CHAPTER 3
PARTICULATE AIR POLLUTION IN ARMIDALE

3.1 Introduction

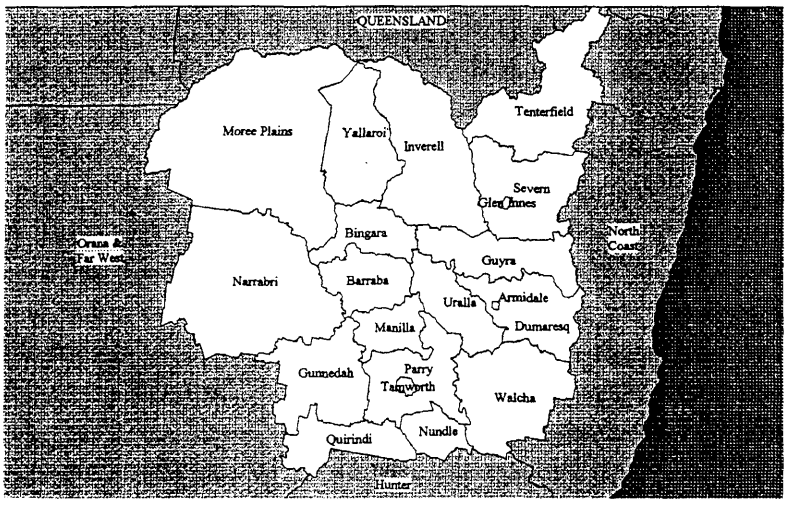
The main objective of this chapter is to assess particulate air pollution in Armidale. The chapter begins with a geographical description of Armidale, its climate and demographic profile, in so far as these relate to particulate air pollution. Sources of air pollution, methods of monitoring pollution and local records of pollution-related data are reviewed. Similar studies, both local and overseas, are also reviewed to provide a comparative assessment of air pollution in Armidale.

3.2 Armidale: An Introduction

3.2.1 Geographical Location

Armidale is in the Northern Tablelands of the New England Region of New South Wales (NSW), Australia. The Tablelands are bordered by the Hunter region to the south, Queensland to the north, the Orana region to the west and the North Coast to the east (Figure 3.1).

Figure 3.1: Geographical Location of Armidale



The city of Armidale is located 1,080 metres above sea level on the New England highway, 570 km north of Sydney, 470 km south of Brisbane and approximately 170 km inland from the Pacific Ocean. The total area is 34.5 sq km. The economy is largely based on education and agriculture, the latter mostly grazing and wool production.

3.2.2 Demographic Profile

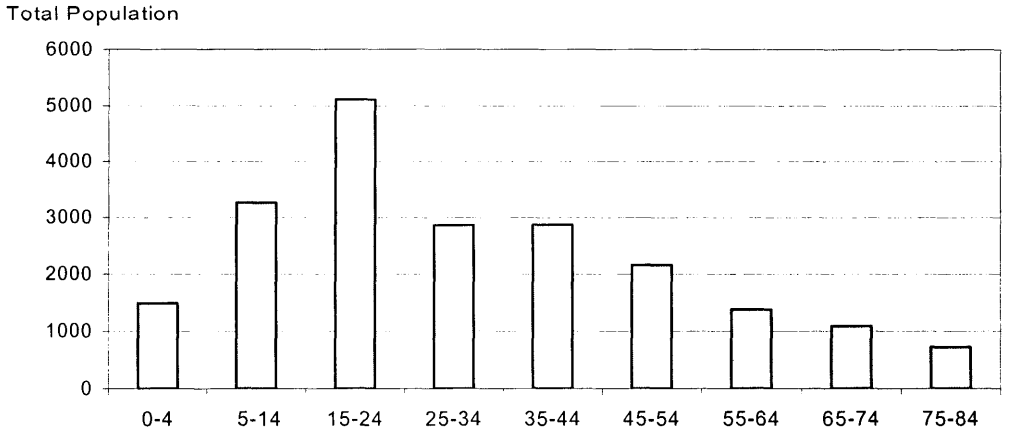
According to the 1996 census Armidale's total population was 21,331, of which 10,159 were male and 11,172 female. The age distribution is given in Table 3.1 and Figure 3.2.

Table 3.1: Population Age Structure in Armidale

Population Age Group	Total Population	Percentage
0-4	1498	7.0
5-14	3276	15.4
15-24	5127	24.0
25-34	2875	13.5
35-44	2879	13.5
45-54	2173	10.2
55-64	1389	6.5
65-74	1101	5.2
75-84	737	3.5
85+	276	1.3

Source ABS, 1997

Figure 3.2: Population and Age Structure in Armidale



Source ABS, 1997

Children under 15 years of age represented around 23 per cent of the population, slightly higher than the NSW average (22 per cent). People aged 65 years and over represented around 10 per cent in comparison to 12 per cent of the total NSW population. The high proportion of teenage and young adults (around 24 per cent of the total population) was possibly due to the student population at the University of New England.

Aboriginal and Torres Strait Islander people constituted 4.8 per cent of the total population in Armidale, higher than the NSW average of 1.2 per cent (ABS 1997).

In Armidale, 8,872 persons were in the labour force: 7,844 persons employed and 1,028 unemployed. The unemployment rate was 11.6 per cent for Armidale compared to 6.1 per cent for NSW (ABS 1997).

The median household income for Armidale was $559 per week and for NSW it was $500-$699 (ABS 1997).

The overall age standardised mortality rates for both males and females in New England in 1988-1992 were significantly higher than NSW as a whole (Table 3.2). The major causes of death for New England residents between 1988-1992 were circulatory diseases and cancer, which is similar to elsewhere in NSW.

Table 3.2: Age Standardised Mortality Rates (per 1,000 people), 1988-1992

	Male	Female
New England	10.6	6.0
NSW	9.5	5.7

Source: NSW Health Department, July 1996; New England Area Profile.

3.2.3 Climate

The New England climate is determined by both terrestrial and atmospheric factors. The main terrestrial factors affecting climate are distance from the coast, distance from the eastern escarpment, relief, altitude and latitude (Tucker 1991). Distance from the coast and the escarpment relate to lower rainfall and higher ranges of temperature, while local rainfall, temperature, and wind patterns are largely determined by local relief. Altitude and latitude

are largely responsible for the maximum and minimum temperatures recorded in the New England Region (Tucker 1991).

During summer, the New England Region is predominantly influenced by an easterly airflow consisting mainly of marine air from the Tasman Sea. In the winter, cool dry air masses affect the region, derived from the continental interior or from the southern ocean. Winds are mainly westerlies and south westerlies (Tucker 1991).

Armidale experiences cold winters and mild summers. The (103 year average) mean temperature for July, the coldest month, is 6.5° C and the mean temperature for January is 20.4° C (Burr 1994). Frosts are experienced between April and October.

The (130 year) average annual rainfall is 787.6 mm (Burr 1994), with a late spring to summer dominance. Average weekly rainfall and temperatures are given in Figures 3.3 and 3.4 (source: Armidale Dumaresq Council, 1995). Maximum rainfall occurs in summer, related to topical cyclones associated with migration into northern Australia of the Inter Topical Convergence Zone and the widespread thunderstorm activity which this promotes. A secondary source of moisture accompanies the passage of cold fronts, especially in winter, which usually originate in sub-polar depressions to the South. This can result in snow.

Figure 3.3: 30 Year Average Weekly Rainfall in Armidale

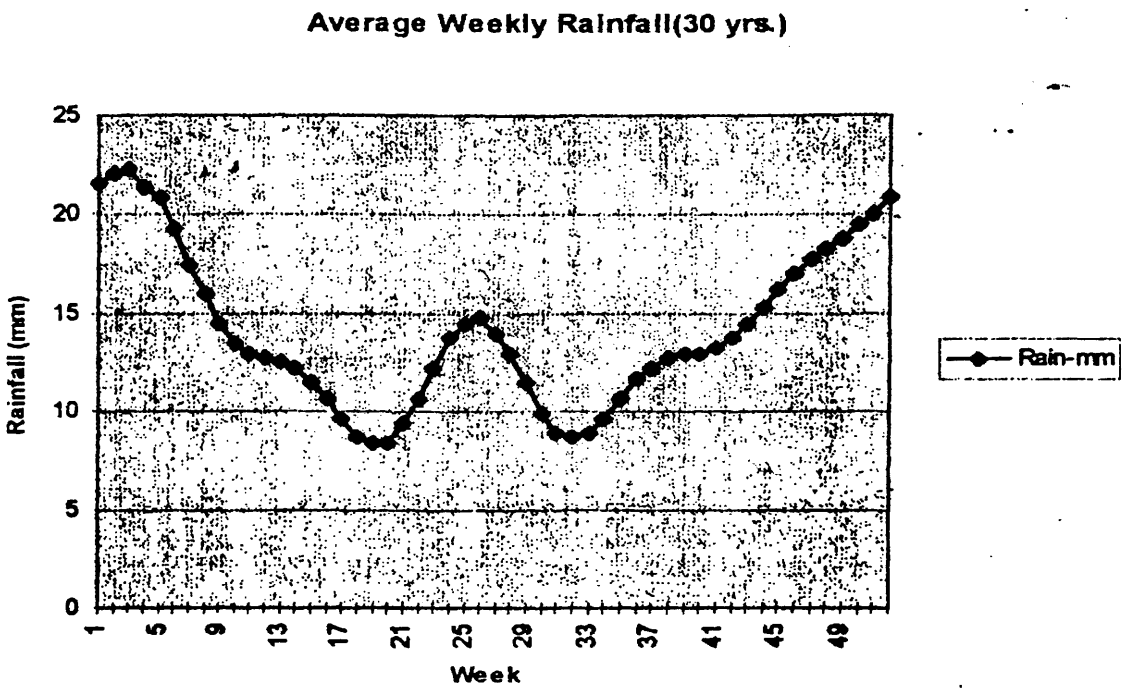
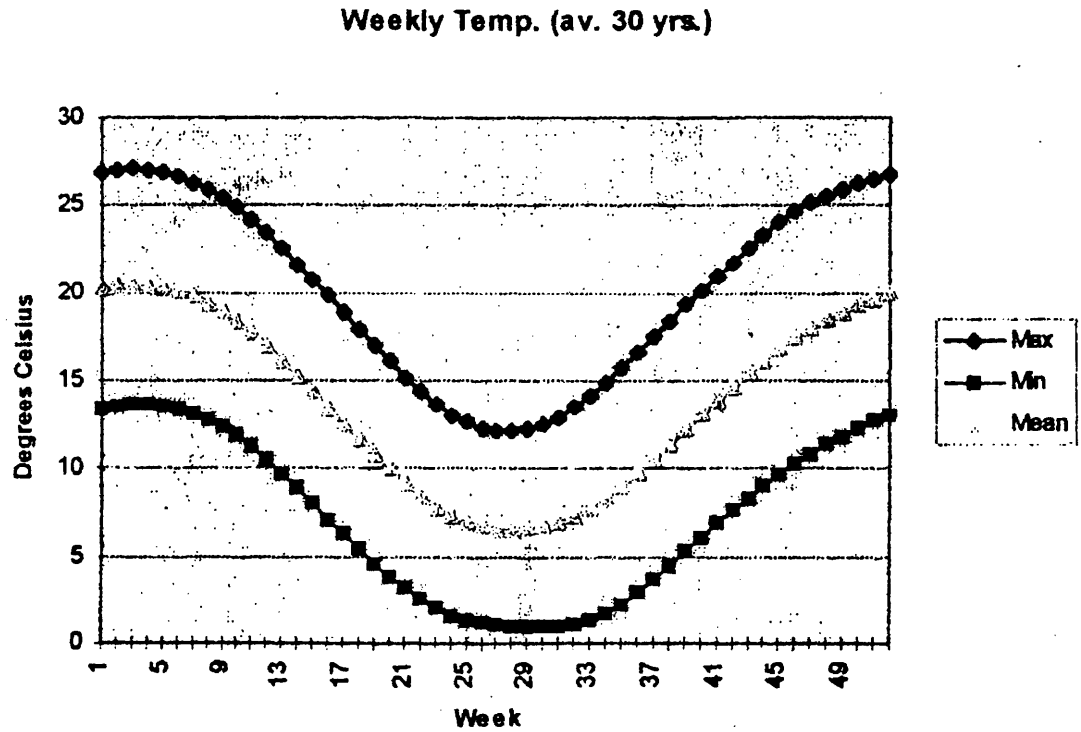


Figure 3.4: Average Weekly Temperature in Armidale



3.3 Particulate Air Pollution

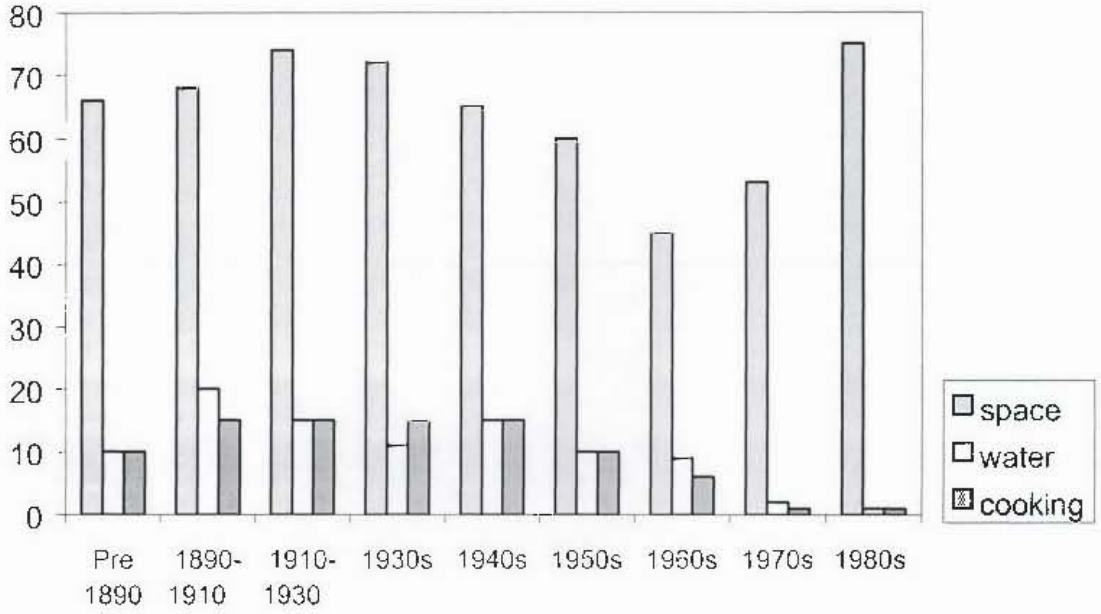
3.3.1 Wood Heaters and Woodsmoke

The use of wood as a source of residential heating has increased in North America and Europe since 1973, and in Australia since 1978 (Quraishi 1987). There are several reasons for the popularity of wood heaters. Firstly, a large increase in oil price in the late 1970s triggered a shift away from oil towards solid-fuel (Quraishi 1987, Wall 1997). Secondly, research and development into wood heater design in the last two decades has culminated in 60 to 70 per cent efficient systems, making wood heaters economically attractive as well as aesthetically appealing. Thirdly, modern wood cutting equipment has made firewood more easily available (Roberts and Lin 1998).

