by calculating the annual changes in the number of cases for each health endpoint using Equation 1 for each year of data provided. The annual changes in the number of cases (cases at current levels – cases at background level) for each endpoint were calculated using annual average  $PM_{2.5}$  concentrations, a constant background concentration, the  $\beta$  coefficient derived from the endpoint's dose-response function, and the annual baseline incidence rate for the endpoint specific to the city. The baseline incidence rates were first converted to annual rates by multiplying the daily incidence rates by the total number of days in the year.



As for the short-term health outcomes, the annual health outcomes for the three years of PM<sub>2.5</sub> data were averaged to provide estimates of the average annual number of cases of each health endpoint attributable to PM<sub>2.5</sub>.

### 2.2.3 STATISTICAL UNCERTAINTY IN HEALTH EFFECTS

A 95% confidence interval has been calculated around the health effect estimates based on the 95% confidence intervals provided with the dose-response data.

### 2.2.4 SENSITIVITY ANALYSES

Two sensitivity analyses have been performed as part of this study to determine the effect of different possible input values or different assumptions on the health effect estimates. The results from both sensitivity analyses are presented in Section 5 of the report.

#### *Sensitivity to Background PM<sub>2.5</sub>*

The sensitivity of the health effect estimates to different assumptions about background PM<sub>2.5</sub> levels has been investigated by repeating the calculations using two alternative values for the background level. The results from this analysis are presented in Section 5.2.2.

#### *Sensitivity to Mathematical Form of the Dose-Response Relationship*

A second sensitivity analysis has been undertaken to determine the effect of using a different mathematical form for the dose-response relationship. Instead of the exponential form of the dose-response relationship, a linear form of the equation is considered in which the change in the number of cases of a given health endpoint  $\Delta y$  corresponding to a change in concentration  $\Delta x$  is represented by:

$$\Delta y = m \Delta x \quad [5]$$

Here  $m$  is the slope of the linear relationship, with units of health outcomes per  $\mu\text{g}/\text{m}^3$ .

The dose-response functions provided by the NEPC Project Team are expressed as a percentage increase in health outcomes associated with a  $10 \mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub> concentration, denoted  $z$  in Section 2.2.2. Assuming that the percentage increase in health outcomes is relative to the baseline health incidence  $y$ , the value of  $m$  to be used in Equation 5 for a given health endpoint was calculated according to

$$m = (z/100) \cdot y \cdot 1/\Delta x \quad [6]$$

Equation 6 was used to recalculate the health outcomes at current levels of exposure for a number of health endpoints using the dose-response data provided by the NEPC Project Team and a value for  $\Delta x$  of  $10 \mu\text{g}/\text{m}^3$ .

## 2.3 Scenario Evaluation

A number of scenarios have been modelled in which the  $\text{PM}_{2.5}$  concentrations were scaled down to be at or below a specified level (referred to as a 'scenario' level). The scenario levels are 35, 30, 25 and  $20 \mu\text{g}/\text{m}^3$  for peak 24-hour  $\text{PM}_{2.5}$ , and 10, 8 and  $5 \mu\text{g}/\text{m}^3$  for annual average  $\text{PM}_{2.5}$ . The methods used to scale down the  $\text{PM}_{2.5}$  concentrations are described in Section 2.3.1 below.

For each scenario, the reductions in  $\text{PM}_{2.5}$ -associated health effects that would result from scaling down the  $\text{PM}_{2.5}$  concentrations have been assessed by re-computing the health effects (using the procedure in Section 2.2.2) after the adjustment to the concentrations. These were then compared to the health effects associated with current (unadjusted)  $\text{PM}_{2.5}$  concentrations.

For the 24-hour scenario levels, the reductions in  $\text{PM}_{2.5}$ -associated health effects have been estimated for the short-term health endpoints in Table 1, S1 – S6. For the annual average scenario levels, the reductions in  $\text{PM}_{2.5}$ -associated health effects have been estimated for the long-term health endpoints L1 – L3.

### 2.3.1 SIMULATING $\text{PM}_{2.5}$ CONCENTRATIONS THAT ARE AT OR BELOW A PROPOSED SCENARIO LEVEL

#### 24-Hour Levels

To adjust daily 24-hour  $\text{PM}_{2.5}$  concentrations so that they are at or below a 24-hour scenario level ( $scl_d$ ), a linear rollback procedure has been used. The rollback procedure consists of the following steps:

1. For each city, determine the maximum 24-hour  $\text{PM}_{2.5}$  concentration  $MAX_d$  from the monitoring sites used in the exposure assessment and for the entire three year period of data.
2. Compute a multiplication factor  $f_d$  for each city with which all daily  $\text{PM}_{2.5}$  concentrations are be adjusted in order to be at or below the 24-hour scenario level  $scl_d$ :

$$f_d = (scl_d - x_0) / (MAX_d - x_0)$$

where  $x_0$  is the background  $\text{PM}_{2.5}$  concentration.

3. If  $x_{day}$  represents the composite 24-hour  $PM_{2.5}$  concentration for a city on a given day, the rolled-back concentration, denoted  $x_{day_{adj}}$ , is given by:

$$x_{day_{adj}} = x_0 + (x_{day} - x_0) * f_d$$

Only the above-background concentrations were adjusted using this procedure. For each city, all daily 24-hour concentrations for the three year period were adjusted using the same multiplication factor  $f_d$ . However, a different value of  $f_d$  was determined for each city since the concentration reductions required for each scenario depend on the current air quality in each city. The values for the four cities ranged between 0.75 and 1.0 for the 24-hour scenario level of  $35 \mu g/m^3$ , and between 0.38 and 0.56 for the 24-hour scenario level of  $20 \mu g/m^3$ .

In the USEPA methodology the multiplication factor determined in Step 2 above is based on the highest monitor-specific 98<sup>th</sup> percentile concentration, rather than the highest monitor-specific peak concentration determined in Step 1. In the present study the peak concentration was used, because the Consultancy Brief specified scenario levels that *peak*  $PM_{2.5}$  values were to be reduced to. Furthermore, the USEPA risk analysis is based on only a single year of data, rather than three. Since the present analysis is based on a three-year period, the multiplication factor for each city was based on the data for all three years.

#### *Annual Levels*

The procedure used to adjust annual average concentrations so that they are at or below an annual average scenario level ( $scl_a$ ) is similar to that used for the 24-hour scenario levels, and consists of the following steps:

1. For each city, determine the maximum annual average  $PM_{2.5}$  concentration  $MAX_a$  from the monitoring sites used in the exposure assessment and for the three year period of data.
2. Compute a multiplication factor  $f_a$  for each city with which the annual average concentrations for the three years are be adjusted in order to be at or below the annual scenario level  $scl_a$ :

$$f_a = (scl_a - x_0) / (MAX_a - x_0)$$

where  $x_0$  is the background  $PM_{2.5}$  concentration.

3. If  $x_{ann}$  represents the composite annual average  $PM_{2.5}$  concentration for a city for one year, the rolled-back annual average concentration, denoted  $x_{ann_{adj}}$ , is given by:

$$x_{ann_{adj}} = x_0 + (x_{ann} - x_0) * f_a$$

As for the 24-hour concentrations, only the above-background annual average concentrations were adjusted using this procedure. Since the

present analysis is based on a three-year period, rather than a single year as in the USEPA risk analysis, the same multiplication factor  $f_a$  was used for all three composite annual average concentrations. As for the 24-hour scenario levels, a different value of  $f_a$  was determined for each city. The values for the four cities ranged between 0.85 and 1.0 for the annual average scenario level of  $10 \mu\text{g}/\text{m}^3$ , and between 0.031 and 0.050 for the annual average scenario level of  $5 \mu\text{g}/\text{m}^3$ .

### 3. INPUT DATA

For the exposure assessment, current PM<sub>2.5</sub> data from population-oriented monitoring sites are required for Sydney, Melbourne, Brisbane and Perth. Both 24-hour and annual average data are required. Estimates are also needed of the size of the population exposed to PM<sub>2.5</sub> living in each city.

In addition, for both parts of the health risk characterisation, the following analysis inputs are required:

- estimates of background PM<sub>2.5</sub> appropriate to each location;
- dose-response functions from epidemiological studies which provide estimates of the relation between the health endpoints in Table 1 and PM<sub>2.5</sub> concentration;
- baseline health effects incidence rates corresponding to current levels of PM<sub>2.5</sub>, for each health endpoint in Table 1, and for each city.

Section 3.1 describes the air quality information used in this study. Section 3.2 presents the baseline health effect incidence rates for the health endpoints in Table 1, for each city. The dose-response data are presented in Section 3.3.

#### ***3.1 Air quality data***

Daily 24-hour average and annual average PM<sub>2.5</sub> concentrations determined using gravimetric methods (TEOM and ANSTO sampler) for Sydney, Brisbane and Perth were provided to EPA Victoria by the NEPC Project Team. The data were obtained from the NSW Environment Protection Authority, the Queensland Environmental Protection Agency and the WA Department of Environmental Protection respectively. Data were also available for Melbourne from the Victorian monitoring network. Since this study is restricted to the metropolitan regions of Sydney, Melbourne, Brisbane and Perth, additional PM<sub>2.5</sub> data provided for the Illawarra and Lower Hunter regions in New South Wales and Bunbury in Western Australia have been excluded from the exposure assessment.

Table 3.1 summarises the data availability including a description of the nature of each monitoring site from which data have been obtained. Any data exclusions and modifications are discussed below.

#### *Use of Data from Population-Oriented Monitoring Sites Only*

The USEPA methodology requires that only monitoring data from population-oriented monitors are included in the spatial averaging procedures. All but one monitoring station listed in Table 3.1 satisfy this criterion, and are therefore included in the study. The exception is the Brisbane CBD monitoring station, which has been designated as 'Peak'. The NEPM process specifically excludes 'Peak' monitoring sites. The

Brisbane CBD station is sited within the central business district of Brisbane and the PM<sub>2.5</sub> levels recorded there are not considered representative of the levels to which the general population would be exposed. Thus data from the Brisbane CBD monitoring station have been excluded.

Table 3.1. PM<sub>2.5</sub> monitoring data.

<i>City</i>	<i>Period</i>	<i>Monitoring Station</i>	<i>Description</i>	<i>PM<sub>2.5</sub> Method</i>
Sydney	1999 - 2001	Liverpool	Mixed residential / commercial	TEOM
		Lidcombe	Mixed residential / commercial	TEOM
		Woolooware	Residential	TEOM
		Richmond	Semi-rural / residential	TEOM
		Westmead	Residential	TEOM
		Earlwood	Residential	TEOM
Melbourne	1998 - 2000	Footscray	Residential / industrial	TEOM
		Alphington	Residential	TEOM
		Brighton	Residential	TEOM
Brisbane	1999 - 2001	Brisbane CBD	Peak	ANSTO
		Rocklea	Residential/ light industrial	TEOM + ANSTO
		Springwood	Residential	TEOM
Perth	1999 - 2001	Caversham	Semi-rural	TEOM
		Duncraig	Residential	TEOM

### *PM<sub>2.5</sub> Instrumentation*

For consistency, only PM<sub>2.5</sub> data obtained using TEOM instrumentation have been used in this study. TEOM data were provided for all jurisdictions. As can be seen from Table 3.1, PM<sub>2.5</sub> data collected using ANSTO samplers were provided for the Brisbane CBD and Rocklea monitoring stations in Brisbane. Since data from Brisbane CBD have already been excluded because they are not considered representative of general exposure, this affects only ANSTO sampler data collected at the Rocklea monitoring station for which TEOM data are also available.

All states except Queensland have used the TEOM PM<sub>10</sub> default settings in their TEOMs to monitor PM<sub>2.5</sub>. A function was built in to the TEOMs by the manufacturers in order to make PM<sub>10</sub> readings match more closely with the US Federal Reference method (HiVol). The function is  $y=1.03x + 3.0$ , where  $x$  is the original measurement and  $y$  the adjusted concentration. As a result, all TEOM data from Sydney, Melbourne and Perth used in this study have been adjusted by default using the  $y=1.03x + 3.0$  function. The exception is the TEOM data from Queensland. Since the correlation between PM<sub>2.5</sub> TEOM data and the reference method (USEPA) is not known, it is not certain how the default adjustment should be corrected. However, to ensure consistency with the other states, the Rocklea and

Springwood TEOM data used in this study have been adjusted using the same function,  $y=1.03x + 3.0$ .

### *Data Influenced by Major Bushfires*

Very high 24-hour  $PM_{2.5}$  concentrations were observed during bushfires in Sydney (25-31 December 2001) and south-east Queensland (Rocklea monitoring station, 7-13 October 2001).

To properly assess typical exposure experienced in Australian cities, representative data are needed. The data influenced by these major fires are clearly not representative of typical current levels. For this reason, a second set of  $PM_{2.5}$  data have been created for Sydney and Brisbane in which the data associated with major bushfires (Sydney 25-31 December 2001 and Brisbane 7-13 October 2001) have been excluded. The health risks associated with current levels of exposure to  $PM_{2.5}$  have been estimated for both sets of data for these two cities.

Using a simple scaling or “rollback” technique, the inclusion of high readings from a rare event would result in unrealistically low scenario concentrations. This would underestimate the general population exposure in all scenarios. Hence the scenario evaluation using the “rollback” technique has been performed only using the  $PM_{2.5}$  data sets in which the data influenced by major bushfires have been excluded.

Where long term health end point calculations have been done both with and without bushfire-affected data, annual averages were required for both situations. The original annual average data provided by the NEPC Project Team were derived from 1-hour averages; these constitute the “with bushfire” annual average values. To estimate the annual average after excluding data, since the original 1-hour data were not available, the exclusion was performed on the 24-hour data, and an annual average was then derived from the 24-hour averages. The true 1-hour-derived annual average was then estimated by applying a small correction factor to account for the difference in data capture rates. The factor was simply derived from the ratio of the original (supplied) annual average to the annual average obtained from 24-hour values (all data included).

## **3.2 Baseline Health Effects Incidence Data**

The per-capita daily rates for all endpoints listed in Table 1 for all four cities are summarised in Table 3.2.

The Sydney baseline incidence rates for endpoints S1-S3 and L1 were provided to the NEPC Project Team by the Australian Bureau of Statistics, and for S4 - S6 by NSW Health. The baseline data for endpoints L2 and L3 for all cities were also provided to the NEPC Project Team by the Australian Bureau of Statistics. For Melbourne, the mortality data were

provided by ABS, and the hospital admissions data were provided by the Victorian Department of Human Services. All data for Brisbane and Perth were obtained by the NEPC Project Team from the relevant State health authorities. For Brisbane, the relevant population is that of the 'Brisbane City' Statistical Subdivision as defined by the Australian Bureau of Statistics. For Perth the relevant population was assumed to be that of the Perth Statistical Division. The source data are given in Appendix 1.

Table 3.2. Per-capita baseline incidence rates expressed as daily incidence per 100,000.

<i>End-point</i>	<i>Description</i>	<i>Age group</i>	<i>Sydney</i>	<i>Melbourne</i>	<i>Brisbane</i>	<i>Perth</i>
<b>Short Term Mortality</b>						
S1	All cause	All ages	1.65	1.85	1.98	1.62
S2	Respiratory	All ages	0.14	0.15	0.19	0.16
S3	Cardiovascular	All ages	0.72	0.81	0.92	0.66
<b>Hospital Admissions</b>						
S4	Asthma	All ages	0.84	0.62	0.73	0.69
S5	Cardiovascular disease	Elderly	16.93	15.63	17.37	17.60
S6	COPD	Elderly	3.37	1.28	2.35	2.98
<b>Long Term Mortality</b>						
L1	All cause	All ages	1.65	1.85	1.98	1.62
L2	Lung cancer	All ages	0.094	0.093	0.102	0.105
L3	Cardiopulmonary disease	All ages	0.85	0.76	0.85	0.75

### **3.3 Dose-Response Relationships**

Table 3.3 summarises the dose-response information for each health endpoint in Table 1. The data were provided by the NEPC Project Team. The dose-response relationships are presented as a percentage increase per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  concentration, together with a 95% confidence interval.

The dose-response relationships have been used to derive a value of the  $\text{PM}_{2.5}$  coefficient  $\beta$  with a 95% confidence interval for each endpoint, assuming a log-linear form of the dose-response function (see Section 2.2.2).



Table 3.3. Dose-response data for the health endpoints specified in Table 1.

<i>End-point</i>	<i>Description</i>	<i>Age group</i>	<i>% Increase per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub></i>	<i>β</i>
<b>Short Term Mortality</b>				
S1	All cause	All ages	2.3 (1.3, 3.3)	0.0023 (0.0013, 0.0032)
S2	Respiratory	All ages	8.6 (5.2, 12.4)	0.0083 (0.0051, 0.0117)
S3	Cardiovascular	All ages	1.04 (0.15, 1.94)	0.0010 (0.0002, 0.0019)
<b>Hospital Admissions</b>				
S4	Asthma	All ages	2.6 (1, 4.2)	0.0026 (0.0010, 0.0041)
S5	Cardiovascular disease	Elderly	1.7 (1, 2.4)	0.0017 (0.0010, 0.0024)
S6	Chronic obstructive pulmonary disease	Elderly	2.0 (0.4, 3.8)	0.0020 (0.0004, 0.0037)
<b>Long Term Mortality</b>				
L1	All cause	All ages	6 (2, 11)	0.0058 (0.0020, 0.0104)
L2	Lung cancer	All ages	14 (4, 23)	0.0131 (0.0039, 0.0207)
L3	Cardiopulmonary disease	All ages	9 (3, 16)	0.0086 (0.0030, 0.0148)

## 4. RESULTS

### 4.1 Sydney

#### 4.1.1 SYDNEY EXPOSURE ASSESSMENT

A map displaying the physical boundaries of the Sydney Statistical Division as defined by the Australian Bureau of Statistics is shown in Figure E1a. The location of each relevant monitoring site as listed in Table 3.1 is also indicated. Population data for the Sydney Statistical Division are given in Table E1a.

Table E1a. Population of the Sydney Statistical Division, 2001.

<i>Age Group</i>	<i>Population</i>
0-14	798,826
65+	472,821
All ages	3,997,321

Severe bushfires led to unusually high PM<sub>2.5</sub> levels at all Sydney monitoring stations between 25 and 31 December 2001. Analyses are presented both including and excluding the bushfire affected data.

Frequency graphs of the daily 24-hour PM<sub>2.5</sub> data are presented in Figures E1b(1) and E1b(2) for 1999, 2000 and 2001. Following the methodology outlined in Section 2.1, the graphs display the composite PM<sub>2.5</sub> concentrations, obtained by averaging the daily data from the Liverpool, Lidmore, Woolooware, Richmond, Westmead and Earlwood monitoring stations.

Figures E1c(1) and E1c(2) display the inverse cumulative frequency distributions for 1999, 2000 and 2001, linking the PM<sub>2.5</sub> concentration  $x$  with the percentage of days on which the concentration was higher than  $x$ .

Tables E1b-d summarise the results for 1999, 2000 and 2001 respectively. Shown in these tables are the maximum 24-hour average PM<sub>2.5</sub> concentrations at each monitoring site and the maximum composite 24-hour PM<sub>2.5</sub> concentration for each year. Note that the maximum composite PM<sub>2.5</sub> concentration is not necessarily an average of the maximum concentrations at the individual monitoring sites. This is because the maximum concentrations at the monitoring sites were not always all recorded on the same day of the year.

To give a further indication of the frequency distribution of the daily PM<sub>2.5</sub> concentrations, the number of days per year on which the concentration was higher than each of the 24-hour scenario levels (20, 25, 30 and 35 µg/m<sup>3</sup>) are also reported in Tables E1b-d.

Table E1e displays the 3-year overall maximum 24-hour average PM<sub>2.5</sub> concentrations for 1999-2001, and the total number of days on which the

concentration was higher than each 24-hour scenario level during this period.

Table E1b. 24-Hour PM<sub>2.5</sub> results for Sydney, 1999.

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
<i>Maximum 24-hour PM2.5 (µg/m3)</i>	25.4	26.2	23.2	33.1	25.3	27.6	25.1
<i># Days &gt; 20 µg/m3</i>	5	7	1	3	3	10	1
<i># Days &gt; 25 µg/m3</i>	1	1	0	1	1	4	1
<i># Days &gt; 30 µg/m3</i>	0	0	0	1	0	0	0
<i># Days &gt; 35 µg/m3</i>	0	0	0	0	0	0	0

Table E1c. 24-Hour PM<sub>2.5</sub> results for Sydney, 2000.

