Risk Assessment for Environmental Health in Adelaide Based on Weather, Air Pollution and Population Health Outcomes

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Table of Contents

SECTION I LITERATURE REVIEW AND STUDY DESIGN ...........................................1

Chapter 1 Literature review .............................................................................3
  1.1 Introduction ............................................................................................3
  1.2 Weather and human health .................................................................4
    1.2.1 The temperature-health relationship .........................................4
    1.2.2 Vulnerable groups .......................................................................7
    1.2.3 International studies ....................................................................7
    1.2.4 Australian studies .......................................................................8
  1.3 Air pollution and human health ...........................................................9
    1.3.1 Air pollution impacts on morbidity and mortality ......................9
      1.3.1.1 What are the ‘adverse effects of air pollution’? .................10
    1.3.2 Vulnerable groups .......................................................................12
    1.3.3 Major air pollutants ....................................................................12
      1.3.3.1 Particulate matter ..................................................................13
      1.3.3.2 Ozone ..................................................................................20
      1.3.3.3 Carbon monoxide ..................................................................20
      1.3.3.4 Nitrogen dioxide ......................................................................21
      1.3.3.5 Sulphur dioxide .......................................................................23
    1.3.4 Air pollution in Australia ...............................................................24
  1.4 Temperature and air pollution associations ............................................30
1.5 Climate change ............................................................................................................. 31
1.6 Methodological issues ................................................................................................. 33
  1.6.1 Study designs ........................................................................................................ 33
    1.6.1.1 Panel studies ................................................................................................. 33
    1.6.1.2 Cohort studies ............................................................................................ 33
    1.6.1.3 Ecological studies ....................................................................................... 34
  1.6.2 Confounding and bias .......................................................................................... 38
1.7 Gaps in current knowledge ......................................................................................... 39
Chapter 2 Study design and methodology ....................................................................... 42
  2.1 Introduction ............................................................................................................... 42
    2.1.1 Background ..................................................................................................... 42
    2.1.2 The study region ............................................................................................ 43
  2.2 Aims and objectives of the study ............................................................................. 46
  2.3 Research questions .................................................................................................. 47
  2.4 Framework of the study ......................................................................................... 47
  2.5 Methodology used in the study .............................................................................. 49
    2.5.1 Data collection and analysis ........................................................................... 49
    2.5.2 Heat-health studies ....................................................................................... 49
    2.5.3 Air pollution studies ...................................................................................... 49
    2.5.4 Health risk assessment .................................................................................. 51
SECTION II THE EFFECT OF EXTREME HEAT ON HEALTH IN ADELAIDE ................... 54
Chapter 3 The effect of heatwaves on renal morbidity .................................................. 56
  3.1 Introduction .............................................................................................................. 56
  3.2 Methods .................................................................................................................. 59
  3.3 Results ..................................................................................................................... 61
  3.4 Discussion ............................................................................................................... 67
Chapter 4 The effect of heatwaves on mental health ..................................................... 73
  4.1 Introduction .............................................................................................................. 73
  4.2 Methods .................................................................................................................. 74
  4.3 Results ..................................................................................................................... 76
  4.4 Discussion ............................................................................................................... 85
Chapter 5 A spatial analysis of ambulance callouts during heatwaves ............................ 96
  5.1 Introduction .............................................................................................................. 96
5.2 Methods .......................................................................................................................... 98
5.3 Results ............................................................................................................................ 100
5.4 Discussion ...................................................................................................................... 108

Chapter 6 Heatwaves and hospital emergency department visits ......................... 116
6.1 Introduction .................................................................................................................... 116
6.2 Methods ......................................................................................................................... 118
6.3 Results ............................................................................................................................ 119
6.4 Discussion ...................................................................................................................... 123

SECTION III THE IMPACT OF AIR POLLUTION ON HEALTH IN ADELAIDE .......... 129
Chapter 7 Particulate air pollution and cardiorespiratory hospital admissions ........ 131
7.1 Introduction .................................................................................................................... 131
7.2 Methods ......................................................................................................................... 134
7.3 Results ............................................................................................................................ 136
7.4 Discussion ...................................................................................................................... 145

Chapter 8 The interactive and confounding effects of temperature and air pollution .. 152
8.1 Introduction .................................................................................................................... 152
8.2 Methods ......................................................................................................................... 154
8.3 Results ............................................................................................................................ 156
8.4 Discussion ...................................................................................................................... 162

SECTION IV USING THE EVIDENCE ............................................................................. 168
Chapter 9 Health risk assessment for environmental stressors in Adelaide ........... 170
9.1 Introduction .................................................................................................................... 170
9.2 Issue identification ....................................................................................................... 173
9.3 Hazard assessment ...................................................................................................... 175
   9.3.1 Hazard identification .............................................................................................. 175
      9.3.1.1 Effects of heat on human health ...................................................................... 176
      9.3.1.2 Effect of air pollution on human health .......................................................... 177
   9.3.2 Dose-response assessment .................................................................................... 178
      9.3.2.1 The heat-health exposure-response relationship ........................................... 178
      9.3.2.2 The air pollution-health exposure-response relationship ............................. 179
      9.3.2.3 Sensitive subpopulations ................................................................................ 181
   9.3.3 Exposure assessment ............................................................................................. 182
10.3.2.2 Heatwave studies ................................................................. 222
10.3.2.3 Air pollution studies .............................................................. 223

10.4 Challenges faced in the study ......................................................... 224
10.4.1 Data .................................................................................. 225
  10.4.1.1 Definition and acquisition of data ....................................... 225
  10.4.1.2 Quality of data ................................................................ 226
10.4.2 Methodology ....................................................................... 227
  10.4.2.1 Overdispersion ............................................................... 227
  10.4.2.2 Case crossover analysis .................................................. 227
  10.4.2.3 Risk of errors ................................................................. 228

10.5 Significance of the study .............................................................. 229

10.6 Policy implications and recommendations ...................................... 230
  10.6.1 Government policy and guidelines ....................................... 230
  10.6.2 Infrastructure .................................................................... 231
  10.6.3 Resource allocation ............................................................. 232
  10.6.4 Organisational practice ....................................................... 232
  10.6.5 Community participation and engagement ........................... 234

10.7 Further research ....................................................................... 235
  10.7.1 Heat-health relationship and climate change ............................ 235
    10.7.1.1 Threshold temperatures ................................................ 235
    10.7.1.2 Adaptive capacity during heatwaves ............................... 235
    10.7.1.3 The effect of race on heat-susceptibility ........................... 236
    10.7.1.4 Heat stress in the workplace .......................................... 237
    10.7.1.5 The 2009 Adelaide heatwave ......................................... 238
    10.7.1.6 The effect of medications on the risk of heat illnesses ....... 238
    10.7.1.7 Heatwaves and infectious diseases ................................ 239
    10.7.1.8 Extension of study investigating thermal impacts and renal outcomes .240
    10.7.1.9 Do persons with diabetes have increased susceptibility to heat? ........ 241
    10.7.1.10 The effect of climate change on rural and remote communities ........... 241
    10.7.1.11 Psychological aspects of, and responses to, heat and climate change .243
    10.7.1.12 Climate change impacts on the criminal justice system .......... 243
  10.7.2 Air pollution and health ......................................................... 244
    10.7.2.1 More Adelaide based studies required ............................ 244
10.7.2.2 Spatial analysis of air pollution-related health effects.........................245
10.7.2.3 The heatwave-air quality association.......................................................246
10.7.2.4 Identification of subgroups susceptible to air pollution ......................247
10.7.2.5 The cardiovascular effects of PM ...........................................................247
10.7.2.6 The acute health effects of exposure to bushfire smoke ....................247
10.7.2.7 The acute health effects of exposure to dust ........................................248
10.7.3 Summary ..................................................................................................249

10.8 Conclusion .....................................................................................................250

APPENDICES ........................................................................................................251
A Differences between postcodes and postal areas ...........................................252
B Publications during candidature ....................................................................255

REFERENCES ........................................................................................................260
List of tables

Table 1.1  Examples of Australian outdoor air pollution studies published 2000 to 2008.......................................................... 26
Table 2.1  Fictional example data set for case-crossover analysis.......................... 52
Table 2.2  Exposure on the index day from Table 2.1, is compared with that of control days................................................................. 53
Table 3.1  Summary statistics of daily temperatures (°C) for Adelaide, 1995-2006........ 62
Table 3.2  The incidence rate ratio (IRR) of hospital admissions for renal disease during heatwave periods compared to non-heatwave periods in the warm season................................................................. 64
Table 3.3  Age-specific regression results showing the incidence rate ratio of hospital admissions of males with acute renal failure (ARF) during heatwave periods compared to non-heatwave periods during the warm season........................................................................................................... 66
Table 3.4  The effect of heatwaves on hospital admissions for renal disorders and comorbidities, showing incidence rate ratios (IRR), 95% confidence intervals (CI) and P-values......................................................................................................................... 67
Table 4.1  Cause-specific mental and behavioural disorder hospital admissions associated with heatwaves in Adelaide 1993-2006 showing incidence rate ratio (IRR) and 95% confidence intervals (CI).................................................................................. 78
Table 4.2  Cause-specific hospital admissions stratified by age and sex, showing incidence rate ratio (IRR) and 95% confidence intervals (CI)............................... 81
Table 4.3  Details of mental and behavioural mortalities associated with heatwaves showing incidence rate ratio (IRR), 95% confidence interval (CI) and P values....................................................................................................................... 84
Table 4.4  Medications that increase the risk of heat stroke in persons with MBDs.... 88
Table 5.1  Proportion of ambulance attendances in Adelaide in each age group during heatwaves, the warm season and over the entire study period, 1993-2005................................................................. 100

Table 5.2  Incidence rate ratios (IRR) and 95% confidence intervals (CI) for postcodes with increases in ambulance attendances during heatwaves 1993-2005...................................................................................... 106

Table 5.3  Incidence rate ratios (IRR) and 95% confidence intervals (CI) for presenting conditions in selected postcodes during heatwaves compared to non-heatwave periods .......................................................... 107

Table 6.1  Incidence rate ratios (IRR) and P-values for regression analyses of ED presentations during heatwaves compared to non-heatwave periods........ 121

Table 6.2  Incidence rate ratios (IRR) and 95% confidence intervals (CI) for postcodes with statistically significant (P < 0.05) increases in ED visits during heatwaves .................................................................................................. 122

Table 7.1  Summary statistics of air pollutants and hospital admissions during the cool and warm seasons, on days of smoke and dust, and across the whole study period ........................................................................ 139

Table 7.2  Percent increase in risk, with 95% confidence intervals (CI) for all-cause hospital admissions per 10 μg/m³ increase in PM ................................................................................. 140

Table 7.3  Percent increase in risk, with 95% confidence intervals (CI) for respiratory admissions per 10 μg/m³ increase in PM ............................................................................. 141

Table 7.4  Percent increase in risk, with 95% confidence intervals (CI) for cardiovascular admissions / 10 μg/m³ increase in PM .............................................................................. 142

Table 8.1  The effect of temperature and pollutants on emergency department visits in uni-and multi-variable models showing odds ratios (OR) and P-values ........................................................................ 158

Table 8.2  The effect of temperature and ozone on high temperature (> 35.3°C) and high ozone (≥ 41 ppb) on ED visits in uni-and multi-variable models showing odds ratios (OR) and P-values ........................................................................ 159
Table 8.3      Overall summary of the effect on emergency department (ED) visits of air pollution and temperature determined using interaction models in a case-crossover analysis........................................................................................................ 160

Table 8.4      Overall summary of the effect on mortalities of air pollution and temperature, determined using interaction models in a case-crossover analysis........................................................................................................ 161

Table 9.1      Bushfire alert categories based on PM$_{10}$ monitoring and visibility ........................................ 181

Table 9.2      Percent changes in the number of days of very high and extreme fire weather using different global warming scenarios (Low and High) for 2020 and 2050, relative to 1990 for Australia and Adelaide......................... 192

Table 9.3      Likely health responses to an increase in heatwaves and air pollution in Adelaide................................................................. 202