Wood is seen as an alternative to conventional sources of energy for residential heating, especially in areas with easy access to forests. Whilst wood is a renewable resource, this advantage is offset by the increased air pollution from wood heating devices compared with devices fuelled by oil and gas. Cooper *et al.* (1981) reported particulate and organic carbon emission rates as much as one to two orders of magnitude larger in wood heating devices than in oil or gas heating units. On a per unit heat output basis, heating with wood generates 20 times more particles than oil and 50 times more than gas (Rau 1989).

As in the other cities and towns, a sharp increase in residential wood heating has been documented for Armidale (Figure 3.5). Wood is readily available around Armidale, partly due to "die back". While there are no firm estimate, our survey revealed that about 30 per cent of households that burn wood collect their own fire wood. According to Wall (1997) there are 3,500 slow combustion and 600 fireplaces operating in the Armidale area. ADC's 1996 survey showed that about 55 per cent of households used wood for winter heating in comparison, the EPA survey found that the State-wide figure was only 25 per cent (Roberts and Lin, 1998). It is probable that many of that 55 per cent do not have any other source of heating. Armidale residents consume approximately 17,000 tonnes of fuel wood each year (Wall 1997). Around 150 tonnes of firewood is burned on a cold night, which produces up to 2 tonnes of particulate matter (Wall 1997).

Figure 3.5: Proportion of Armidale Dwellings in which Wood is the Main Source of Energy for Space Heating, Cooking and Water Heating



Source: Wall, 1995

Armidale's woodsmoke problem is aggravated by local atmospheric conditions. Armidale is located in a shallow depression, at a relatively high elevation (range 960 to 1,080 meters above sea level), sea breezes are absent and high-pressure cells dominate in winter. The result is frequent clear, calm and cold winter nights. These conditions create temperature inversions, caused by long-wave radiation loss from the ground on a cloudless night (Linacre and Hobbs 1977), in which rapid cooling of the ground causes surface air to cool more quickly than the air above (Lynn 1976). The result is that atmospheric dispersion is poor during cloudless winter nights.

During the months of April to October, night time temperature inversions are frequent in Armidale. These inversions trap smoke and prevent its dispersion. Trapped woodsmoke accumulates under cold air to form a "brown haze", which is often visible in the morning. It usually disperses by around 10 am as the day warms up and the climatic inversion resolves.

Another significant source of particulate emissions is the boiler at the University of New England. At maximum operation it consumes about 17 t/day of coal and contributes about 6 per cent to particle loading during a very cold night (Wall 1997).

Pollution from oil and gas are considered negligible, particularly since only about15 per cent of Armidale residents use oil for interior heating and 12 per cent use gas (Wall 1997). Particles of biogenic origin including starch, spores, pollen, and fungal hyphae are negligible in winter.

Fugitive dusts and road dusts are other potential sources of airborne particulates (Sexton *et al.* 1984, Zib 1984). In the United States 90 per cent of national particulate emissions originate from fugitive dust (Zip 1984), which includes wind erosion and travelling on unpaved roads. On a national scale, the contribution of dust to total particles in Australia is significant (Zip 1984), yet from a night time Armidale perspective, dust is unlikely to contribute to haze at all. For the same reason, motor vehicles are thought to contribute insignificant amounts of particulates but they do contribute about 25 per cent of the carbon monoxide. Other sources of emission are negligible in Armidale. In the absence of other significant local industry, residential wood combustion (RWC) is considered to be responsible for the remainder.

The brown haze problem occurs in all wood burning towns and cities in which firewood is burned and atmospheric dispersion is poor. Examples include Launceston, Canberra, and Lithgow, as well as some cities and towns in North America (such as Nevada, Oregon, Denver, Montana, and Colorado). This brown haze problem degrades visibility and is a potential health threat.

3.3.2 Monitoring Woodsmoke

Air pollution can be monitored by gravimetric methods such as high volume samplers, or by light scattering methods such as nephelometers.

The nephelometer works on the principle that light passing through the atmosphere diminishes mostly by scattering and absorption by fine particulates (particles between 0.1 and 2.5 microns in size). Preheated air is continuously drawn into the nephelometer and

illuminated by a light source. A photo-multiplier detects the intensity of scattered light, which increases with increasing concentrations of fine particles (Ayres *et al.* 1999). NSW cities such as Sydney, Wollongong and Newcastle use nephelometers to determine the aerosol scattering coefficient (bsp).

Nephelometer readings are reported in units of 10^{-4} m^{-1}. In Australia levels have been found to be generally with in the range 0.001 to 10 (10^{-4} m^{-1}) The reading can be interpreted as the proportion of light lost per unit distance eg a reading of 2 is a typical reading for a hazy atmosphere, and represents a loss of light through particle scattering of 0.02 per cent in one metre (Roberts 1995). A reading of 2 or more is considered high in terms of the SPI (Sydney Air Pollution Index). It is important to note that SPI is based on a visibility goal of 11 km and not a particular "health" goal (Ainsworth 1996, Roberts 1995).

The EPA began monitoring air pollution in Armidale in April 1995 using a nephelometer, placed within the grounds of the Armidale swimming pool centre. Measurements were also taken on a second machine situated in east Armidale. A mobile nephelometer was also used to allow a broader indication of particulate pollution across the city.

For a high volume air sampler, air is drawn in at a constant flow rate (about $70m^3$/hr). Particulates larger than 10 microns (PM10) are trapped by an impaction plate. Smaller particles follow the airstream through vent tubes and are collected on a pre-weighed filter. After sampling, the density of particles less than PM10 is calculated at standard temperature and pressure conditions. Finally, this density can be calibrated against near-by nephelometer readings, although this technique is labour intensive (Roberts 1995).

In 1995 in addition to the nephelometer placed within the grounds of the Armidale Swimming pool centre, a high volume air sampler was set on the same location to establish a correlation between the nephelometer and the volume of fine particles (Roberts and Lin 1998). Simultaneous 3-h readings from the nephelometer and high volume sampler were obtained on seven occasions. A regression relationship between bsp and PM10 was derived; it was linear and highly significant (PM 10 = 19.01 * bsp + 9.02) (Wall 1997).

3.3.3 Air Pollution Index

In Sydney, three pollution indices are calculated – O_3, NO_2, and fine particles. Sydney's air pollution is described as low (L), if the nephelometer coefficient is less than 1.05, medium (M) if coefficient is in between 1.05 and 2, and high (H) if it is more than 2.1. The nephelometer coefficient used is the highest hourly value. The corresponding index has been calculated for Armidale, based only on the particulate measurement, assuming O_3 and NO_2 small enough not to influence the index. In Armidale, the scales were rounded and two additional categories were added (Table 3.3).

Table 3.3: Armidale Air Quality Index

Category	Low	Medium	High	Very High	Extreme
Neph coefficient (Max 1-hour average)	<1	1-2	2-4	4-8	>8
Equivalent PM2.5 $\mu g/m^3$ (max 1-hour average)	<20	20-40	40-80	80-160	>160

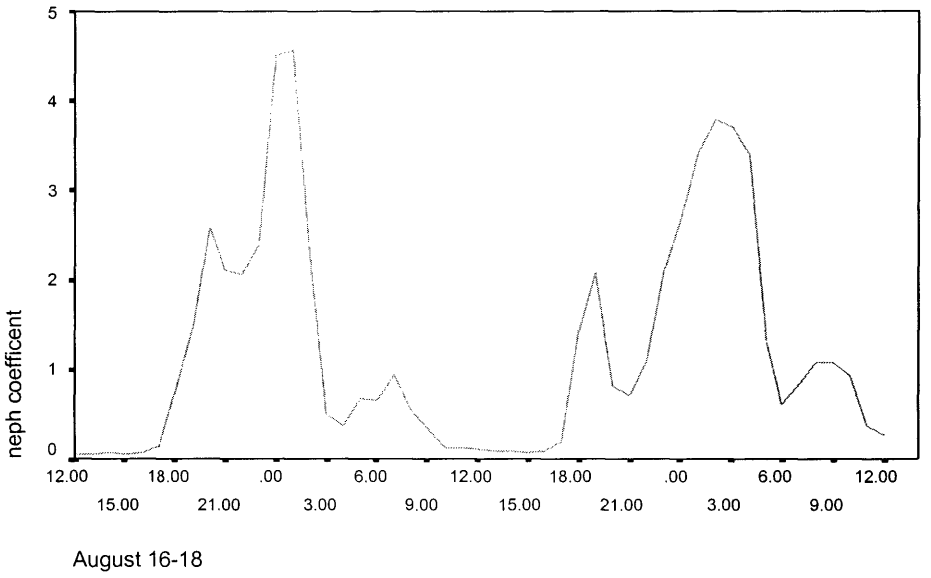
Sources: 1 ADC Air Quality Technical Working Committee
2. Armidale Air Quality Group

3.3.4 Pollution Pattern

In winter in Armidale, particulate pollution follows a particular pattern. Fine particle levels starts to build up from late afternoon as fires are ignited. High levels are experienced around midnight and into the early morning. This temporal pattern is characteristic of the onset of both stable atmospheric conditions and wood heating on cold, clear evenings. The peak is probably related to the common practice of loading the fire at night and cutting back the oxygen supply when retiring to bed. This establishes a smouldering fire, which lasts for many hours. A second increase in particulates occurs on cold still mornings between 6 to 8 am when people reactivate or re-light their wood heaters. Figure 3.6 shows the fluctuation in particulate level for the period 16 to 18 August 1999.

Figure 3.6: Fluctuation of Particulate Pollution

August 16-18, 1999



This is a "classic" profile of particulates when a period of low temperature and low wind speed is experienced. It is evident from the graph that particulate levels fall to almost zero by midday. Around this time the temperature has risen, wind speed may have increased, home-owners have shutdown their fires, and the inversion layer dissipated.

During periods of high wind speed and temperature, particulate pollution in Armidale decreases. This could reflect either lower use of wood heaters or particulates being dispersed by winds. Particulate pollution does, however, increase when wind speed and temperature decrease. It appears that low wind speed is the dominant factor required for an accumulation of particulates. That is, wood fires are consistently burnt in Armidale's cold winter periods, but particulates are unlikely to accumulate over the city unless wind speeds are low.

Particulate pollution fluctuation has also been observed in US wood-burning towns. Typically, night time levels in Waterbury, Vermont exceed afternoon levels by 5 to 10 fold (Sexton *et al.* 1984), and in Portland, Oregon 3 to 6 hour peak concentrations during evenings contribute up to 50 per cent of the mean 24-hour concentration (Khalil *et al.* 1983). Makut and Fry (1992) gave the hourly variation of nephelometer coefficients for two different locations in the Puget Sound Region, Washington. They noticed a substantial increase in bps frequently exhibited at the residential site (Lake Forest Park) in the evening

(around 7 pm), which peaked around midnight. In contrast, in the industrial valley (Duwamish) a daytime peak dominated, mainly associated with industrial activity.

3.4 Monitoring Particulate Air Pollution in Armidale

3.4.1 EPA/Armidale Dumaresq Council Records

ADC established a monitoring program for woodsmoke particulates in association with the EPA in winter 1995. The EPA monitored nephelometer observations until the end of the 1997 winter season. Council then acquired the (Belfort) nephelometer and conducted the monitoring activity for the 1998 and 1999 winter seasons. In 1999, Council moved their nephelometer from the swimming pool centre to the Council Chambers in the CBD.

Table 3.4 contains a summary of the last three years winter air pollution records.

Table 3.4: Particulate Air Pollution in Armidale 1997-99
(Maximum 24 hour nephelometer coefficient)

Year	Minimum	Maximum	Mean	St. Deviation
1997	0.44	8.37	3.95	1.77
1998	0.11	9.55	3.22	2.53
1999	0.10	8.30	2.04	1.95

Sources: calculated from EPA /ADC data.

Figure 3.7 and Table 3.5 compare particulate air pollution data for the 1995 through 1999 winters. On average, more than 50 per cent of Armidale's winter days fall in the high to extreme category of particulate pollution level. In 1995, around 60 per cent of winter days fell in the high to extreme category, in 1996 it was 68 per cent, 84 per cent in 1997, 47 per cent in 1998 and 42 per cent in 1999. Table 3.6 shows minimum winter temperatures in Armidale.

Figure 3.7: Particulate Pollution in Armidale
1997, 1998, and 1999

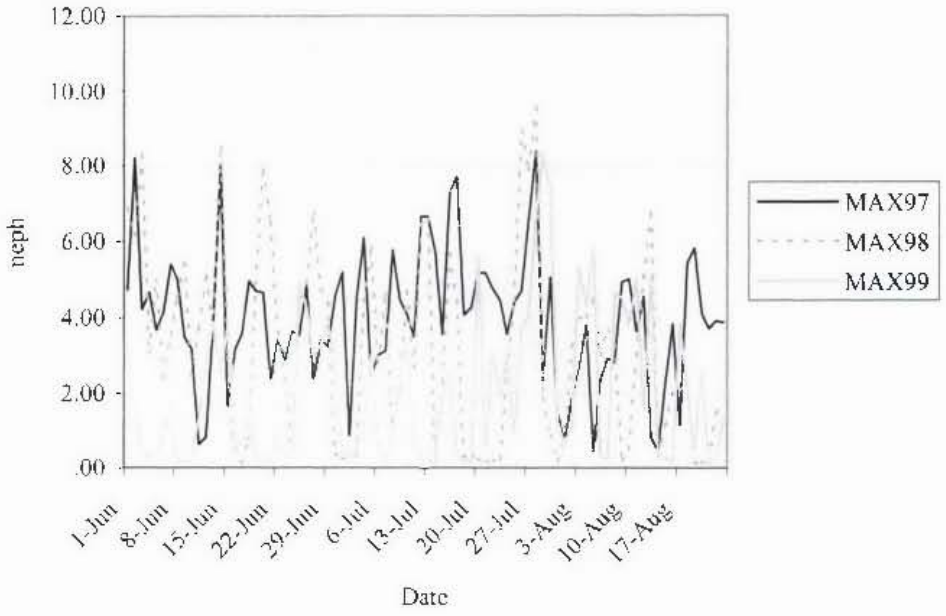


Table 3.5: ADC Nephelometer Records
Winter Months: 1995 - 1999

(Highest hourly average for 24 hour period)

Month	**Number of days**														
	2<c<4					**4<c<6**					**c>6**				
	95	**96**	**97**	**98**	**99**	**95**	**96**	**97**	**98**	**99**	**95**	**96**	**97**	**98**	**99**
April	2	8	4	3	-	0	2	0	0	-	0	0	0	0	-
May	6	8	6	11	-	3	9	6	4	-	6	1	2	3	-
June	2	6	10	8	7	4	11	10	8	1	8	1	5	4	0
July	2	6	12	3	11	6	16	14	7	3	10	1	4	2	0
August	14	9	15	7	5	3	13	6	2	11	5	1	2	3	1
Sept	8	6	11	0	-	2	5	0	0	-	0	0	0	0	-