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
<i>Maximum 24-hour PM2.5 (µg/m3)</i>	45.1	45.1	33.2	*	*	35.4	37.6
<i># Days &gt; 20 µg/m3</i>	12	3	7	*	*	6	6
<i># Days &gt; 25 µg/m3</i>	5	2	3	*	*	3	3
<i># Days &gt; 30 µg/m3</i>	2	1	2	*	*	2	2
<i># Days &gt; 35 µg/m3</i>	2	1	0	*	*	1	1

(\*) insufficient data (< 75% of the year) available

Table E1d(1). 24-Hour PM<sub>2.5</sub> results for Sydney, 2001 (December 2001 [bushfires excluded](#)).

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
<i>Maximum 24-hour PM2.5 (µg/m3)</i>	24.4	24.8	23.7	*	*	26.6	23.3
<i># Days &gt; 20 µg/m3</i>	9	2	7	*	*	19	3
<i># Days &gt; 25 µg/m3</i>	0	0	0	*	*	2	0
<i># Days &gt; 30 µg/m3</i>	0	0	0	*	*	0	0
<i># Days &gt; 35 µg/m3</i>	0	0	0	*	*	0	0

(\*) insufficient data (< 75% of the year) available

Table E1d(2). 24-Hour PM<sub>2.5</sub> results for Sydney, 2001 (December 2001 [bushfires included](#)).

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
<i>Maximum 24-hour PM2.5 (µg/m3)</i>	118.6	82.9	81.9	*	*	81.7	93.7
<i># Days &gt; 20 µg/m3</i>	15	7	9	*	*	26	10
<i># Days &gt; 25 µg/m3</i>	6	4	2	*	*	8	7
<i># Days &gt; 30 µg/m3</i>	6	4	1	*	*	6	7
<i># Days &gt; 35 µg/m3</i>	6	4	1	*	*	5	6

(\*) insufficient data (< 75% of the year) available

Table E1e(1). 24-Hour PM<sub>2.5</sub> results for Sydney, 1999-2001 (December 2001 bushfires excluded).

3 Year Period	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM <sub>2.5</sub> (µg/m <sup>3</sup> )	45.1	45.1	33.2	*	*	35.4	37.6
# Days > 20 µg/m <sup>3</sup>	26	12	15	*	*	35	10
# Days > 25 µg/m <sup>3</sup>	6	3	3	*	*	9	4
# Days > 30 µg/m <sup>3</sup>	2	1	2	*	*	2	2
# Days > 35 µg/m <sup>3</sup>	2	1	0	*	*	1	1

(\*) insufficient data (< 75% of the year) available

Table E1e(2). 24-Hour PM<sub>2.5</sub> results for Sydney, 1999-2001 (December 2001 bushfires included).

3 Year Period	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM <sub>2.5</sub> (µg/m <sup>3</sup> )	118.6	82.9	81.9	*	*	81.7	93.7
# Days > 20 µg/m <sup>3</sup>	32	17	17	*	*	42	17
# Days > 25 µg/m <sup>3</sup>	12	7	5	*	*	15	11
# Days > 30 µg/m <sup>3</sup>	8	5	3	*	*	8	9
# Days > 35 µg/m <sup>3</sup>	8	5	1	*	*	6	7

(\*) insufficient data (< 75% of the year) available

The annual average PM<sub>2.5</sub> concentrations derived from hourly data at each monitoring site and the composite annual average concentrations are shown in Tables E1f(1) and E1f(2).

The supplied annual averages included the bushfire-affected data, and are shown in Table E1f(2).

To estimate the annual averages excluding the bushfire, daily averages without the 25-31 December 2001 data were averaged. Then a correction factor was applied to account for the difference between 1-hour derived annual averages and 24-hour derived annual averages. The results are shown in Table E1f(1).

Table E1f(1). Estimated annual average PM<sub>2.5</sub> results for Sydney (Dec 2001 bushfire excluded)

Year	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
1999	9.7	10.0	8.1	6.7	9.9	10.2	9.1
2000	10.4	9.9	9.6	*	*	10.3	10.1
2001	10.8	10.5	10.9	*	*	10.7	10.4

Table E1f(2). Annual average PM<sub>2.5</sub> results for Sydney (Dec 2001 bushfires included)

Year	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
1999	9.7	10.0	8.1	6.7	9.9	10.2	9.1
2000	10.4	9.9	9.6	*	*	10.3	10.1
2001	11.8	11.1	11.1	*	*	11.6	11.4

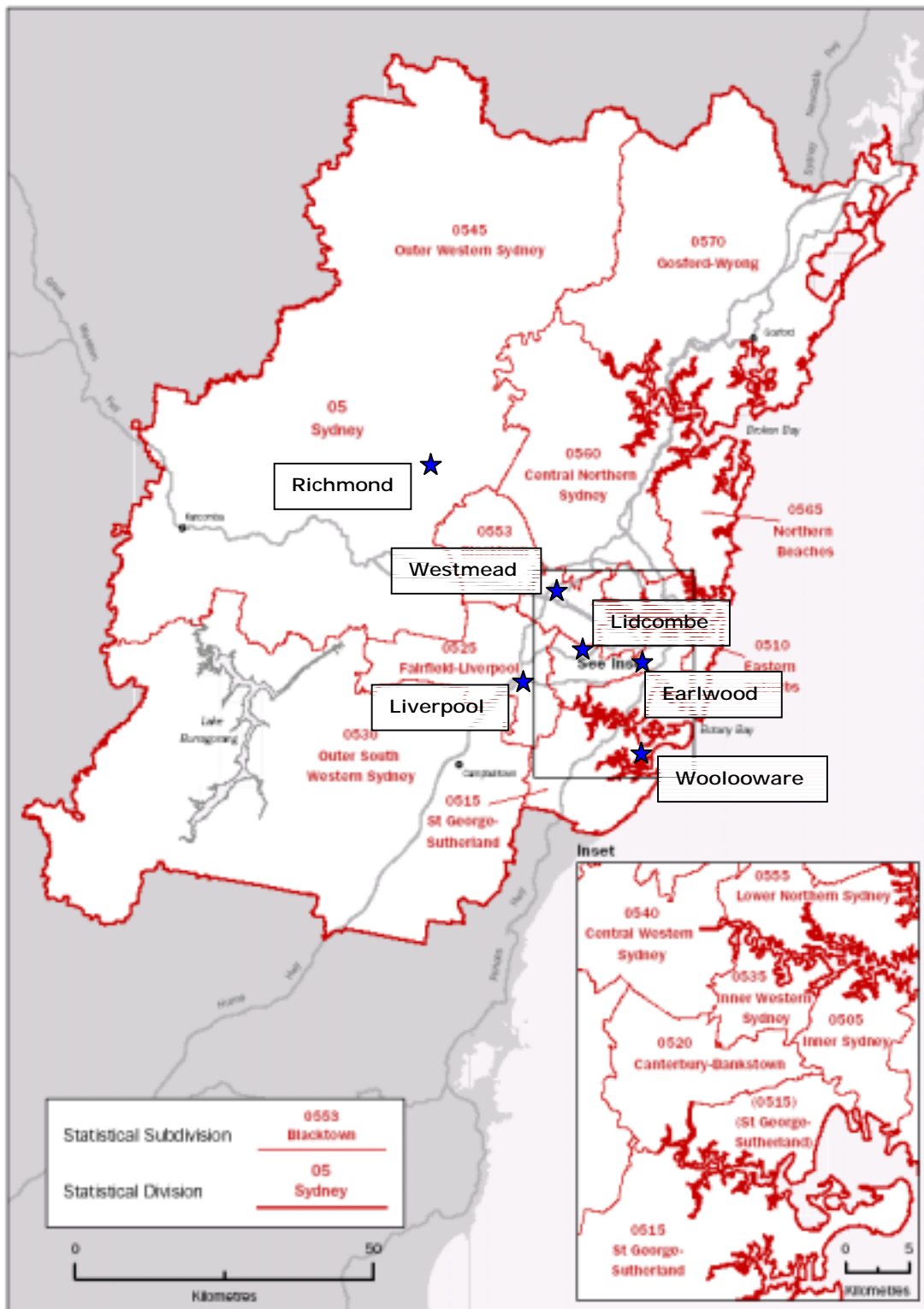


Figure E1a. Map of the Sydney Statistical Division. The locations of the PM<sub>2.5</sub> monitoring stations are indicated with blue stars.

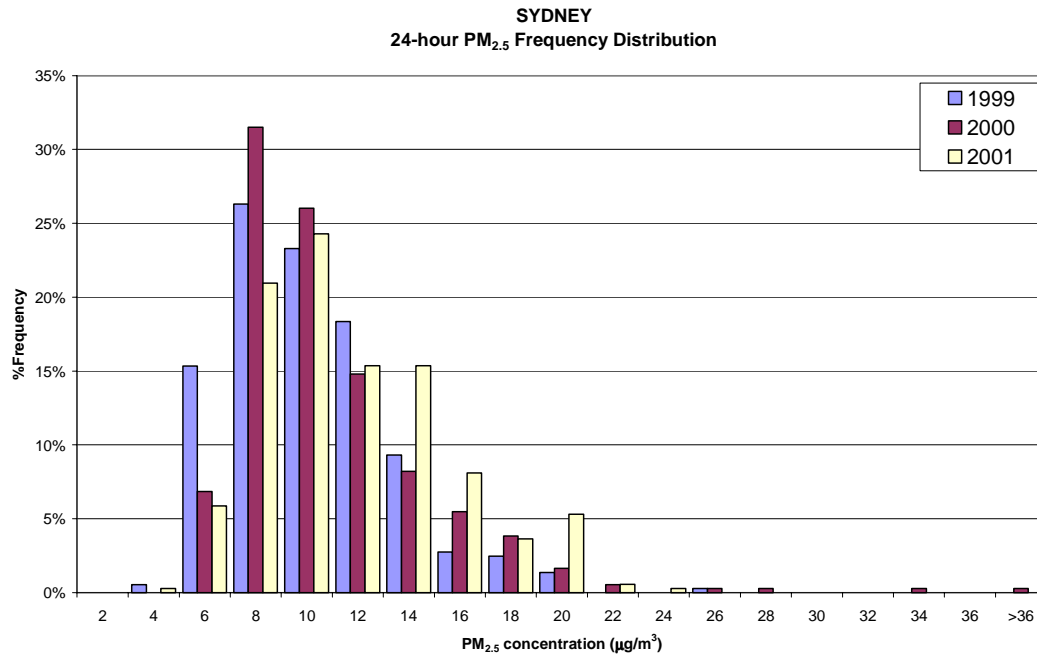


Figure E1b(1). Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Sydney for 1999, 2000 and 2001. Bushfire-affected data (25-31 December 2001) have been excluded. The concentrations for the frequency distribution on the X-axis denote concentration bins of X-2 to X µg/m<sup>3</sup>.

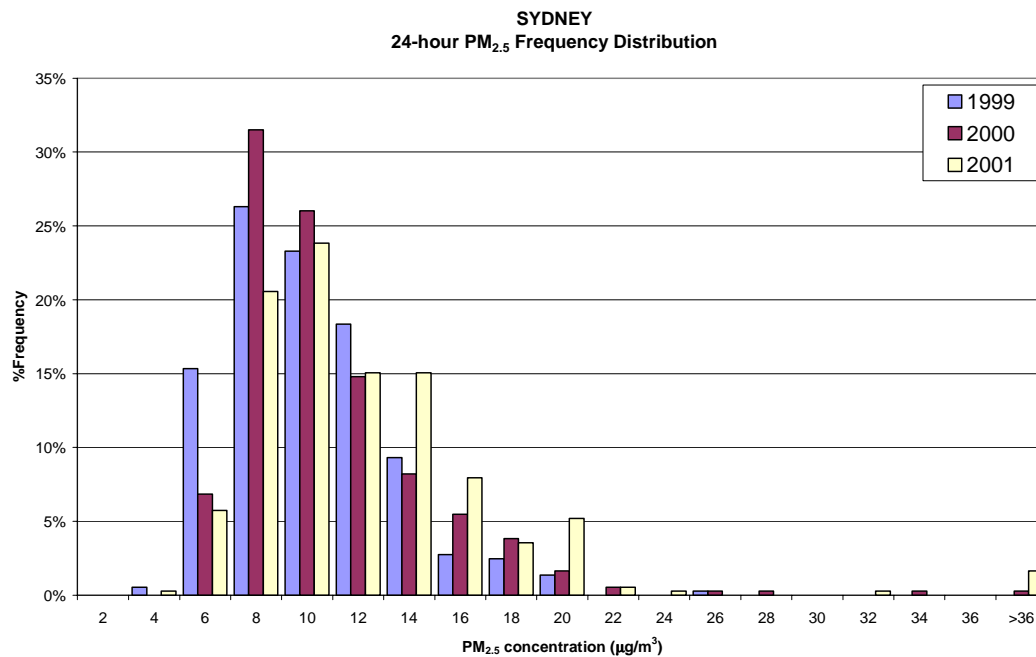


Figure E1b(2). Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Sydney for 1999, 2000 and 2001. Bushfire-affected data (25-31 December 2001) have been included. The concentrations for the frequency distribution on the X-axis denote concentration bins of X-2 to X µg/m<sup>3</sup>.

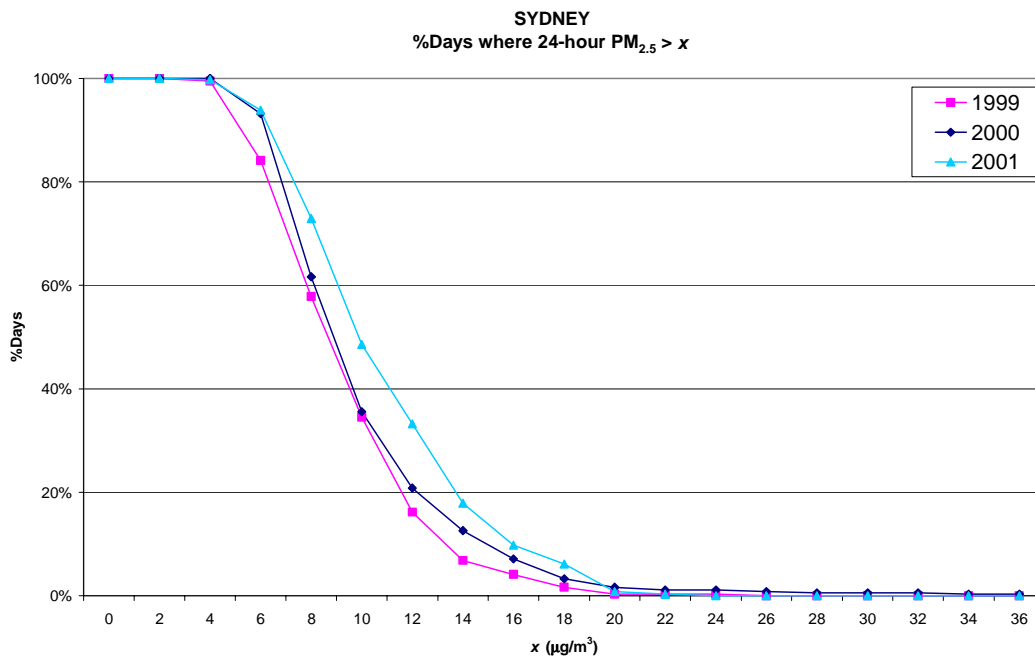


Figure E1c(1). Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour  $PM_{2.5}$  concentrations in Sydney for 1999, 2000 and 2001. Bushfire-affected data (25-31 December 2001) have been excluded.

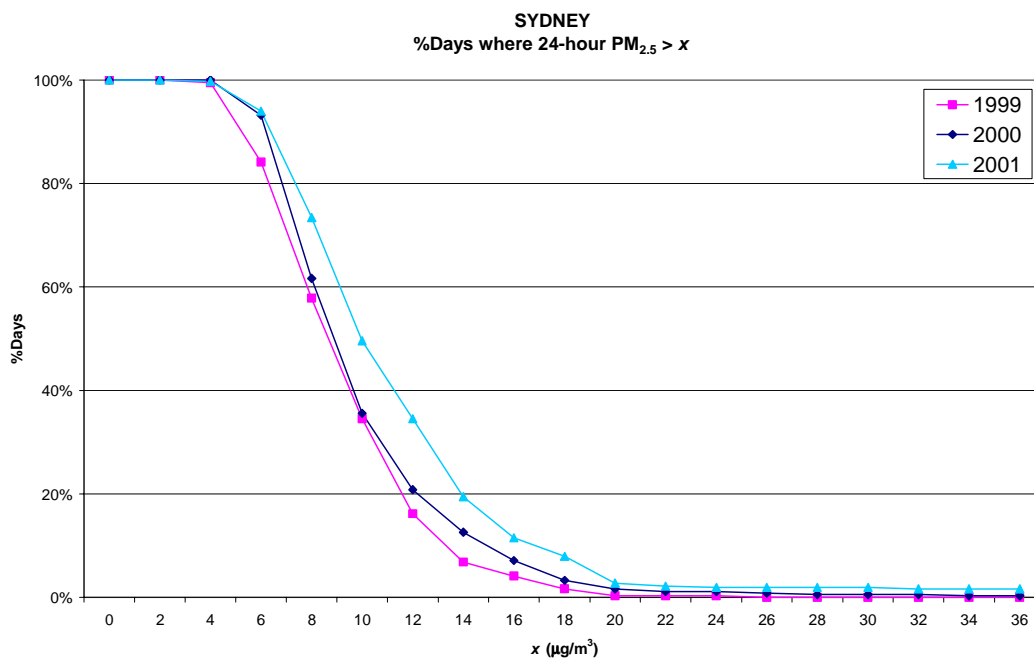


Figure E1c(2). Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour  $PM_{2.5}$  concentrations in Sydney for 1999, 2000 and 2001. Bushfire-affected data (25-31 December 2001) have been included.

#### **4.1.2 SYDNEY RISK ASSESSMENT**

Table R1.1 lists the estimated health outcomes attributable to PM<sub>2.5</sub> concentrations above the background level in the Sydney region. The results are presented both as absolute values (number of cases per year) and as a proportion of the usual baseline incidence. The ranges in brackets represent uncertainty due to the 95% confidence interval on the dose response relationship. The results are averages over the three-year study period.

In the first part of the table, the major bushfire impact of 25-31 December 2001 has been excluded. The last two rows show the same results with the bushfires included.

#### **4.1.3 SYDNEY SCENARIO ASSESSMENT**

Table S1.1 lists the results of re-running the risk analysis for Sydney with modified concentrations as described in the methodology section. Results are presented for 7 scenarios: 4 modifications to the 24-hour PM<sub>2.5</sub> concentrations, and 3 modifications to the annual average PM<sub>2.5</sub> concentrations.

The results are presented as the total number of health outcomes avoided per year, averaged over the three-year study period. Figures S1.1 and S1.2 present the same information graphically for the 24-hour and annual average scenario levels respectively.

Data affected by the major bushfires of 25-31 December 2001 have been excluded from this analysis.



Table R1.1 - PM<sub>2.5</sub> Risk Assessment Results - Annual Health Outcomes attributable to above-background PM<sub>2.5</sub> - SYDNEY

	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
Estimated cases per year 95% conf. interval	274 ( 156 - 389 )	81 ( 50 - 114 )	55 ( 8 - 101 )	157 ( 61 - 251 )	246 ( 146 - 346 )	58 ( 12 - 108 )	699 ( 240 - 1237 )	88 ( 27 - 136 )	527 ( 183 - 893 )
Estimated cases as % of baseline rate 95% conf. interval	1.1% ( 0.6% - 1.6% )	4.0% ( 2.5% - 5.6% )	0.5% ( 0.1% - 1.0% )	1.3% ( 0.5% - 2.0% )	0.8% ( 0.5% - 1.2% )	1.0% ( 0.2% - 1.9% )	2.9% ( 1.0% - 5.1% )	6.4% ( 2.0% - 9.9% )	4.3% ( 1.5% - 7.2% )
<b>With bushfires:</b>									
Cases per year	290	85	58	167	262	61	743	93	560
As % of baseline	1.2%	4.2%	0.6%	1.4%	0.9%	1.1%	3.1%	6.8%	4.5%

NB: confidence intervals shown above are based on statistical uncertainty in the dose-response relationships.