Table 9.4      Evidence that an increase in adverse health outcomes in Adelaide will occur in association with an increase in exposure to heatwaves or air pollution........................................................................................................ 203

Table 9.5      NEPM standards and goals for O$_3$ and PM$_{10}$ and advisory reporting standards for PM$_{2.5}$........................................................................................................ 209

Table 9.6      Example of a risk communication strategy for air pollution, outlining cautionary advice and actions for low and high bushfire smoke alert levels........................................................................................................ 213

Table 10.1     EPA air quality summary for 3rd February 2009................................................................. 246
# List of figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Factors affecting the progression from heat exposure to heat-related death</td>
<td>6</td>
</tr>
<tr>
<td>1.2</td>
<td>The air pollution health effects pyramid</td>
<td>10</td>
</tr>
<tr>
<td>1.3</td>
<td>Particulate matter size distribution</td>
<td>14</td>
</tr>
<tr>
<td>1.4</td>
<td>Pathophysiological mechanisms of cardiovascular toxicity of particulate matter</td>
<td>17</td>
</tr>
<tr>
<td>1.5</td>
<td>Simplified representation of the generation of secondary pollutants from NO₂ precursors</td>
<td>21</td>
</tr>
<tr>
<td>1.6</td>
<td>The death rate during the London fog increased in accordance with the rise in SO₂ concentrations</td>
<td>23</td>
</tr>
<tr>
<td>2.1</td>
<td>A: Location of Adelaide, South Australia; B: metropolitan Adelaide</td>
<td>44</td>
</tr>
<tr>
<td>2.2</td>
<td>Topographical map showing the Adelaide airshed and surroundings</td>
<td>45</td>
</tr>
<tr>
<td>2.3</td>
<td>Annual increase in temperatures for the Adelaide region</td>
<td>46</td>
</tr>
<tr>
<td>2.4</td>
<td>Flowchart outlining the framework of the study</td>
<td>48</td>
</tr>
<tr>
<td>3.1</td>
<td>Monthly mean maximum temperatures in Adelaide and days over 40°C.</td>
<td>59</td>
</tr>
<tr>
<td>3.2</td>
<td>Relation between daily maximum ambient air temperature and smoothed daily hospital admissions for renal disease excluding dialysis (N00-N39)</td>
<td>63</td>
</tr>
<tr>
<td>3.3</td>
<td>Plot of IRRs of admissions for renal (including dialysis), renal (excluding dialysis), and acute renal failure (ARF) during heatwave periods compared to non heatwave periods in the warm season</td>
<td>65</td>
</tr>
<tr>
<td>3.4</td>
<td>The relationship between daily hospital admissions for ARF and temperature during February and March 2004 when 3 heatwaves were recorded</td>
<td>66</td>
</tr>
<tr>
<td>Figure 4.1</td>
<td>Point estimates with 95% confidence intervals for the risk during heatwaves compared to non-heatwave periods, of hospital admission for mental, behavioural and cognitive disorders</td>
<td>79</td>
</tr>
<tr>
<td>Figure 4.2</td>
<td>Exposure-response relationships between daily maximum air temperature and hospital admissions for mental disorders</td>
<td>83</td>
</tr>
<tr>
<td>Figure 4.3</td>
<td>Proposed model for the association between heat and mental illness</td>
<td>87</td>
</tr>
<tr>
<td>Figure 4.4</td>
<td>A: Map showing hospital admissions with a principal diagnosis of schizophrenia, schizotypal and delusional disorders, metropolitan Adelaide 2004-2005. B: Index of Relative Socio-economic Disadvantage, metropolitan Adelaide, 2001</td>
<td>90</td>
</tr>
<tr>
<td>Figure 5.1</td>
<td>The relationship between SAAS callouts and $T_{max}$ during the warm season</td>
<td>101</td>
</tr>
<tr>
<td>Figure 5.2</td>
<td>A: map of callouts per postcode; B: Adelaide metropolitan area</td>
<td>102</td>
</tr>
<tr>
<td>Figure 5.3</td>
<td>Pattern of presenting conditions for ambulance callouts across the metropolitan area during heatwaves</td>
<td>103</td>
</tr>
<tr>
<td>Figure 5.4</td>
<td>A: Map of callouts per 1,000 population; B: Strategic industrial areas in the Adelaide metropolitan area</td>
<td>104</td>
</tr>
<tr>
<td>Figure 5.5</td>
<td>A: Callouts heatwaves: warm season; B: Per population heatwaves: warm season</td>
<td>105</td>
</tr>
<tr>
<td>Figure 5.6</td>
<td>Adelaide’s north western suburbs, showing the concentration of industries in the area</td>
<td>107</td>
</tr>
<tr>
<td>Figure 6.1</td>
<td>Change in emergency department visits across metropolitan Adelaide during heatwave periods compared to non-heatwave periods</td>
<td>123</td>
</tr>
<tr>
<td>Figure 7.1</td>
<td>Map of the Adelaide Air Quality Index sites and regions, showing the Netley monitoring site</td>
<td>135</td>
</tr>
<tr>
<td>Figure 7.2</td>
<td>Air quality indices for Adelaide 2002 to 2006</td>
<td>137</td>
</tr>
<tr>
<td>Figure 7.3</td>
<td>PM$_{2.5}$ concentrations peaked on 25 January 2006 when bushfire smoke blanketed Adelaide</td>
<td>138</td>
</tr>
<tr>
<td>Figure 7.4</td>
<td>PM$_{10}$ concentrations peaked during a dust storm over Adelaide</td>
<td>138</td>
</tr>
<tr>
<td>Figure 7.5</td>
<td>The association between PM$_{2.5}$ and hospital admissions over the study period and by season</td>
<td>144</td>
</tr>
</tbody>
</table>
Figure 7.6  Effect estimates for the association between PM$_{10}$ and hospital admissions over the study period and by season................................. 145

Figure 8.1  Diagrammatic representation of interaction. C, additive effect of the impact of components A and B. D, interaction with a synergistic effect. E, interaction with an antagonistic effect........................................... 154

Figure 8.2  Three-dimensional plot of deaths against PM$_{10}$ and maximum temperature.................................................................................. 157

Figure 8.3  The relationship between weather and air pollution, classified as very good, good, fair, poor or very poor using an Air Quality Index (AQI), during a 3 week period in January-February 2009............................................ 166

Figure 9.1  Conceptual model of risk........................................................................ 171

Figure 9.2  Comparative risk assessment model in the context of climate change, with definitions of attributable and avoidable disease burden.............. 172

Figure 9.3  Risk assessment model........................................................................... 173

Figure 9.4  Point estimates and 95% confidence intervals for mortality related to a 10 µg/m3 increase in PM$_{10}$, black smoke (BS) and PM$_{2.5}$ from European and U.S. studies......................................................................................... 180

Figure 9.5  Components of vulnerability to climate change.................................... 182

Figure 9.6  An increase in exposure may pose an unacceptable risk in a population in which A, a proportion is sensitive, or B, no unacceptable risk in a less sensitive population......................................................................................... 183

Figure 9.7  The effect of increasing mean temperatures on temperature extremes.................................................................................................................. 184

Figure 9.8  CSIRO and Australian Bureau of Meteorology projections of the probability of exceeding annual warming thresholds relative to 1990, based on the spread of climate model results using mid, low and high emission scenarios......................................................................................... 185

Figure 9.9  A range of CSIRO simulated annual temperature anomalies for Adelaide and the Mt. Lofty Ranges regions smoothed by an 11-year running mean.................................................................................................................. 186
Figure 9.10  Graph showing the mean number of days per month in Adelaide ≥ 35°C and the monthly mean maximum temperatures................................................. 187
Figure 9.11  Model of photochemical smog formation over the Adelaide airshed at different times (11.50, 12.50, 14.00 and 15.00 hrs) during a summer day.................................................................................................................. 190
Figure 9.12  Simulated rainfall anomalies using 11 models, for the Adelaide and Mt Lofty ranges region from 1850 to 2100................................................................. 191
Figure 9.13  Fire potential map 2009-10.......................................................................................................................... 193
Figure 9.14  Smoke trails (arrowed) extending into SA, from Victorian bushfires........ 193
Figure 9.15  The age structure for SA 2006 and 2056....................................................................................................... 196
Figure 9.16  Adelaide’s projected population to 2056 using 5 different models........ 196
Figure 9.17  Mid range estimates for heat–related deaths in Australian capital cities including Adelaide, for the present, 2020 and 2050.................................................. 198
Figure 9.18  Flowchart showing some of the possible environmental effects of climate change in southern Australia, and the impact on public health.... 205
Figure 9.19  Examples of risk communication during the 2009 Adelaide heatwave..... 211
Figure 9.20  The proportion of Australian respondents concerned about climate change......................................................................................................................... 214
Figure 10.1  How Adelaide’s A, ozone and B, PM$_{2.5}$ levels compare with other cities around the world.......................................................... 220
Figure 10.2  Example of a hydration chart................................................................. 233
Figure 10.3  South Australian hospitalisation rates for Aboriginal and non-Aboriginal peoples 2003-04 to 2006-07. Hospitalisations for A, renal disease; and B, mental health conditions......................................................... 237
Figure 10.4  The possible association between mental conditions and the onset of renal disease during periods of extreme heat............................................. 242
Figure 10.5  Long range transportation of dust in Australia September 2009.............. 249
List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>µm</td>
<td>Micrometre (micron) = $10^{-6}$ metres</td>
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<tr>
<td>ABS</td>
<td>Australian Bureau of Statistics</td>
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<tr>
<td>ARF</td>
<td>Acute renal failure</td>
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<td>AQI</td>
<td>Air quality index</td>
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<tr>
<td>CBD</td>
<td>Central business district</td>
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<tr>
<td>CI</td>
<td>Confidence interval</td>
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<tr>
<td>CNS</td>
<td>Central nervous system</td>
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<tr>
<td>CO</td>
<td>Carbon monoxide</td>
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<tr>
<td>CO$_2$</td>
<td>Carbon dioxide</td>
</tr>
<tr>
<td>COPD</td>
<td>Chronic obstructive pulmonary disease</td>
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<tr>
<td>CSIRO</td>
<td>Commonwealth Scientific and Industrial Research Organisation</td>
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<tr>
<td>CVD</td>
<td>Cardiovascular disease</td>
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<tr>
<td>ED</td>
<td>Hospital emergency department</td>
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<tr>
<td>EPA</td>
<td>Environment Protection Authority</td>
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<tr>
<td>ICD</td>
<td>International classification of diseases</td>
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<td>IHD</td>
<td>Ischaemic heart disease</td>
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<td>IPCC</td>
<td>Intergovernmental Panel on Climate Change</td>
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<tr>
<td>m$^3$</td>
<td>Cubic metre</td>
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<tr>
<td>MBD</td>
<td>Mental and behavioural disorders</td>
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<td>MI</td>
<td>Myocardial infarction</td>
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<tr>
<td>NEPC</td>
<td>National Environment Protection Council</td>
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<td>NEPM</td>
<td>National Environment Protection Measure</td>
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<td>NO$_2$</td>
<td>Nitrogen dioxide</td>
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<tr>
<td>NO$_x$</td>
<td>Nitrous oxides</td>
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<tr>
<td>NPI</td>
<td>National Pollutant Inventory</td>
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<tr>
<td>O$_3$</td>
<td>Ozone</td>
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<td>°C</td>
<td>Degree Celsius</td>
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<td>PM</td>
<td>Particulate matter</td>
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<td>PM$_{10}$</td>
<td>Particles with an equivalent aerodynamic diameter ≤ 10 µm</td>
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<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>PM$_{2.5}$</td>
<td>Particles with an equivalent aerodynamic diameter $\leq 2.5 , \mu m$</td>
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<tr>
<td>ppb</td>
<td>Parts per billion</td>
</tr>
<tr>
<td>ppm</td>
<td>Parts per million</td>
</tr>
<tr>
<td>SA</td>
<td>South Australia</td>
</tr>
<tr>
<td>SAAS</td>
<td>South Australian Ambulance Service</td>
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<tr>
<td>SES</td>
<td>State Emergency Service</td>
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<tr>
<td>SO2</td>
<td>Sulphur dioxide</td>
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<tr>
<td>TEOM</td>
<td>Tapered Element Oscillating Microbalance</td>
</tr>
<tr>
<td>U.S.</td>
<td>United States of America</td>
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<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
</tbody>
</table>
Abstract

**Background**

The progression of climate change may have wide ranging and varied implications for population health. Climatologists predict increases in heatwaves, droughts and bushfires for Australia, with health consequences including a potential rise in heat-related illnesses and adverse effects from increases in some air pollutants. Epidemiological evidence of the impact of temperature extremes and air pollution on morbidity and mortality at the local level is essential to identify site specific characteristics of vulnerable sub-populations and in projections of future scenarios. This study aimed to assess the impact of weather and air pollution on population health outcomes in Adelaide, and to inform decision makers on likely health impacts of climate change.