Sources: ADC, EPA data

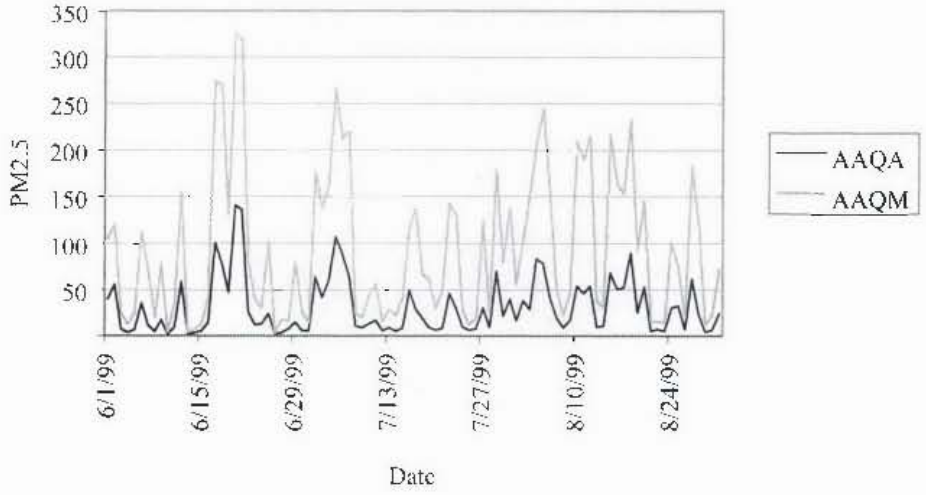
Table 3.6: Minimum Temperature Data for Armidale
Winters of 1997, 1998 and 1999

	Average Minimum			**Lowest Minimum**			**Cold mornings (total # days below 5.0 degrees)**		
	1999	1998	1997	1999	1998	1997	1999	1998	1997
June	1.3	1.8	-0.3	-8.2	-3.0	-	20	25	29
July	2.5	2.9	-1.6	-4.0	-5.9	-8.0	23	21	28
Aug	0.6	3.4	-2.6	-6.3	-6.9	-8.4	24	19	31
Total							**67**	**65**	**88**

3.4.2 Armidale Air Quality Group Records

Air quality in Armidale is also recorded by AAQG using a Radiance Research Reeve Analytical Orthogonal nephelometer calibrated for PM2.5 (one scattering coefficient unit is between 20 to 23 $\mu g/m^3$ of PM2.5, Robinson 1999). This nephelometer is situated in the East Armidale residential area, where wood is burnt for winter heating, and has remained there throughout the study period. Figure 3.8 gives particulate pollution records for East Armidale for the winter of 1999 (maximum 1-hour (AAQM) and daily average (AAQA)).

Figure 3.8: Particulate Air Pollution in East Armidale
(June - August 1999)



3.4.3 Comparison of Air Pollution Data

ADC launched an awareness campaign concerning the health implications of woodsmoke, aimed at improving the community's operation of their wood heaters, promoting alternative fuel sources, and promoting energy efficiency. There is some indication of improvement in the recorded wood-smoke particulate levels, particularly in 1998 and 1999 compared with 1997. This *may* suggest that the ADC education campaign has improved the community's wood-heating behaviour.

There is an alternative explanation, however, that must also be considered. Weather conditions in Armidale during June, July and August of 1998 and 1999 were somewhat milder and drier than normal (Table 3.6), so there may have been less need to use wood fires. In winter 1999, day temperatures were slightly higher than normal, averaging 13.3 degrees – 0.5 degree higher than the long-term average. More importantly, overnight temperatures were considerably higher than normal, averaging 1.5 degrees – 1.3 degrees higher than the long-term average. Minimum temperatures dropped below 5.0 degrees on 67 days compared to 78 days for the long-term average.

Another possible explanation for the apparent improvement in pollution levels in 1999 is that ADC moved the site of their nephelometer from the swimming pool area to Council's office in 1999. This office is located in the CBD area, which is relatively chimney free. Without calibration, it would be foolhardy to attempt any direct comparisons using EPA/ADC data between 1999 and the previous two years.

There are substantial differences between ADC (CBD) and AAQG (East Armidale) particulate air pollution readings. Table 3.7 compares the daily average and daily maximum readings for east Armidale and the CBD from July to August 1999.

Table 3.7: Comparison Between CBD (ADC Data) and East Armidale (AAQG Data) Air Pollution Data (PM2.5)

	Minimum	Maximum	Mean	Std. Deviation
AAQG daily average (24-h)	1.71	140.46	31.70	31.39
AAQG daily maximum (1-h)	3.17	325.94	96.18	83.70
ADC daily average (24-h)	1.26	44.82	13.83	12.14
ADC daily maximum (1-h)	2.25	186.75	46.08	44.50

ADC = Armidale Dumaresq Council/EPA data

AAQG = Armidale Air Quality Group data

In the 1999 winter monitoring period, mean daily average PM2.5 for the CBD was 13.8 and for East Armidale was 31.7. When the maximum daily average in CBD was 44.8, it was 140 in East Armidale. Comparative results are also shown in Figures 3.9 and 3.10.

Figure 3.9: Daily Average Particulate Pollution in CBD and East Armidale
(June - August 1999)

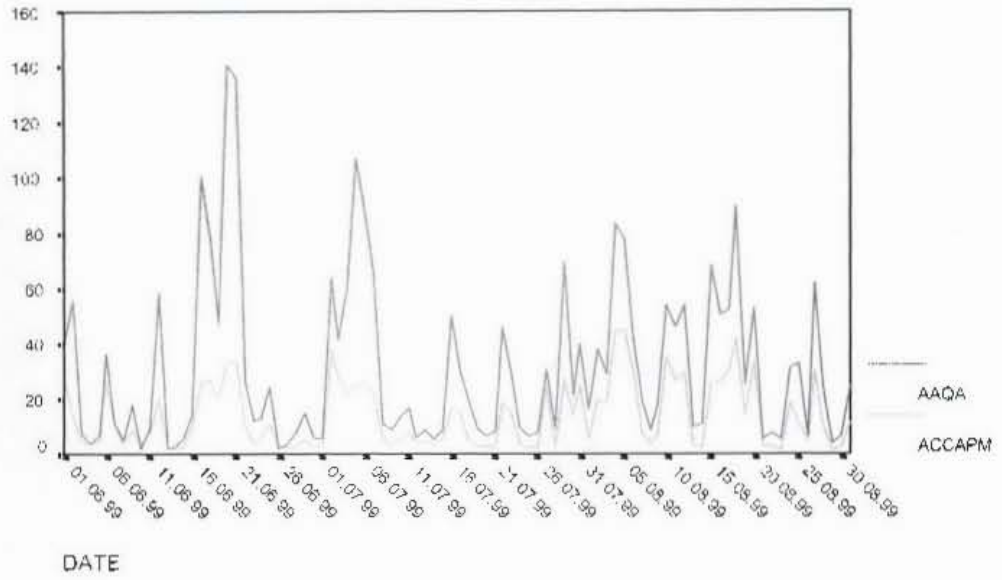
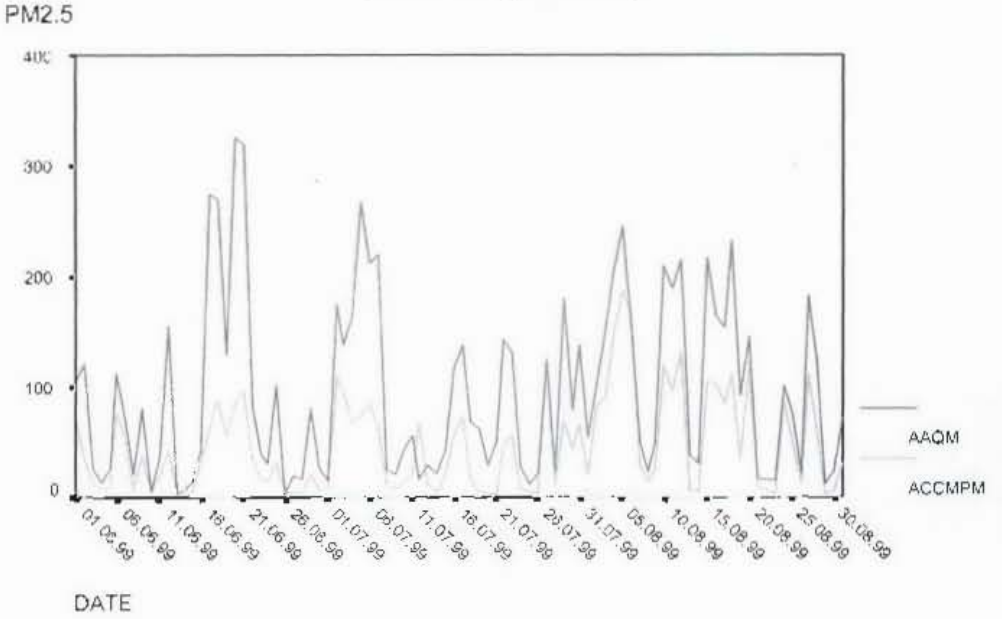


Figure 3.10: Daily Maximum Particulate Air Pollution in CBD and East Armidale
(June - August 1999)



A probable reason for the differences between CBD and East Armidale data may be the location of the monitoring devices. AAQG's nephelometer is located in the residential area where wood is burnt for winter heating and the ADC/EPA monitor is located in the CBD area, which is a comparatively chimney-free area. The change in monitoring location alone may account for ADC's lower winter nephelometer readings in 1999.

Larson and Koenig (1994, p134) noted that 'the Klamath Falls studies emphasise ... the location of the air-monitoring device. There is up to a four-fold difference between various parts of town, with the highest reading in the residential areas'. The Armidale Air Quality Group revealed that 'in places, average pollution levels were found to increase four-fold within 40 meters' (Robinson 1997) when they used a portable nephelometer to measure the variation in woodsmoke pollution in Armidale.

Considering all these possibilities it is premature to suggest that any of these results indicate either (a) the frequency or (b) the intensity of high particulate episodes in Armidale is being reduced due to modified community behaviour, although publicity since monitoring started may have raised community awareness of the issues.

3.4.4 Population Exposure

To estimate population exposure for Armidale, a pollution mapping exercise using six north/south transects of the city was carried out by AAQG (Robinson 1997, 1998, 2001). A portable Radiance Research nephelometer was used to measure the variation in woodsmoke pollution on 14 winter nights in 1996. Pollution levels varied considerably, according to location, ranging from scattering coefficients (bsp) of less than $1x10^{-4}$ to $8.7x10^{-4}$, the latter representing an average over 14 measurement of 174 $\mu g/m^3$ of PM2.5 (Robinson 1997).

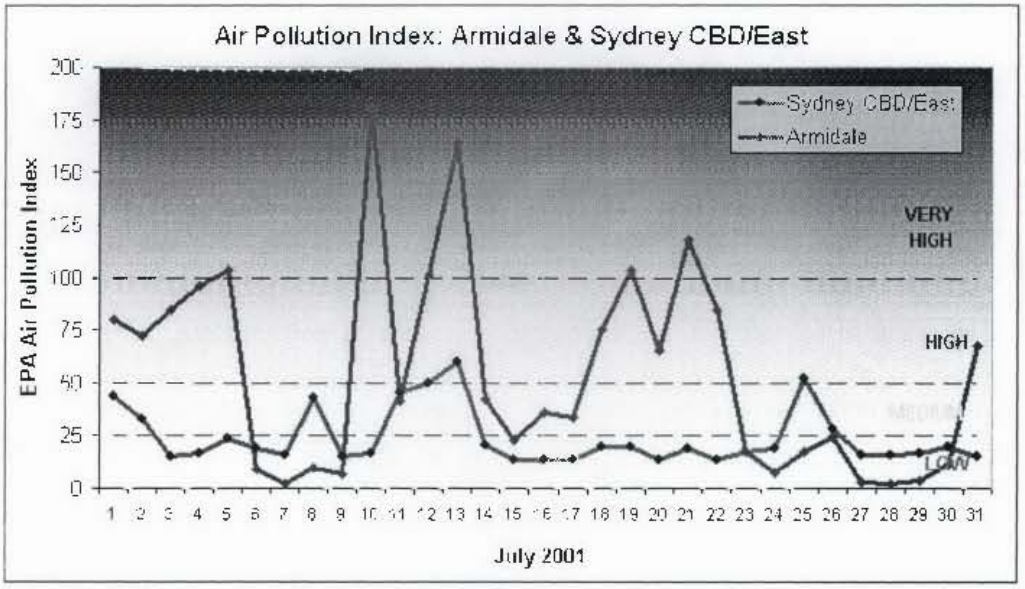
The study found that the average nephelometer coefficient for the whole area was 4.08. Averaged over the 14 nights, the ADC/EPA nephelometer and East Armidale nephelometer scattering coefficients were 3.35 and 8.68 respectively. Measured pollution on the 14 nights near the East Armidale monitor was, on average, 2.56 times higher than at the CBD.

The study suggested that the ratio of average pollution on the 14 nights for any/all locations in Armidale compared with the CBD monitor could be used as an indicator of the ratio of monthly averaged pollution levels for those locations. After adjusting for possible discrepancies, the study suggested that the estimated exposure for the town as a whole was neph coefficient 1.02 for the 6 months from April to September (equivalent to a 6-monthly average PM2.5 pollution exposure of $20.4\mu g/m^3$), 0.28 (5.6 PM2.5) in October as heater use declines, and no pollution in the summer months (Robinson 1997).

3.5 Comparison of Armidale Air Pollution Readings with other Cities

Figure 3.12 compares the Sydney East/CBD Regional Pollution Index (RPI) in July 2001 with Armidale data. The Air Pollution Index in Armidale recorded high to extreme readings for around 50 per cent of the nights in July 2001. During the same period, Sydney's air pollution index only recorded high readings on two nights.

Figure 3.11: Sydney and Armidale Air Pollution Data



Source: Armidale Air Quality Group

Air pollution data in Armidale can be compared with other wood burning towns and cities experiencing winter inversions. Sexton *et al.* (1984) obtained a TSP value of 80µg/m3 in Waterbury, Vermont. Only 28 per cent of the 350 households in Waterbury (population 2,000) used wood as their main form of energy. In Elverum, Norway (population 10,000) a maximum level of 101µg/m^3 was recorded for PM2.5 in 1982 (Ramdahl *et al.* 1984). Launceston in Tasmania (population 95,000), where almost 70 per cent of residents use fuel wood, recorded up to 250µg/m^3 for PM10 in July 1991 (Todd 1992). As shown in Figure 3.8, Armidale experienced maximum higher than these even during the relatively mild winter of 1999.

3.6 Compliance with the National Standard

Australia adopted a National Environment Protection Measure (NEPM) in 1998 that sets national ambient air quality standards for all states and territories. The standard covers six major pollutants - particulate (PM10), O_3, SO_2, NO_2, CO and lead (National Environment Protection Council, NEPC 1997). The NEPM air standard envisages that ambient concentrations of PM10 (over 24 hours) should not exceed 50μg/m^3 on more than 5 occasions per year. However, there is a strong community concern for considering PM2.5 standard instead of PM10, since PM2.5 is more relevant to adverse health effects. The NEPC Technical Review Panel recommended a limit of 25μg/m^3 of PM2.5 and the Health Review Panel 20-25μg/m^3 (NEPC 1997). In December 2000, NEPC initiated a review to determine whether a new ambient air quality standard for PM2.5 was needed in Australia, and the feasibility of developing such a standard (NEPC 2001).