Table S1.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - SYDNEY

€	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
<b>Scenario</b>									
Scenario: 24h PM2.5 <35	70	21	14	40	63	15			
Scenario: 24h PM2.5 <30	104	31	21	60	94	22			
Scenario: 24h PM2.5 <25	138	41	28	79	125	29			
Scenario: 24h PM2.5 <20	172	51	35	99	155	36			
Scenario: Annual PM2.5 <10							100	12	75
Scenario: Annual PM2.5 <8							329	39	247
Scenario: Annual PM2.5 <5							677	80	510

Figure S1.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - SYDNEY

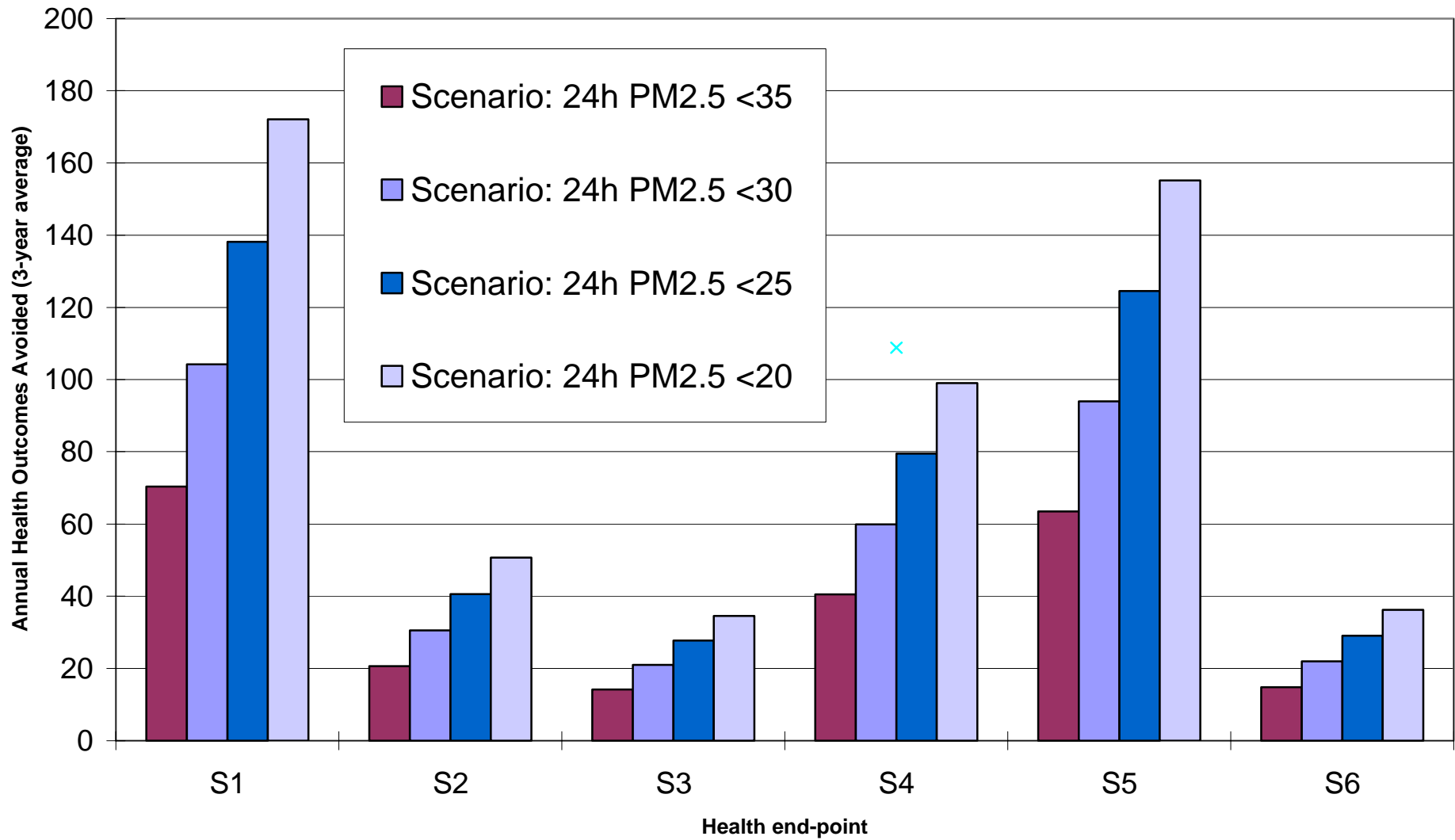
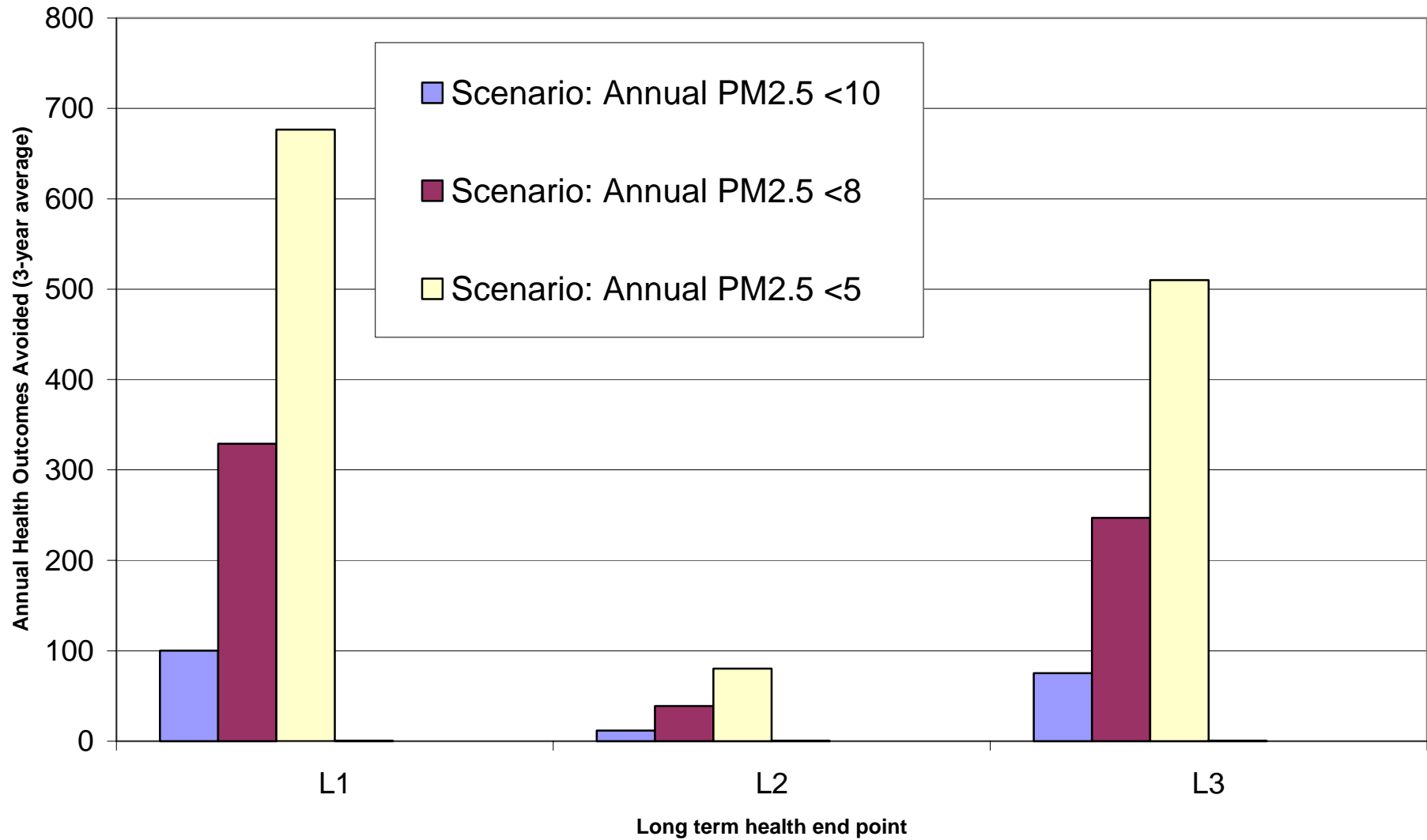


Figure S1.2 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year -SYDNEY



## 4.2 Melbourne

### 4.2.1 MELBOURNE EXPOSURE ASSESSMENT

A map displaying the physical boundaries of the Melbourne Statistical Division as defined by the Australian Bureau of Statistics is shown in Figure E2a. The location of each relevant monitoring site as listed in Table 3.1 is also indicated. Population data for the Melbourne Statistical Division are given in Table E2a.

Table E2a. Population of the Melbourne Statistical Division, 2001.

<i>Age Group</i>	<i>Population</i>
0-14	660,378
65+	406,797
All ages	3,366,542

Frequency graphs of daily 24-hour PM<sub>2.5</sub> data are presented in Figure E2b for 1998, 1999 and 2000. As for Sydney, the graphs display the composite PM<sub>2.5</sub> concentrations, obtained by averaging the data obtained at Footscray, Alphington and Brighton monitoring stations. The inverse cumulative frequency distributions are displayed in Figure E2c.

The 24-hour average PM<sub>2.5</sub> results for 1998, 1999, 2000 and the three years combined are shown in Tables E2b-e respectively. The annual average PM<sub>2.5</sub> concentrations derived from hourly data at each monitoring site and the composite annual average concentrations are shown in Table E2f.

Table E2b. 24-Hour PM<sub>2.5</sub> results for Melbourne, 1998.

	Alphington	Brighton	Footscray	<b>Composite</b>
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	34.0	31.0	35.7	33.2
<i># Days &gt; 20 µg/m<sup>3</sup></i>	18	17	8	15
<i># Days &gt; 25 µg/m<sup>3</sup></i>	5	4	2	4
<i># Days &gt; 30 µg/m<sup>3</sup></i>	1	2	1	1
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	1	0

Table E2c. 24-Hour PM<sub>2.5</sub> results for Melbourne, 1999.

	Alphington	Brighton	Footscray	<b>Composite</b>
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	24.5	30.5	22.5	24.9
<i># Days &gt; 20 µg/m<sup>3</sup></i>	9	6	2	4
<i># Days &gt; 25 µg/m<sup>3</sup></i>	0	1	0	0
<i># Days &gt; 30 µg/m<sup>3</sup></i>	0	1	0	0
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	0	0

Table E2d. 24-Hour PM<sub>2.5</sub> results for Melbourne, 2000

	Alphington	Brighton	Footscray	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	43.9	29.9	*	30.8
<i># Days &gt; 20 µg/m<sup>3</sup></i>	7	4	*	5
<i># Days &gt; 25 µg/m<sup>3</sup></i>	5	2	*	4
<i># Days &gt; 30 µg/m<sup>3</sup></i>	2	0	*	2
<i># Days &gt; 35 µg/m<sup>3</sup></i>	1	0	*	0

(\*) insufficient data (< 75% of the year) available

Table E2e. 24-Hour PM<sub>2.5</sub> results for Melbourne, 1998-2000

	Alphington	Brighton	Footscray	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	43.9	31.0	35.7	33.2
<i># Days &gt; 20 µg/m<sup>3</sup></i>	34	27	10	24
<i># Days &gt; 25 µg/m<sup>3</sup></i>	10	7	2	8
<i># Days &gt; 30 µg/m<sup>3</sup></i>	3	3	1	3
<i># Days &gt; 35 µg/m<sup>3</sup></i>	1	0	1	0

Table E2f. Annual average PM<sub>2.5</sub> results for Melbourne (µg/m<sup>3</sup>)

Year	Alphington	Brighton	Footscray	Composite
1998	9.8	9.2	9.1	9.4
1999	9.5	8.9	8.6	9.0
2000	9.2	6.9	*	8.0

(\*) insufficient data (< 75% of the year) available

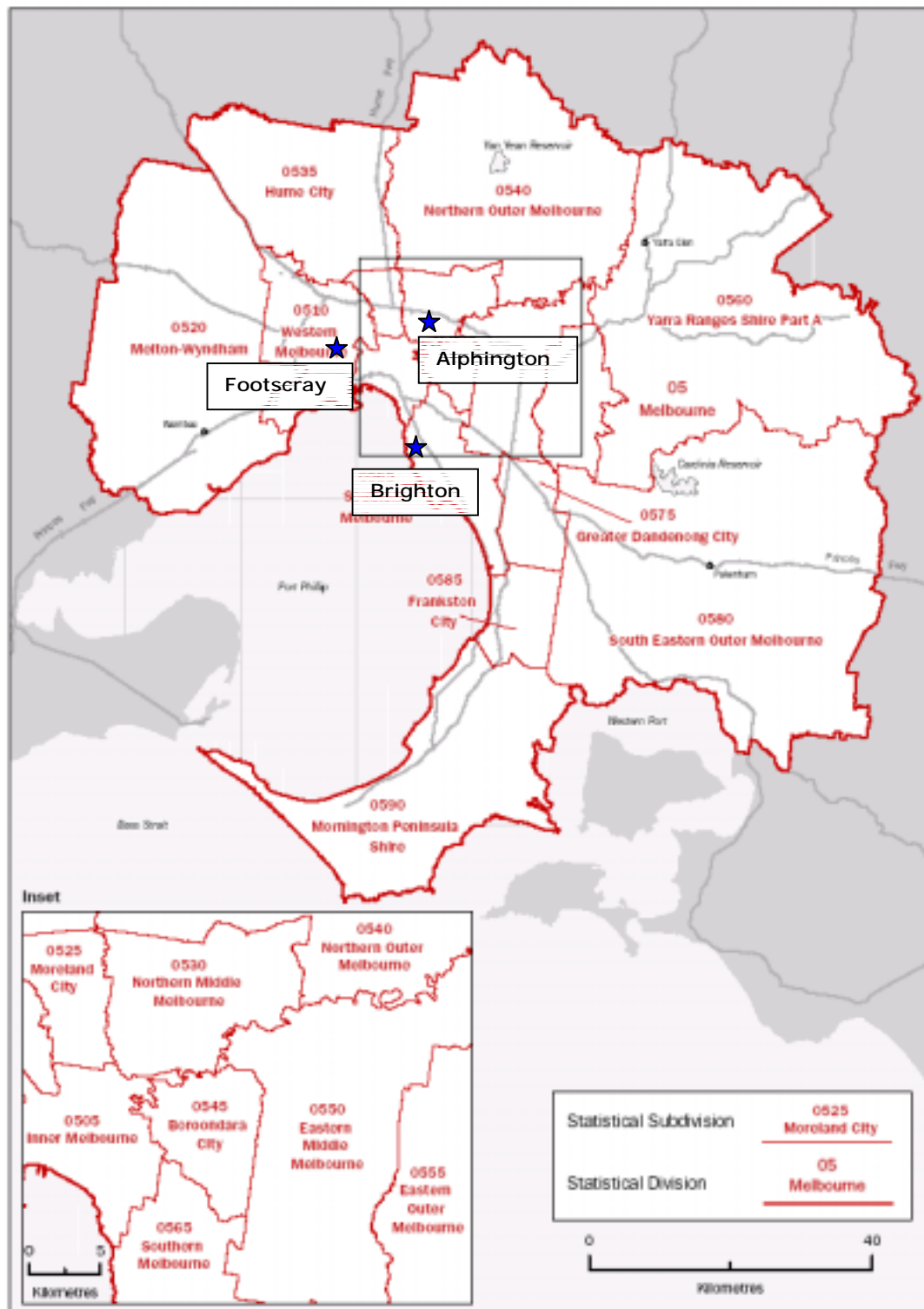


Figure E2a. Map of the Melbourne Statistical Division. The locations of the PM<sub>2.5</sub> monitoring stations are indicated with blue stars.

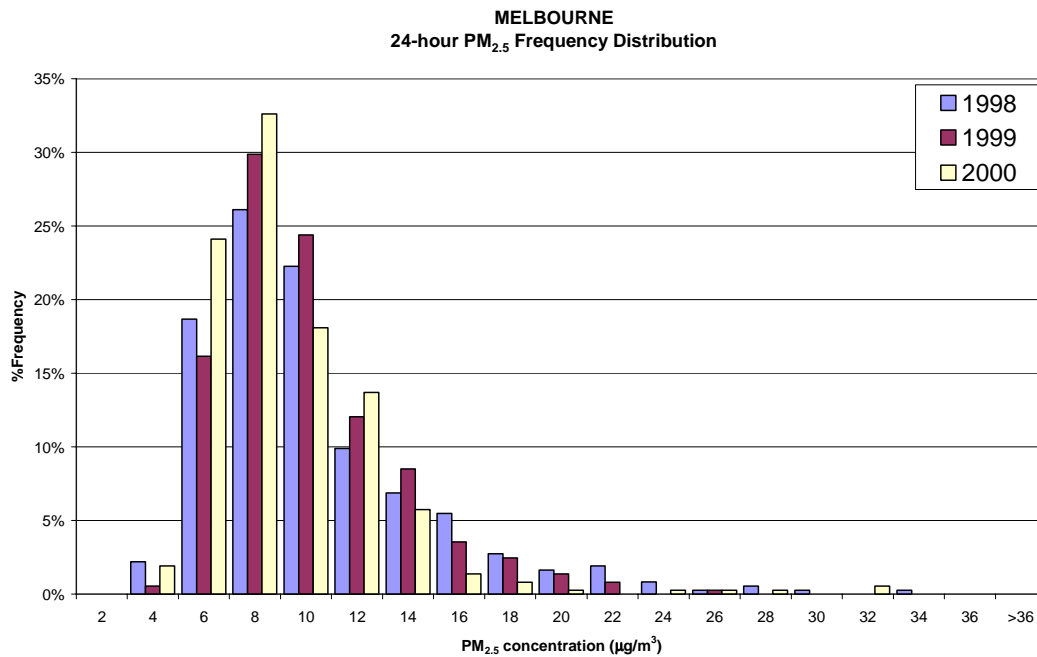


Figure E2b. Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Melbourne for 1998, 1999 and 2000. The concentrations for the frequency distribution on the X-axis denote concentration bins of  $X-2$  to  $X$   $\mu\text{g}/\text{m}^3$ .

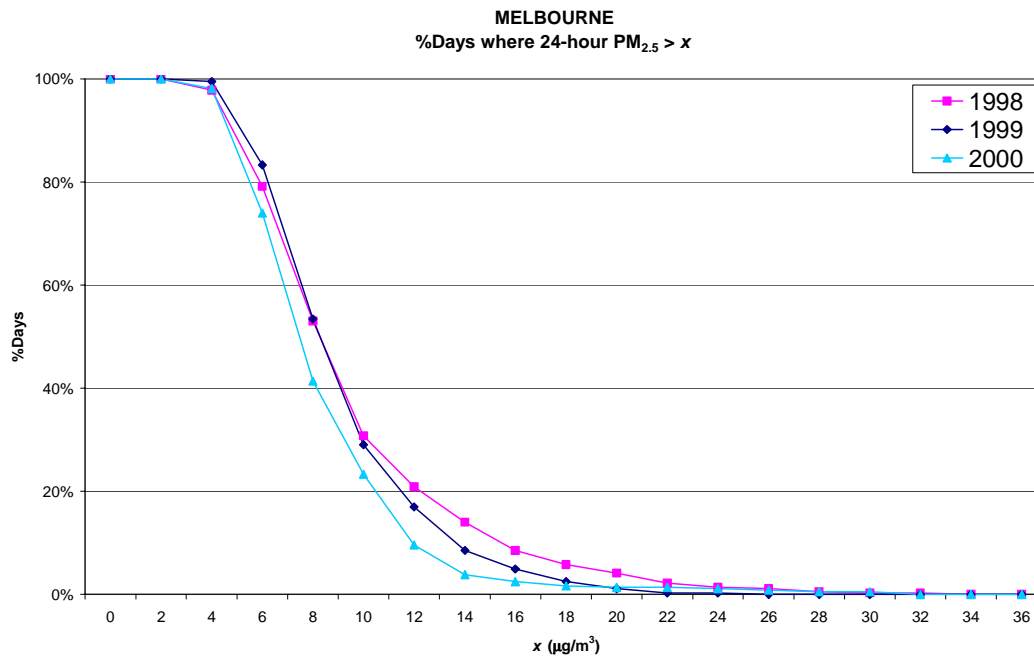


Figure E2c. Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour PM<sub>2.5</sub> concentrations in Melbourne for 1998, 1999 and 2000.



## 4.2.2 Melbourne Risk Assessment

Table R2.1 lists the estimated health outcomes attributable to above-background PM<sub>2.5</sub> in the Melbourne region, both as absolute values (number of cases per year) and as a proportion of the usual baseline incidence. The results are averages over the three-year study period.