**Method**

Health outcome, meteorological and air quality data for periods of up to 12 years were used to assess the environmental health impact of heatwaves and air pollution on morbidity in Adelaide. The first part of the study investigated the impact of heatwaves, defined as being three or more consecutive days of maximum temperatures 35°C or above, on hospital admissions, ambulance callouts and emergency department visits using a case series approach. Spatial analytical techniques were used to identify regions at increased risk in the metropolitan area. The second part of the study investigated, using case-crossover analysis, the effect of air pollution on cardiovascular and respiratory health outcomes. Finally, an evidence based environmental health risk assessment for Adelaide was formulated using a climate change perspective.

**Results**

Heatwaves have a noticeable effect on population health in Adelaide. Findings showed ambulance callouts increased by 3.6% during heatwaves in Adelaide, with
some industrial and disadvantaged suburbs identified as heat-sensitive regions of the metropolitan area. Persons with mental and behavioural disorders were found to be susceptible to heat extremes with hospitalisations increasing by 7.3% during heatwaves compared to non-heatwave periods. Hospital admissions for renal disease and acute renal failure were increased by 10.0% and 25.5% respectively during heatwaves and heat-related presentations at emergency departments increased almost 3-fold compared to non-heatwave periods in the warm season. Despite Adelaide’s air quality comparing well with cities elsewhere, airborne particulate matter had a noticeable effect on health, more so in the cool season, with a 4.5% increase in cardiovascular hospitalisations associated with an increase of 10 µg/m$^3$ in fine particles.

**Conclusion**

Mounting evidence points towards a continued rise in global temperatures and more intense and frequent heatwaves. Findings from this study suggest that in the absence of adaptation and acclimatisation of the local population, there may be a disproportionate increase in heatwave-related morbidity; however the effect on air pollution-related morbidity is less clear. In a warming climate the adverse cardiovascular health effects of air pollution observed in the cool season in Adelaide may decrease, but overall may be modified by the health impacts of increased exposure to bushfire smoke and dust.

**Policy Implications**

Findings from this study have helped inform policies for extreme heat emergency plans for South Australia and may be of interest to the government and non-government sectors concerned with formulating local and national air quality guidelines.
Statement

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xix
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Alana Hansen
January 2010
Introduction

The influence of environmental stressors on human health has been of interest to researchers for decades. Due to metabolic homeostasis, the healthy human body generally adapts well to natural changes in the surrounding environment. However, age, underlying illness, predisposition and social factors can limit adaptive capabilities, rendering individuals and populations susceptible to negative health outcomes associated with environmental exposures.

Since industrialisation, anthropogenic activities have contributed to increasing concentrations of atmospheric pollutants with history providing considerable evidence of their detrimental effect on health. Additionally climatologists claim the gradual build up of greenhouse gases in the atmosphere is contributing to changes in global weather patterns. Extreme weather events in the form of storms, floods, droughts, cold snaps and heatwaves, can have devastating short- and long-term health consequences for affected communities and susceptible individuals. Predictions of a climate change induced increase in some or all of these extreme events should be cause for great public health concern.

Whilst many epidemiological studies have been conducted to investigate the health impacts of air pollution and extreme weather, most have been undertaken in the major cities of North America and Europe. Many research groups, often collaboratively, have undertaken studies incorporating large study populations, some exposed to high levels of ambient air pollution or rare temperature extremes. Whilst evidence of environmental health impacts on a global scale is imperative and forms the basis for international guidelines, local evidence is also crucial to enable public health strategists to formulate targeted intervention strategies at the community level. With Australia’s unique climate, lifestyle, demographics and geography, health responses may differ substantially from those in locations in the Northern Hemisphere.
The aim of this thesis was to characterise the public health impacts of extreme weather and air pollution in Adelaide, the capital city of the state of South Australia. The city has a temperate climate with long hot summers, mild winters and low rainfall. Heatwaves are common in Adelaide, with maximum temperatures above 35°C occurring on average 17 days per annum at present, with climate change scenarios predicting future increases in very hot days. Levels of air pollutants are generally relatively low although guideline exceedances do occur, often in association with bushfires or dust events. The impact on public health is yet to be assessed. A retrospective analysis of health outcomes associated with changes in ambient temperature and air quality was undertaken as part of this thesis, and susceptible subpopulations in Adelaide identified, thus providing an evidence base for an environmental health risk assessment, with implications for public health policy.

It should be noted that the terms “climate” and “weather” are often used interchangeably throughout the thesis, but generally “climate” refers to long term meteorological conditions, whereas “weather” refers to daily conditions or those over a period of several days.

The focus of the study was on conditions relevant to South Australia, and hence discussions and studies on the effects of extreme temperatures are limited to extreme heat, as very cold conditions are rare in Adelaide. Similarly, some pollutants in the Adelaide air shed are more prevalent than others which have decreased to low levels in the last few decades due to enforced regulations on vehicle design and industry emissions. The main pollutants of interest in this study were thus be particulate matter (PM\textsubscript{10} and PM\textsubscript{2.5}), nitrogen dioxide and ozone.

This thesis is formulated in four sections. Section I comprises two chapters, the first of which is a comprehensive literature review of international and Australian studies investigating the effects of weather and air pollution on human morbidity and mortality. Air pollutants are discussed in turn as well possible temperature-pollutant interactions, vulnerable populations, methodological issues concerned with various study designs, and gaps in current knowledge. The health impacts of climate change are addressed and whilst not the principal focus of the thesis, are a recurring theme
in discussions of extreme heat and future scenarios. Section I concludes with a chapter outlining the aims and objectives of the study, research questions, framework of the study and methodologies used.

Section II comprising four chapters, provides details of studies investigating the health effects of heatwaves in Adelaide. Chapter 3 focuses on renal disease whilst Chapter 4, mental health morbidity and mortality during heatwaves. Chapters 5 and 6 seek to identify potentially heat-sensitive regions in the metropolitan area, incorporating spatial analysis into studies investigating the impact of extreme heat on ambulance callouts and syndromic surveillance of hospital emergency department visits.

The theme of Section III is air pollution in Adelaide. Chapter 7 summarises a study on the effect of air pollution on cardiorespiratory hospitalisations, whereas Chapter 8 investigates the complex interrelationships that can occur between individual air pollutants, and between temperature and air pollution.

Section IV combines the evidence gained from an extensive body of literature, together with results from studies undertaken within the candidature, to speculate on future scenarios. Chapter 9 uses a risk assessment framework to assess the future impact of the aforementioned environmental stressors on population health in Adelaide. Chapter 10 concludes the thesis with a general discussion and summary of the previous chapters highlighting the key findings, limitations, the public health significance of this work, policy implications and suggestions for future research.
SECTION I

LITERATURE REVIEW AND STUDY DESIGN
Introduction

This first section of the thesis, comprising two chapters, provides a general background and introduction to the relevant issues whilst providing a basis for the main study.

Chapter 1 is a comprehensive literature review of recently published international and Australian studies concerning weather and air pollution as environmental health stressors. The epidemiological evidence is presented and discussed in the context of climate variation, and vulnerable subgroups of the population identified. Gaps in current knowledge are also addressed.

Chapter 2 gives a general outline of the study design complete with aims and objectives, research questions, the general framework of the study, and a justification of the chosen study methods.
Chapter 1

Literature review

1.1 Introduction

This chapter, the first of two in this section, is a comprehensive literature review of studies relating to the two main themes of the thesis - weather and air pollution, and their impact on human health.

The chapter begins with a general review of literature concerning the association between weather and health, with the focus mainly on exposure to high environmental temperatures and heat-susceptible subgroups of the population. Indirect health effects of weather variability including the influence on infectious diseases does not form part of the discussion.

A review of the air pollution literature follows. It should be noted however, that this only briefly touches the surface of the comprehensive body of literature regarding the health impacts of air pollution that has been published in the last few decades. A PubMed search using the search term ‘air pollution’ revealed a total of 34,168 articles and hence due to the limitations of this thesis, only a general overview of the main concepts is be presented herein. Each of the major pollutants, their sources and health effects are discussed, with reference to both international and Australian studies. The focus is on cardiopulmonary health effects, and although it is recognised there are a growing number of studies investigating the association between air pollution and birth outcomes, these are not discussed in the thesis.

The interactive effects of temperature and air pollution are addressed followed by the likely impact on health of a changing global climate. Methodology can vary
considerably between research groups, often making comparisons between study findings problematic. The main study designs used in environmental epidemiology are explained, along with issues concerning confounding and bias.

Finally, gaps in the international and Australian literature are identified, confirming there is much we are yet to understand about environment-health associations. The present study aimed to address some of these issues with relevance to variations in climate and air pollution in Adelaide.

1.2 Weather and human health

Weather can have a marked influence on the physiological and psychological well being of humans. Exposure to heat, cold, humidity, wind and rain can affect the behaviour of individuals and their health, the extent of which may depend on an ability to physically and metabolically adapt to the surrounding environment. When weather conditions become extreme, the adaptive capacity of individuals will vary according to a range of health, social and demographic determinants. Perhaps the most significant environmental variable in this context is ambient temperature, and its impact on population health outcomes in terms of morbidity and mortality is discussed below.

1.2.1 The temperature-health relationship

The relationship between temperature and mortality has been described as J-\(^1,2\) or U shaped\(^3,4\) with lowest mortality at moderate temperatures, rising with increasing or declining temperatures. Cold related deaths often occur due to respiratory diseases and arterial thrombosis, the latter resulting from the blood become more concentrated in the cold, increasing the likelihood of clotting.\(^5\) Unlike Europe and the United States, exposure to cold extremes is relatively rare in Australia, and is not discussed further in this thesis.