Particulate air pollution data show that on average around 50 per cent of Armidale's winter days fall in the high to extreme air pollution index category. In 1999, the hourly maximum PM2.5 pollution in the CBD reached 186μg/m^3 and 325μg/m^3 in East Armidale. The PM2.5 readings are much higher than the NEPM PM10 standard, and compare very unfavourably with the PM2.5 standard of 20-25μg/m^3 recommended by the Technical Review and Health Review panels.

Clearly, the concentration of particles in Armidale is reaching levels high enough to be of significant health concern. Given this, and based on the review of the health effects of particulate air pollution (Chapter 2), there is an urgent need for a more detailed assessment of the health implications of woodsmoke in Armidale and the associated economic costs.

CHAPTER 4
COST OF PARTICULATE AIR POLLUTION IN ARMIDALE: A THEORETICAL ESTIMATE

4.1 Introduction

An important objective of this study is to estimate, in dollars, the economic benefits of better health resulting from a reduction in particulate air pollution in Armidale. The health effects and economic valuation that follow is based on dose-response relationship and air pollution data for Armidale available at the present time. This chapter develops a range of economic costs associated with the current level of air pollution within Armidale. The figures could also be interpreted as potential public benefits from institutional or technical options for reducing current levels of pollution. Several important terms are introduced here and specific definitions (based on the World Health Organisation's publication 1982) provided to help with the analysis.

Air pollution exposure estimation, used to make exposure assessments and health-effect analyses, 'is the determination of the distribution of pollutants to humans as a function of time and location' (WHO 1982, p7). Such assessments provide pointers to the characteristics of the population or sub-groups exposed to particular pollutants and the conditions of exposure. On the other hand, the purpose of health-effect studies is 'to determine at what level a given exposure implies an unacceptable risk to human health' (WHO 1982, p7).

Two critical measures are important for our study. One is *exposure* and the other is *dose*. The former quantifies the degree of 'contact between the pollutant and the outer or inner surface of the human body' (WHO 1982, p7). To be more precise, 'air pollution exposure estimation is the determination of the concentration of air pollutants in the inhaled air as a function of time and space coordinates, in which the space coordinates define the position of the exposed individual, particularly his nose and mouth' (WHO 1982, p7). Thus, the association between the concentration of high air pollution and their likely harmful effect on the wellbeing of the human population is quantifiably reflected by this measure.

Introducing physiological considerations into the analysis, *dose*, in contrast denotes 'the amount of air pollutant that crosses one of the body boundaries' (WHO 1982, p7) like the epithelium in the nose or lungs.

Two other concepts are also important to the understanding of the effects of air pollutants – *integrated exposure* and *average exposure*. Time and degree of pollutant concentration are used in estimating integrated exposure, which is fundamentally the product of the two provided the concentration stays constant during the duration of the exposure. This measure is directly linked to the *average exposure,* which facilitates its calculation. More specifically, average exposure is the value of the integrated exposure divided by the mean time. Various average exposure times to air pollution are used, for example, *total exposure* often refers to 24-hour exposure and *occupational exposure* to an 8-hour work period.

To estimate the adverse health effects and economic value associated with air pollution, the following three factors must be determined:

i. dose-response relationship,
ii. the exposed population, and
iii. economic valuation of the health endpoint.

Each of the above steps is difficult and requires extensive data and analysis. Furthermore, each of the steps has uncertainties that complicate the interpretation and use of the resulting estimates (Krupnick and Portney 1991, Hall *et al.* 1992).

Estimating human exposure to air pollution is a rapidly developing research area. As exposure has been defined as the amount of a particular physical or chemical agent that reaches the target, estimates of human exposure are usually based on measurements that treat the human being as the target. Methodological approaches, technical skills, and instruments used to estimate exposure to different air pollutants are, however, still in the development stage.

4.2 Dose-Response Relationship

In determining the dose-response relationship, the first step is to estimate the effects of air pollution on various health outcomes. In many studies, dose functions that relate health impacts of air pollution are adopted from published epidemiological literature. These functions allow expected change in health effects to be estimated for a population with a given change in pollution levels.

Various studies, over a period of two decades, have reported an association between exposure to inhaled particles and adverse health effects, including mortality, cardio-respiratory hospital admissions, respiratory related emergency room visits, decrease in lung function, aggravated asthma and restricted activity days. While particulate air pollution generally had the largest effects on respiratory disease mortality, effects on cardiovascular mortality were also observed (Dockery and Pope 1994).

Studies on particulate air pollution and human health discussed in Chapter 2 form the basis for developing the dose-response relationship in the present work.

In epidemiological studies several alternative measures of particulate matter have been used such as TSP, PM10, PM2.5, BS and CoH.

The most recent research has sought to discover whether observed effects are directly related to particles, to some closely correlated but unmeasurable factor, or else to some specific component of the particles such as sulphur or acidic compounds. The convergence of epidemiological results suggests a clear role for particulates, especially, fine particles such as PM2.5, in triggering numerous health effects. Current research indicates that smaller particles are more dangerous than larger particles (Dockery *et al.*1993, Marrack 1995, Pope *et al.*1995, Abramson and Beer 1998).

PM2.5 can freely reach the gas exchange epithelia in the alveoli where they can do the most damage (Larson and Koenig 1994). Exposure to PM2.5 rather than PM10 has generally been found to be a better predictor of adverse health effects such as increased long-term mortality (Dockery and Pope 1994) across a variety of climates and mixtures with other

pollutants. PM2.5 pollution does not settle readily under gravitation but moves with the general air stream (Larson and Koenig 1994).

Schwartz *et al* (1996) in their six US cities study suggested that increased daily mortality is specifically associated with particle mass constituents under 2.5μg/m^3. The proportion of PM2.5 in PM10 depends on the source of the pollutants.

In Armidale the main sources of particulate pollution is residential wood burning. In wood smoke, almost all the particles are PM2.5, which means that measured concentrations of PM10s are roughly equal to the concentration of PM2.5 for this pollutant (Larson and Koenig 1994). This was also confirmed for Armidale (Robinson 1999). Therefore, it would be more appropriate to use PM2.5 data to calculate the health effects of particulate air pollution in Armidale.

A limited number of epidemiological studies are available that relate PM2.5 pollution data to health effects. While a few studies have been carried out using PM2.5 data and mortality effects (Dockery *et al.* 1993; Pope *et al.* 1995, Schwartz *et al* 1996), studies involving PM2.5 air pollution data and morbidity effects are limited. The main work in this area was done by Ostro (Ostro *et al.* 1983; Ostro 1987; Ostro and Rothschild 1989).

4.2.1 Mortality Coefficient

Dickey (1997) showed a short-term effect of a 2 per cent increase in mortality for each 10μg/m^3 increase in PM2.5. That is, a unit increase (1μg/m^3) in PM2.5 will increase mortality by 0.2 per cent. Earlier, Dockery *et al.* (1993) included socio-economic factors such as smoking, body mass and education in their study of six US cities and found that an increase of 10μg/m^3 of annual PM2.5 increases death rates by 14 per cent, compared to only 1 per cent increase for a 10μg/m^3 increase in daily PM10. In a larger study, Pope *et al.* (1995) associated air pollution data between 1979 and 1983 to death rates from 1983 to 1989. The study found a 7 per cent increase in mortality rate for a 10μg/m^3 increase in PM2.5. Schwartz *et al.* (1996) found that a 10μg/m^3 increase in two-day mean PM2.5 was associated with a 1.5 per cent increase in total daily mortality. Robinson *et al.* (1998), analysing the Dockery *et al.* and Pope *et al.* studies, suggested using the average of the two studies, a 1 per cent increase in mortality for an additional 1μg/m^3 of PM2.5 pollution.

Based on the above literature, a lower bound of 0.2 and an upper bound of 1 per cent increase in mortality rate for a unit increase in PM2.5 were used for the present study.

The relationship between mortality and air pollution can be written as follows:

Per cent change in mortality = b * change in PM2.5

The absolute change in mortality then can be written as:

Absolute change in mortality = b * ΔPM2.5 * CMR * exposed population * 1/100

where b is the dose response relationship,

ΔPM2.5 is the change in PM2.5

CMR the crude mortality rate, and

the factor 1/100 converts percentages to absolute numbers.

4.2.2 Morbidity Coefficient

Studies of air pollution episodes have confirmed an association between particulate air pollution and morbidity effects (Brunekreff *et al.* 1995). The economic value of many health effects cannot as yet be estimated, often because of the unknown dose-response relationship between PM2.5 exposure and morbidity effects. Some studies have measured mainly pulmonary function and are not appropriate for valuation purpose.

Estimates of morbidity using restricted activities days (RAD) was considered most appropriate for the present study. RAD include days spent in bed, days missed from work, and other days when activities are significantly restricted due to illness. Ostro (1987) examined the relationship between adult RAD in a two-week period and fine particles (PM2.5) in the same two-week period from 47 metropolitan areas in the United States. RAD data came from the National Centre for Health Statistics' Health Interview surveys (taken annually). Fine particle data were estimated from airport data in each area. Separate regression estimates were obtained for six years, 1976-1981. A statistically significant relationship between fine particles and RAD was found in each year, supporting earlier findings. Ostro (1994, Jakarta) selected the average of the six coefficients to calculate a central, and derived upper and lower estimates from the range in the coefficients over the

six-years. The form of the estimated relationship was such that the coefficient for fine particles gave the percentage change in RAD associated with unit change in fine particles. Specifically, the result indicates the following relationship between RAD and fine particles:

> Change in RAD per adult per year = b * annual RAD * change in fine particles (PM2.5)

The estimates for b were 0.0076 (high), 0.0048 (central), and 0.0034 (low). The central estimate shows that a 0.48 per cent change in annual RAD is associated with a $1\mu g/m^3$ change in PM2.5.

Ostro's summary was used to calculate health benefits for Jakarta (Ostro 1994), for Turkey (Zaim 1997), for the UK (Pearce and Crowards 1996) and also for Chile (Eskeland 1997). The dose-response coefficient derived by Ostro (1994) will be used to assess the RAD effects of particulate pollution in Armidale.

4.2.3 Problems of Dose-Response Function

Dose-response functions (DRF) are mainly derived from data linking changes in PM2.5 concentrations to health effects in US cities. Transferring these DRF to calculate health effects of particulate air pollution in Armidale relies on a number of assumptions relating to the validity of the epidemiology underlying the DRFs and the validity of transferring the results.

According to Pearce and Crowards (1996), the main critical factors appear to be:

i. Threshold: there is no threshold below which PM2.5 concentrations are harmless and not a cause of mortality.
ii. Linearity: the dose-response function is linear, so that the relationship is the same at all levels of particulate pollution.
iii. Transferability: the conditions in US cities and measurements taken there are comparable to Australian cities and towns.
iv. Biological Pathways: there is some *biological pathway* by which particulate matter affects health.

v. Confounding factors: the possible presence of confounding factors does not alter the relationship.

Thresholds

Current evidence does not support the existence of threshold levels and the World Health Organisation has "confirmed" that there are no safe levels for particulate air pollution (WHO 1995). Schwartz (1994d) found no support for thresholds in his Philadelphia study. Earlier, Desvousges *et al.* (1993) assumed the absence of thresholds because the linear and exponential functional forms generally used by researchers showed no marked attenuation of effects at low levels. Neither Ostro's (1984) work on 14 London winters nor Baruch's (1998) Australian study found safe levels of pollution.

Significantly, studies have reported a significant effect of particulate pollution on mortality even where levels were lower than USEPA or WHO standards (Ponka *et al.* 1998; Schwartz 1994d; Desvousges *et al.*1993; Dockery *et al.* 1993). These standards are thus not expected to offer any realistic approximation to a threshold.

Linearity

If the concentration-to-mortality relationship was nonlinear, it would be difficult to transform the dose-response relationship from one level of particulate air pollution to another. Dockery *et al.* (1993) hinted that the relationship was more or less linear. Morgan *et al.* (1998), following on from his Sydney study, indicated the existence, even if at low levels, of a linear does-response relationship between particulates and daily mortality.

Transferability

Within a locality, the statistical relationships between daily mortality in a specified area and daily concentrations of particulate pollution monitored at several outside locations provide an estimate of dose-response relationships. Such results unreservedly assume a similarity in the distribution of baseline factors such as health status, occupational exposure and time spent in outdoor activities. These factors have be considered when undertaking similar studies in other areas.

The demographic characteristics of the population, their lifestyle and housing features that affect their vulnerability to air pollutant effects may also influence the relationship between mortality-morbidity and air pollution concentrations (Chesnut 1997). Thus, the examination of health profiles of the population, lifestyle, housing and other demographic characteristics will be useful in any study of a similar nature.

This study uses DRF obtained from relatively affluent, industrialised cities in primarily temperate climate zones whose demographic characteristics, lifestyle and health status match those in Australia. While Australia has a relatively warmer climate, Morgan *et al.* (1998) suggested that the extent of association and the linear dose-response relationship between particles and daily mortality in Australia (Sydney) were comparable to results reported internationally. Another factor to consider, however, is the difference in pollution sources between the industrialised cities and Armidale, a country town. This factor reduces transferability. Yet, previous studies on health effects of particulates have confirmed consistent relationships across various sources of particulate pollution.

Biological Pathways

Uncertainties exist about the biological pathway for particulate matter affecting human health. Reviewing various epidemiological studies, Utell and Samet (1993) argued that the biological pathway was unknown, but Seaton *et al.* (1995) suggested the possibility of particulate clouds containing acidic particles travelling and persisting in time in human bodies. Alveolar inflammation may be caused by small particles and may affect and aggravate lung disease and coagulability of blood. The condition of people affected by respiratory problems may worsen due to exposure to pollution. Available evidence 'is consistent with the requirements of tests for causality: the relationship is consistent, specific, produces a dose-response relationship and fits other known facts' (Ostro, reported in Pearce and Crowards 1996, p 613).

Confounding Factors

The extent to which DRF adequately control all variables affecting health status is critical to understanding the problem. Apart from such factors as smoking and diet, "social status" may serve as an important factor in determining ill health. It may be that exposure to particulate pollution is correlated with social status and illness. Such an assumption, however, needs

precise testing with panel data embracing income, other measures of social status, and indoor and outdoor pollution concentrations. Time series studies may help avoid such confounding factors because demographic characteristics remain fairly constant over time (Ostro, in Pearce and Crowards 1996).

4.3 The Exposed Population

The next consideration for the present study is the estimation of the exposed population. For certain pollution-related health effects this may include the entire exposed population; for other effects there may be particularly sensitive sub-groups such as children or asthmatics.

Repace *et al.* (1980) stated that there are two possible methods for estimating the exposure of a person (or a population) to air pollution:

i. Field studies, using personal air pollution monitoring devices to measure an individual's exposure (or the exposure of a sufficiently large population sample); and
ii. Mathematical models, which consider both pollution level at different spatial points and the activities of persons over time to estimate the exposure. To estimate air pollution exposure, the model should be capable of describing the interactions between pollutant concentrations and the activity patterns of an individual or a group of individuals in space and time (WHO 1982).