## 4.2.3 Melbourne Scenario Assessment

Table S2.1 lists the results of re-running the risk analysis for Melbourne with modified concentrations as described in the methodology section.

The results are presented as the total number of health outcomes avoided per year, averaged over the three-year study period. Figures S2.1 and S2.2 present the same information graphically for the 24-hour and annual average scenario levels respectively.

Table R2.1 - PM<sub>2.5</sub> Risk Assessment Results -Annual Health Outcomes attributable to above-background PM<sub>2.5</sub> - MELBOURNE

	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
Estimated cases per year	207	60	41	78	157	15	524	58	316
95% conf. interval	( 118 - 294 )	( 37 - 83 )	( 6 - 77 )	( 30 - 124 )	( 93 - 220 )	( 3 - 28 )	( 179 - 929 )	( 18 - 91 )	( 110 - 538 )
Estimate cases as % of baseline rate	0.9%	3.2%	0.4%	1.0%	0.7%	0.8%	2.3%	5.1%	3.4%
95% conf. interval	( 0.5% - 1.3% )	( 2.0% - 4.5% )	( 0.1% - 0.8% )	( 0.4% - 1.6% )	( 0.4% - 0.9% )	( 0.2% - 1.5% )	( 0.8% - 4.1% )	( 1.6% - 8.0% )	( 1.2% - 5.8% )

NB: confidence intervals shown above are based on statistical uncertainty in the dose-response relationships.

Table S2.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - MELBOURNE

€	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	Mortality	Mortality	Mortality	Hospital Admissions	Hospital Admissions	Hospital Admissions	Mortality	Mortality	Mortality
	All cause	Respiratory	Cardiovascular	Asthma	Cardiovascular disease	COPD	All cause	Lung cancer	Cardio-pulmonary disease
<b>Scenario</b>									
Scenario: 24h PM2.5 <35	48	14	10	18	36	4			
Scenario: 24h PM2.5 <30	74	21	15	28	57	5			
Scenario: 24h PM2.5 <25	101	29	20	38	77	7			
Scenario: 24h PM2.5 <20	128	37	26	48	97	9			
Scenario: Annual PM2.5 <10							0	0	0
Scenario: Annual PM2.5 <8							191	23	115
Scenario: Annual PM2.5 <5							504	61	304

Figure S2.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - MELBOURNE

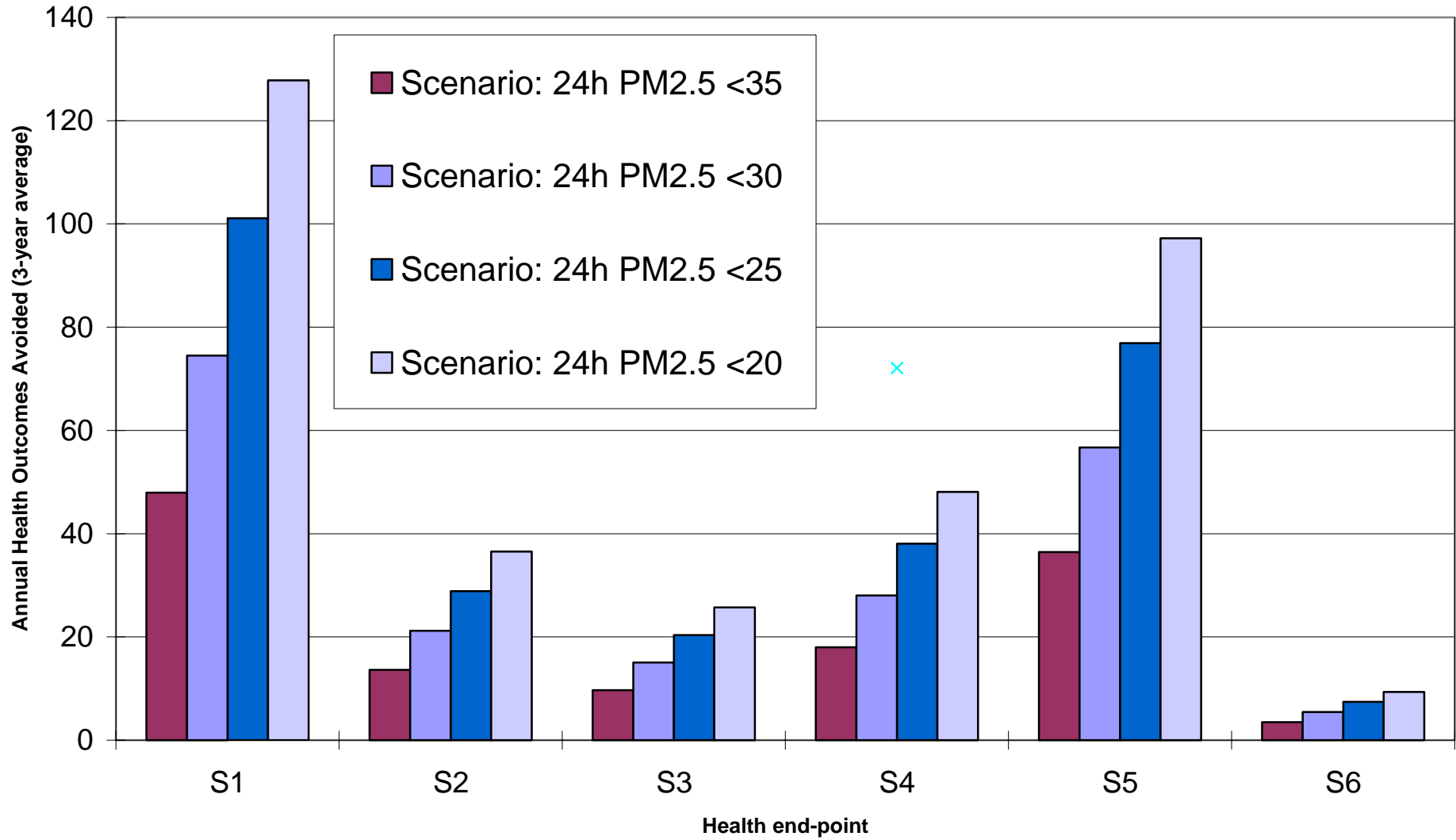
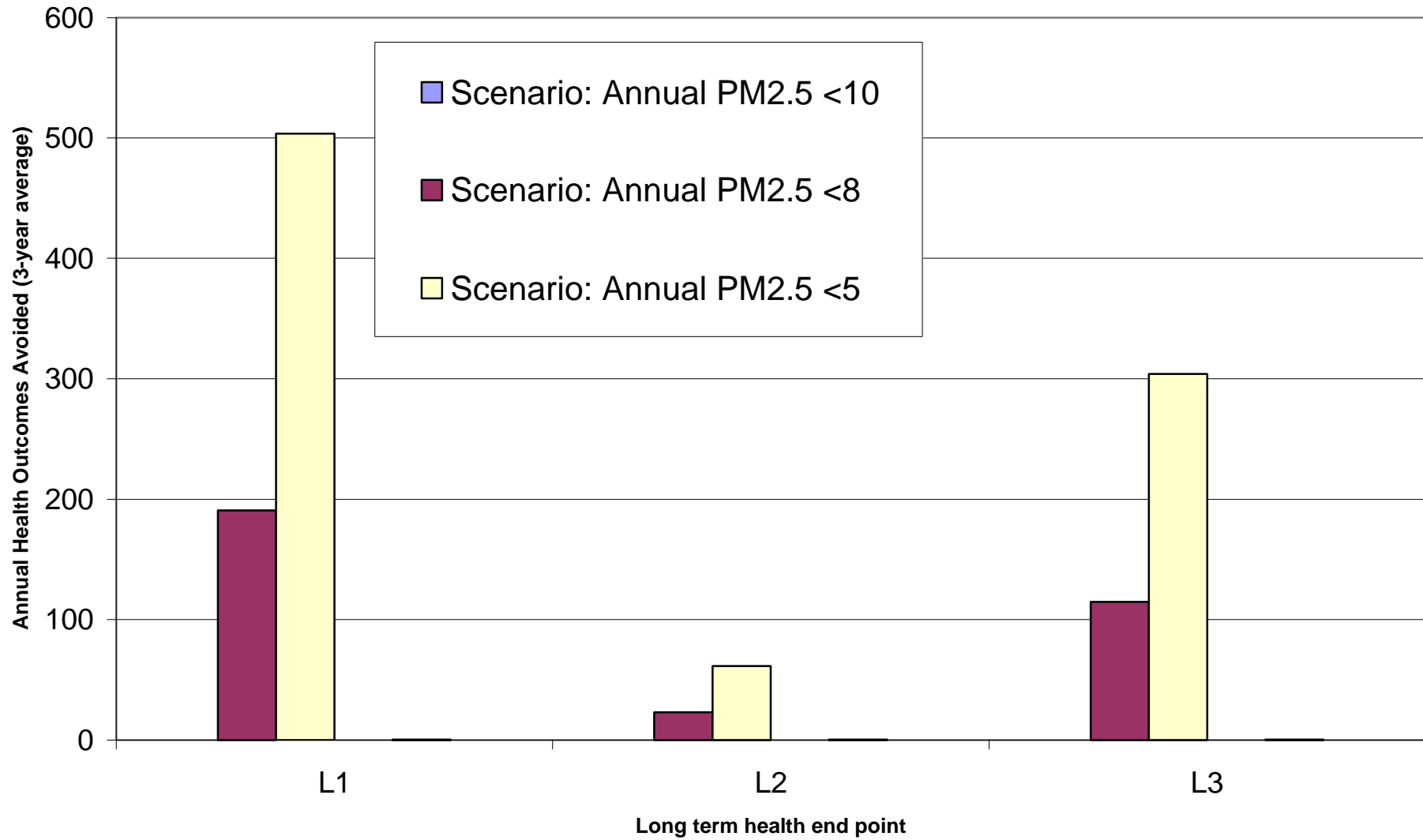


Figure S2.2 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - MELBOURNE



## 4.3 Brisbane

### 4.3.1 BRISBANE EXPOSURE ASSESSMENT

A map displaying the physical boundaries of the Brisbane Statistical Division as defined by the Australian Bureau of Statistics is shown in Figure E3a. The location of each relevant monitoring site as listed in Table 3.1 is also indicated. Population data for the Brisbane Statistical Division are given in Table E3a.

Table E3a. Population of the Brisbane Statistical Division, 2001.

<i>Age Group</i>	<i>Population</i>
0-14	337,963
65+	178,349
All ages	1,627,535

Frequency graphs of daily 24-hour  $PM_{2.5}$  data are presented in Figures E3b(1) and E3b(2) for 1999, 2000 and 2001. As for the other cities, the graphs display the composite  $PM_{2.5}$  concentrations, obtained by averaging the TEOM data obtained at Rocklea and Springwood monitoring stations.

Note that the Brisbane TEOM data as originally provided was inconsistent with all other Australian TEOM data, in that the standard USEPA correction function ( $y=1.03x+3.0$ ) had been removed from the TEOM instruments. To make the data comparable with all other Australian data, the USEPA function was applied. NB: The USEPA correction function was derived for  $PM_{10}$ , and there is no evidence that it applies to  $PM_{2.5}$ ; however most TEOM users do not modify the function when setting up instruments to measure  $PM_{2.5}$ , and so until a more detailed study can be done, it is best to retain the use of this function.

Bushfires (7-13 October 2001) gave rise to an extremely high 24-hour  $PM_{2.5}$  concentration at Rocklea on 9 October 2001.

The inverse cumulative distributions for 1999, 2000 and 2001 are displayed in Figures E3c(1) and E3c(2).

The 24-hour average  $PM_{2.5}$  results for 1999, 2000, 2001 and the three years combined are shown in Tables E3b-e respectively. Results with and without the bushfire data (7-13 October 2001) are provided.

Table E3b. 24-Hour  $PM_{2.5}$  results for Brisbane, 1999.

	Rocklea	Springwood	Composite
<i>Maximum 24-hour <math>PM_{2.5}</math> (<math>\mu g/m^3</math>)</i>	17.9	26.0	22.0
<i># Days &gt; 20 <math>\mu g/m^3</math></i>	0	1	1
<i># Days &gt; 25 <math>\mu g/m^3</math></i>	0	1	0
<i># Days &gt; 30 <math>\mu g/m^3</math></i>	0	0	0
<i># Days &gt; 35 <math>\mu g/m^3</math></i>	0	0	0

Table E3c. 24-Hour PM<sub>2.5</sub> results for Brisbane, 2000.

	Rocklea	Springwood	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	41.5	37.5	37.6
<i># Days &gt; 20 µg/m<sup>3</sup></i>	8	19	14
<i># Days &gt; 25 µg/m<sup>3</sup></i>	3	10	6
<i># Days &gt; 30 µg/m<sup>3</sup></i>	3	6	1
<i># Days &gt; 35 µg/m<sup>3</sup></i>	2	2	1

Table E3d(1). 24-Hour PM<sub>2.5</sub> results for Brisbane, 2001 (October 2001 [bushfires excluded](#))

	Rocklea	Springwood	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	23.9	23.0	22.5
<i># Days &gt; 20 µg/m<sup>3</sup></i>	4	6	3
<i># Days &gt; 25 µg/m<sup>3</sup></i>	0	0	0
<i># Days &gt; 30 µg/m<sup>3</sup></i>	0	0	0
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	0

Table E3d(2). 24-Hour PM<sub>2.5</sub> results for Brisbane, 2001 (October 2001 [bushfires included](#))

	Rocklea	Springwood	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	97.8	23.0	58.7
<i># Days &gt; 20 µg/m<sup>3</sup></i>	7	6	6
<i># Days &gt; 25 µg/m<sup>3</sup></i>	3	0	3
<i># Days &gt; 30 µg/m<sup>3</sup></i>	3	0	2
<i># Days &gt; 35 µg/m<sup>3</sup></i>	3	0	1

Table E3e(1). 24-Hour PM<sub>2.5</sub> results for Brisbane, 1999-2001 (October 2001 [bushfires excluded](#))

	Rocklea	Springwood	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	41.5	37.5	37.6
<i># Days &gt; 20 µg/m<sup>3</sup></i>	12	26	18
<i># Days &gt; 25 µg/m<sup>3</sup></i>	3	11	6
<i># Days &gt; 30 µg/m<sup>3</sup></i>	3	6	1
<i># Days &gt; 35 µg/m<sup>3</sup></i>	2	2	1

Table E3e(2). 24-Hour PM<sub>2.5</sub> results for Brisbane, 1999-2001 (October 2001 [bushfire included](#))

	Rocklea	Springwood	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	97.8	37.5	58.7
<i># Days &gt; 20 µg/m<sup>3</sup></i>	15	26	21
<i># Days &gt; 25 µg/m<sup>3</sup></i>	6	11	9
<i># Days &gt; 30 µg/m<sup>3</sup></i>	6	6	3
<i># Days &gt; 35 µg/m<sup>3</sup></i>	5	2	2

The annual average PM<sub>2.5</sub> concentrations derived from hourly data at each monitoring site and the composite annual average concentrations are shown in the following tables.

The supplied annual averages included all data (including the bushfire), this information (corrected for the USEPA function, see Section 3.1) is shown in Table E3f(2).

To estimate the annual averages excluding the bushfire, daily averages without the 7-13 October 2001 data were averaged. Then a correction factor was applied to account for the difference between 1-hour derived annual averages and 24-hour derived annual averages. The results are shown in Table E3f(1).

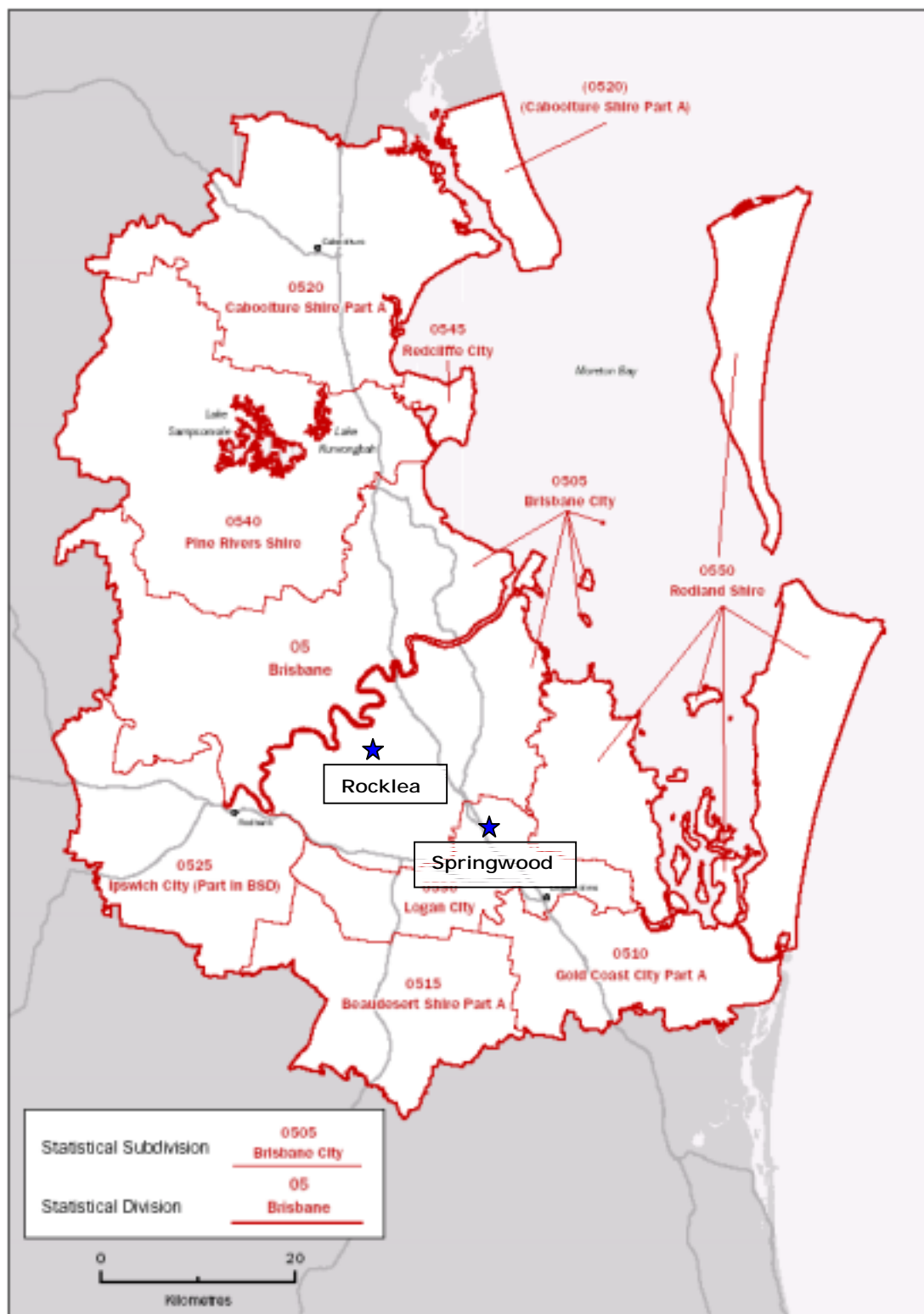
Table E3f(1). Estimated annual average PM<sub>2.5</sub> results for Brisbane (October 2001 bushfires excluded) ( $\mu\text{g}/\text{m}^3$ )

Year	Rocklea	Springwood	Composite
1999	8.2	7.4	7.8
2000	8.9	9.6	9.2
2001	8.2	8.4	8.3

Table E3f(2). Annual average PM<sub>2.5</sub> results for Brisbane (October 2001 bushfires included) ( $\mu\text{g}/\text{m}^3$ )

Year	Rocklea	Springwood	Composite
1999	8.2	7.4	7.8
2000	8.9	9.6	9.2
2001	8.7	8.5	8.6





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Figure E3a. Map of the Brisbane Statistical Division. The locations of the PM<sub>2.5</sub> monitoring stations are indicated with blue stars.