Heat-related mortalities however often occur in Australia and are of growing public health concern worldwide as climate change progresses. The health impacts of heat exposure have been the subject of much research in recent years\(^1,4,6-12\) due largely to the
A devastating European heatwave of 2003 which resulted in well over 30,000 excess deaths in Western Europe\textsuperscript{13} including 14,802 in France, 2,099 in Portugal and 2,045 in the UK.\textsuperscript{14}

Thermoregulation is the process of maintaining stable core body temperature between 36.2°C and 38.2°C. In response to heat exposure the hypothalamus initiates a compensatory effect involving the increase of blood flow to the skin allowing cooling to occur by sweating.\textsuperscript{15,16} Failure of the thermoregulatory system can lead to hyperthermia and dehydration, defined as a loss of body water of more than 3\% of body weight.\textsuperscript{16} Heat stress, presenting as perceived discomfort and physiological strain, can easily progress to heat illnesses (Figure 1.1) such as heat exhaustion and/or heat stroke aided by increased production of inflammatory cytokines and altered expression of heat shock proteins.\textsuperscript{17} Heat exhaustion can be a mild to moderate illness with symptoms including headache, dizziness, muscle cramps, nausea, weakness and fainting.\textsuperscript{17,18} Heat stroke on the other hand, is a serious condition that occurs when body temperature exceeds 40°C and central nervous system involvement manifests as delirium or convulsions.\textsuperscript{17} Symptoms may progress to include liver or renal failure\textsuperscript{19,20} and subsequent multi-organ system dysfunction.\textsuperscript{18} Heat stroke has a fatality rate of approximately 33\%\textsuperscript{21} and death often occurs rapidly. Heat-related death often occurs prior to seeking medical attention or reaching hospital\textsuperscript{22,23} and most of these deaths generally occur at the individual’s place of residence.\textsuperscript{24} Those who survive heat stroke can be left with residual damage to the brain\textsuperscript{17} or other organs, increasing the risk of early mortality.\textsuperscript{25} Reports following the French heatwave show heat stroke mortality rates at 28 days and 2 years as high as 58\% and 71\% respectively.\textsuperscript{26}

However, many deaths during heatwaves are not due to classic heat stroke but to the cardiovascular consequences of heat stress. Thermoregulation requires an increase in cardiac output, placing strain on the circulatory system, particularly in those with previous heart or lung disease. Loss of salt and body water can result in haemoconcentration, leading to cerebral or cardiac thrombosis.\textsuperscript{5,16} As a consequence, deaths due to heat are often not recorded as heat-related\textsuperscript{16,27} especially if they occur as a result of exacerbation of prior conditions. The reported incidence of heat-related illnesses is thus often imprecise with heatstroke being grossly underdiagnosed.\textsuperscript{28}
Whilst the association between heatwaves and increased mortality has been substantiated by many studies, relatively few have assessed the impact of extreme heat on morbidity. In 1995 heatwaves were associated with an increase of 11% in hospital admissions in Chicago,\textsuperscript{19} and a non-significant increase in London.\textsuperscript{22} A recent study reported an excess of 1,182 hospitalisations and 16,166 emergency department (ED) visits in association with the Californian heatwave of 2006.\textsuperscript{29} Heatwaves of several consecutive days of extreme heat may be of more health concern than isolated hot days as it was found in a small observational study conducted in Adelaide that heat-related illnesses often become apparent in hospital emergency departments following three to four consecutive days of high temperatures.\textsuperscript{30} As well as hospital visits and admissions, ambulance callouts have also been shown to increase during heatwaves.\textsuperscript{6,31}

NOTE:
This figure is included on page 6 of the print copy of the thesis held in the University of Adelaide Library.

\textbf{Figure 1.1:} Factors affecting the progression from heat exposure to heat–related death.  
\textit{Source:} Adapted from Kovats & Hajat.\textsuperscript{25}
1.2.2 Vulnerable groups

Those most vulnerable to the effects of heat include the elderly, persons with chronic or underlying medical conditions, the socio-economically disadvantaged, and those living in social isolation or widowed. The elderly are particularly susceptible as they frequently meet more than one of these criteria. Thermoregulation is often impaired in older persons and acclimatisation to heat, which is usually quite rapid in the young, may take more than a week in the elderly. Studies have shown that from about 50 years of age, the risk of heat-related mortality increases, often being higher in women. As well as physiological reasons, the financial and social limitations that often accompany ageing can be predictors of heat susceptibility. Some studies have also shown race/ethnicity to be a risk factor for heat susceptibility which may have implications in Australia for Indigenous persons and migrants.

Additionally it is known that many drugs can affect the thermoregulatory process. These include medications used to treat disorders such as hypertension, depression, diabetes, cerebrovascular or cardiovascular disorders and Parkinson’s disease. In particular, many medications used in psychiatry can render individuals more susceptible to heat, contributing to the vulnerability of those with mental disorders in whom heat stroke is common.

1.2.3 International studies

In August 2003, Europe experienced its hottest summer for several hundred years. The World Health Organization estimates there were at least 27,000 excess deaths across Europe during that time compared to previous summers, other reports placed the death toll much higher. Some 14,800 heat-related deaths occurred in France alone, with 4,866 occurring in and around Paris. Most of the decedents were aged 75 years and over and excess mortality was 15% higher in women than in men of comparable age. The main causes of excess mortality during the European heatwave were diseases of the cardiovascular, respiratory, genitourinary and nervous systems as well as endocrine and mental disorders. There was a relative increase in deaths at home of 74%, and in retirement homes 91%.

Nursing homes and hospitals in Europe were not routinely air-
conditioned at the time, a contributing factor to the high number of heat victims who were elderly or infirm.  

Previous heatwaves in Chicago in 1995 and Los Angeles in 1963 resulted in an increase in mortalities of 147% and 172% respectively, with once again most occurring in those aged over 75 years of age. The high number of mortalities associated with the European and the United States heatwaves combined with the relatively small increases in hospital admissions, suggests that many deaths occurred in vulnerable and isolated people before they had the opportunity to seek medical attention.

1.2.4  Australian studies

Unlike many Northern Hemisphere countries, extreme heat is common in most parts of Australia and populations are generally more acclimatised to hot spells. Nevertheless, heat-related mortalities occur each summer in Australia and New Zealand, and estimates suggest that 40% of the weather-related deaths recorded between 1803 and 1992 were due to heatwaves. A New Zealand study showed that in Christchurch, each 1°C increase in temperature above 20.5°C was associated with a 1% increase in all-cause mortality and 3% increase in respiratory mortality. Similarly an Australian study has calculated threshold temperatures and dose-response relationships for persons aged over 65 years. The authors showed that heat attributable mortality in the elderly increases as daily maximum temperatures exceed 20°C in Sydney, 28°C in Brisbane and 34°C in Darwin and Cairns.

Although some research on heat–related mortality has recently been undertaken in Sydney and Brisbane, Adelaide investigations are gaining particular credibility in the area of heatwave research. A recent study of the effect of heatwaves in Adelaide showed a 2.6 fold increase in mental health related mortality in the 65-74 age group but no overall increase in all-cause mortality in any age group. Ambulance use and hospital admissions were however increased in this study indicating that morbidity may be a more informative heat-health indicator than mortality rates as, unlike many overseas countries, air conditioning and adaptive behaviours are common mitigating factors in Australia. Nevertheless the recent severe heatwave of early 2009 may have
been, according to press reports, responsible for 347 deaths in Victoria,\textsuperscript{52} and an unusually high number of deaths, yet to be finalised, in Adelaide.\textsuperscript{53}

\section*{1.3 Air Pollution and human health}

The atmosphere contains a heterogeneous mix of naturally occurring and anthropogenically derived gaseous, liquid and solid components, some of which may be harmful to health in undesirable amounts. Sources of air pollution include ‘mobile’ (e.g. vehicular traffic), ‘stationary’ (e.g. power plants) and ‘area’ (e.g fireplaces) sources.\textsuperscript{54} Information about air quality in an airshed is obtained by measuring ambient concentrations of pollutants at monitoring stations,\textsuperscript{55} the locations of which are determined on the basis of pollutant distribution and potential human exposures.\textsuperscript{56} Isolating separate health effects can be problematic given individual pollutants rarely occur in isolation,\textsuperscript{57,58} hence the major pollutants (to be discussed below) are often considered as indicators of the air pollution mixture.\textsuperscript{59}

\subsection*{1.3.1 Air pollution impacts on morbidity and mortality}

With increased urbanisation and greenhouse gas emissions, air quality is currently a growing global public health concern, although reports of the negative effect of heavily polluted air on health date back as far as the 17\textsuperscript{th} century.\textsuperscript{60} More recently in December 1952, the dense air pollution of the London smog\textsuperscript{61} caused several thousand excess deaths\textsuperscript{62,63} with unusually high mortality rates persisting for over two months.\textsuperscript{64} Similar events, albeit to a lesser extent, occurred in the Meuse Valley (Belgium) in 1930 and Donora (Pennsylvania) in 1948.\textsuperscript{64} Numerous studies since that time have investigated and confirmed the adverse health effects of acute exposures to air pollution, and the World Health Organisation (WHO) has claimed that urban air pollution contributes to the deaths of some 800,000 people annually.\textsuperscript{65} Furthermore in Australia it is estimated that motor vehicle pollution alone contributed to the premature deaths of 900-2000 early deaths in 2000.\textsuperscript{66}
1.3.1.1 What are the ‘adverse effects of air pollution’?

A considerable body of literature discusses the ‘adverse health effects’ of acute air pollution exposure, a term which may require clarification. As defined by the American Thoracic Society (ATS), adverse health effects of air pollution range in severity from eye, nose and throat irritation, through to a range of respiratory complications, lung cancer and increased mortality.67 Adverse respiratory effects can take the form of mild interference of normal activity to progressive respiratory dysfunction and death, with the frequency of the outcome generally inversely proportional to the severity68 (Figure 1.2). The recognised list of effects includes factors contributing to diminished quality of life or change in clinical status, with recent additions including social and psychological categories.67 Strong evidence11,69-74 indicates that cardiovascular disease should also be added to this extensive list of air pollution-related adverse health effects.

![Figure 1.2: The air pollution health effects pyramid. Source: Reproduced with permission from Airnet WG 4.75](image)

Although ingestion and skin absorption can occur, the major route of exposure to gaseous and particulate air pollutants is via inhalation into the respiratory tract and thus respiratory diseases are often chosen as the most relevant outcomes for investigation.76
The average adult breathes approximately 10 million times per year\textsuperscript{77} and generally has little control over the air they inhale or the pollutants penetrating into the airways. The respiratory tract consists of the nasopharyngeal region, the tracheobronchial region and the pulmonary region, where the respiratory bronchioles in the lung branch into alveolar ducts and tiny alveolar sacs.\textsuperscript{78} With some 300 million alveoli in the adult lung\textsuperscript{55} a large alveolar surface area of up to 150 m\textsuperscript{2} \textsuperscript{78} is available for aerosol deposition.

The respiratory bioavailability of aerosols is determined by several factors including their physicochemical characteristics and distribution, with smaller particles able to penetrate deeper into the respiratory tract. Particles of 5-30 µm in aerodynamic size can enter the nasopharyngeal region, those between 1-5 µm may be deposited in the tracheobronchial region, and those smaller than 1 µm may reach the alveoli.\textsuperscript{78} Deposition and rate of clearance can be determined by other factors including the surface area, shape, density, conductivity and solubility of aerosols. Additionally meteorological factors such as temperature, humidity and wind speed will also influence the fate of inhaled pollutants.\textsuperscript{78}

The bulk of the published literature discusses the acute health effects of air pollution without mention of chronic effects. A lag time between exposure and absorption may result in an extended period of time before any adverse health outcome is evident. Conversely, chronic exposures can produce immediate effects after each exposure, as well as long term effects.\textsuperscript{79} Whilst several studies investigate lagged effects of a few days post exposure, few investigate longer term effects. Zanobetti and colleagues\textsuperscript{80} found that cardiovascular and respiratory effects persisted for over a month after exposure to air pollution, claiming that one and two day lags underestimate the impact of exposure. Dockery et al\textsuperscript{81} conducted a benchmark study involving a 14-16 year follow-up of over 8,000 persons in six U.S. cities incorporating personal information including smoking habits, occupational exposure, body-mass index, and level of education of participants. By comparing mortalities and air pollution levels between cities, the authors found that after adjusting for confounders, long-term exposure to air pollution was significantly associated with the risk of death from cardiopulmonary disease and lung cancer.
Overall, individual- and multi-city studies world-wide report small but (mostly) statistically significant increases in all-cause mortality and/or morbidity associated with airborne pollution. Risk estimates vary but approximate 1% per unit increase in pollutant concentration, often with higher risk estimates for cardiovascular and respiratory outcomes. The strength of the evidence and consistency of the findings are such that exposure-response curves identified in the major studies have become the basis upon which international air quality guidelines have been set, often with a non threshold approach.82

1.3.2 Vulnerable groups

For a variety of reasons, certain sub-groups of the population are more sensitive to air pollutants than others. The literature has identified several vulnerable groups including the elderly,69,71,83,84 young children,77,85,86, individuals with pre-existing illness,86 especially diabetes87 or advanced heart or lung disease,70 are particularly at risk. Additionally, some persons may be genetically hypersensitive to certain xenobiotics, and may respond adversely at common exposure levels.67

As many of those who succumb to the effects of air pollution or extreme temperatures are elderly, frail or very ill, it has been suggested that exposure in these individuals is merely bringing forward the time of death. ‘Harvesting’, or ‘mortality displacement’,88,89 terms used for the temporal advancement of death, refer to an increase in mortality followed by an associated decrease. This phenomenon, and its implication for interpretation of measures of effect, has been reported and discussed in several studies.83,88-93

1.3.3 Major air pollutants

The highest emissions of air pollutants tend to be in densely populated urban areas94 where heavy traffic and industrial emissions lower air quality. The major pollutants of concern are particulate matter and the gaseous components - ozone, nitrogen dioxide, carbon monoxide and sulphur dioxide. In a meta-analysis of 109 studies from cities
around the world, it has been shown using single pollutant models that these individual pollutants are all significantly associated with mortality.83

In June 1998 Australia’s National Environment Protection Council (NEPC) introduced the National Environment Protection Council (Ambient Air Quality) Measure (Air NEPM). The NEPC established a national set of guidelines for the measurement and reporting of key air pollutants which pose a risk to health, and defined standards based on toxicological and epidemiological studies.56,94 Pollutants addressed by Air NEPM are:

- particulate matter less than 10 µm in aerodynamic diameter (PM$_{10}$)
- carbon monoxide
- sulphur dioxide
- ozone
- nitrogen dioxide
- lead

Additionally air toxics (a group of hazardous and sparsely distributed volatile organic compounds including formaldehyde, polycyclic aromatic hydrocarbons, toluene, benzene and xylenes), can be detrimental to human health94 but to date exposure data have been limited. Lead levels, although a serious air quality issue in Port Pirie, South Australia, have declined markedly in Adelaide following the introduction of unleaded petrol in the 1980s, resulting in levels well below the NEPM standard.94 Neither lead nor air toxics were included in the present study and are not discussed further in this review.