Many studies (e.g. Ostro 1994, Pearce and Crowards 1996, Zaim 1997) used the total population of the study area as the exposed population. On the other hand Hall *et al.* (1992, 1994), developed a regional human exposure model, known as the REHEX II model to estimate the population's exposure to concentrations above the California standard. This model accounts for the spatial and temporal pollution patterns across the region, and the amount of time that different groups in the population spend indoors, outdoors or in transit.

In the context of Armidale, it is appropriate to consider the total population as the exposed population. The rationale behind this is that in Armidale, particulate levels build up from late afternoon, and peak between 11 p.m. to 2 a.m. Particles levels then fall to almost zero by 10 a.m. the next day (Roberts and Lin 1998). Day time exposures, when most people are

not at home and pollutant levels generally lower, may not be a significant consideration for Armidale compared to night-time exposures, since most people stay inside during the highly polluted time (late night).

The main source of particulate pollution in Armidale is residential wood burning. Particles resulting from the combustion process are generally below PM2.5. These fine particles readily infiltrate residential buildings with indoor levels similar to levels immediately outside the structure (Brauer *et al.* 1989, Hall *et al.* 1994). In Armidale, the location of the residence is probably the most important factor in calculating the exposed population.

Average annual population exposure is available from two sources: NSW Environmental Protection Authority (EPA) / Armidale Dumaresq Council (ADC) and the Armidale Air Quality Group (AAQG). According to EPA/ADC nephelometer readings, the average annual mean exposure was neph coefficient 0.5756 with a maximum mean annual exposure of 1.819 for the year 1998. In terms of PM2.5, the average annual exposure of particulate air pollution is around 11.5$\mu g/m^3$ PM2.5.

Using a portable radiance research nephelometer, AAQG estimated the population exposure for Armidale at 20.4$\mu g/m^3$ from April to September, 5.6$\mu g/m^3$ in October and assumed no particulate pollution from November to March. Their average annual exposure is thus 10.67$\mu g/m^3$ PM2.5 (details in chapter 3). Hence the AAQG estimate is close to that of the EPA/ADC. Although it therefore makes little difference, which is selected, to be conservative, the lower of the two is used in the remainder of this study.

4.4 Economic Valuation of Health

To obtain a valuation for health effects, results from the epidemiological literature, which link air pollution to illness, were merged with the results from the economic literature, which place a value on mortality and morbidity.

Economists use two approaches to find a dollar value for a premature death. The first, the human capital approach, is based on measuring the economic productivity of the individual at risk. The second is based on an individual's willingness to pay to reduce his/her risk of

death. It assumes that individuals treat longevity as any other good and that it is possible to estimate the value they place on life expectancy by looking at the trade offs they make between reductions in the risk of death and other goods whose value can be measured in mortality terms (Chapter 2).

Fisher *et al.* (1989), Miller (1989), and Viscusi (1992, 1993) compiled data on recent empirical studies of valuation of a statistical life. Fisher *et al.* (1989) presented a range of values from US$1.6 million to US$8.5 million per life, but placed more confidence in the lower bound. Miller (1989) suggested a value between US$1.4 and US$4.3 million (in 1994 dollars). Viscusi's (1993) values ranged from US$3.2 million to US$7.5 million. Chestnut (1995) summarised the VSL estimates from these studies (Table 4.1). The Consumer Product Safety Commission US currently use Viscusi's mid point value of $US 5 million for each life saved (Miller *et al.* 1997) Hall *et al.* (1994) used US$3.1, US$4.2 and US$9.5 million as low, middle and upper values.

Table 4.1: Empirical Value of a Statistical Life

Researcher	**Value of a statistical life in US$ million (in 1994 dollars)**	
	Lower	**Upper**
Fisher *et al.* (1989)	2	11
Miller *et al.* (1989)	1	4
Cropper and Freeman (1991)	2	7
Viscusi (1992)	3	8
Hall *et al.* (1994)	3	9

Premature deaths tend to be concentrated among the elderly. Society may not be willing to pay as much to prevent a premature death among the elderly as it would among young workers. The underlying issue is: how long would life be extended in the absence of air pollution? In some cases, the extension of life may be short, but in others it could be considerable. For example, given appropriate rehabilitation, sufferers of cardiac diseases typically survive many years. Therefore US$4.0 million as an upper bound and US$1.0 million as a lower bound appear reasonable estimates of the representative value of a life.

In contrast to studies to estimate the value of averted death, few studies have examined how much people are willing to pay for a reduction in morbidity symptoms.

Although the average lost wage for one day has been used for the restricted activities day, the willingness to pay function was nonlinear in the number of days for which the symptoms were avoided (Loehman *et al.* 1979).

4.5 Results and Discussion

Using 10.67μg/m^3 for the average PM2.5 concentration and total deaths of 177 persons in Armidale (based on a mortality rate of 8.3 per 1000 and total population 21,331), and mortality dose-response functions of 0.2 and 1 per cent for each 1μg/m^3 change in PM2.5 as lower and upper bounds, the total number of deaths in Armidale attributable to PM2.5 exposure could be calculated using the formula:

$$\Delta H_{MT} = b * \Delta PM2.5 * CMR * POP * 1/100$$

Using the lower bound, the estimate is:

$$\Delta H_{MT} = 0.2*10.67*177*1/100$$
$$= 3.77 \approx 4$$

And using the upper bound:

$$\Delta H_{MT} = 1*10.67*177*1/100$$
$$= 18.88 \approx 19$$

The lower and upper bounds, using coefficients of 0.2 and 1.0, are 4 and 19 deaths respectively per year due to particulate air pollution in Armidale.

The above results are comparable with findings of Robinson (1998, 2001). Using a population exposure of 8.5μg/m^3 for Armidale and 1 per cent increase in mortality for each additional annual 1μg/m^3 of PM2.5, Robinson (1988) estimated that 8.5 per cent of deaths occurred due to air pollution. Later, using a population exposure of 10μg/m^3, Robinson

(2001) estimated the overall mortality impact for Armidale from woodsmoke pollution as 10 per cent. Robinson's values in 1998 and 2001 translate into 15 and 18 deaths respectively. Both numbers are within the range established by the present study.

In terms of monetary values (Table 4.1), US$1 million and US$4.0 million could be assumed as lower and upper values, which will be around A$1.6 million and A$6.4 million (exchange rate US$1=A$1.6). Using the upper and the lower estimates for both mortality coefficient and value of life, the following estimates are obtained.

For upper bounds of mortality estimates of 19 deaths per year, the economic costs are between A$30.4 million (lower bound) and A$121.6 million (upper bound).

For the lower bound of mortality estimates of 4 deaths per year, the economic cost are between A$6.4 million (lower bound) and A$25.6 million (upper bound).

For calculating morbidity effects, restricted activity days (RAD) are first estimated.

$$\Delta Hi = b * \Delta PM2.5 * POP_i * 1/100$$

where ΔHi is the change in population risk of RAD,
b is the slope of the dose-response curve,
POP_i is the population at risks of health effect i,
$\Delta PM2.5$ is the change in air pollution under consideration, and
the factor 1/100 converts percentages to absolute numbers.

Therefore, the upper bound of the RAD coefficient produces

$$\Delta Hi = 0.76*10.67*21{,}330*1/100$$
$$= 1729.69 \text{ person days}$$

The central estimate (0.48) gives 1092.44 person days and the lower estimate (0.34) 773.80 person days of RAD due to particulate air pollution.

The value of a RAD is based on daily wage rates. While not everyone who experiences RAD will miss a day of work, wage rate is considered to reflect the average value of time to this population. Some of those affected will be children who do not earn wages. Some who

would otherwise work, or use the day for other purposes, must forgo other activities to take care of an ill child. Consequently, all RAD are valued at the same rate.

The direct annual loss of output caused by particulate air pollution is now computed by multiplying the estimated days lost due to sickness by the average daily wage in Australia, which is $122.4 per day (ABS 2000). Therefore, the cost for RAD lies in the range $94,713 (773.8*$122.4) to $211,715 (1729.7*122.4) per year.

The economic cost of the current levels of particulate air pollution in Armidale, with the very conservative estimate (lower bounds) of the total mortality and morbidity effects (RAD), will be in the order of $6.5 million ($6.4million + $94,713) to $25.7 million ($25.6 million + $94,713) per year.

Particulate pollution has a dominating impact on the total economic cost. However, the results should be viewed as a general estimate only because of the uncertainties and assumptions that exists for both public health and economic values related to air pollution. The uncertainties associated with the results include uncertainty surrounding the transferability, measurement of the population at risk and uncertainties about the unit value of a statistical life. In recognition of these uncertainties, estimates of the upper and lower coefficient 'b' provide the range within which actual health effects are likely to fall. However, in the absence of DRF for many health effects of PM2.5, these estimates could underestimate the true damage to human health significantly.

The above estimates could be viewed as conservative since an individual's actions, the "cost of illness", and the cost of "pain and suffering" have not been included. Furthermore, air pollution is usually associated with other, non-health-related effects including aesthetics, quality of life, material damage, etc, which have not been considered. Consequently, these estimates probably understate the true social costs of health damage due to particulate air pollution in Armidale.

CHAPTER 5
PARTICULATE AIR POLLUTION AND RESPIRATORY SYMPTOMS IN ARMIDALE: CLINICAL EVENT STUDY

5.1 Introduction

Epidemiological studies have consistently shown a relationship between particulate air pollution and exacerbation of illness in people with respiratory disease. Morbidity studies measuring the effects of air pollution on respiratory illness and asthma are recognised as a valid measure of the impact of air pollution in Australia (Voigt *et al.* 1998; Rennick and Jarman 1992; Jalaludin 1996). The survey of clinical events presented in this chapter explores the impact of particulate air pollution on the proportion of respiratory visits to general practitioner (GP) clinics in Armidale.

Armidale was selected as an ideal place to examine the potential adverse health effects of woodsmoke, since air pollution in the town is mostly generated by emissions from wood heaters. Low temperatures in winter, together with relatively cheap wood fuel, encourage people to burn wood for space heating. As much as two tonnes of particulate can be emitted into the air over Armidale on a very cold night (ADC 1995, Wall 1995). Incomplete combustion results in increased quantities of pollutants, which become trapped in Armidale's frequent temperature inversions (see Chapter 4).

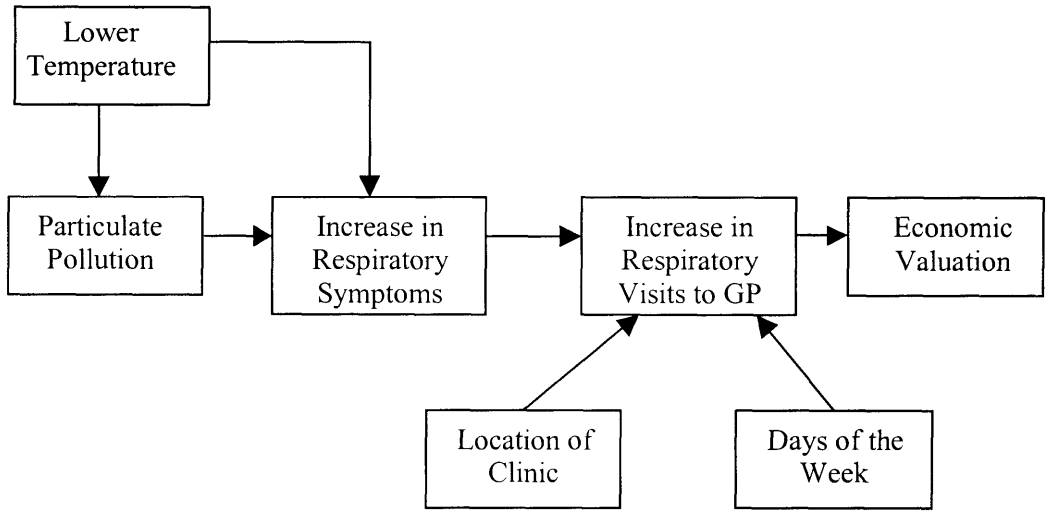
Ponka *et al.* (1991) and Pope (1989, 1991) used data on hospital admissions and emergency room visits to examine respiratory morbidity. However, hospital data require a large population to identify relationships between air pollution and respiratory illness. As Armidale is only a small town (population 21,300) it seemed appropriate to use local GP data to identify such a relationship.

There are both direct and indirect links between lower temperatures and increases in respiratory illnesses. Temperature certainly plays a primary role in the generation of particulate air pollution, since low temperatures encourage people to light up their wood

heaters, leading to increased particulate pollution. Lower temperatures may also directly trigger an increase in respiratory illness or asthma attacks.

The day of the week may also be important in establishing the pattern of respiratory consultations, since GP clinics are closed on weekends. Any increase in respiratory patients in GP clinics may, in part, be confounded by a "weekend effect". In particular, we could expect increases on Mondays and Fridays. Further, the location of GP clinics might have an important influence on increases in respiratory patient visits. Figure 5.1 shows the impact of temperature on particulate pollution causing physical effects and which incur economic costs.

Figure 5.1: Links and Some Key Variables



Thus, the factors: minimum temperature, particulate air pollution, location of the GP clinic and day of the week were incorporated as conditional variables in the model for examining clinical events.

Only the effect of particulate air pollution on respiratory symptoms is being examined at this stage. This chapter contains a brief discussion about the sources of data and a description of the data. These data were analysed using pair-wise correlation and bivairate analysis. A logistic regression model was then used to identify whether any relationship existed between particulate air pollution and the proportion of patients with respiratory symptoms visiting local general practitioners' clinics.

The economic cost of particulate air pollution will be estimated in Chapter 6.

5.2 Air Pollution, Weather and Respiratory Symptoms Data

5.2.1 Particulate Air Pollution and Weather Data

Particulate air pollution data for the winter of 1999 (June to August) were obtained from the Armidale Air Quality Group (AAQG), Armidale Dumaresq Council (ADC) and the NSW Environmental Protection Authority (EPA). The AAQG data were measured in the East Armidale residential area, whilst the ADC nephelometer reading was taken in the council chamber in the CBD area, a relatively chimney-free area. Usually the air pollution reading at ADC is about half the AAQG's reading (see Chapter 3). Air pollution data were collected for both the daily (24-hour) mean concentration and daily maximum (1-hour) concentration.

Weather data for Armidale were obtained from the Bureau of Meteorology-NSW Regional Office. The Bureau of Meteorology weather station is located at Armidale Airport, 1084m above sea level and at the western end of the valley. This station records minimum, maximum, dew point and average daily temperatures, wind speed, wind vector speed and wind direction at 20-minute intervals.

5.2.2 Respiratory Symptoms Data

Respiratory symptom data were collected from Armidale GP clinics. There are 26 GPs in Armidale, practising in 8 clinics in the city. The number of GPs per clinic varies from 1 to 6 with some working only part-time.

The New England Division of General Practice in Armidale facilitates communication with local GPs. Before the survey, a description was published in the Division's newsletter, to inform GPs of the study. Subsequently, a letter together with a sample form was sent to all Armidale GPs explaining the objectives of the survey, the procedures, the data requirements and the time frame.