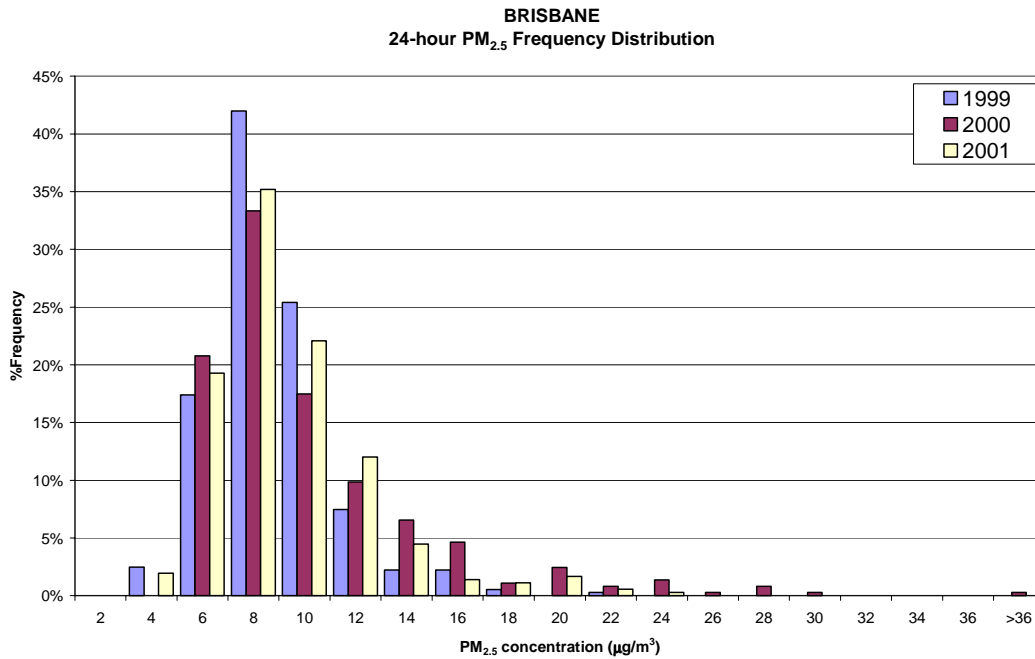


Figure E3b(1). Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Brisbane for 1999, 2000 and 2001. The concentrations for the frequency distribution on the X-axis denote concentration bins of X-2 to X µg/m<sup>3</sup>. Bushfire-affected data (7-13 October 2001) have been excluded.

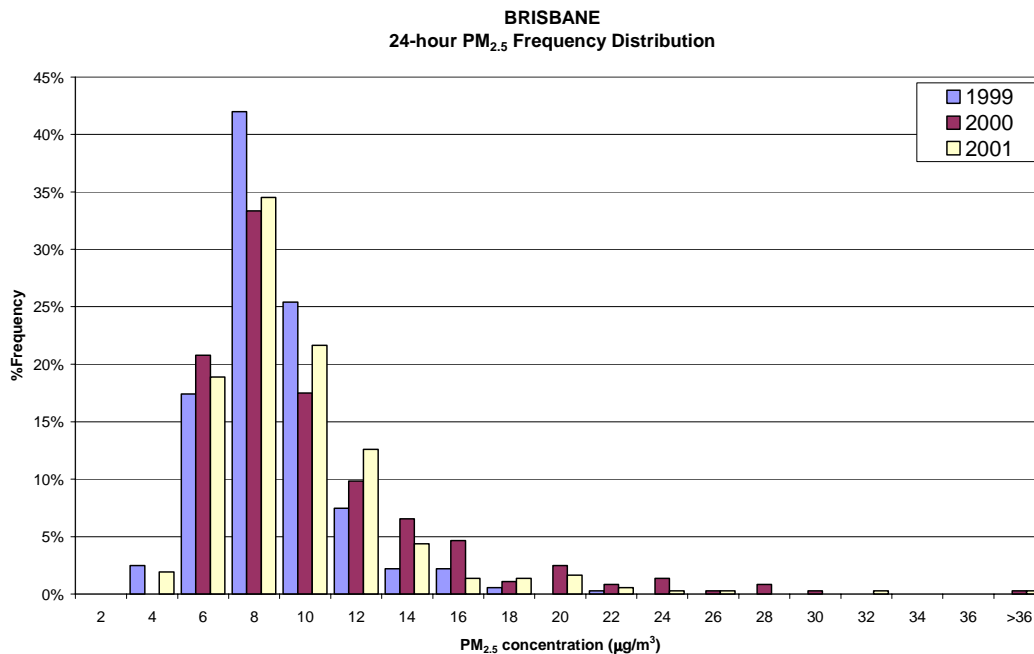


Figure E3b(2). Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Brisbane for 1999, 2000 and 2001. The concentrations for the frequency distribution on the X-axis denote concentration bins of X-2 to X µg/m<sup>3</sup>. Bushfire-affected data (7-13 October 2001) are included on this plot.

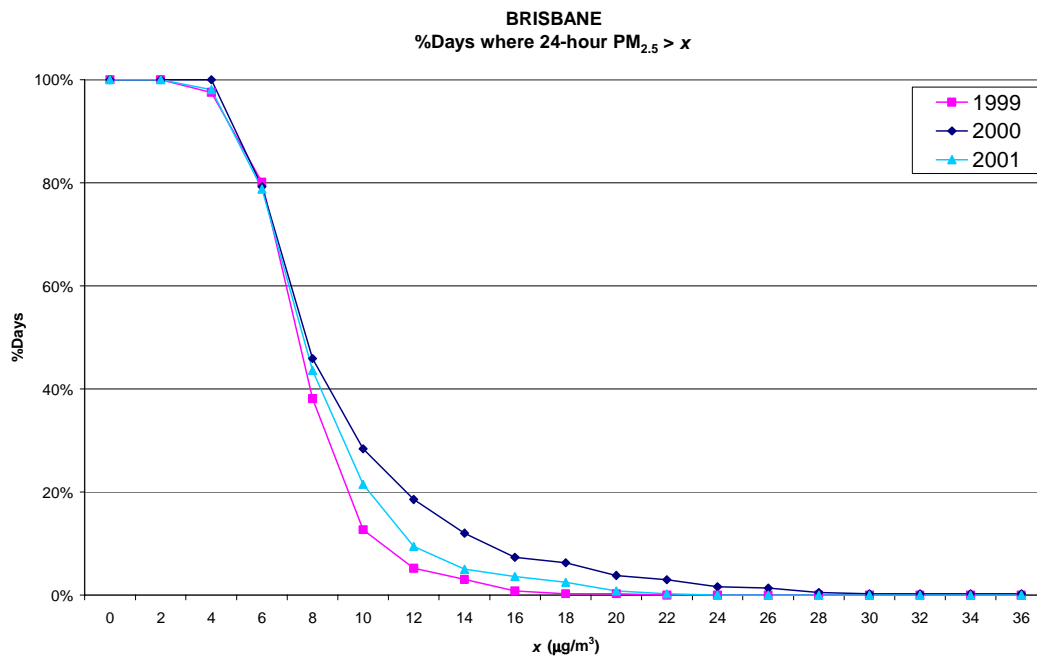


Figure E3c(1). Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour  $PM_{2.5}$  concentrations in Brisbane for 1999, 2000 and 2001. Bushfire-affected data (7-13 October 2001) have been excluded.

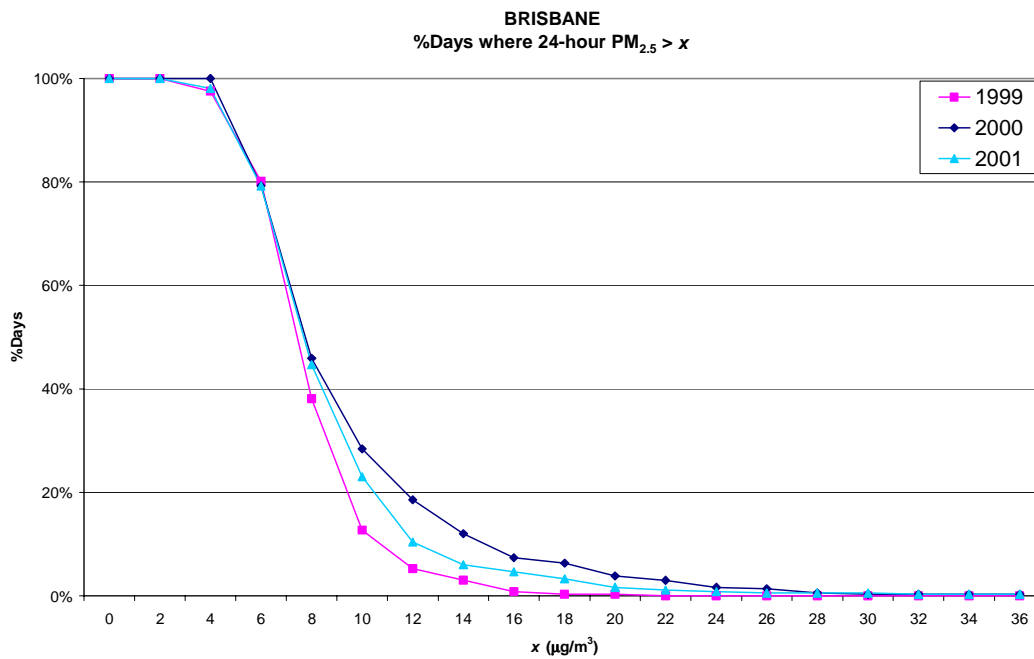


Figure E3c(2). Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour  $PM_{2.5}$  concentrations in Brisbane for 1999, 2000 and 2001. Bushfire-affected data (7-13 October 2001) are included.

### **4.3.2 Brisbane Risk Assessment**

Table R3.1 lists the estimated health outcomes attributable to above-background PM<sub>2.5</sub> in the Brisbane region, both as absolute values (number of cases per year) and as a proportion of the usual baseline incidence. The results are averages over the three-year study period.

The results in the first part of the table show the health outcomes with the effects of the 7-13 October 2001 bushfires excluded. The last two rows show the same results with the bushfires included.

### **4.3.3 Brisbane Scenario Assessment**

Table S3.1 lists the results of re-running the risk analysis for Brisbane with modified concentrations as described in the methodology section.

Results are presented as the total number of health outcomes avoided per year, averaged over the three-year study period. Figures S3.1 and S3.2 present the same information graphically for the 24-hour and annual average scenario levels respectively.

Data affected by the major bushfires of 7-13 Oct 2001 have been excluded from this analysis.

Table R3.1 - PM<sub>2.5</sub> Risk Assessment Results - Annual Health Outcomes attributable to above-background PM<sub>2.5</sub> - BRISBANE

	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
Estimated cases per year 95% conf. interval	97 ( 55 - 138 )	32 ( 20 - 45 )	20 ( 3 - 38 )	40 ( 16 - 64 )	69 ( 41 - 97 )	11 ( 2 - 21 )	247 ( 85 - 439 )	28 ( 9 - 44 )	156 ( 54 - 266 )
Estimated cases as % of baseline rate 95% conf. interval	0.8% ( 0.5% - 1.2% )	2.9% ( 1.8% - 4.1% )	0.4% ( 0.1% - 0.7% )	0.9% ( 0.4% - 1.5% )	0.6% ( 0.4% - 0.9% )	0.7% ( 0.1% - 1.3% )	2.1% ( 0.7% - 3.7% )	4.7% ( 1.4% - 7.2% )	3.1% ( 1.1% - 5.3% )
<b><i>With Bushfires:</i></b> Cases per year As % of baseline	99 0.8%	33 3.0%	21 0.4%	41 1.0%	71 0.6%	11 0.7%	252 2.1%	29 4.8%	160 3.2%

NB: confidence intervals shown above are based on statistical uncertainty in the dose-response relationships.

Table S3.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - BRISBANE

€	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
<b>Scenario</b>									
Scenario: 24h PM2.5 <35	19	6	4	8	13	2			
Scenario: 24h PM2.5 <30	32	11	7	13	23	4			
Scenario: 24h PM2.5 <25	45	15	10	19	32	5			
Scenario: 24h PM2.5 <20	58	19	12	24	42	7			
Scenario: Annual PM2.5 <10							0	0	0
Scenario: Annual PM2.5 <8							82	9	51
Scenario: Annual PM2.5 <5							237	28	150

Figure S3.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - BRISBANE

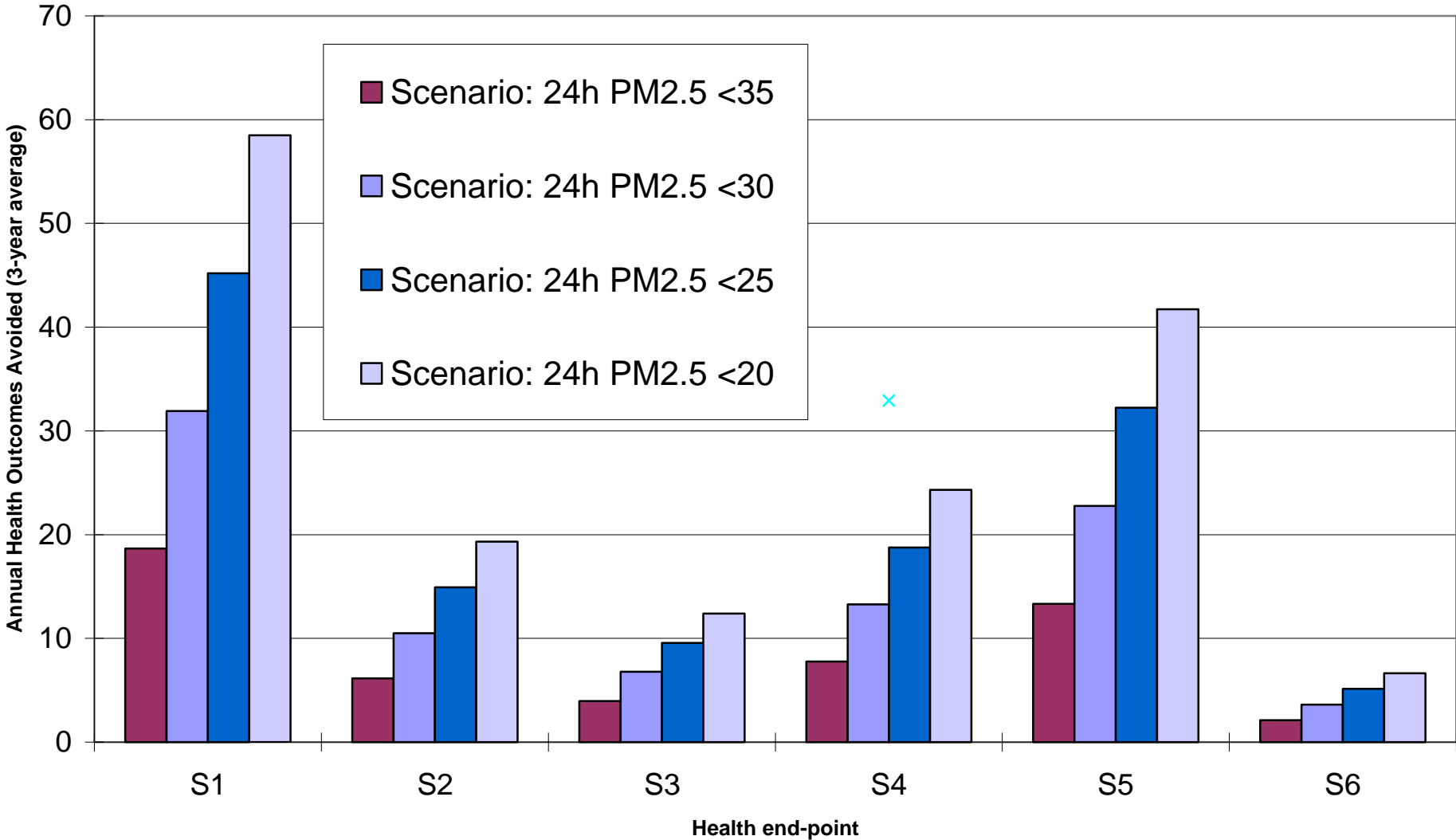
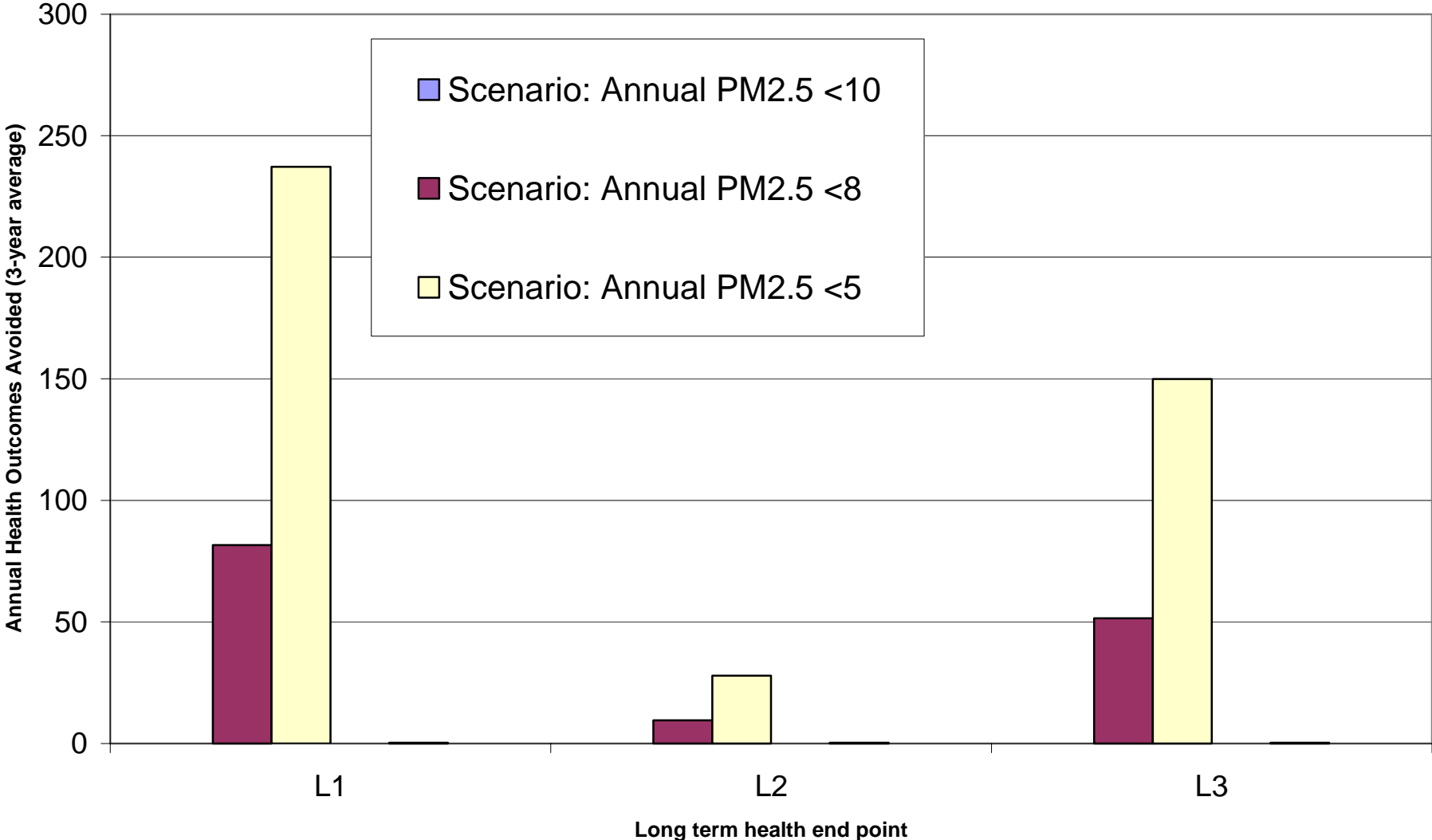


Figure S3.2 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - BRISBANE





## 4.4 Perth

### 4.4.1 PERTH EXPOSURE ASSESSMENT

A map displaying the physical boundaries of the Perth Statistical Division as defined by the Australian Bureau of Statistics is shown in Figure E4a. The location of each relevant monitoring site as listed in Table 3.1 is also indicated. Population data for the Perth Statistical Division are given in Table E4a.

Table E4a. Population of the Perth Statistical Division, 2001.

<i>Age Group</i>	<i>Population</i>
0-14	274,349
65+	150,861
All ages	1,339,993

Frequency graphs of daily 24-hour PM<sub>2.5</sub> data are presented in Figure E4b for 1999, 2000 and 2001 respectively. As for the other cities, the graphs display the composite PM<sub>2.5</sub> concentrations, obtained by averaging the data obtained at Caversham and Duncraig monitoring stations. The inverse cumulative frequency distributions are displayed in Figure E4c.

The 24-hour average PM<sub>2.5</sub> results for 1999, 2000, 2001 and the three years combined are shown in Tables E4b-e respectively. The annual average PM<sub>2.5</sub> concentrations derived from hourly data at each monitoring site and the composite annual average concentrations are shown in Table E4f.