1.3.3.1 Particulate matter

Particulate matter (PM) can be of organic or inorganic origin and includes airborne dust particles, soot and hydrocarbons from combustion processes, metal residues, fibres, and sulphate, chloride or nitrate compounds,91 as well as biological components and adsorbed volatile and semi volatile organic compounds.95 Primary particulates are emitted directly into the air whereas secondary sources result from the transformation of gases.74 Sources are mainly motor vehicle69 or industry emissions, wood or biomass burning82,96 and wind-blown dust.
Classification of PM is by size as seen in Figure 1.3 which shows relative biological sizes. Total suspended particulate matter (TSP) refers to particles with an aerodynamic diameter less than 50 µm. Particulates less than 10 µm are termed coarse particles or thoracic particles, those less than 2.5 µm (PM$_{2.5}$) fine particles, and those between 2.5 µm and 10 µm ‘the coarse fraction’. The collective term for fine and coarse particulate matter less than 10 µm in aerodynamic diameter is PM$_{10}$. Recently, ultrafine particles with diameter 0.01-0.1 µm have been recognized as a significant component of air pollution, as have accumulation particles (0.1-1.0 µm) formed by the coagulation of ultrafine particles.

Concentrations of PM$_{10}$ and TSP are measured using a Tapered Elemental Oscillating Microbalance (TEOM) device or by the use of a high volume sampler. As samplers are unable to determine precise size differentiation, particle size usually refers to a 50% cut point relative to a specific aerodynamic diameter. An older method of measurement incorporates a light scattering technique using nephelometry. Calculation of PM$_{10}$ concentrations from TSP measurements using conversion factors has been reported however inaccurate estimates may be produced due to the heterogeneity of particulate size and shape (Mr. R. Mitchell, EPA, pers. comm.)
18/7/06). Concentrations of PM$_{2.5}$ are also measured using a TEOM, with routine monitoring undertaken in recent years only. Some retrospective studies have used conversion factors to calculate PM$_{2.5}$ concentrations from available PM$_{10}$ and/or TSP concentrations, but once again imprecise conversion factors may lead to some inaccuracies. Coarse and fine particles are of respirable size and have been implicated in a range of health problems including the onset of respiratory and cardiovascular diseases.

**PM-associated respiratory disease**

With ease of inhalation deep into the lungs, PM is often linked with the onset of respiratory disease and associations have been reported in numerous studies with both morbidity$^{85,86,103-106}$ and mortality.$^{4,58,70}$ A New Zealand study showed that an increase in PM$_{10}$ of 10 µg/m$^3$ was associated with a 1% (95% CI 0.5-2.2%) increase in all-cause mortality and a 4% (95% CI 1.5-5.9%) increase in respiratory mortality.$^4$ There is substantive evidence that short term exposure to PM exacerbates pre-existing pulmonary symptoms with subsequent decline in lung function.$^{101}$ Mucociliary clearance of deposited particulates can occur, or clearance via engulfment by alveolar macrophages, invoking inflammatory responses. Removal of particle-laden macrophages from the pulmonary region may take several weeks. Components of PM such as nitrates and sulphates may be responsible for some respiratory outcomes with asthmatics found to be more sensitive to sulphuric acid than non-asthmatics.$^{107}$ Additionally allergens including pollen grains and spores, as well as transition metals, may be included in the particulate mix, contributing to alveolar inflammation and airway permeability, leading to the generation of free radicals.$^{108}$ For a number of reasons, children often display heightened sensitivity to air pollution triggered respiratory illnesses, particularly asthma.$^{109}$

**PM-associated cardiovascular disease**

Despite the seemingly obvious association of particulates with respiratory disease, the majority of adverse effects are often related to the effect on the cardiovascular system.$^{110}$ Although the association between PM and cardiovascular disease has been well documented$^{70-72,74,111}$ the biological pathways between inhalation into the lungs
and eventual effects on the cardiovascular system are yet to be fully understood\textsuperscript{73,74}. It is believed however that following absorption into the body, particulates have the ability to alter cardiovascular haemodynamics, heart rate and heart rate variability, blood pressure and blood coagulability, leading to atherosclerosis, adverse thrombotic and vascular effects, an increased risk of myocardial ischaemia and exacerbation of chronic heart conditions.\textsuperscript{71,74,95,112}

Three complex pathways have been proposed. The first involves effects on autonomic balance in the parasympathetic and sympathetic nervous systems following deposition of PM in the pulmonary airways, and transportation of soluble or very small particles into the systemic circulation. The second involves the consequences of lung inflammation and oxidative stress with pro-oxidative and pro-inflammatory mediators (eg. interleukin-6, fibrinogen, C-reactive protein) being released into the systemic circulation, affecting cardiovascular responses.\textsuperscript{110} A controlled study of wood smoke-PM inhalation found markers of increased inflammation, coagulation and possibly lipid peroxidation in volunteers, suggesting local inflammation in the airways may induce systemic inflammatory changes.\textsuperscript{113} The generation of reactive oxygen species in endothelial cells or heart muscle tissue as a result of pro-inflammatory signalling has also been suggested.\textsuperscript{95} The third pathway proposes translocation of soluble or ultrafine particles across the alveolar-blood barrier into the systemic circulation with direct effects on haemostasis and cardiovascular integrity.\textsuperscript{110,112} Hence the cardiotoxic effects of PM may be as a result of direct effects on the cardiovascular system via translocation into the circulation, or indirect effects via secondary pulmonary-mediated inflammatory responses and oxidative stress. These may be responsible for acute and chronic cardiovascular effects respectively.\textsuperscript{74} The pathophysiological pathways of toxicity are represented diagrammatically in Figure 1.4. It has also been suggested that long term effects are the result of exacerbation and acceleration of pre-existing pulmonary disease, specifically chronic obstructive pulmonary disease. Thus it is likely that no single pathway is responsible for PM-induced cardiovascular morbidity and mortality, and that multiple complex interactions occur which culminate in ill health.\textsuperscript{101}
Figure 1.4: Pathophysiological mechanisms of cardiovascular toxicity of particulate matter.

*Inhaled ambient air particles increase production of reactive oxygen species (ROS) in the airways and lung alveoli and stimulate local inflammatory reaction in the lungs. The ROS and pro-inflammatory cytokines released into the blood stream affect autonomic cardiac control (heart rate, heart rate variability, and cardiac contractility), blood pressure, vascular tone and reactivity, blood coagulability, and progression of atherosclerosis. Ultrafine particles may translocate into the circulation and induce oxidative stress and pro-inflammatory changes directly in the cardiac muscle and vasculature. Lung- and circulation-mediated and direct pathophysiological mechanisms exacerbate myocardial ischemia and increase cardiovascular mortality. CRP = C-reactive protein; IL = interleukin; TNF = tumor necrosis factor. Figure illustration by Rob Flewell. * Source: Reproduced with permission from the Journal of the American College of Cardiology (Simkhovich et al. p. 723)\textsuperscript{55}
Effects of PM on mortality and life expectancy

Numerous studies from different continents have documented the effects of PM on mortality and the consensus is that no threshold exists below which concentrations have been considered to be safe.\textsuperscript{110} Some studies have actually found the slope of the concentration-response function to be steeper at lower than higher concentrations.\textsuperscript{101}

A review of Asian studies found an increase of 0.4\%-0.5\% in all-cause mortality associated with every 10 \( \mu g/m^3 \) rise in PM\(_{10}\) levels\textsuperscript{114} whereas a large multi-city European study estimated the increase to be 0.6\%.\textsuperscript{84} Additionally, the World Health Organization (Europe Office) has shown that long-term exposure to high concentrations of particulate pollution may lead to significant reductions in life expectancy\textsuperscript{77} estimating an association with approximately 100,000 deaths and 725,000 years of life lost per year in Europe.\textsuperscript{77} It has been suggested that the lowering of PM\(_{10}\) levels by 5 \( \mu g/m^3 \) would prevent up to 7,700 deaths annually in Western and Eastern European cities.\textsuperscript{92} Also in Europe, a study incorporating 23 cities estimated that 16,929 premature deaths could be prevented annually if long-term exposure to PM\(_{2.5}\) were reduced to 15 \( \mu g/m^3 \), this figure converting to an increase in life expectancy at age 30, of between one month and more than two years.\textsuperscript{115}

In the United States, a study across six cities showed that a 10 \( \mu g/m^3 \) increase in PM\(_{2.5}\) was associated with a 1.5\% increase in daily deaths with no evidence of a threshold.\textsuperscript{97} The authors predicted that a reduction of 10 \( \mu g/m^3 \) in the ambient concentration of PM\(_{2.5}\) could result in approximately 36,000 fewer early deaths annually in the U.S.\textsuperscript{97} More recently, a study in 51 U.S. metropolitan areas estimated that a decrease of 10 \( \mu g/m^3 \) in PM\(_{2.5}\) may be associated with an increase in mean life expectancy of 0.61 ± 0.2 years.\textsuperscript{116}

Pope et al\textsuperscript{101} has estimated, based on U.S. figures, that with a 10 \( \mu g/m^3 \) increase in PM\(_{2.5}\) equating to an approximate 1\% increase in mortality, a 50 \( \mu g/m^3 \) short term rise in PM\(_{2.5}\) would result in an average of 1.2 deaths per day in a population of 1 million people (approximately the size of the city of Adelaide). Despite the small numbers, this is still of major public health concern.
Traffic based PM

Fine and ultrafine particles from combustion sources are of particular health concern as they are present in high numbers in the air, and are readily able to penetrate deep into the respiratory tract, posing even more of a concern than PM$_{10}$. Associations between fine particles and respiratory and cardiovascular diseases have been reported with linear relationships found between concentrations and mortality. Vehicle exhaust is a major source of fine and ultrafine particles. PM$_{2.5}$ levels strongly correlate with traffic density and highest concentrations of ultrafine particles have been found to occur during rush-hour traffic. The PM-related health effects for persons residing near roadways has been investigated in several studies and a link between traffic exposure and impaired cardiovascular and respiratory health has been established. Schwartz et al found there to be no threshold in the association between traffic particles and daily deaths, and estimated the difference between 10 µg/m$^3$ and 20 µg/m$^3$ in mean PM$_{2.5}$ concentrations would equate to 130 additional early deaths per year in a population of similar size to Adelaide (approximately one million). Additionally a study in the Netherlands found the adjusted risk of cardiopulmonary mortality was nearly double for persons living within 100 metres of a freeway or 50 metres of a major road, concluding that long-term exposure to traffic pollution may shorten life expectancy.