Following this correspondence, 15 general practitioners from 6 clinics agreed to participate. Using the form, the total number of medical visits and respiratory morbidity diagnoses were collected for each working day, Monday to Friday.

GPs recorded their diagnoses observing the following classification (a) total visits (respiratory and non-respiratory) for that day; (b) respiratory visits due to acute upper respiratory symptoms; (c) respiratory visits due to acute lower respiratory symptoms; (d) respiratory visits due to chronic lower respiratory symptoms; (e) visits due to asthma; and (f) visits due to respiratory infection. For each day the number of medical visits with each diagnosis was totalled across clinics.

The recorded data reflect medical visits in general and do not discriminate between first visits and follow up visits. Consequently, the data capture the number of visits, not the number of illness episodes. The health end-points studied represent patients who were successful in obtaining a GP's attention and who were diagnosed with respiratory illness. Of course, some people do not seek treatment, some may purchase medicine from a pharmacy, and some may go to the hospital emergency clinic. Also it may not have been possible to get an appointment with a doctor, due to time limitation or capacity.

Data were collected during the Southern Hemisphere winter from 1 June to 20 August, a total 12 weeks, excluding weekends. This period was chosen because, as previously mentioned, it is the period in which wood pollution is significant in Armidale.

5.3 Description of Data

Data obtained in this survey were initially analysed using the Statistical Package for Social Science (SPSS). Univariate statistics, including frequency distribution, mean and standard deviation were used to report descriptive finding of the variables. To examine the relationship between the dependent and independent variable, bivariate analysis and the correlation coefficient were used. In the study period, the total number of patient visits was 9,481 of whom 1,370 presented with respiratory illness, or 14.4 per cent of total visits. Table 5.1 provides the general descriptive statistics during the study sample period.

Table 5.1: Summary Statistics: Air Pollution and Temperature Data

1 June to 20 August, 1999

	Mean	Std. deviation	Minimum	Maximum
Daily Minimum Temperature	0.98	4.48	-8.00	9.00
Daily average particulate pollution (AAQG) PM2.5	31.82	31.39	1.71	140.46
Daily average particulate pollution (ADC) PM2.5	13.93	12.14	1.26	44.82
Daily maximum particulate pollution (AAQG) PM2.5	96.18	83.76	3.17	325.94
Daily maximum particulate pollution (ADC) PM2.5	46.08	44.50	2.25	186.75
Daily average particulate pollution (ADC +AAQG) PM2.5	22.90	21.21	1.98	86.77
Daily maximum particulate pollution (ADC +AAQG) PM2.5	21.13	61.32	3.17	215.91

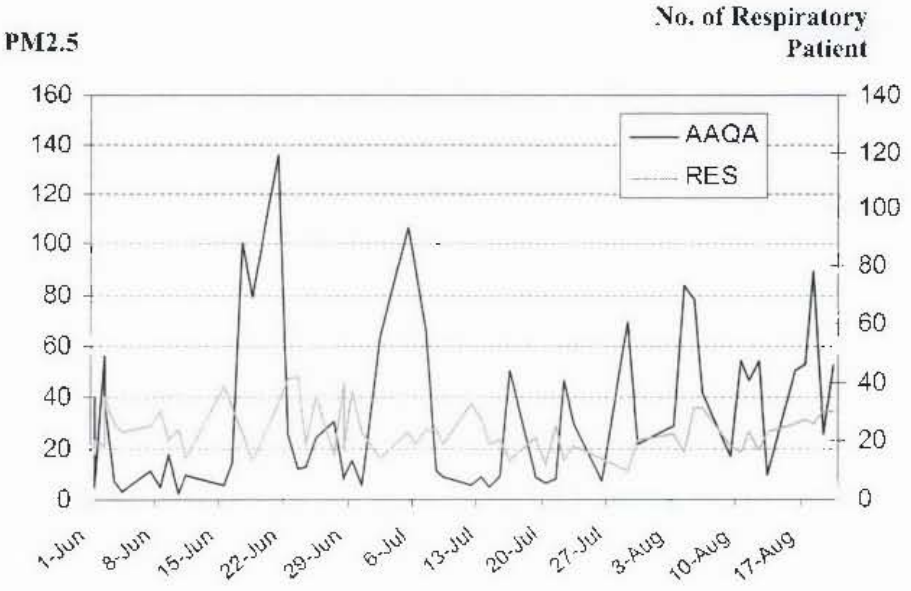
AAQG= Armidale Air Quality Group, ADC= Armidale Dumaresq Council

5.4 Preliminary Analysis

To observe the time-series behaviour of particulate air pollution and number of respiratory visits, the sequence of data from June 1999 to August 1999, was plotted in Figure 5.2.

Reasonably complete daily data on particulate air pollution and weather were available during this study period. Table 5.2 contains correlation coefficients for selected variables.

Figure 5.2: Particulate Air Pollution and Respiratory Visits Data



Particulate air pollution was negatively related to temperature, which supports the proposition that the wood used for heating was an important source. Also there was a fairly strong correlation between Armidale Air Quality Group and Armidale Dumaresq Council data.

Table 5.2: Coefficients of Correlation of Selected Variables

	Minimum Temperature	Maximum AAQG	Average AAQG	Maximum ADC	Average ADC
Minimum Temperature	1.00	-0.669(.000)	-0.619(.000)	-0.516(.000)	-0.578 (.000)
Max. AAQG		1.00	0.955(.000)	0.788(.000)	0.884 (.000)
Ave. AAQG			1.00	0.705(.000)	0.848 (.000)
Max. ADC				1.0	0.982(.000)
Ave. ADC					1.00

* Figure in parentheses indicates p values

The pair-wise Pearson correlation coefficient was calculated for both the total number of respiratory patients and air pollution and the proportion of respiratory patients and air pollution. Pollution data included same day, 1-day lag, 2-day lag, 3-day lag and 4-day lag. Only particulate pollution 2-day lagged was consistently associated with both proportion of respiratory visits and total number of respiratory visits to the local GP clinics. Table 5.3 shows the correlation between respiratory visits and air pollution.

Table 5.3: Correlation Between Respiratory Visits and Air Pollution

	Proportion of respiratory visits	Total number of visits
Ave.AAQG-2-day lag	0.227 (.000)	0.252 (.000)
Max.AAQG, 2-day lag	0.192 (.001)	0.222 (.000)
Ave.ADC, 2-day lag	0.208 (.000)	0.207 (.000)
Max.ADC, 2-day lag	0.163 (.005)	0.142 (.015)
Ave.(AAQG+ADC), 2-day lag	0.299 (.000)	0.248 (.000)
Max.(AAQG+ADC), 2-day lag	0.191 (.001)	0.206 (.000)

* Figure in parentheses indicates p values

Total respiratory visits significantly correlate with 2-day lagged air pollution levels. The proportion of respiratory visits correlate with 2-day lagged particulate air pollution.

Data were also analysed with bivariate (cross-tabulation) analysis. For this analysis, particulate air pollution was categorised according to the Armidale Air Pollution Index (Chapter 3) as low, medium, high, very high and extreme.

Using contingency table analysis, a statistically significant relationship was found between categorised air pollution and all respiratory visits. A strong relationship was found with 2-day lagged air pollution, Chi-square = 17.101, p=0. 001 (the contingency coefficient 0.041 with significance level 0.001). 3-Day lagged categorical air pollution was related to respiratory visits (Chi-square = 15.755 and p = 0.001; contingency coefficient = 0.041, p = 0.001).

Respiratory illness was also analysed in conjunction with day of the week. The calculated result was not statistically significant (Chi-square = 3.997, p = 0.5550).

Location of the clinic had a statically significant association with respiratory visits (Contingency coefficient = 0.105, p = 0.000).

Asthma consultations were statistically related to air pollution levels with a 3 day-lag (Chi-square = 13.557, p = 0.004; Contingency coefficient = 0.038, p = 0.004).

5.5 Particulate Air pollution and Respiratory Clinic Visits: Analysis of Proportions

Data from the clinical events were analysed using a statistical model and the results are presented in the following pages. The main emphasis of the analysis was to investigate the impact of particulate pollution on the proportion of respiratory visits to local GP clinics. The model seeks to explain the proportion of patients with respiratory symptoms in terms of particulate air pollution, together with conditioning variables, daily minimum temperature, location of the clinic and day of the week. Other information on patient demographics (age, gender, socio-economic background,etc.) would have been highly desirable, but it was considered too demanding to ask the GPs to provide.

Since there are multiple observations for the same values of independent variables, we analyse proportions rather than individual responses, using a modification of logistic regression. This is standard in social sciences and econometrics. However, for expository purposes, it will be developed from first principles.

5.5.1 Modified Logistic Regression Model

Let p_i, π_i and n_i be the observed proportion, the unobserved "true" proportion and the number of people in the sample, respectively, for group "i". The fundamental result, which forms the basis for using p_i to make inferences about π_i is

$$p_i \sim N(\pi_i , \ \pi_i (1 - \pi_i)/n_i) \tag{5.1}$$

Equivalently, we may write,

$$p_i = \pi_i + \varepsilon_i \tag{5.2}$$

where ε_i is a random variable which is normally distributed, with

$$E (\varepsilon_i) = 0 \tag{5.3}$$

and

$$Var(\varepsilon_i) = \pi_i (1 - \pi_i)/n_i \tag{5.4}$$

As the variance of ε_i depends on the characteristics π_i and n_i of the i^{th} group, it follows that ε_i is heteroskedastic.

The basic assumption, which underlies the model we will use, is that π_i is determined by K observable "conditioning variables", such as level of air pollution, clinic attended, day of the week, etc.

Denoting the values of the conditioning variables which characterise the i^{th} group as X_{1i}, X_{2i}, ... ,X_{Ki} we assume that π_i depends on a linear combination $X_i'\beta$ of the Xs, given by

$$X_i'\beta = \beta_0 + \beta_1 X_{1i} + \beta_2 X_{2i} + \ldots + \beta_K X_{Ki}$$

where

$$X_i' = (1, X_{1i}, X_{2i}, \ldots X_{Ki})$$

and

$$\beta = (\beta_0, \beta_1, \beta_2, \ldots, \beta_K)'$$

β is an unknown vector that has to be estimated. Thus, we will assume that

$$\pi_i = F(X_i'\beta) \qquad (5.5)$$

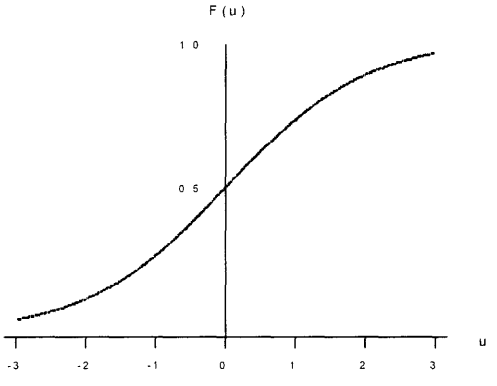
where, F(.) is some (unspecified) function.

Because the proportion π_i has the property that $0 \leq \pi_i \leq 1$, the function F(.) must have the characteristic that it takes any value of $X_i'\beta$ and produces a number in the range (0,1). The form of F(.) that we will use is known as the "logistic function", defined as

$$F(u) = \frac{\exp(u)}{1+\exp(u)} \qquad (5.6)$$

and illustrated in Figure 5.3.

Figure 5.3: The Logistic Function



Combining (5.2), (5.5) and (5.6) we have the regression model

$$p_i = \frac{\exp(X_i'\beta)}{(1+\exp(X_i'\beta))} + \varepsilon_i \qquad (5.7)$$

This model is nonlinear in the unknown parameters β and may be estimated by nonlinear least squares, after adjusting for the heteroskedasticity of ε_i, given by (5.4).

However, using Taylor's expansion (Greene 1997) (5.7) can be approximated by the *linear* model

$$y_i = X_i'\beta + \varepsilon_i \qquad (5.8)$$

where

$$y_i = \ln(p_i/(1-p_i)) \qquad (5.9)$$

The unknown parameters β are estimated by a three-step procedure as follows:

Step 1: Estimate (5.8) by ordinary least squares and obtain the model-based predictions of y_i $(i = 1,2,3,\ldots, N)$ denoted by $\hat{y}_i$.

Step 2: Defining

$\hat{\pi}_i = \exp(\hat{y}_i)/(1+\exp(\hat{y}_i))$

and the weights

$w_i = [n_i\hat{\pi}_i(1-\hat{\pi}_i)]^{1/2}$,

the variables y_i and X_i are weighted by w_i to obtain y_i^* and X_i^*.

Step 3: The unknown parameters β are then estimated by regressing y_i^* on X_i^*.

Once the unknown parameters have been estimated, the model may be used for three main purposes:

1. To test whether the coefficients of pollution are significantly different from zero. This is a test of the causal effect of pollution on respiratory symptoms.
2. Given knowledge of the values of the conditioning variables, the proportion of people suffering respiratory complaints can be predicted. Specifically,

$$\hat{\pi}_i = \frac{\exp(X_i'\hat{\beta})}{[1+\exp(X_i\hat{\beta})]}, \qquad (5.10)$$

where $\hat{\beta}$ is the estimate of β, and $\hat{\pi}_i$ the prediction of π_i.

3. The increase in the proportion π_i of patients with respiratory illnesses due to a unit increase in air pollution can be estimated. It can be shown that if $\hat{\beta}_1$ is the estimated coefficient of air pollution,

$$\frac{\partial \hat{\pi}_i}{\partial(\text{APP})} = \hat{\beta}_1 \hat{\pi}_i (1 - \hat{\pi}_i) \qquad (5.11)$$

where APP is the measure of air pollution.

In all, six models were examined. The models differed only according to the explanatory variable used to measure particulate air pollution. The models in terms of the measures of particulates are defined as follows:

Table 5.4: Measurement of Air Pollution for Different Models

Model	Air Pollution Variables
1	Average daily air pollution from AAQG
2	Average daily air pollution from ADC/EPA
3	Mean average daily air pollution AAQG+ADC/EPA
4	Maximum daily air pollution AAGQ
5	Maximum daily air pollution ADC/EPA
6	Mean maximum daily air pollution AAGQ+ADC/EPA

Detailed definitions of all variables are given in Table 5.5.