Table E4b. 24-Hour PM<sub>2.5</sub> results for Perth, 1999.

	Caversham	Duncraig	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	20.3	26.3	19.8
<i># Days &gt; 20 µg/m<sup>3</sup></i>	1	5	0
<i># Days &gt; 25 µg/m<sup>3</sup></i>	0	2	0
<i># Days &gt; 30 µg/m<sup>3</sup></i>	0	0	0
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	0

Table E4c. 24-Hour PM<sub>2.5</sub> results for Perth, 2000.

	Caversham	Duncraig	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	20.1	22.2	19.4
<i># Days &gt; 20 µg/m<sup>3</sup></i>	1	1	0
<i># Days &gt; 25 µg/m<sup>3</sup></i>	0	0	0
<i># Days &gt; 30 µg/m<sup>3</sup></i>	0	0	0
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	0

Table E4d. 24-Hour PM<sub>2.5</sub> results for Perth, 2001

	Caversham	Duncraig	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	31.8	27.0	29.3
<i># Days &gt; 20 µg/m<sup>3</sup></i>	1	10	3
<i># Days &gt; 25 µg/m<sup>3</sup></i>	1	4	1
<i># Days &gt; 30 µg/m<sup>3</sup></i>	1	0	0
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	0

Table E4e. 24-Hour PM<sub>2.5</sub> results for Perth, 1999-2001

	Caversham	Duncraig	Composite
<i>Maximum 24-hour PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	31.8	27.0	29.3
<i># Days &gt; 20 µg/m<sup>3</sup></i>	3	16	3
<i># Days &gt; 25 µg/m<sup>3</sup></i>	1	6	1
<i># Days &gt; 30 µg/m<sup>3</sup></i>	1	0	0
<i># Days &gt; 35 µg/m<sup>3</sup></i>	0	0	0

Table E4f. Annual average PM<sub>2.5</sub> results for Perth (µg/m<sup>3</sup>)

Year	Caversham	Duncraig	Composite
1999	7.2	8.6	7.9
2000	7.4	8	7.7
2001	7.6	8.6	8.1

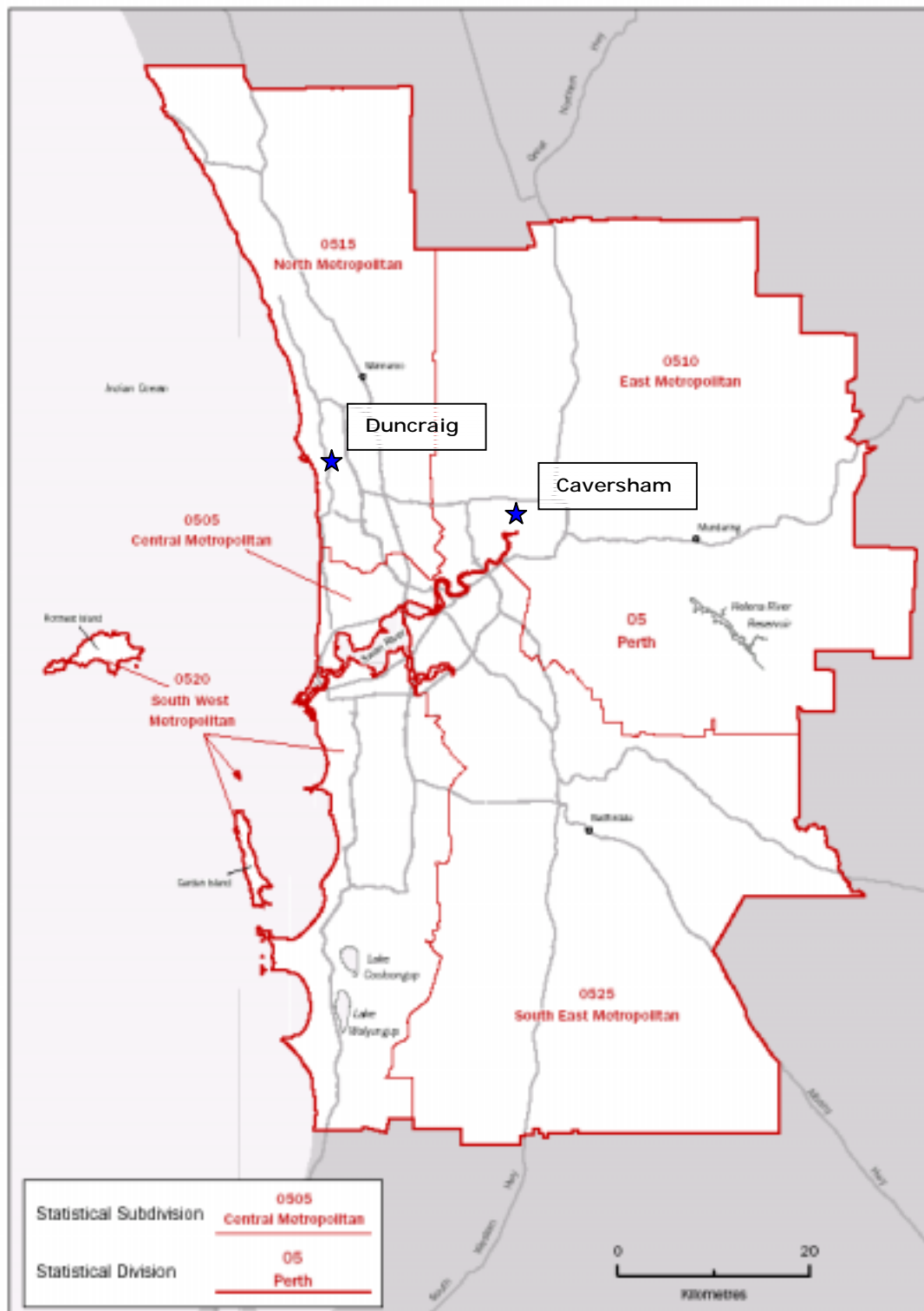


Figure E4a. Map of the Perth Statistical Division. The locations of the PM<sub>2.5</sub> monitoring stations are indicated with blue stars.

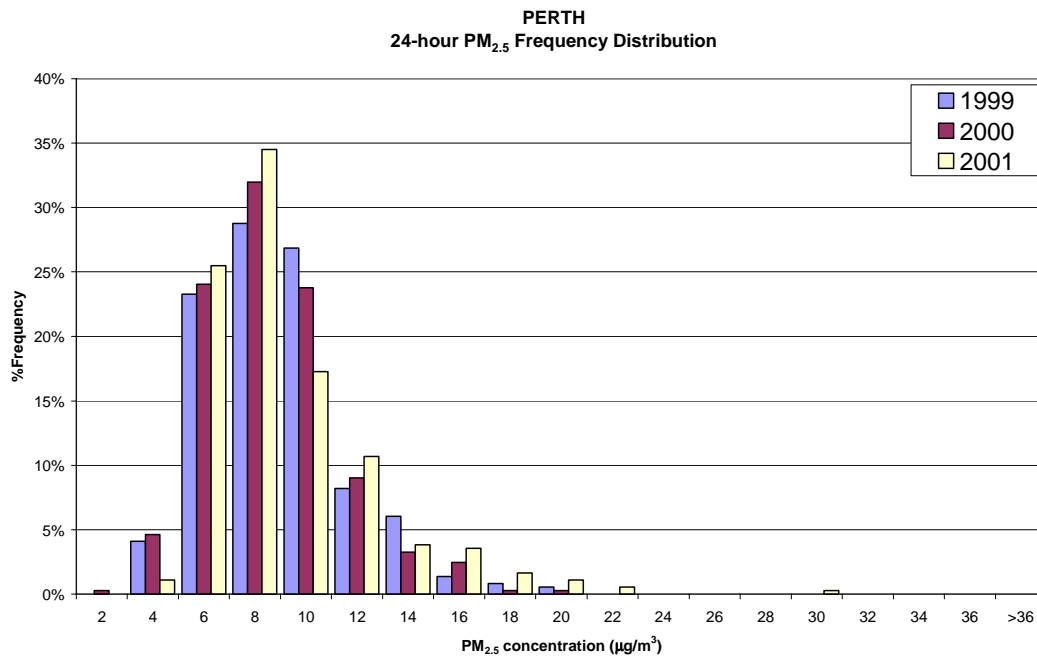


Figure E4b. Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Perth for 1999, 2000 and 2001. The concentrations for the frequency distribution on the X-axis denote concentration bins of  $X-2$  to  $X$  µg/m<sup>3</sup>.

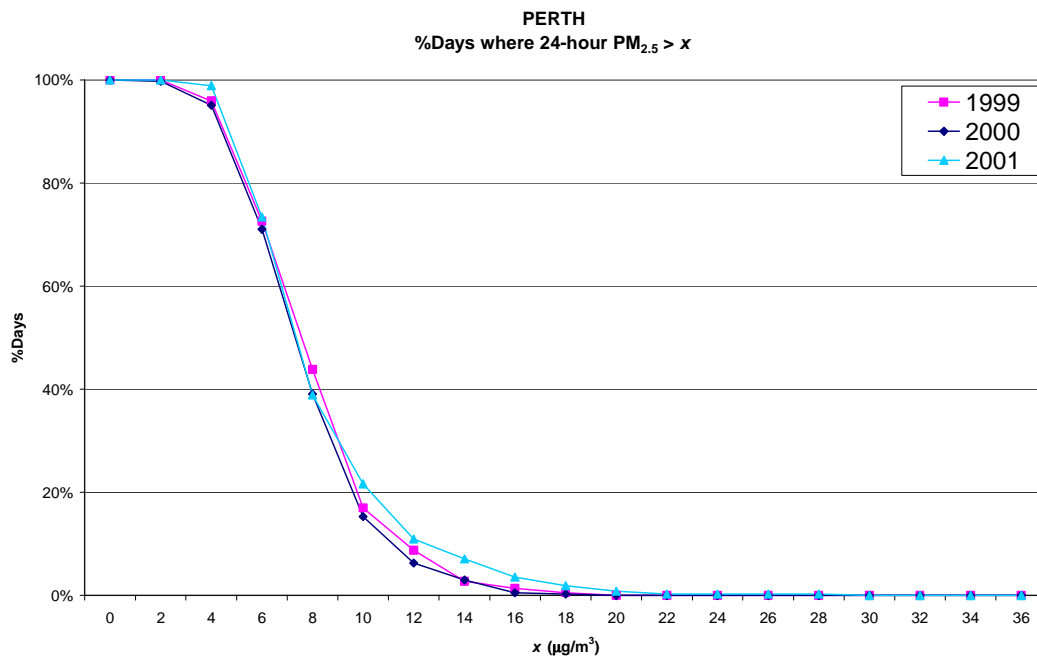


Figure E4c. Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour PM<sub>2.5</sub> concentrations in Perth for 1999, 2000 and 2001.

#### **4.4.2 PERTH RISK ASSESSMENT**

Table R4.1 lists the estimated health outcomes due to above-background PM<sub>2.5</sub> in the Perth region, both as absolute values (number of cases per year) and as a proportion of the usual baseline incidence. The results are averages over the three-year study period.

#### **4.4.3 PERTH SCENARIO ASSESSMENT**

Table S4.1 lists the results of re-running the risk analysis for Perth with modified concentrations as described in the methodology section.

The results are presented as the total number of health outcomes avoided per year, averaged over the three-year study period. Figures S4.1 and S4.2 present the same information graphically for the 24-hour and annual average scenario levels respectively.

Table R4.1 - PM<sub>2.5</sub> Risk Assessment Results - Annual Health Outcomes attributable to above-background PM<sub>2.5</sub> - PERTH

	Short Term Health Endpoint						Long Term Health Endpoint		
	<i>S1</i>	<i>S2</i>	<i>S3</i>	<i>S4</i>	<i>S5</i>	<i>S6</i>	<i>L1</i>	<i>L2</i>	<i>L3</i>
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
Estimated cases per year 95% conf. interval	55 ( 32 - 79 )	20 ( 12 - 27 )	10 ( 1 - 19 )	27 ( 10 - 42 )	50 ( 30 - 71 )	10 ( 2 - 19 )	142 ( 48 - 252 )	20 ( 6 - 32 )	97 ( 34 - 165 )
Estimated cases as % of baseline rate 95% conf. interval	0.7% ( 0.4% - 1.0% )	2.5% ( 1.5% - 3.5% )	0.3% ( 0.0% - 0.6% )	0.8% ( 0.3% - 1.3% )	0.5% ( 0.3% - 0.7% )	0.6% ( 0.1% - 1.1% )	1.8% ( 0.6% - 3.2% )	4.0% ( 1.2% - 6.2% )	2.6% ( 0.9% - 4.5% )

NB: confidence intervals shown above are based on statistical uncertainty in the dose-response relationships.

Table S4.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - PERTH

€	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
<b>Scenario</b>									
Scenario: 24h PM2.5 <35	0	0	0	0	0	0			
Scenario: 24h PM2.5 <30	4	1	1	2	4	1			
Scenario: 24h PM2.5 <25	14	5	3	7	13	3			
Scenario: 24h PM2.5 <20	25	9	5	12	23	4			
Scenario: Annual PM2.5 <10							0	0	0
Scenario: Annual PM2.5 <8							22	3	15
Scenario: Annual PM2.5 <5							135	19	92

Figure S4.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - PERTH

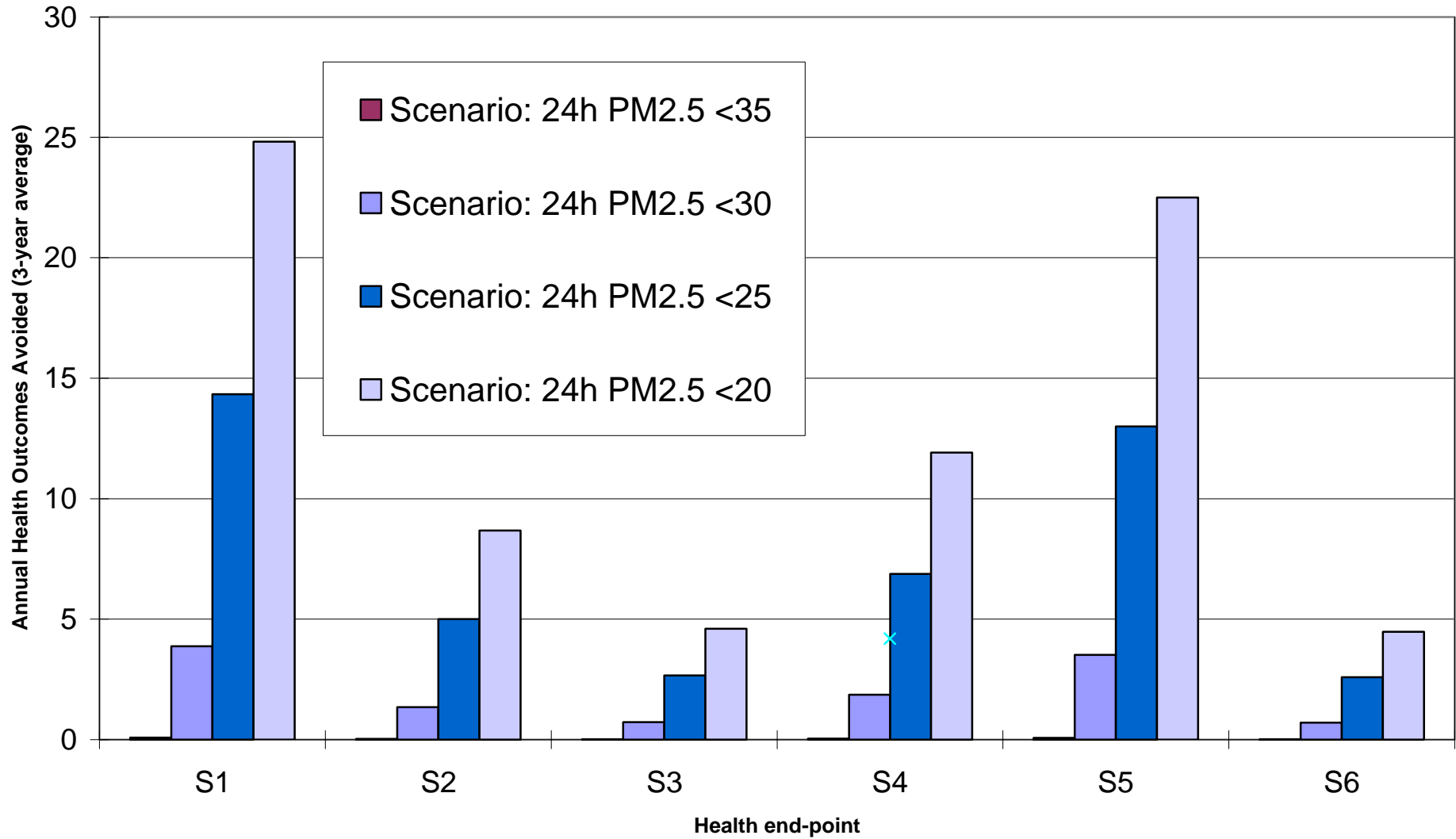
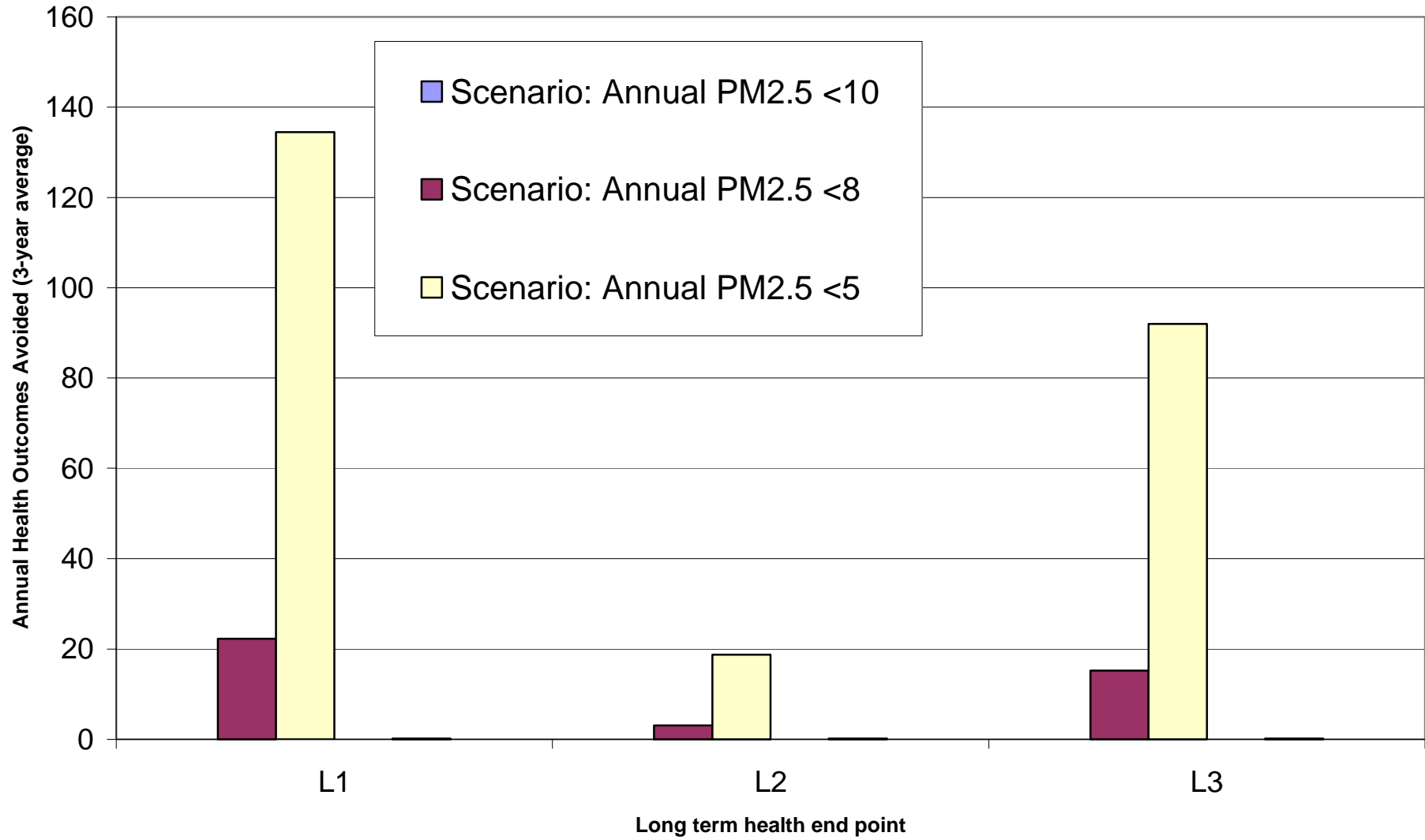




Figure S4.2 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year - PERTH



## 4.5 Summary

### 4.5.1 EXPOSURE SUMMARY

Frequency distributions for the combined 24-hour data for three consecutive years are plotted for all four cities in Figure E5a. The corresponding inverse cumulative frequency distributions are displayed in Figure E5b, linking  $PM_{2.5}$  concentration  $x$  with the percentage of days on which the concentration was higher than  $x$ .