A study of the economic costs of the health impact of transport emissions in Australia estimates that in 2000 there were between 900 and 4,500 cases of cardiovascular or respiratory disease, with an economic cost of $0.4 billion to $1.2 billion; and that between 900 and 2,000 early deaths, costed at between $1.1 billion to $2.6 billion, could be attributed to traffic related pollution. Other estimates are even higher. It has been claimed that “mortality due to air pollution is higher than the road toll” with 2,400 deaths each year in Australia caused by air quality issues, and 1,700 caused by motor vehicle accidents. In summary, a substantial body of evidence points to the negative effects of PM on cardiorespiratory morbidity and mortality with smaller particles likely posing the greatest health concerns.
Black carbon is emitted as a result of fossil fuel or biomass burning, and studies have shown more than 90% resides in the PM$_{2.5}$ fraction of particulate matter.\textsuperscript{122} There is evidence that several of the components of PM$_{2.5}$ including those of a carbonaceous nature, contribute to cardiovascular mortality.\textsuperscript{123}

### 1.3.3.2 Ozone

Tropospheric ozone (O$_3$) is a photochemical oxidant formed by the chemical interaction of nitrogen oxides (NO$_x$) and reactive hydrocarbons. With formation being dependent on strong sunlight, ground level O$_3$ is more prevalent during summer\textsuperscript{58,70,94,103} and is a major component of photochemical smog. Ambient O$_3$ concentrations are usually measured using an ultraviolet photometric analyzer.\textsuperscript{100}

As well as causing eye irritation,\textsuperscript{91} O$_3$ exposure can produce oxidative stress in lung cells,\textsuperscript{103} causing inflammation in the lung and nasal passages\textsuperscript{124} and a temporary reduction in lung function.\textsuperscript{91} Previous respiratory conditions can be exacerbated by O$_3$\textsuperscript{58,61,94,103,124} as evidenced by a Brisbane study showing a dose-response relationship between O$_3$ concentration and asthma/respiratory disease with little evidence of a threshold.\textsuperscript{124} The authors estimated that O$_3$ was associated with approximately 4-10% of hospital admissions for asthma.

Nevertheless there are conflicting reports in the literature regarding the effect of O$_3$ on human health. Whilst some studies have shown significant relationships of O$_3$ levels with mortality\textsuperscript{83,111} and morbidity,\textsuperscript{103} particularly in the elderly,\textsuperscript{62,125} others found no effect of elevated O$_3$ levels on rates of death\textsuperscript{70} or hospital admissions.\textsuperscript{57} Similarly, some studies have reported an association between O$_3$ and cardiovascular disease,\textsuperscript{86,111,126} and yet others found no association.\textsuperscript{69}

### 1.3.3.3 Carbon monoxide

Carbon monoxide (CO) is produced by the incomplete combustion of fossil fuels. Vehicle emissions are the main source of CO in urban areas, and concentrations can depend on local topography, vehicle density and weather conditions.\textsuperscript{91} Ambient CO concentrations, measured using a non-dispersive infrared analyzer,\textsuperscript{100} have been reported to be at higher levels during the winter months.\textsuperscript{58}
The health effects of CO stem from its ability to bind to haemoglobin producing carboxyhaemoglobin,\textsuperscript{91} with the affinity of haemoglobin for CO being approximately 200-250 times that for oxygen.\textsuperscript{107} Studies have found increases in ambient CO concentrations to be associated with an increase in all-cause mortality\textsuperscript{83} and cardiovascular disease.\textsuperscript{57,69,91,127} Biological plausibility stems from the inhibition of oxygen uptake through the bloodstream by CO causing tissue hypoxia, affecting the cardiovascular system, the brain and exercising skeletal muscle.\textsuperscript{107} Consequently exposure can increase the risk of angina in the presence of ischaemic heart disease as well as affecting eyesight and mental concentration.\textsuperscript{67,94}

1.3.3.4 Nitrogen dioxide

Nitrogen dioxide (NO\textsubscript{2}) is a byproduct of fossil fuel combustion and a precursor of secondary air pollutants including O\textsubscript{3}, and particulates containing nitric acid (HNO\textsubscript{3}). When there is an abundance of NO, (as in busy city centres) O\textsubscript{3} is “scavenged”, regenerating NO\textsubscript{2} and oxygen\textsuperscript{128} (Figure 1.5). Nitrogen oxides which yield outdoor NO\textsubscript{2} are mainly from motor vehicle emissions and hence ambient concentrations of NO\textsubscript{2} can be an indicator of traffic pollution.\textsuperscript{84} Levels of are generally higher in winter,\textsuperscript{58,124} as domestic unflued gas heaters and cookers are major contributors of NO\textsubscript{2} to indoor air pollution.\textsuperscript{129} Measurement of ambient NO\textsubscript{2} and other oxides of nitrogen (NO\textsubscript{x}) is performed using gas-phase chemiluminescence.\textsuperscript{100}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.5.png}
\caption{Simplified representation of the generation of secondary pollutants from NO\textsubscript{2} precursors. \textit{Source:} Adapted from WHO.\textsuperscript{128}}
\end{figure}

NOTE:
This figure is included on page 21 of the print copy of the thesis held in the University of Adelaide Library.
The main toxic effects of NO$_2$ involve the respiratory system where hyper-responsiveness in the airways is increased.\textsuperscript{91} With high short term exposures mainly occurring near major roads,\textsuperscript{130} exposure has been shown to exacerbate respiratory conditions\textsuperscript{124} including asthma, particularly in the elderly.\textsuperscript{57} Children are also affected as demonstrated in a study involving Australian and New Zealand cities, where a 6.0\% increase in hospital admissions for asthma in children aged 5-14 years was associated with a 5.1 ppb increase in 24-hour NO$_2$ levels, independent of other pollutants.\textsuperscript{85} Similarly, indoor NO$_2$ levels have been positively associated with the exacerbation of asthma symptoms in children.\textsuperscript{131} As well as respiratory effects, an increase in NO$_2$ concentrations has been found to be associated with cardiovascular disease.\textsuperscript{69,91,104,125} Associations have also been found with mortality\textsuperscript{83,90,111,132} particularly when ambient concentrations exceed a threshold of 100 ug/m$^3$\textsuperscript{132} [To avoid confusion between ppb and ug/m$^3$, note 1 ug/m$^3$ NO$_2$ equates to $5.32 \times 10^{-4}$ ppm, i.e. 0.532 ppb.\textsuperscript{107}] In June 2009, the U.S. Environmental Protection Agency proposed to strengthen the NO$_2$ air quality standards to protect public health, particularly the health of sensitive subpopulations.\textsuperscript{130} The proposal aims to establish a 1-hour standard at 80-100 ppb whilst retaining the current annual average of 53 ppb. It is also proposed that monitors be placed near major urban roads to measure peak concentrations. The National Ambient Air Quality Standard will cover all NO$_x$ although it is NO$_2$ that is of the greatest health concern. The review of the primary standard for NO$_2$ is due for completion by January 2010.\textsuperscript{130}

As seen in Figure 1.5, NO$_x$ react with other compounds to form small particulates,\textsuperscript{130} and correlations seen in multi-pollutant models between the effects of NO$_2$ and particulates can be suggestive of a confounding\textsuperscript{84} or synergistic effect between the two pollutants with both having similar emission sources and health outcomes.\textsuperscript{104,111} Similarly, NO$_x$ reacts with volatile organic compounds to form O$_3$ under the right conditions\textsuperscript{130} and considerations should be given to the control of confounding in multipollutant models where NO$_2$, PM and O$_3$ are covariables.
1.3.3.5 Sulphur dioxide

Sulphur dioxide (SO$_2$), emissions are mainly derived from the industrial combustion of sulphur-containing fossil fuels.$^{91}$ Compared to some major industrial cities overseas, SO$_2$ levels in Australian cities are quite low,$^{58,125}$ particularly since the transfer of industrial fuel use in Australia from oil to gas.$^{56}$ Levels are likely to be highest in locales adjacent to certain major industrial sites. Ambient concentrations of SO$_2$ are measured by a process which involves fluorescing SO$_2$ molecules present in the air, then measuring the emitted light using a photomultiplier.$^{100}$

With SO$_2$ being oxidized to sulphuric acid in the presence of humidity,$^{91}$ it is a respiratory irritant which can elicit bronchoconstriction$^{91,94}$ and has been linked with the onset of asthma in children.$^{85}$ Responses are usually rapid and short lived, and include wheezing, shortness of breath and reduced ventilatory capacity.$^{107}$ Some authors have associated increases in respiratory hospital admissions$^{85}$ and mortality$^{83,90}$ with increased SO$_2$ concentrations, whilst others have reported marginal$^{62}$ or no significant attributable health effects.$^{4,124}$ High SO$_2$ levels during the London fog of 1952 paralleled the high mortality rate as shown in Figure 1.6. In this thesis SO$_2$ will not be a pollutant of major interest due to the relatively low levels recorded in Adelaide.

**NOTE:**
This figure is included on page 23 of the print copy of the thesis held in the University of Adelaide Library.

*Figure 1.6:* The death rate during the London fog increased in accordance with the rise in SO$_2$ concentrations. *Source:* Reproduced with permission from P Lioy.$^{133}$
1.3.4 Air pollution in Australia

The main urban air pollution sources in Australia are motor vehicle emissions, industrial emissions, home heating sources and bushfires. Additionally drought-related dust storms can occasionally cause extreme pollution events. In 1998 the National Environment Protection Council (NEPC) established the National Environment Protection Council (Ambient Air Quality) Measure (Air NEPM), a national set of standards and goals for the six “criteria air pollutants” which pose a risk to health: PM\textsubscript{10}, CO, SO\textsubscript{2}, photochemical oxidants (as O\textsubscript{3}), NO\textsubscript{2} and lead. An advisory reporting standard for PM\textsubscript{2.5} was later established in 2003. Air toxics are a group of hazardous pollutants which include formaldehyde, polycyclic aromatic hydrocarbons, toluene, benzene and xylenes. A NEPM for air toxics was established in 2004, providing the impetus for monitoring in hot spots where previously exposure data had been scarce.

During the period 1991 to 2001, downward trends were observed in four of the six criteria pollutants: lead, CO, SO\textsubscript{2}, NO\textsubscript{2}, indicating increasing air quality in the major Australian cities. However concentrations of O\textsubscript{3} and PM have not declined to the same extent, exceeding the NEPM standards on several occasions. Nevertheless, urban air quality in Australia is generally much better than in many other parts of the world and since the introduction of recent emissions controls, major urban centres now report levels well below the standards for most pollutants. In a meta-analysis of studies from 109 cities around the world, it was shown that compared to cities in Canada, United States, South America, Western Europe and Asia, mean air pollution concentrations in Australian and New Zealand cities were the lowest for PM\textsubscript{10}, NO\textsubscript{2} and SO\textsubscript{2}. Carbon monoxide concentrations were considerably lower than those in South America but in line with other countries, whereas O\textsubscript{3} concentrations were comparable to those of Asian and Western European cities but considerably lower than in California, South America, and Eastern Europe.

Generally, the majority of Australian air pollution studies have yielded similar findings to those undertaken in other countries, where meta-analyses have shown increases in daily mortality of 0.5% (U.S.) to 0.6% (Europe) and for cardiovascular hospital admissions, increases of 0.5% (Europe) to 1.1% (U.S.), per 10µg/m\textsuperscript{3} increment in
PM$_{10}$. The effects on respiratory admissions were also of a similar magnitude. However a review of 43 ozone-associated mortality studies worldwide showed exposure-response risk estimates for all-season data were highest for Melbourne and Brisbane.\textsuperscript{139} Table 1.1 summarizes findings from a collection of recent (from 2000) Australian studies investigating cardiovascular or respiratory effects of exposure to ambient air pollution. Between-city heterogeneity may be accounted for by geographical, climatic and demographic differences together with variations in pollution mixtures.
Table 1.1: Examples of Australian outdoor air pollution studies published 2000 to 2008.