Table 5.5: Definitions of Variables

Variables	Description
Y	Proportion of respiratory visits to GP's clinic
N	Total number of patients
Temp	Minimum temperature (same day)
Temp1	Minimum temperature (1-day lag)
Temp2	Minimum temperature (2-day lag)
Temp3	Minimum temperature (3-day lag)
Temp4	Minimum temperature (4-day lag)
AP	Air pollution PM2.5 (AAQG, average, same day)
AP1	Air pollution PM2.5 (AAQG, , average ,1-day lag)
AP2	Air pollution PM2.5 (AAQG, , average 2-day lag)
AP3	Air pollution PM2.5 (AAQG, , average,3-day lag)
AP4	Air pollution PM2.5 (AAQG, , average 4-day lag)
ADCAPM	Air pollution PM2.5 (ADC, average same day)
ADCAPM1	Air pollution PM2.5 (ADC, average, 1-day lag)
ADCAPM2	Air pollution PM2.5 (ADC, average, 2-day lag)
ADCAPM3	Air pollution PM2.5 (ADC, average, 3-day lag)
ADCAPM4	Air pollution PM2.5 (ADC, average, 4-day lag)
AVEAIRP	Air pollution PM2.5 (AAQG+ADC, average, same day)
AVEAIR1	Air pollution PM2.5 (AAQG+ADC, average, 1-day lag)
AVEAIR2	Air pollution PM2.5 (AAQG+ADC, average, 2-day lag)
AVEAIR3	Air pollution PM2.5 (AAQG+ADC, average, 3-day lag)
AVEAIR4	Air pollution PM2.5 (AAQG+ADC, average, 4-day lag)
MAXAAQ	Air pollution PM2.5 (AAQG, daily maximum, same day)
MAXAAQ1	Air pollution PM2.5 (AAQG, daily maximum, 1-day lag)
MAXAAQ2	Air pollution PM2.5 (AAQG, daily maximum, 2-day lag)
MAXAAQ3	Air pollution PM2.5 (AAQG, daily maximum, 3-day lag)
MAXAAQ4	Air pollution PM2.5 (AAQG, daily maximum, 4-day lag)
MAXADC	Air pollution PM2.5 (ADC, daily maximum, same day)
MAXADC1	Air pollution PM2.5 (ADC, daily maximum, 1-day lag)
MAXADC2	Air pollution PM2.5 (ADC, daily maximum, 2-day lag)
MAXADC 3	Air pollution PM2.5 (ADC, daily maximum, 3-day lag)
MAXADC 4	Air pollution PM2.5 (ADC, daily maximum, 4-day lag)
MAX	Air pollution PM2.5 (AAQG+ADC, daily maximum, same day)
MAX1	Air pollution PM2.5 (AAQG+ADC, daily maximum, 1-day lag)
MAX2	Air pollution PM2.5 (AAQG+ADC, daily maximum, 2-day lag)
MAX 3	Air pollution PM2.5 (AAQG+ADC, daily maximum, 3-day lag)
MAX 4	Air pollution PM2.5 (AAQG+ADC, daily maximum, 4-day lag)
C1	1 if clinic 1; 0 otherwise
C2	1 if clinic 2; 0 otherwise
C3	1 if clinic 3; 0 otherwise
C4	1 if clinic 4; 0 otherwise
C5	1 if clinic 5; 0 otherwise
D1	1 if Monday; 0 otherwise
D2	1 if Tuesday; 0 otherwise
D3	1 if Wednesday; 0 otherwise
D4	1 if Thursday; 0 otherwise

5.5.2 Results

The results were consistent across all models. For this reason, a description is given only of the estimation and final form arising from Model 3. This model used mean average daily air pollution data from Armidale Air Quality Group and Armidale Dumaresq Council.

The regressor variables fall into four groups, as follows:

i. A group of variables to determine the effect of particulate air pollution, both contemporaneous and lagged up to four days;
ii. Variables to capture the effect of temperature;
iii. Variables to capture the "clinic effect"; and
iv. Variables to capture the "day of the week" effect.

The variables in iii and iv were dummy variables. The initial regression, which included all the above variables, indicated that the day of the week effects were not significant. A formal test of the coefficients of the day of the week variables confirmed this.

A second regression was then estimated, omitting these variables. This was the final form of the model. The temperature variables, though only one was significant, were retained for the theoretical reasons discussed previously. Because of the strong correlation between temperature and air pollution (Table 5.2), incorrect omission of these variables could result in the coefficients of air pollution being biased. Also, inclusion of temperature variables enables us to separate the pollution effect from the confounding temperature effect.

The results from the estimated model show that particulate air pollution affects the proportion of respiratory patients with a two-day delay. The p-value 0.017 associated with this coefficient is statistically significant at the 5 per cent level.

Model 3 results are given in Table 5.6; the detailed results of other models are in Appendix A.

Table 5.6: Results from Model 3

Mean Average Daily Air Pollution, AAQG+ADC/EPA

Variables	Estimated Coefficient	Standard Error	T-Ratio	P-Value
Temp	0.89236E-02	0.1586E-01	0.5626	0.574
Temp1	-0.77054E-02	0.1513E-01	-0.5091	0.611
Temp2	-0.12994E-01	0.1545E-01	-0.8408	0.401
Temp3	0.26901E-01	0.1280E-01	2.101	0.037
Temp4	-0.98493E-02	0.1230E-01	-0.8007	0.424
AP	-0.10145E-02	0.2283E-02	-0.4444	0.657
AP1	-0.56048E-03	0.2678E-02	-0.2093	0.834
AP2	0.66414E-02	0.2757E-02	2.409	0.017
AP3	0.17781E-02	0.2922E-02	0.6085	0.543
AP4	0.28869E-02	0.2653E-02	1.088	0.277
C1	-0.39537	0.1159	-3.410	0.001
C2	-0.83328	0.1365	-6.105	0.000
C3	-0.12127	0.1203	-1.008	0.314
C4	-0.18553E-01	0.1542	-0.1203	0.904
C5	-0.21845	0.1747	-1.250	0.212
Constant	-1.7558	0.1602	-10.96	0.000

As mentioned earlier, these results could be used for three main purposes:

1. We see that the coefficient of AP2 is positive and statistically significant. This supports the proposition that air pollution is a causal factor in respiratory illnesses. Furthermore, as particulate pollution increases, the proportion of respiratory patients increases. This is the key result.

2. From equation (5.10)

$$\hat{\pi}_i = \frac{\exp(X_i'\hat{\beta})}{[1+\exp(X_i\hat{\beta})]},$$

for any values of the regressors, the proportion of respiratory patients can be estimated. For example, if temperature and air pollution are set at average values, we obtain

$\hat{\pi}_i = 0.1809$ for the base clinic

$\hat{\pi}_i = 0.1295$ for clinic 1

$\hat{\pi}_i = 0.0880$ for clinic 2

$\hat{\pi}_i = 0.16364$ for clinic 3

$\hat{\pi}_i = 0.1782$ for clinic 4

$\hat{\pi}_i = 0.15077$ for clinic 5

$\hat{\pi}_i = 0.14844$ (average proportion)

Thus, at average values of minimum temperature and air pollution, about 14.8 per cent of all patients had respiratory illness. This estimate agrees with the proportion 14.4% of all visits, noted earlier.

3. From equation (5.10)

$$\frac{\partial \hat{\pi}_i}{\partial(\text{APP})} = \hat{\beta}_1 \hat{\pi}_i (1 - \hat{\pi}_i)$$

At average values for air pollution and temperature, π_i will increase by 0.0008326 for a one unit increase in air pollution. That is, there will be an increase in the proportion of respiratory patients by about 0.08 per cent in response to a 1 $\mu g/m^3$ increase in air pollution. Given that the difference between the mean and maximum levels of air pollution is 64 $\mu g/m^3$, the proportion of respiratory patients would increase from 14.8 per cent to 20 per cent following this increase. However, it is also worthwhile to look at the increase in the proportion of the respiratory patients for a one unit standard deviation increase in air pollution. From Table 5.1, the standard deviation is 21.21 units of air pollution; we thus multiply the right hand side of the equation (5.1) by 21.21. Then at average values, and for an increase of one standard deviation in the value of air pollution, we obtain

$$\frac{\partial \hat{\pi}_i}{\partial(\text{APP})} = 0.0177$$

That is, if the air pollution increases by one standard deviation, the proportion of respiratory patients will increase by about 1.8 per cent, from 14.8% to 16.6%.

Alternatively, the response to an increase in air pollution could be expressed as an elasticity.

$$\text{Elasticity} = \frac{\text{APP}}{\hat{\pi}_i} \frac{\partial \hat{\pi}_i}{\partial(\text{APP})}$$

$$= \frac{22.9 \times 0.0008326}{0.14844}$$

$$= 0.128$$

That is, a 1% increase in air pollution results in a 0.13% increase in the proportion of respiratory patients.

5.6 Discussion

The *Analysis of proportion* model used in this study gave a statistically significant association between air pollution (PM2.5) and proportion of respiratory symptoms requiring GPs attention. Respiratory visits were significantly correlated with 2-day lagged particulate air pollution in Armidale. At average values of minimum temperature and air pollution, about 14.8 per cent of all patients had respiratory illnesses associated with PM2.5.

These findings are consistent with investigations overseas and in Australia. Schwartz and Neas (2000), Neas *et al.* (1999) found the effects of PM2.5 stronger than the coarse fraction PM10-2.5 on the respiratory health of children. Schwartz *et al.* (1993) established a convincing association between emergency room visits for asthma and fine particles in Seattle, US where a considerable percentage of fine particles were produced by residential wood burning as in Armidale. Koenig *et al.*(1993) found a significant association between fine particles and acute respiratory irritation in young asthmatic children in Seattle. Ostro *et al.* (1991) observed similar associations for particulate pollution and asthma attacks in Denver.

Pope (1991) and Pope and Dockery (1992) observed a significant decline in lung function, increases in symptoms of respiratory disease, and the use of asthma medication with

elevated levels of particulate pollution. Dockery *et al.* (1982) and Schwartz (1989) found a decrease in pulmonary function with particulate pollution.

Australian studies found similar associations between air pollution and respiratory illness. Voigt *et al.* (1998) showed a significant relationship between airborne particles, nitrogen dioxide and respiratory morbidity in the Latrobe Valley. Rennick and Jarman (1992) found that susceptible children could have asthma attacks triggered by visibility-reducing particulates. Lewis *et al.* (1998) in a cross-sectional study of primary school children in New South Wales, reported a consistent association between particulate pollution and symptoms of night-time cough and chest colds.

Delfino *et al.* (1997), Roemer *et al.* (1993), Ostro (1998) and Pope and Dockery (1992) reported that lags of two days or more rather than concurrent exposure are strongly related to the health end point. In Armidale, respiratory visits were significantly correlated with 2-day lagged particulate air pollution. The lag may be due either to delay in seeking medical care or to the pathogenesis of the respiratory effects of particulate pollution.

The day of the week was a statistically significant variable in some studies (Krupnick *et al.* 1990; Ostro 1998). However, no significant influence was detected between the day that visits were made to the clinic in the present study.

Use of local clinics for gathering information for the study had both advantages and disadvantages. One of the principal advantages was that GPs who completed the record of visits were familiar with filling out special diagnostic forms. Studies that use data on hospital admissions or emergency room visits often face difficulties in terms of accuracy and consistency of coding and compliance (Ostro 1998).

The main disadvantage in data from local clinics, as well as hospital visits, is that the action of visiting a clinic is ultimately a subjective choice, which can be influenced by factors such as attention to illness, severity of illness and seeking an appointment with the GP. Furthermore, the clinics are only open on weekdays and during normal working hours and some patients may be discouraged from seeking medical attention from the GP.

CHAPTER 6
ECONOMIC COST OF PARTICULATE AIR POLLUTION IN ARMIDALE: CLINICAL EVENT SURVEY

6.1 Introduction

Chapter 5 examined the clinical events arising from the effect of particulate air pollution on respiratory symptoms. A model relating proportions of respiratory patients to relevant variables was estimated. This model demonstrated a statistically significant and positive effect of air pollution on the proportion of respiratory visits to GP clinics.

Chapter 6 assesses the economic costs associated with particulate air pollution in Armidale. The essential feature of the analysis in this chapter is the decomposition of the total number of respiratory patients into those caused by air pollution and those due to all other causes. The estimated number due to air pollution is then multiplied by the estimated cost.

It is important to note that while Chapter 5 used proportion of respiratory visits, Chapter 6 considers the number of respiratory patients visiting GP clinics to establish the relationships between particulate air pollution and respiratory morbidity. The results in Chapter 5 are based only on the survey data, reflecting a self-selective sample who chose to visit a GP clinic, but the results in Chapter 6 are extrapolated to provide an indication of likely effects on the population in Armidale.

The present chapter is based on data described in Chapter 5. It was observed from the clinical events study that there was little variation in the results obtained by using different sources of air pollution data. Therefore, only the set of data reflecting the mean of average daily air pollution recorded by Armidale Air Quality Group (AAQG) and Armidale Dumaresq Council (ADC) has been considered in the model to assess the economic cost.

6.2 Analytical Method-Poisson Regression

In conventional regression analysis, we have a *continuous* (dependent) variable and we seek to explain its variation. This is done by postulating that the value the variable takes is

determined to a large extent by some other (regressor) variables. The regression model shows how the average of the dependent variable is determined by the values of regressor variables. Poisson regression is the analogous statistical tool for the analysis of *count data.*

In this context, the variable we seek to explain is the number of patients per day who visit clinics with respiratory complaints. As with the previous analysis, we assume that this number is determined, in part, by the value of particulate air pollution, the minimum temperature, and the particular clinic chosen. Variables that measure these three types of determinants become the regressor variables, denoted collectively by X. The average number of respiratory patients, denoted as n_r, is the dependent variable.

The Poisson regression model relates the dependent variable to the regressor variables through the function

$$n_r = \exp(X'\beta) \qquad (6.1)$$

where exp(.) is the exponential function, and $X'\beta$ is a linear combination of the regressor variables. In particular, we assume that

$$X'\beta = \beta_0 + \beta_1 x + \delta_1 C_1 + \ldots + \delta_5 C_5 + \varphi_0 T + \varphi_1 T_1 + \ldots + \varphi_4 T_4, \qquad (6.2)$$

where x is particulate air pollution lagged 2 days, C_1 to C_5 are dummy variables relating to the five clinics other than the base clinic (clinic 0), T is the minimum temperature and T_1 to T_4 are the minimum temperatures lagged one to 4 days, respectively.

Poisson regression results, giving estimates of the unknowns β_0 to φ_4, are in Table 6.1.

In interpreting the results, two features should be noted. Firstly, the coefficient in which we are mainly interested is β_1, associated with air pollution (x). Its value of 0.00693 means that a one unit increase in air pollution, with all other factors unchanged, leads to a 0.693 per cent increase in the number of respiratory patients. Alternatively, a one standard deviation (21.12 units) increase in air pollution results in a $21.12 \times 0.693 = 14.6$ per cent increase.

Table 6.1: Results of the Estimated Poisson Regression

Variables	Estimated Coefficient	Standard Error	p-Value
X	.006932700624	.0017534828	.0001
T	.01446733972	.0079275040	.0680
T_1	-.002879004122	.010993110	.7934
T_2	.007623046597	.0091210813	.4033
T_3	.001239448514	.0081392957	.8790
T_4	.02348748284	.0080579779	.0036
C_1	.7603741646	.082613673	.0000
C_2	.0188430185	.098001030	.8475
C_3	.2577885870	.082315776	.0017
C_4	-.1112521826	.110356050	.3134
C_5	-.4957227478	.123205370	.0001
Constant	1.186992520	.085438055	.0000

Secondly suppose, for example, we are interested in the average number of respiratory patients attending clinic 5 on a day in which air pollution is 24 units, the minimum temperature 2 degree Celsius and has been for the last 4 days. In this example, the variables in the model have the following values:

$$x = 24,\ C_1 = C_2 = C_3 = C_4 = 0,\ C_5 = 1,\ T = T_1 = T_2 = T_3 = T_4 = 2$$

Thus,

$$\begin{aligned} n_r &= \exp[1.187 + (0.00693) \times 24 + (0.0145) \times 2 + (-0.0029) \times 2 + (0.0076) \times 2 \\ &\quad + (0.0012) \times 2 + (0.0235) \times 2 + (-0.4957) \times 1] \\ &= \exp(0.9454) \end{aligned}$$

ie: $n_r = 2.574$ patients

and so the average number of respiratory patients at clinic 5 on all days with these characteristics is 2.57.