Table E5a summarises the results from Tables E1e, E2e, E3e and E4e for the composite daily  $PM_{2.5}$  concentrations for all three years and all four cities. It can be seen from this table that the majority of concentrations are below the 24-hour scenario levels of 20, 25, 30 and 35  $\mu g/m^3$ .

Table E5a. Composite 24-Hour  $PM_{2.5}$  results for Sydney, Melbourne, Brisbane and Perth for each combined three-year period.

<i>Monitor</i>	<i>Maximum 24-hour <math>PM_{2.5}</math> (<math>\mu g/m^3</math>)</i>	<i>#Days &gt;20<math>\mu g/m^3</math></i>	<i># Days &gt;25 <math>\mu g/m^3</math></i>	<i># Days &gt;30 <math>\mu g/m^3</math></i>	<i># Days &gt;35 <math>\mu g/m^3</math></i>
Sydney composite 1999-2001	<b>37.6</b>	<b>10</b>	<b>4</b>	<b>2</b>	<b>1</b>
Sydney composite 1999-2001 (fires included)	93.7	17	11	9	7
Melbourne composite 1998-2000	<b>33.2</b>	<b>24</b>	<b>8</b>	<b>3</b>	<b>0</b>
Brisbane composite 1999-2001	<b>37.6</b>	<b>18</b>	<b>6</b>	<b>1</b>	<b>1</b>
Brisbane composite 1999-2001 (fires included)	58.7	21	9	3	2
Perth composite 1999-2001	<b>29.3</b>	<b>3</b>	<b>1</b>	<b>0</b>	<b>0</b>

Table E5b summarises the composite annual average PM<sub>2.5</sub> concentrations for all cities. Three annual scenario levels for PM<sub>2.5</sub> have been considered: 5, 8 and 10 µg/m<sup>3</sup>.

Table E5b. Composite annual average PM<sub>2.5</sub> results for Sydney, Melbourne, Brisbane and Perth. For Melbourne, Year 1 = 1998, Year 2 = 1999 and Year 3 = 2000. For the other cities Year 1 = 1999, Year 2 = 2000 and Year 3 = 2001.

<i>Monitor</i>	<i>Year 1 annual average PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	<i>Year 2 annual average PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>	<i>Year 3 annual average PM<sub>2.5</sub> (µg/m<sup>3</sup>)</i>
Sydney composite	<b>9.1</b>	<b>10.1</b>	<b>10.4</b>
Sydney composite (fires included)	9.1	10.1	11.4
Melbourne composite	<b>9.4</b>	<b>9.0</b>	<b>8.0</b>
Brisbane composite	<b>7.8</b>	<b>9.2</b>	<b>8.3</b>
Brisbane composite (fires included)	7.8	9.2	8.6
Perth composite	<b>7.9</b>	<b>7.7</b>	<b>8.1</b>

**ALL CITIES - 3 YEAR PERIOD 24-hour PM<sub>2.5</sub> Frequency Distribution**

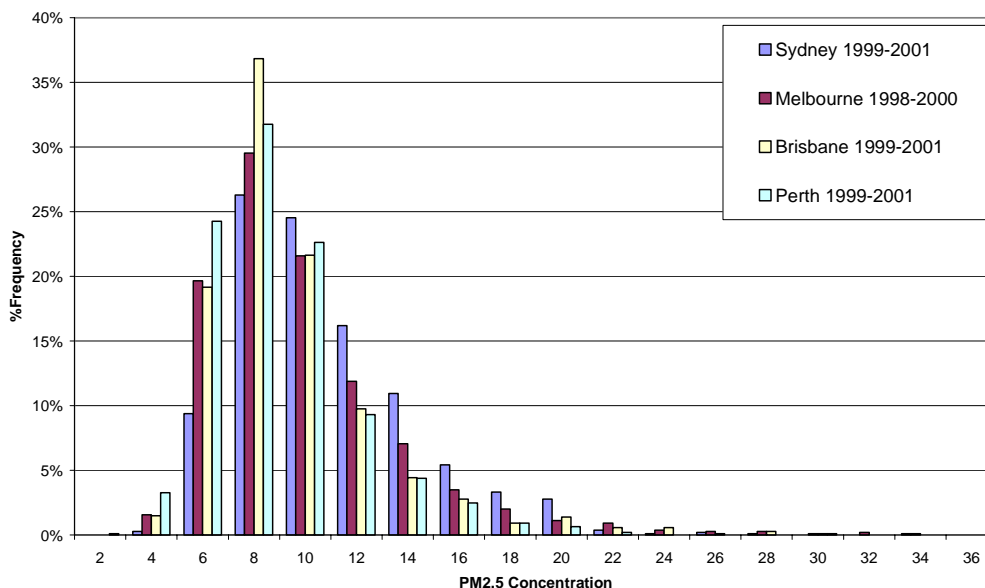


Figure E5a. Frequency distribution of composite daily 24-hour PM<sub>2.5</sub> concentrations in Sydney, Melbourne, Brisbane and Perth for each combined three year period (1998-2000 for Melbourne, 1999-2001 all other cities). The concentrations for the frequency distribution on the X-axis denote concentration bins of X-2 to X µg/m<sup>3</sup>. Bushfire-affected data (Sydney, 25-31 December 2001; Brisbane, 7-13 October 2001) have been excluded.

**ALL CITIES - 3 YEAR PERIOD % Days where 24-hour PM<sub>2.5</sub> > x**

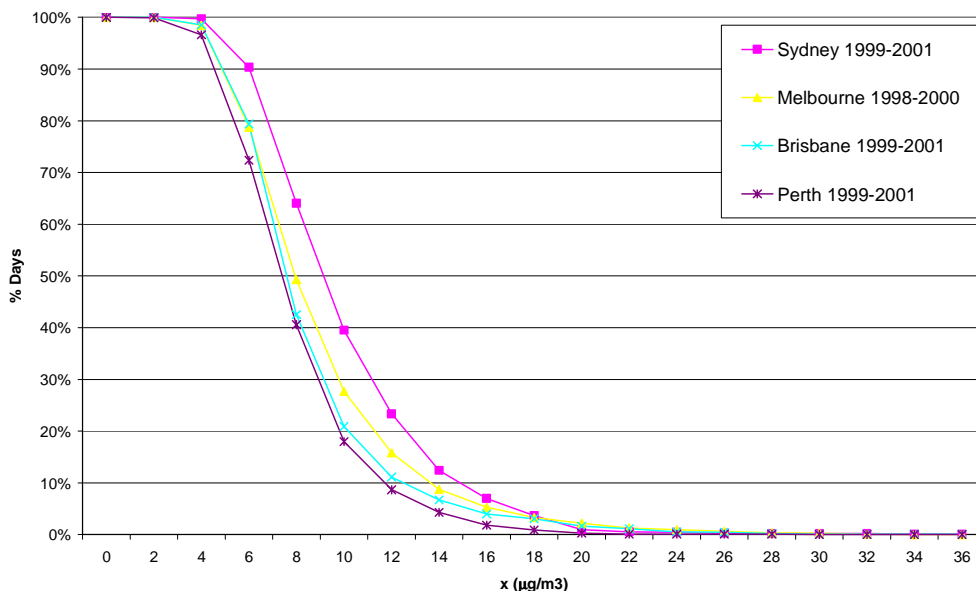


Figure E5b. Inverse cumulative frequency distribution (percentage of days above each concentration level) of composite daily 24-hour PM<sub>2.5</sub> concentrations in Sydney, Melbourne, Brisbane and Perth for each combined three year period (1998-2000 for Melbourne, 1999-2001 all other cities). Bushfire-affected data (Sydney, 25-31 December 2001; Brisbane, 7-13 October 2001) have been excluded.

#### **4.5.2 RISK ASSESSMENT SUMMARY**

Table R5.1 lists the annual health outcomes from Tables R1.1, R2.1, R3.1 and R4.1, together with a total for the four cities. Results are averages over the three-year study period.

It is important to note that these data are estimates only. As is detailed in the section on uncertainties, numerous assumptions and approximations are required to arrive at these results.

The first set of results in the table exclude the effects of the major bushfires in Brisbane and Sydney in 2001, the last two rows give the results with the bushfire-affected data included.

#### **4.5.3 SCENARIO ASSESSMENT SUMMARY**

Table S5.1 lists the estimates of the health outcomes avoided for each scenario from Tables S1.1, S2.1, S3.1 and S4.1, together with totals for the four cities. Results are averages over the three-year study period.

Again it is important to note that these data are only estimates. The main sources of uncertainty surrounding these estimates are discussed in Section 5.



Table R5.1 - PM<sub>2.5</sub> Risk Assessment Results - Annual Health Outcomes attributable to above-background PM<sub>2.5</sub>  
- ALL CITIES

	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
Sydney	274	81	55	157	246	58	699	88	527
Melbourne	207	60	41	78	157	15	524	58	316
Brisbane	97	32	20	40	69	11	247	28	156
Perth	55	20	10	27	50	10	142	20	97
<b>TOTAL</b>	<b>632</b>	<b>193</b>	<b>127</b>	<b>302</b>	<b>523</b>	<b>94</b>	<b>1611</b>	<b>195</b>	<b>1096</b>
<i>Including 2001 Major Bushfires:</i>									
Sydney	290	85	58	167	262	61	743	93	560
Brisbane	99	33	21	41	71	11	252	29	160

Table S5.1 - PM<sub>2.5</sub> Scenario Assessment - Health Outcomes Avoided per year – ALL CITIES

	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
€ <b>Scenario</b>									
Scenario: 24h PM2.5 <35	137	40	28	66	113	20			
Scenario: 24h PM2.5 <30	214	64	43	103	177	32			
Scenario: 24h PM2.5 <25	299	89	60	143	247	44			
Scenario: 24h PM2.5 <20	383	115	77	183	317	57			
Scenario: Annual PM2.5 <10							100	12	75
Scenario: Annual PM2.5 <8							624	74	428
Scenario: Annual PM2.5 <5							1552	188	1056



## 5. SOURCES OF UNCERTAINTY

In order to perform the risk analyses described in this report it was necessary to make a number of assumptions. Some of these assumptions relate to the general methodology, whereas others are more specific to this work.

The USEPA methodology (USEPA, 2002) for particulate matter risk analysis was followed in this study as a requirement of the consultancy. There are several underlying assumptions inherent in the application of this methodology, not least being the assumption that it is possible to estimate human exposure to PM<sub>2.5</sub> using data from outdoor ambient monitors. It is also assumed that the exposure experienced by all people living within a city can be estimated from PM<sub>2.5</sub> data averaged across the monitoring network of that city, following the approach used in epidemiological studies.

Many of the assumptions made in the risk analyses give rise to uncertainties in the health risk estimates. Furthermore, the risk estimates are only as good as the inputs to the analyses – that is, the dose-response functions, the air quality data, the baseline incidence rates, and the population sizes. The key uncertainties in the risk analyses are discussed in more detail below.

### 5.1 Dose-Response Functions

The dose-response functions for the selected health endpoints are a key component of the risk analyses. The statistical uncertainty associated with the dose-response relationships has been used to calculate a confidence interval around the risk estimates for each health endpoint. The confidence intervals are presented alongside the health risk estimates in Section 4. Figure U2.1 displays the confidence intervals for the health risks at current levels of exposure in Melbourne. The plots for the other three cities are very similar and are not shown here.

Note that the confidence interval surrounding a health risk estimate expresses the range within which the true risk is likely to fall, *if the statistical uncertainty surrounding the pollutant coefficient estimate ( $\beta$ ) were the only uncertainty in the analysis*. However, there are other important sources of uncertainty associated with the dose-response functions which are much more difficult to quantify. The quality of the risk assessment depends, in part, on:

- the accuracy of the dose-response functions, e.g. whether they are unbiased estimates of the relationship between the health response and ambient PM<sub>2.5</sub> concentration in the epidemiological study locations;
- the applicability of the dose-response functions to Australian cities, given that they were estimated elsewhere;

- the applicability of the dose-response functions to TEOM PM<sub>2.5</sub> data, given that they may have been estimated using PM<sub>2.5</sub> data obtained using other instrumentation;
- the extent to which the dose-response relationships apply beyond the range of PM<sub>2.5</sub> concentrations from which they were estimated.

Each of these issues is discussed in more detail below.

### **5.1.1 ACCURACY OF THE DOSE-RESPONSE FUNCTIONS**

Since the dose-response functions have been empirically estimated in epidemiological studies, there is uncertainty surrounding these estimates. Any confounding variables which may have been omitted from an epidemiological study may cause the dose-response relationships to be overestimated. Such confounding variables can include the effect of other pollutants; exposure to other health risks, such as smoking and occupational exposure; demographic characteristics, including age, sex, socioeconomic status, and access to medical care; and the population health status independent of PM<sub>2.5</sub> levels.

Another source of uncertainty is the assumed mathematical form of the dose-response relationships. In the epidemiological studies, and in the present study, it is assumed that the mathematical form of the dose-response function is log-linear. However, the statistical significance of coefficients in the estimated dose-response functions does not necessarily mean that the log-linear form of the function is the best model of the true dose-response relationship. In order to test the sensitivity of the health estimates to the assumed mathematical form of the dose-response relationship, the health effect calculations have been repeated for a subset of the data using a linear dose-response relationship (see Section 2.2.4). The health effects at current levels of exposure for Melbourne were recalculated for the three endpoints S1, S5 and L1. The results are shown in Figure U2.2. The largest difference in the health outcomes using a linear form of the dose-response relationship (relative to the exponential form) was 4% for L1.

### **5.1.2 APPLICABILITY OF DOSE-RESPONSE FUNCTIONS TO AUSTRALIAN CITIES**

All dose-response functions used in the risk analyses have been estimated in locations outside Australia. The accuracy of the results therefore depends on the assumption that the relation between ambient PM<sub>2.5</sub> and a given population health response is the same in Sydney, Melbourne, Brisbane and Perth as in the epidemiological study locations.

It is most likely that the dose-response relationship for a given endpoint is not the same everywhere, for a variety of reasons. The individual's response to PM<sub>2.5</sub> exposure may be different in different locations, due to different patterns of behaviour such as time spent outdoors. The

composition of PM<sub>2.5</sub>, and the characteristics of the population exposed to air pollution may also vary from location to location. For example, populations in the epidemiological study locations may have more or fewer people susceptible to a condition such as asthma than the populations of Sydney, Melbourne, Brisbane or Perth. There is also likely to be variation in the dose-response relation from one Australian city to another. Confounding pollutant concentrations may also be different in Australia compared with the epidemiological study locations.

### **5.1.3 APPLICABILITY OF DOSE-RESPONSE FUNCTIONS TO TEOM PM<sub>2.5</sub> DATA**

All Australian PM<sub>2.5</sub> data used for the health risk analyses were TEOM based. There is some uncertainty surrounding the risk estimates resulting from any possible bias in the TEOM PM<sub>2.5</sub> data used in this study which may not have been present in the PM<sub>2.5</sub> data used in the epidemiological studies. It is known that TEOM measurements may underestimate PM<sub>2.5</sub> concentrations compared with manual sampling methods, due to the potential loss of semi-volatile components from heating the inlet air stream.

### **5.1.4 EXTRAPOLATION BEYOND OBSERVED PM<sub>2.5</sub> LEVELS**

Although a dose-response function describes the theoretical relationship between ambient PM<sub>2.5</sub> concentrations and a given health endpoint for all possible PM<sub>2.5</sub> levels, the estimation of a dose-response function is based on real ambient PM<sub>2.5</sub> values that are limited to the range of PM<sub>2.5</sub> concentrations in the location in which the study was conducted. The actual shape of the dose-response function is not known outside the observed air quality range.

The risk analyses assume that the estimated dose-response functions adequately represent the true dose-response relation down to the background level and below. It is possible that for a given health endpoint the background level used in this study is below the lowest observed PM<sub>2.5</sub> level in the corresponding epidemiological study. The risk estimates for the lowest concentrations considered are therefore more uncertain than the estimates for concentrations in the middle of the range of the epidemiological study data.

The dose-response relationships may also be less certain towards the upper end of the concentration range considered in the risk analyses. In particular, the high PM<sub>2.5</sub> concentrations observed during major bushfires in Sydney and Brisbane may lie outside the range of the PM<sub>2.5</sub> concentrations observed in the epidemiological studies. The health risk estimates based on the PM<sub>2.5</sub> data which included the data influenced by major bushfires may therefore be subject to greater uncertainties than the estimates based on the PM<sub>2.5</sub> data in which the data influenced by the major fires were excluded.

There is no known minimum PM<sub>2.5</sub> concentration (i.e. threshold) below which PM<sub>2.5</sub> is not associated with health effects. If there is such a threshold, the estimated health risks may be overestimated.

## **5.2 Air Quality Data**

### **5.2.1 SPATIAL AVERAGING PROCEDURE**

The method of averaging the PM<sub>2.5</sub> concentration data across a city's monitoring network in the exposure assessment is similar to the methods used to characterise ambient air quality in most epidemiological studies. It is possible that this method results in biased measures of population exposure, either in the assessment location or in the epidemiological study location. This bias may be different in Australian cities compared with the epidemiological study locations.

### **5.2.2 CHOICE OF BACKGROUND LEVELS**

The health outcomes calculated in the risk assessment are those associated with PM<sub>2.5</sub> levels above background.

The background PM<sub>2.5</sub> concentration is defined as the 'natural' background, excluding all anthropogenic contributions. Note that the concentration and composition of background PM<sub>2.5</sub> can vary with geographic location, from monitoring site to monitoring site; with season of the year; and with meteorological conditions which affect the emissions and secondary production of biogenic or geogenic species to the background (USEPA, 1996b).

In this study a background concentration has been estimated by taking the mean of the 5<sup>th</sup> percentile 24-hour PM<sub>2.5</sub> concentration in each city. The same background value was then used in the risk assessment for each city. There are a number of uncertainties associated with this choice of background level:

- the estimated background level may include contributions from anthropogenic sources of PM<sub>2.5</sub>, in addition to natural sources;
- the background PM<sub>2.5</sub> concentration may not be the same in Sydney, Melbourne, Brisbane and Perth;
- there may be seasonal or daily variation in background PM<sub>2.5</sub>.

In order to investigate the effect of some of these uncertainties on the results of the risk assessment, two alternative background concentrations were also considered.

One alternative background level was obtained by considering only the contribution to PM<sub>2.5</sub> from sea-spray, which is usually measured as sea-salt. Based on measurements of the sea-salt component of PM<sub>2.5</sub> over a

7-year period in Mascot, Sydney (Cohen 1999), the background level due to sea-salt was estimated at  $0.96 \mu\text{g}/\text{m}^3$ . An Australia-wide study (Ayers *et. al.* 1998) showed that the sea-salt component of  $\text{PM}_{2.5}$  (as measured by Na, Cl and Br) does vary from city to city, but in general is of the order of  $0.4 - 1.0 \mu\text{g}/\text{m}^3$ .

A second alternative background level was obtained by including all soil-derived elements as well as sea-salt. Using this method, an alternative  $\text{PM}_{2.5}$  background level is  $1.61 \mu\text{g}/\text{m}^3$  (Cohen 1999).

Note that other natural sources of fine particles exist (for example, secondary particles derived from biogenic sources), however there are insufficient data to quantify these levels.

The health effects at current levels of exposure were recalculated for Melbourne using the two alternative background values of  $1.61$  and  $0.96 \mu\text{g}/\text{m}^3$ . The results are shown in Table U2.2 and Figure U2.3.