<table>
<thead>
<tr>
<th>Author</th>
<th>Scope</th>
<th>Pollutants</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barnett, A.</td>
<td>7 cities in</td>
<td>PM$<em>{10}$, PM$</em>{2.5}$</td>
<td>Significant effects of PM$<em>{2.5}$, PM$</em>{10}$, b$_{sp}$, NO$_2$, SO$_2$ on child respiratory health. For 5-14 year olds, a 6% (95% CI 0.2-12.1%) (\uparrow) in asthma admissions per 5.1 ppb (\uparrow) in NO$<em>2$; a 1.9% (95% CI 0.1-3.8%) (\uparrow) in respiratory admissions per 7.5 µg/m$^3$ (\uparrow) in PM$</em>{10}$. No associations with CO or O$_3$.</td>
</tr>
<tr>
<td>et al. 2005$^{85}$</td>
<td>Aust &amp; NZ</td>
<td>b$_{sp}$, CO, NO$_2$, SO$_2$, O$_3$</td>
<td></td>
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<td></td>
<td>1998-2001</td>
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<tr>
<td>Barnett, A.</td>
<td>7 cities in</td>
<td>PM$<em>{10}$, PM$</em>{2.5}$</td>
<td>In the elderly ((\geq) 65 years) all pollutants except O$_3$ were associated with CV admissions, with (\uparrow) of 2.2% (95% CI 0.9-3.4%), 3.0% (95% CI 2.1-3.9%) &amp; 1.3% (95% CI 0.6-2.0%) per interquartile increase in CO, NO$<em>2$ &amp; PM$</em>{2.5}$ respectively. Largest (\uparrow) for cardiac failure in association with both NO$<em>2$ (6.9% (\uparrow) per 5.1 ppb unit (\uparrow)) &amp; CO (6.0% (\uparrow) per 0.9 ppm (\uparrow)). Found greater impact of PM$</em>{2.5}$ on cardiac admissions in cities with less humidity. Suggested no confounding by temperature.</td>
</tr>
<tr>
<td>et al. 2006$^{69}$</td>
<td>Aust &amp; NZ</td>
<td>CO, NO$_2$, O$_3$</td>
<td></td>
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<td></td>
<td>1998-2001</td>
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<tr>
<td>Bennett, C.</td>
<td>Melbourne</td>
<td>PM$_{2.5}$</td>
<td>Two combined cohorts of adults (N = 1446), were recruited &amp; given questionnaires regarding adverse respiratory symptoms. Mean time of 5.2 years between baseline &amp; follow up. Most associations between PM$_{2.5}$ exposure &amp; a range of adverse respiratory symptoms over time were non-significant.</td>
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<tr>
<td>et al. 2007$^{736}$</td>
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<tr>
<td>Chen, L.</td>
<td>Brisbane</td>
<td>PM$_{10}$</td>
<td>Found an (\uparrow) in daily respiratory hospitalisations to be associated with (\uparrow) PM$<em>{10}$. For an (\uparrow) in PM$</em>{10}$ from low to medium or high, there was an (\uparrow) in respiratory admissions of 9-19%. The association with PM$_{10}$ was found to be stronger during bushfire periods than non-bushfire periods.</td>
</tr>
<tr>
<td>et al. 2006$^{410}$</td>
<td></td>
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<tr>
<td>Chen, L.</td>
<td>Brisbane</td>
<td>PM$_{10}$, NO$_2$, O$_3$</td>
<td>Reported a 4.0% (\uparrow) (95% CI 1.1-6.9%) in respiratory emergency admissions associated with an (\uparrow) of 10 µg/m$^3$ in PM$_{10}$, using a spatial approach, weaker after adjusting for O$_3$ (2.6%, 95% CI 1.0-5.5%), not affected by NO$_2$. Non-spatial approach yielded weaker estimates. Associations between PM &amp; respiratory emergency admissions were stronger in areas with heavy traffic.</td>
</tr>
<tr>
<td>et al. 2007$^{718}$</td>
<td></td>
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<tr>
<td>Erbas, B</td>
<td>Melbourne</td>
<td>PM$_{10}$, NO$_2$, O$_3$</td>
<td>Reported associations between PM$<em>{10}$ &amp; childhood asthma emergency department presentations with strongest being RR = 1.17 (95% CI 1.05-1.31) in inner Melbourne, per (\uparrow) in PM$</em>{10}$ from the 10th to 90th centile. Associations with NO$_2$ &amp; O$_3$ varied.</td>
</tr>
<tr>
<td>et al. 2005$^{411}$</td>
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b$_{sp}$: particle scattering coefficient used as a measure of particle concentration in nephelometry$^{32}$; CV: cardiovascular; PEFR: peak expiratory flow rates; RR: rate ratio; \(\uparrow\): increase.
Table 1.1 (cont.): Examples of Australian outdoor air pollution studies published 2000 to 2008.

<table>
<thead>
<tr>
<th>Author</th>
<th>Scope</th>
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<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hanigan, I.</td>
<td>Darwin</td>
<td>PM$_{10}$</td>
<td>Found mostly positive associations between PM$<em>{10}$ from vegetation fire smoke &amp; respiratory admissions, with a 4.81% $\uparrow$ (95% CI -1.04-11.01%) per 10μg/m$^3$ $\uparrow$ in PM$</em>{10}$ with higher effect estimates for Indigenous people compared to non-Indigenous. Mostly negative estimates for CV conditions.</td>
</tr>
<tr>
<td>et al. 2008$^{42}$</td>
<td>1996-2005</td>
<td></td>
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<tr>
<td>Hinwood, A.</td>
<td>Perth</td>
<td>PM$<em>{10}$, PM$</em>{2.5}$, b$_{10}$ CO, NO$_2$, O$_3$</td>
<td>Small number of associations evident between particles, NO$<em>2$ &amp; CO with respiratory diseases, COPD, pneumonia, asthma &amp; CVD hospitalisations. Modelled PM$</em>{2.5}$ was associated with respiratory disease, pneumonia &amp; asthma. A 1 ppb $\uparrow$ in NO$_2$ was associated with a 0.6% $\uparrow$ in respiratory admissions &amp; 0.4% $\uparrow$ in CVD admissions in over 65 year olds. No associations observed with O$_3$.</td>
</tr>
<tr>
<td>Hu, W. et al.</td>
<td>Sydney</td>
<td>PM$_{10}$, NO$_2$, O$_3$, CO, SO$_2$</td>
<td>Reported an $\uparrow$ in daily mortality of 0.9% (95% CI 0.6-1.3%) per °C $\uparrow$ in maximum temperature &amp; a 22% (95% CI 6.4-40.5%) $\uparrow$ per 1 ppb in daily average SO$_2$. Interaction effects observed. When temperature &gt; 32°C &amp; SO$_2$ &gt; 0.315 ppb, mortality increased 7.3% &amp; 12.1% respectively.</td>
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<tr>
<td>2008$^{49}$</td>
<td>1994-2004</td>
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<td>Jalaludin, B.</td>
<td>Sydney</td>
<td>PM$_{10}$, NO$_2$, O$_3$, pollen, alternaria</td>
<td>Studied the effects of bushfire smoke on peak expiratory flow rates (PEFR) in 32 children with wheeze &amp; found no significant association between bushfire periods &amp; evening PEFR nor between mean PM$_{10}$ &amp; evening PEFR, but a negative association in children without bronchial hyper-reactivity ($\beta$ coefficient = -0.1029).</td>
</tr>
<tr>
<td>et al. 2000$^{43}$</td>
<td>1994</td>
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<tr>
<td>Jalaludin, B.</td>
<td>Sydney</td>
<td>PM$_{10}$, NO$_2$, O$_3$, pollen, alternaria</td>
<td>In a cohort of 125 children with a history of wheezing, a negative association was found between PEFR &amp; same-day mean daytime O$_3$ ($\beta$ coefficient 0.88), with a dose response relationship. No association with same-day daily maximum O$_3$. Effects greater in children with bronchial hyper-reactivity &amp; doctor diagnosis of asthma.</td>
</tr>
<tr>
<td>et al. 2000$^{44}$</td>
<td>1994</td>
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<tr>
<td>Jalaludin, B.</td>
<td>Sydney</td>
<td>PM$_{10}$, NO$_2$, O$_3$, pollen, alternaria</td>
<td>In an 11-month longitudinal study, found an association between PM$_{10}$ &amp; doctor visits for asthma (RR=1.11, 95% CI 1.04-1.19) &amp; between NO$_2$ &amp; prevalence of wet cough (RR = 1.05, 95% CI 1.003-1.10), in a final cohort of 125 children with a history of wheeze. No association found between O$_3$ &amp; respiratory symptoms, asthma medication use &amp; doctor visits for asthma. Unable to show adverse health effects of ozone in children with a history of wheezing.</td>
</tr>
<tr>
<td>et al. 2004$^{45}$</td>
<td>1994</td>
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Table 1.1 (cont.): Examples of Australian outdoor air pollution studies published 2000 to 2008.

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</tr>
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<tbody>
<tr>
<td>Jalaludin, B. et al. 2006&lt;sup&gt;27&lt;/sup&gt;</td>
<td>Sydney 1997-2001</td>
<td>PM&lt;sub&gt;2.5&lt;/sub&gt;, PM&lt;sub&gt;10&lt;/sub&gt;, NO&lt;sub&gt;2&lt;/sub&gt;, NO, O&lt;sub&gt;3&lt;/sub&gt;, SO&lt;sub&gt;2&lt;/sub&gt;, Co</td>
<td>PM, NO, CO &amp; SO&lt;sub&gt;2&lt;/sub&gt; associated with increased ED attendances for CVD, cardiac disease &amp; ischaemic heart disease in the elderly. No associations with O&lt;sub&gt;3&lt;/sub&gt;. Effects were generally greater in the cool period except for O&lt;sub&gt;3&lt;/sub&gt;. Found a 1.26% (95% CI 0.56-1.96%) ↑ in CVD attendances per interquartile ↑ in PM&lt;sub&gt;2.5&lt;/sub&gt;.</td>
</tr>
<tr>
<td>Jalaludin, B. et al. 2008&lt;sup&gt;46&lt;/sup&gt;</td>
<td>Sydney 1997-2001</td>
<td>PM&lt;sub&gt;2.5&lt;/sub&gt;, PM&lt;sub&gt;10&lt;/sub&gt;, NO&lt;sub&gt;2&lt;/sub&gt;, NO, O&lt;sub&gt;3&lt;/sub&gt;, SO&lt;sub&gt;2&lt;/sub&gt;, Co</td>
<td>Investigated ED visits for asthma in children. In children 1-4 yrs, 3% ↑ (95% CI 1.8-4.2) in asthma visits per interquartile range ↑ in NO&lt;sub&gt;2&lt;/sub&gt;, 1.4% ↑ (95% CI 0.7-2.1) for PM&lt;sub&gt;0.5&lt;/sub&gt; &amp; PM&lt;sub&gt;2.5&lt;/sub&gt;. Similar PM&lt;sub&gt;0.5&lt;/sub&gt; &amp; PM&lt;sub&gt;2.5&lt;/sub&gt; results for those aged 5-9 yrs. For 10-14 yr olds, 1.2% ↑ (95% CI 0.01-2.5) for PM&lt;sub&gt;2.5&lt;/sub&gt;; 2.8% ↑ (95% CI 0.8-4.9) for CO. Effects greater in the warm months for all pollutants except CO.</td>
</tr>
<tr>
<td>Johnston, F. et al. 2002&lt;sup&gt;105&lt;/sup&gt;</td>
<td>Darwin 2000</td>
<td>PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Found a significant ↑ (adjusted RR 1.20 (95% CI 1.09-1.34)) in asthma emergency presentations with each 10ug/m&lt;sup&gt;3&lt;/sup&gt; ↑ in PM&lt;sub&gt;10&lt;/sub&gt; during the bushfire season. Greater association on days when levels were &gt; 40ug/m&lt;sup&gt;3&lt;/sup&gt; (adjusted RR 2.39 (95% CI 1.46-3.90)) compared to baseline days of PM&lt;sub&gt;10&lt;/sub&gt; &lt; 10ug/m&lt;sup&gt;3&lt;/sup&gt;.</td>
</tr>
<tr>
<td>Johnston, F. et al. 2006&lt;sup&gt;47&lt;/sup&gt;</td>
<td>Darwin 2004</td>
<td>PM&lt;sub&gt;2.5&lt;/sub&gt;, PM&lt;sub&gt;10&lt;/sub&gt;, pollen, spore counts</td>
<td>In a panel of 251 adults &amp; children over a 7 month period, found particulates from vegetation fires (PM&lt;sub&gt;2.5&lt;/sub&gt; &amp; PM&lt;sub&gt;10&lt;/sub&gt;) were associated with the onset of asthma symptoms, daily symptom count &amp; medication use, but not with severe outcomes of asthma attacks, missed school/work due to asthma, or health service attendance.</td>
</tr>
<tr>
<td>Johnston, F. et al. 2007&lt;sup&gt;48&lt;/sup&gt;</td>
<td>Darwin 2000, 2004, 2005</td>
<td>PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Found positive relationship between PM&lt;sub&gt;10&lt;/sub&gt; from vegetation fires, &amp; mainly respiratory rather than CV admissions (OR 1.08, (95% CI 0.98-1.18)). Larger effect observed in Indigenous persons (OR 1.17 (95% CI 0.98-1.40)) in whom there was also an association with ischaemic heart disease (OR 1.71 (95% CI 1.14-2.55)) at lag 3.</td>
</tr>
<tr>
<td>Petroeschevsky, A. et al. 2001&lt;sup&gt;34&lt;/sup&gt;</td>
<td>Brisbane 1987-1994</td>
<td>b&lt;sub&gt;20&lt;/sub&gt;, NO&lt;sub&gt;2&lt;/sub&gt;, SO&lt;sub&gt;2&lt;/sub&gt;, O&lt;sub&gt;3&lt;/sub&gt;</td>
<td>Found that O&lt;sub&gt;3&lt;/sub&gt;, particles &amp; SO&lt;sub&gt;2&lt;/sub&gt; were associated with respiratory hospital admissions. A 1 unit ↑ in b&lt;sub&gt;20&lt;/sub&gt; resulted in a 0.5% ↑ in respiratory admissions in 15-64 age group. Estimated that O&lt;sub&gt;3&lt;/sub&gt; was associated with 4-10% of asthma admissions. A seasonal association of NO&lt;sub&gt;2&lt;/sub&gt; with respiratory admissions was observed.</td>
</tr>
</tbody>
</table>
Table 1.1 (cont.): Examples of Australian outdoor air pollution studies published 2000 to 2008.