In exactly the same way, the model can be used to estimate the average number of respiratory patients for any combination of values of air pollution, clinic and minimum daily temperature.

To estimate the costs due to air pollution, we now use the model to decompose n_r into a part due to air pollution, and the remainder accounting for all other causes of respiratory illness. Of course, this could be done numerically, for any given settting of the regressor variables, by evaluating n_r for a given value of APP, then with APP = 0 and subtracting the results. The following analysis provides some simple, linear formulae approximate the evaluation of pollution induced respiratory complaints.

First, we write $X'\beta$, defined by (6.2), as

$$X'\beta = a + b + c$$

where

$$a = \beta_1 x$$

$$b = \beta_0 + \delta_1 C_1 + \ldots + \delta_5 C_5,$$

$$c = \varphi_0 T + \varphi_1 T_1 + \ldots + \varphi_4 T_4.$$

Note that "a" is determined by the value of air pollution alone, "b" by the particular clinic and "c" by the minimum temperatures.
Then,

$$n_r = \exp(a + b + c) = \exp(a) * \exp(b) * \exp(c).$$

Also, because the absolute values of "a" and "c" are small relative to 1 for all values of the variables, we can approximate exp(a) and exp(c) by (1 + a) and (1 + c), respectively. Thus, n_r can be approximated by

$$n_r = \exp(b) * (1 + a) * (1 + c) = \exp(b) * (a + (1 + c)),$$

because (a * c) is always small enough to be ignored. Thus, finally,

$$n_r = n_{r,x} + n_{r,o}$$

where $n_{r,x}$ is that component of n_r attributable to air pollution alone, given by

$$n_{r,x} = a(\exp(b)) = \beta_1 x \exp(\beta_0 + \delta_1 C_1 + \ldots + \delta_5 C_5). \qquad (6.3)$$

To assist in the interpretation of (6.3), we define $n^{(i)}_{r,x}$ to be the contribution to $n_{r,x}$ due to the i^{th} clinic.

Then

$$n^{(i)}_{r,x} = \beta_1 x \exp(\beta_0 + \delta_i) \qquad (6.4)$$

where $i = 0,1,2,\ldots,5$

and $\delta_0 = 0$.

There is one final factor that we must account for in order to estimate the number of pollution-caused respiratory patients. In most of the six clinics surveyed, only some of the doctors completed the survey. We therefore apply "expansion factors" α_i which adjust the values of $n^{(i)}_{r,x}$ to take this into account. For example, in clinic 2 only 2 of the 5 doctors actually completed the survey. We therefore adjust $n^{(i)}_{r,x}$ for this clinic by multiplying by $\alpha_2 = 5/2 = 2.5$.

These 'expansion factors' are very blunt tools, as they do not account for different doctor practice profiles. Nevertheless, given the limitations on the data, they do provide a method for obtaining a total-clinic estimate.

Finally, we define the estimated average number of pollution induced respiratory patients at clinic i, denoted by $N^{(i)}_{r,x}$, as

$$N^{(i)}_{r,x} = \alpha_i \, n^{(i)}_{r,x} = \alpha_i \beta_1 x \exp(\beta_0 + \delta_i) \qquad (6.5)$$

The method for computing the number of pollution-caused respiratory patients per day for the whole of Armidale, for any given value of air-pollution (x), is simply to add the contributions for each clinic. For example, for clinic 2 with adjustment factor $\alpha_2 = 2.5$, we obtain

$$N^{(2)}_{r,x} = 2.5 \, \beta_1 x \exp(\beta_0 + \delta_2) = 2.5 \, (0.006933) \, x \exp(1.187 + 0.01884)$$

$$= 2.5(0.023153)x = 0.05788x.$$

We note that the above expression is linear in x (the value of air pollution). It follows that over time, the average number of pollution-caused respiratory patients can be obtained by replacing x by its average value, namely $\bar{x} = 22.9$. Thus, the daily average at clinic 2 = (0.05788)*(22.9) = 1.325 patients. This computation was carried out for each clinic, and the results presented in Table 6.2.

Table 6.2: Average Daily Number of Respiratory Patients

Clinic	Expansion factor (α_i)	$N^{(i)}_{r,x}$ when $\bar{x}$ = 22.9
0	2	1.041
1	1	1.113
2	2.5	1.325
3	1.5	1.010
4	5	2.328
5	2	0.634
Total $N_{r,x}$		7.451

The average daily number of respiratory patients (for the survey period) at the six clinics which can be attributed to air-pollution is thus $N_{r,x}$ = 7.45 persons.

Standard Error of the Estimate

Denoting the vector of estimated coefficients collectively by β, the standard error of $N_{r,x}$ is given by

$$\text{se}(N_{r,x}) = \bar{x}\sqrt{\frac{\partial N_{r,x}}{\partial \beta'}\text{cov}(\beta)\frac{\partial N_{r,x}}{\partial \beta}} \qquad (6.6)$$

where cov(β) is the covariance matrix of β. Using the covariance matrix which accompanies the Poisson regression output, this expression can be evaluated to obtain

$$\text{se}(N_{r,x}) = 1.52$$

6.3 Economic Cost

To assess the economic cost, only doctors' fees, costs of medicine and wage losses are considered.

Typically, general practitioners in Armidale charge, on average, $40 per visit (New England Division of General Practice). Allowing for transport costs and medical prescription, a rough estimate of an additional $50 per consultation seems reasonable.

We assume that each visit takes one half day of an adult's time and that such time is valued at $61 based on the national annual wage rate for Australia (ABS 2000). While not every one who seeks medical attention due to respiratory symptoms will miss half a day of work, the wage rate is considered a good reflection of the average value of time for this population. Some patients are children who do not earn wages, however all visits for children include adult supervision, with a consequent potential wage loss.

Therefore, the total cost for each respiratory visit is estimated as

$$\$40 + \$50 + \$61 = \$151$$

The average daily cost of pollution induced respiratory illness (for the survey period) is now obtained by multiplying the average number of respiratory patients $N_{r,x}$ by the cost of a respiratory visit. That is,

$$\text{Average daily cost} = \$151 \times 7.45 = \$1125.$$

The standard error of this cost estimate is $\$151 \times 1.52 = \$229$

6.4 An Alternative Estimate of the Number of Respiratory Patients

In equation (6.3) $n_{r,x}$ was estimated by:

$$n_{r,x} = \beta_1 x \exp(\beta_0 + \delta_1 C_1 + \ldots + \delta_5 C_5).$$

We can exploit the strong relationship that exists between temperature (T) and air pollution x, and estimate a regression relationship of the form

$$x = \gamma_0 + \gamma_1 T,$$

and obtain a relationship between $n_{r,x}$ and T, denoted by $n_{r,T}$, by substituting this latter relationship into $n_{r,x}$. Thus,

$$n_{r,T} = \beta_1(\gamma_0 + \gamma_1 T)\exp(\beta_0 + \delta_1 C_1 + \ldots + \delta_5 C_5), \qquad (6.7)$$

or, equivalently

$$n^{(i)}_{r,T} = \beta_1(\gamma_0 + \gamma_1 T)\exp(\beta_0 + \delta_i) \qquad (6.8)$$

This relationship is useful in at least three ways. First, it provides a method for predicting the number of patients who will report at a given clinic (and by implication, for the whole of Armidale) with pollution induced respiratory illness, based only on the minimum temperature. Because the minimum temperature is routinely reported on the radio, this provides a more convenient method than using a measure of particulate air pollution.

Second, it can be used to estimate the proportion of respiratory patients whose illnesses are caused by pollution.

Third, it enables us to confirm the average number of daily patients over the survey period simply by replacing T in equation (6.8) by the average minimum daily temperature. We discuss these uses now.

The predictive model can be derived as follows:
From (6.8), we may write

$$n^{(i)}_{r,T} = (\theta_0 + \theta_1 T)\exp(\beta_0)\exp(\delta_i) \qquad (6.9)$$

where

$$\theta_0 = \beta_1\gamma_0, \theta_1 = \beta_1\gamma_1, \text{ and } \delta_0 = 0.$$

Thus,

$$N^{(i)}_{r,T} = \alpha_i(\theta_0 + \theta_1 T)\exp(\beta_0)\exp(\delta_i) \qquad (6.10)$$

where α_i is the expansion factor and $N^{(i)}_{r,T}$ is defined analogously to $N^{(i)}_{r,x}$. Equation (6.10) gives the number of pollution affected patients reporting to clinic i.

It follows that the total number of pollution-induced respiratory patients can be estimated by simply adding up over all clinics. This estimate, denoted by $N_{r,T}$, is given by:

$$N_{r,T} = \sum_{i=0}^{5} N^{(i)}{}_{r,T} .$$

That is,

$$N_{r,T} = (\theta_0 + \theta_1 T)\exp(\beta_0)\sum_{i=0}^{5} \alpha_i \exp(\delta_i) \qquad (6.11)$$

The regression x on T yielded γ_0 = 24.681 (2.190) and γ_1 = -2.336 (0.5166), where the quantities in parentheses are the standard errors. Thus, θ_0 = 0.1711 and θ_1 = -0.01619. Furthermore, $\exp(\beta_0)$ = 3.2772 and $\sum_{i=0}^{5} \alpha_i \exp(\delta_i)$ = 14.3195. Therefore, the final predictive relationship is

$$N_{r,T} = (0.1711 - 0.01619\ T) \times 3.2772 \times 14.3195.$$

That is,

$$N_{r,T} = 8.029 - 0.7598\ T \qquad (6.12)$$

This simple linear expression provides a method for obtaining an estimate of the total number of patients reporting to all clinics with pollution induced respiratory illnesses, based only on the minimum temperature T.

From (6.3) it is easily seen that $n_{r,o}$, the component of respiratory patients due to other causes is given by

$$n_{r,o} = [1 + \varphi_0 T + \varphi_1 T_1 + \ldots + \varphi_4 T_4]\exp(\beta_0 + \delta_i),$$

and hence, using obvious notation,

$$N_{r,o} = [1 + \varphi_0 T + \varphi_1 T_1 + \ldots + \varphi_4 T_4]\exp(\beta_0)\sum_{i=0}^{5} \alpha_i \exp(\delta_i) \qquad (6.13)$$

The proportion R_x of pollution-induced respiratory patients to all respiratory patients is given by

$$R_x = N_{r,T} / [\ N_{r,T} + N_{r,o}\ ].$$

Using (6.11) and (6.13) and cancelling the common factor $\exp(\beta_0)\sum_{i=0}^{5}\alpha_i \exp(\delta_i)$

$$R_x = \frac{(\theta_0 + \theta_1 T)}{(\theta_0 + \theta_1 T) + (1 + \varphi_0 T + ... + \varphi_4 T_4)} \qquad (6.14)$$

Of course, this ratio depends on current and lagged minimum temperatures and will vary from day to day. To get an idea of its magnitude, we can evaluate it at the mean temperature (1 June-20 August) 0.987°C. This yields

$R_x = 0.129$

It appears then that particulate air pollution is responsible for around 13 per cent of respiratory visit during 1 June to 20 August.

Equation (6.12) can also be used to verify the value of average daily number of patients (obtained earlier) by evaluating it at the average daily temperature over the sample period, namely 0.987 ^{0}C. Thus,

average $N_{r,T} = 8.029 - 0.7598 \times 0.987 = 7.28$ patients.

This agrees well with the earlier estimate of 7.45 patients.

6.5 Discussion

Chapter 6 estimated the health and economic costs of air pollution in Armidale using GP clinical survey data for the winter of 1999. The chapter also validated the findings in Chapter 5 that there was an association between respiratory-related GP visits and particulate air pollution in Armidale.

Major Findings

i. During the survey period, the average number of respiratory patients due to particulate air pollution in Armidale was 7.45 persons per day.

ii. Approximately 13 per cent of the total respiratory visits to local GP clinics were due to particulate air pollution.

iii. Taking into consideration the costs and expenses arising from such ailments, the average daily economic cost of respiratory symptoms due to particulate air pollution was estimated to be $1,125.

Limitations of Findings

The estimated economic cost is conservative, and it only considers the direct medical costs. Dollar outlays were calculated in terms of doctors' usual charge for clinic visits, cost of drugs, and time loss estimated on the basis of the average wage rate. Related costs, such as X-rays, hospital admission, emergency room visits, alternative medicines and so on, and costs associated with "pain and suffering" have been ignored. Furthermore, data were only available from six of the eight GP clinics, resulting in underestimation of the true effects.

Conceptually, the monetary aspect could be extended further. One important step would be to estimate the *value* of missed schooling and work loss. Contingent valuation techniques might yield estimates of willingness to pay by the people of Armidale for the value of "pain and suffering" due to respiratory symptoms.

The study did not take account of preventive or defensive measures, which could contribute to an underestimation of economic cost. For example, asthma can be controlled with maintenance treatment. Many asthma patients experience mild symptoms and treat themselves with medication instead of reporting to a GP. Such cases are not captured in the GP reports. Asthma also affects people chronically, and the estimate only captures exacerbation due to fluctuations in pollution levels.

As the clinical survey was based on respiratory visits to local GPs, the study excluded hospital admission and emergency room visits. Patients with severe asthma attacks, who normally go to the hospital emergency department rather than to clinics, have not been included. In many cases, asthma imposes significant costs on persons with symptoms and their families. Researchers have used the cost-of-illness method to estimate the direct and indirect costs of asthma prevalence for several developed economies, including the US, Canada and the UK. Barnes *et al.* (1996) tabulated summary measures from nine studies in which direct costs typically contributed 50 to 60 per cent of total costs.

One of the earlier studies assessing the economic costs of air pollution, Ransom and Pope (1995), compared hospital admissions and mortality data before and after the temporary closure of a steel mill in a mountain valley in central Utah. They estimated that the annual increase in hospitalisation costs were US$2 million and more than US$40 million in mortality costs, due to particulate emissions.

Zaim (1997) estimated that by reducing its air pollution to WHO levels from 1993, Turkey would have reduced hospital admissions for respiratory diseases by 5,480, emergency room visits by 112,100, avoided 6.85 million restricted activity days and 73,000 cases of low respiratory symptoms in children 0-12 years of age. The estimated annual economic value of avoiding these effects represented nearly 0.08 per cent of Turkey's 1993 gross national product.

Hall *et al.* (1992) predicted that hypothetical attainment of air pollution standards in the Californian south coast air basin would save 1,600 lives a year. However, the benefits of actual attainment were not known.

It is clear from the above that inclusion of emergency room visits, hospital admissions and mortality would substantially increase the economic cost of particulate air pollution in Armidale.

Implications of Findings

The findings from this study have several implications. One is that an improvement in air quality can lead to both health and economic benefits to society. The economic cost estimated in this study was limited by the available data and should be regarded only as providing a *lower bound.* If additional data on hospital admissions, emergency room visits, willingness to pay and so on were available, a more realistic assessment of economic costs could be obtained.