### **5.2.3 PROCEDURE USED TO SIMULATE $\text{PM}_{2.5}$ CONCENTRATIONS THAT ARE AT OR BELOW A PROPOSED SCENARIO LEVEL**

The pattern and extent of daily reductions in  $\text{PM}_{2.5}$  that would result if all daily  $\text{PM}_{2.5}$  concentrations were to be at or below the 24-hour scenario levels are unknown. A linear rollback procedure has been used in which it is assumed that daily above-background  $\text{PM}_{2.5}$  concentrations are reduced by the same percentage on all days over a three-year period. An examination of historical  $\text{PM}_{2.5}$  data in Philadelphia and Los Angeles in reports prepared for the U.S. Environmental Protection Agency (USEPA, 1996a; USEPA, 2002) indicated that such a procedure may be reasonable. However, there remains uncertainty about the shape of the air quality distribution of daily levels which are at or below a 24-hour  $\text{PM}_{2.5}$  scenario level in each city, which will depend on each city's future air quality control strategies.

## ***5.3 Baseline Health Incidence Data***

The uncertainties in the baseline health incidence data are not likely to be large because baseline incidence data were available for all endpoints in each city. It was therefore not necessary to use incidence data from a different location for any of the endpoints in each city. However, some uncertainties did arise in the health risk estimates due to some assumptions made in using the baseline incidence data. These sources of uncertainty are described below.

### 5.3.1 ESTIMATES OF PER-CAPITA INCIDENCE RATES

For most cities, with the exception of the baseline incidence rates for lung cancer and cardiopulmonary mortality (L2 and L3) which were provided as an incidence rate per 100,000 people, all other baseline incidence rates provided by the NEPC Project Team were expressed as a value per population group. For Sydney most data were provided per 100,000 people. In order to calculate per-capita incidence rates some assumptions were made regarding the size of the population group relevant to each endpoint in each city. There is therefore some uncertainty associated with the per-capita baseline incidence rates presented in Table 3.2. However, the per-capita rates are consistent between the four Australian cities, indicating that this uncertainty is small.

### 5.3.2 LACK OF DAILY HEALTH EFFECTS INCIDENCE RATES

Both ambient  $PM_{2.5}$  concentrations and the number of cases of each health endpoint vary somewhat from day to day, and also from season to season. In estimating the short-term health effects for endpoints S1 to S6, Equation 1 in Section 2.2.2 is evaluated on a daily basis, with the results summed over the days of the year to provide annual estimates. However, since the actual daily baseline incidence rates are not known, Equation 1 is evaluated using an average daily baseline incidence rate. This means that on days when the  $PM_{2.5}$  concentration is higher than average, the actual baseline incidence is underestimated. Similarly, on days when the  $PM_{2.5}$  concentration is below average, the actual baseline incidence rate is overestimated. Both effects may cancel out to some extent however.

### 5.3.3 DISEASE CODES USED IN BASELINE INCIDENCE RATES

A further source of uncertainty in the health risk estimates may be any differences in the diseases included in a health endpoint in an epidemiological study and the diseases included in the baseline health incidence data for that endpoint.

For example, if for a given health endpoint the baseline health incidence data apply to a larger number of diseases than those included in the epidemiological study, the health effects for that endpoint may be overestimated.

## *5.4 Size of the Population Exposed to $PM_{2.5}$*

### 5.4.1 DATE OF POPULATION DATA

The population of each city has been estimated using the Australian Bureau of Statistics 2001 Census data. However, the  $PM_{2.5}$  concentration

data were collected from 1999 to 2001 for Sydney, Brisbane and Perth and from 1998 to 2000 for Melbourne. There is therefore some small uncertainty in the estimates of population exposure.

There may also be some uncertainty associated with any mismatches between the periods during which the baseline health incidence data were collected and the date of the study population (2001).

#### **5.4.2 PHYSICAL BOUNDARY AROUND EXPOSED POPULATION**

The physical boundary for each urban area has been taken to be that of the city's Statistical Division as defined by the Australian Bureau of Statistics. The resulting coverage by PM<sub>2.5</sub> monitors is sparse. The assumption is that the PM<sub>2.5</sub> monitoring data used in the exposure assessment are representative of the PM<sub>2.5</sub> levels within each airshed. The airshed regions are defined in the jurisdictional monitoring plans for the National Environment Protection Measure (NEPM) for Ambient Air Quality. In general, the Statistical Division for each city is a similar size or is smaller than the relevant Air NEPM region. For Sydney, the relevant Air NEPM region is the Sydney Region, which is similar in size to the Sydney Statistical Division. Similarly, the Perth Statistical Division incorporates most of the Perth Region. The Melbourne Statistical Division encompasses metropolitan Melbourne, which is part of the Port Phillip Region defined for the Air NEPM. For Brisbane, the Brisbane Statistical Division is a similar size to the Brisbane Sub-region of the South-East Queensland Region.

**Figure U2.1 Annual Health Outcomes Attributable to Above-Background PM<sub>2.5</sub> - MELBOURNE**  
**Statistical Uncertainty due to Dose Response 95% Confidence Interval**

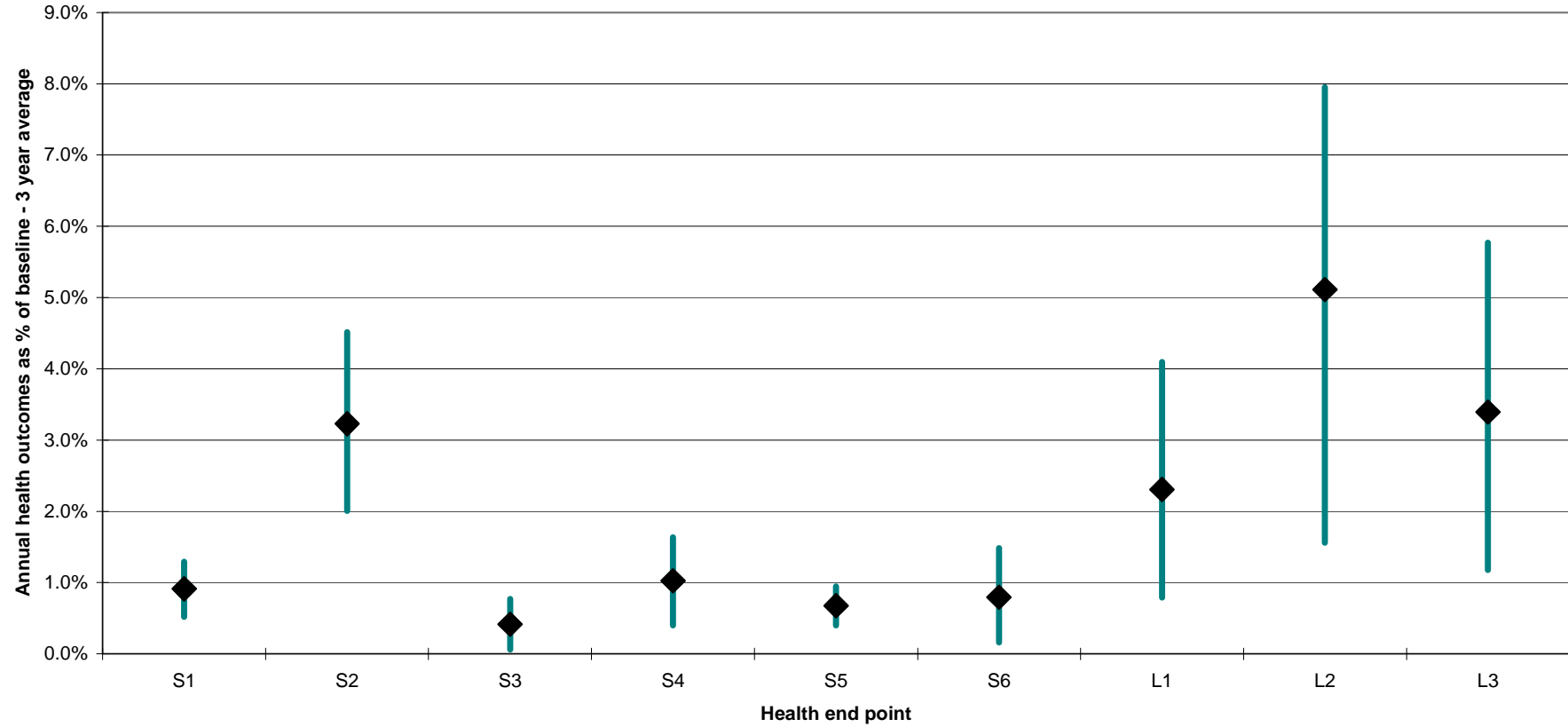




Figure U2.2 - Annual Health Outcomes attributable to above-background PM<sub>2.5</sub> - MELBOURNE  
Effect of Dose-Response Formulation

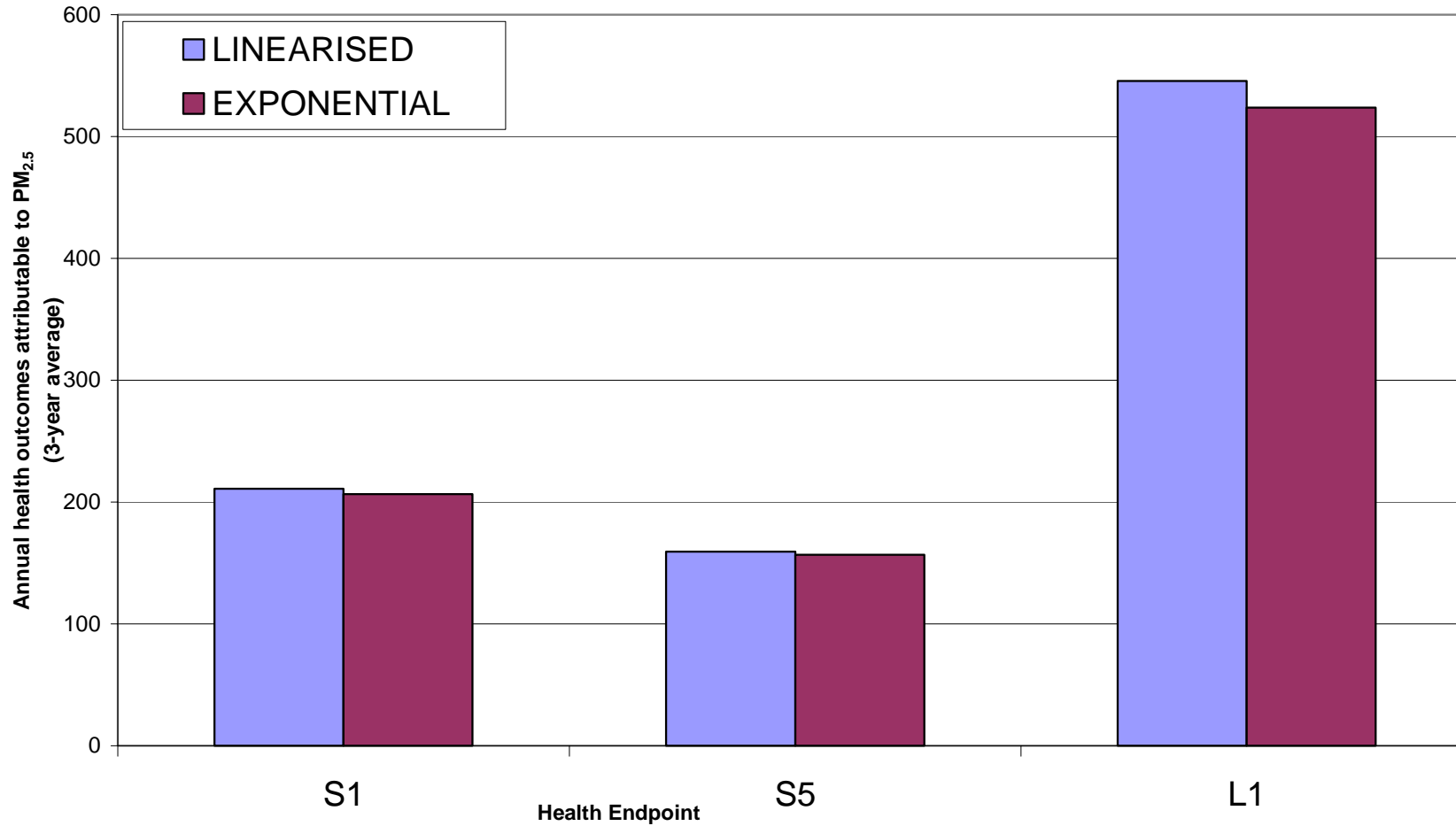
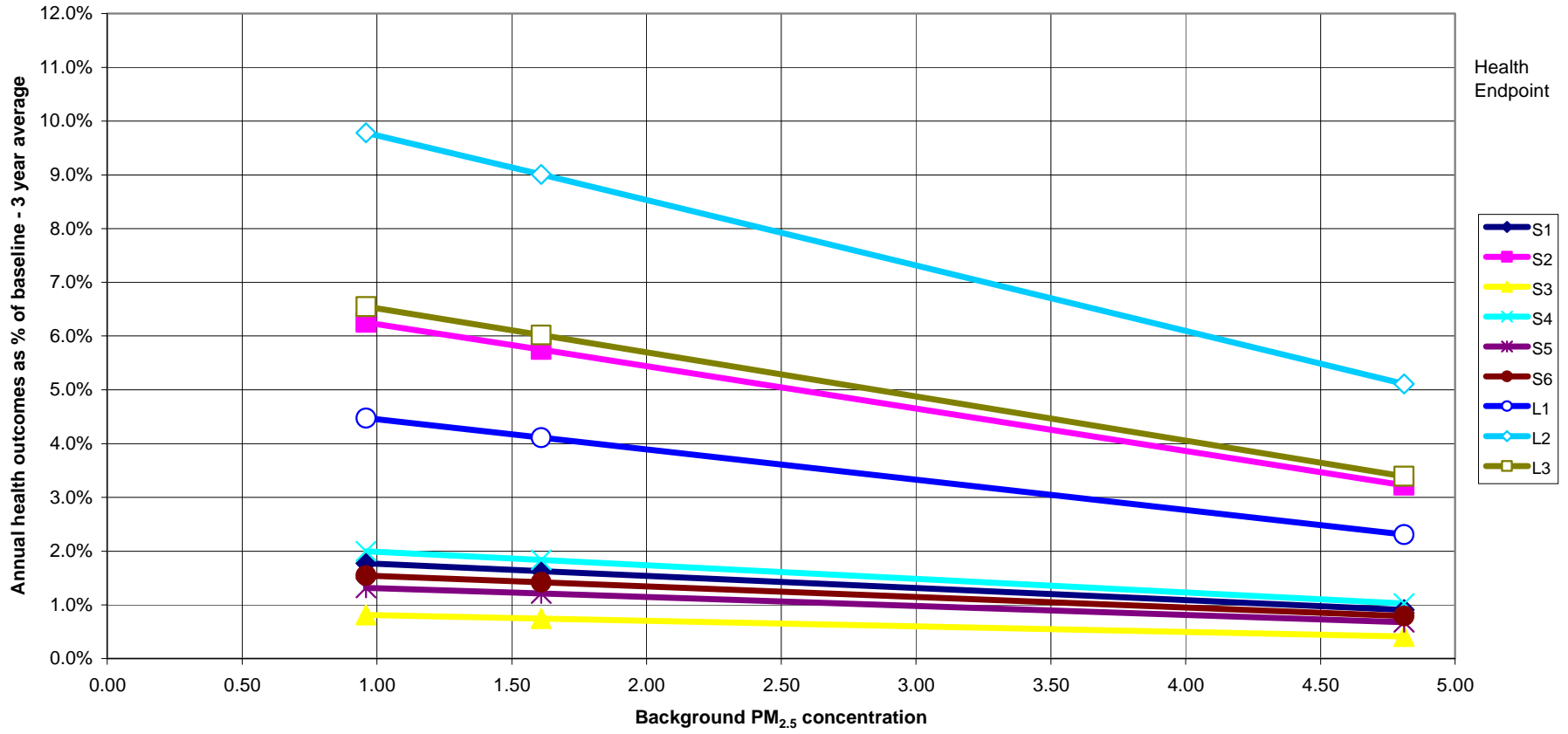


Table U2.2 - PM<sub>2.5</sub> Risk Assessment - Sensitivity of health outcomes attributable to above-background PM<sub>2.5</sub> to background concentration - MELBOURNE

Background Level	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Hospital Admissions</i>	<i>Mortality</i>	<i>Mortality</i>	<i>Mortality</i>
$\mu\text{g}/\text{m}^3$	<i>All cause</i>	<i>Respiratory</i>	<i>Cardiovascular</i>	<i>Asthma</i>	<i>Cardiovascular disease</i>	<i>COPD</i>	<i>All cause</i>	<i>Lung cancer</i>	<i>Cardio-pulmonary disease</i>
	Estimated number of cases per year								
<b>4.81</b>	<b>207</b>	<b>60</b>	<b>41</b>	<b>78</b>	<b>157</b>	<b>15</b>	<b>524</b>	<b>58</b>	<b>316</b>
1.61	370	106	74	139	281	27	933	103	561
0.96	403	115	81	152	306	29	1015	112	610
	Cases as % of baseline rate								
<b>4.81</b>	<b>0.9%</b>	<b>3.2%</b>	<b>0.4%</b>	<b>1.0%</b>	<b>0.7%</b>	<b>0.8%</b>	<b>2.3%</b>	<b>5.1%</b>	<b>3.4%</b>
1.61	1.6%	5.7%	0.7%	1.8%	1.2%	1.4%	4.1%	9.0%	6.0%
0.96	1.8%	6.3%	0.8%	2.0%	1.3%	1.5%	4.5%	9.8%	6.5%

**Figure U2.3 - Annual Health Outcomes Attributable to Above-Background PM<sub>2.5</sub> - MELBOURNE  
Sensitivity to Background Concentration**



## 6. REFERENCES

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Cohen, D.D., 1999, "*Seasonal and Regional Variations in Ambient Fine Particle Concentrations and Sources in New South Wales, Australia: A Seven Year Study.*", International Conference on Urban Climatology Sydney November 1999.

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Appendix 1. Baseline Health Effects Incidence Data: Source data provided by the NEPC Project Team.

Table A1. Baseline health effects incidence data for the short-term health endpoints.

<i>End-point</i>	<i>Description</i>	<i>Age group</i>	<i>Sydney</i>		<i>Melbourne</i>		<i>Brisbane</i>		<i>Perth</i>	
			<i>Daily Incidence<sup>1</sup></i>	<i>Baseline Population</i>	<i>Daily Incidence</i>	<i>Baseline Population</i>	<i>Daily Incidence</i>	<i>Baseline Population<sup>2</sup></i>	<i>Daily Incidence</i>	<i>Baseline Population<sup>3</sup></i>
	<b>Mortality</b>									
S1	All cause	All ages	1.65	100000	55.3	2994280	16	806746	20.2	1244320
S2	Respiratory	All ages	0.138	100000	4.5	2994280	1.5	806746	2	1244320
S3	Cardiovascular	All ages	0.724	100000	24.3	2994280	7.4	806746	8.2	1244320
	<b>Hospital Admissions</b>									
S4	Asthma	All ages	26.4	3130000	18.47	2994280	5.9	806746	8.6	1244320
S5	Cardiovascular disease	Elderly	61.8	365000	56.11	359024	17	97881	23.6	134115
S6	COPD	Elderly	12.3	365000	4.6	359024	2.3	97881	4	134115

1. Sydney baseline data for S1-S3 were provided as annual incidence rates and have been converted to daily incidence rates. The incidence rates are averaged over three years (1998-2000).

2. 'Brisbane City' Statistical Subdivision populations.

3. Perth Statistical Division populations.

Table A2. Baseline health effects incidence data for the long-term health endpoints.

<i>End-point</i>	<i>Description</i>	<i>Age group</i>	<i>Sydney</i>		<i>Melbourne</i>		<i>Brisbane</i>		<i>Perth</i>	
			<i>Annual Incidence</i>	<i>Baseline Population</i>	<i>Annual Incidence</i>	<i>Baseline Population</i>	<i>Annual Incidence</i>	<i>Baseline Population</i>	<i>Annual Incidence</i>	<i>Baseline Population</i>
	<b>Mortality</b>									
L1	All cause <sup>1</sup>	All ages	603.6	100000	20185	2994280	5840	806746	7373	1244320
L2	Lung cancer <sup>2</sup>	All ages	34.3	100000	33.9	100000	37.4	100000	38.4	100000
L3	Cardiopulmonary disease <sup>2</sup>	All ages	309.6	100000	276.7	100000	311.1	100000	275.3	100000

1. Incidence data for endpoint L1 are the daily incidence rates for S1 in Table A1 converted to annual rates for Melbourne, Brisbane and Perth.

2. Incidence rates for endpoints L2 and L3 for all cities and L1 for Sydney are averaged over three years (1998-2000)