<table>
<thead>
<tr>
<th>Author</th>
<th>Scope</th>
<th>Pollutants</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ren, C. et al.</td>
<td>Brisbane</td>
<td>PM$_{10}$, O$_3$</td>
<td>Found PM$<em>{10}$ modified the effects of temperature on cardiorespiratory hospital admissions, all non-external cause mortality &amp; CV mortality. Found temperature effects were more adverse when PM$</em>{10}$ was high. No evidence of interactive effects on CV &amp; respiratory emergency visits.</td>
</tr>
<tr>
<td>Ren, C. &amp; Tong, S.</td>
<td>Brisbane</td>
<td>PM$_{10}$, O$_3$, NO$_2$</td>
<td>Reported interaction between PM$<em>{10}$ &amp; temperature on most cardiorespiratory outcomes with more adverse effects on warm days than cold days. For a 10µg/m$^3$ increase in PM$</em>{10}$, respiratory admissions decreased by 1.67% at low temperatures &amp; increased 3.84% at high temperatures. Maximum temperature modified the association of PM$<em>{10}$ with respiratory hospital admissions &amp; emergency visits, CV mortality &amp; emergency visits, &amp; all cause mortality. Joint effects of PM$</em>{10}$ &amp; maximum temperature appeared synergistic.</td>
</tr>
<tr>
<td>Rodríguez et al.</td>
<td>Perth</td>
<td>CO, NO$_2$, O$<em>3$, PM$</em>{2.5}$, bsp</td>
<td>In a cohort of 263 children followed from birth for 5 years, associations found between NO$_2$ (24h) &amp; cough (OR 1.028 (95% CI 1.002-1.055)); O$<em>3$ (8h) &amp; raised body temperature (OR 1.022 (95% CI 1.008-1.036)); CO (8h) &amp; wheeze/rattle (OR 1.089 (95% CI 0.968-1.226)); PM$</em>{2.5}$ (1h) (OR 1.006 (95% CI 1.000-1.012)) &amp; bsp (1h) (OR 1.170 (95% CI 1.019-1.344)) with cough.</td>
</tr>
<tr>
<td>Simpson, R. et al.</td>
<td>Melbourne</td>
<td>b$<em>{sp}$, PM$</em>{10}$</td>
<td>Positive associations found between PM &amp; respiratory &amp; all-cause mortality in warm season. Found a 1x $10^{-4}$ m$^{-1}$ increase in maximum bsp to be associated with 2.19% (95% CI 0.01-4.43) increase in all-cause mortality, &amp; 10.4% (95% CI 2.44-18.97%) increase in respiratory mortality. Effects of NO$_2$ &amp; O$_3$ were also significant.</td>
</tr>
<tr>
<td>Simpson, R. et al.</td>
<td>4 cities</td>
<td>b$_{sp}$, NO$_2$, O$_3$</td>
<td>Fine particles &amp; NO$_2$ were associated with CV admissions (RR 1.0856 per 1 unit increase in bsp, &amp; RR 1.0023 per 1 ppb increase in NO$_2$) for 3 out of 4 cities. Fine particles, NO$_2$ &amp; O$_3$ had significant effects on elderly respiratory admissions in 3 out of 4 cities. Particles &amp; NO$_2$ had effects on CV admissions on all cities.</td>
</tr>
<tr>
<td>Simpson, R. et al.</td>
<td>4 cities</td>
<td>b$_{sp}$, NO$_2$, O$_3$</td>
<td>Reported a significant effect of particles (b$_{sp}$) &amp; NO$<em>2$ on total mortality with RRs of 1.0281, &amp; 1.0011 per unit increase, respectively. O$<em>3$ was associated with respiratory mortality yielding an RR 1.0022 per unit increase. Meta-analysis results showed non-significant increases in daily deaths associated with PM$</em>{10}$ &amp; PM$</em>{2.5}$.</td>
</tr>
</tbody>
</table>
1.4 Temperature and air pollution associations

Prevailing meteorological conditions often influence the concentration of air pollutants in an airshed.\textsuperscript{150} The temperature-air pollution relationship is evidenced by the prevalence of photochemical smogs in the warmer months\textsuperscript{58} when, in the presence of high temperatures clear skies and light winds, pollutant levels increase\textsuperscript{58} and incoming solar radiation enhances photochemical reactions.\textsuperscript{150,151} Temperature inversions occur when warmer air in the lower atmosphere is displaced by cooler air, trapping pollutants in a blanket layer above the ground, contributing to the formation of smog.

Temperature has been considered a possible confounder in the analysis of air pollution health effects\textsuperscript{83,89} suggesting that a complex mode of interactions may exist between air pollution, weather variables and health outcomes. Some studies have found cardiovascular or pulmonary health outcomes to have significant seasonal associations with air pollution and many have found health effects to be compounded during the warm season.\textsuperscript{58,61,72,103,152} As heat stress can place strain on the heart and exacerbate the cardiotoxicity of chemicals, interactive health effects between temperature and air pollution are biologically plausible. A Sydney study found interactive effects between SO$_2$ and maximum temperature on mortality,\textsuperscript{49} whereas other studies have found no evidence of temperature-air pollution interactions.\textsuperscript{4,69}

Conversely, in a study of temperature and the occurrence of cardiorespiratory disease, Ren et al\textsuperscript{11} claimed that particulate air pollution is a confounder of the association by rendering people more vulnerable to the adverse health effects of temperature. Similarly, others have found temperature to have a significant effect on mortality with a confounding effect by PM$_{10}$ and O$_3$.\textsuperscript{47} Schwartz\textsuperscript{153} on the other hand in a study involving 14 U.S. cities, found no confounding by temperature of the ozone–mortality association.

Generally interactions between individual pollutants and the eventual effect on health outcomes are poorly understood. There may be complex relationships between NO$_2$, O$_3$ and particulates (Figure 1.5) that may be additive, antagonistic (negative interdependence) or synergistic (positive interdependence).\textsuperscript{128} A review of laboratory
studies has found evidence of synergism involving O₃, although most studies were conducted using higher than ambient concentrations.¹⁵⁴ One study investigating hospital admissions in Hong Kong found significant positive interactions “between NO₂, O₃ and PM₁₀, and between O₃ and winter months.”¹⁵⁵ (p. 679) However, overall there is limited information regarding inter-pollutant relationships and the effects on health. This area, warranting further investigation, is discussed later in the thesis.

### 1.5 Climate change

Air pollution is inherently linked to climate change and both share fossil fuel combustion as the basis for their ill-effects. The earth’s climate is naturally dependent on greenhouse gases - carbon dioxide, methane and nitrous oxide, which assist in keeping the atmosphere at an inhabitable temperature. However, industrialisation has seen an unprecedented rise in these gases with atmospheric CO₂ alone increasing by more than 30% since pre-industrial times, trapping more heat in the lower atmosphere and contributing to global warming.⁴⁵ Recent evidence has also suggested that due to its light absorbing properties, black carbon may be the second most important component of global warming after CO₂ in terms of direct radiative forcing.¹⁵⁶

An increasing body of evidence has shown that over the past century (1901-2000), anthropogenic activities have contributed to a global rise of 0.6°C (± 0.2°C) in the mean surface temperature of the earth.³³ The Intergovernmental Panel Climate Change (IPCC) project that by the end of the 21st century temperatures to relative to 1980–1999, will rise by 1.8°C- 4.0°C.¹⁵⁷ Recent observations reported at the Copenhagen Climate Change Congress in 2009, suggest that worst case scenarios are being realised or exceeded however, and that any rise above 2°C will be very difficult for societies to cope with.¹⁵⁸

Australia’s rate per person of greenhouse gas emissions is the highest in the industrialised world,⁹⁴ with sources including emissions from power stations, natural gas processing, agriculture, industry and transport.⁹⁴ Despite global and local efforts to reduce greenhouse gas emissions, the Commonwealth Scientific and Industrial Research Organisation (CSIRO) predicted in 2001 that by 2030 there would be an
increase in CO₂ concentration from 350 ppm (in 1990) to 430-455 ppm, paralleled with a 0.4-2.0°C increase in temperature over most of Australia. Adelaide currently records an average of 17 days per year over 35°C and this number is expected to rise to 29-47 by 2070 under worse case scenarios, fewer if greenhouse gas emissions are abated.

Global warming has implications for population health, both directly and indirectly. It is projected there will be an increase in the number of heatwaves coinciding with increased incidences of thermal stress and temperature-related deaths. Exacerbated by higher humidity and urban air pollution levels, rates of respiratory disease are likely to increase. The susceptible groups will be the elderly and the very young, the infirm, the socially disadvantaged and those without access to adequate air conditioning.

According to the recently published Garnaut Review, impacts on temperature related deaths and hospitalisations in Australia will vary by state. Modest reductions in cold-related deaths will be outnumbered by heat-related deaths in the second half of the 21st century. The 1,100 temperature-related deaths presently recorded each year in Australians aged over 65 years is expected to increase dramatically over time. Given a demographic shift to an older population structure, this figure could reach 4,200-8,000 deaths each year by 2100 if a strong policy action to reduce greenhouse gas emissions is adopted, higher if emissions were to continue unabated.

Population health may also be affected in other ways. Continued depletion of stratospheric ozone may result in increased exposure to UV-B radiation, a major cause of skin and eye damage. Higher incidences of vector-borne and food-borne disease are predicted with changing ecosystems, and rising sea-levels, and increases in the number of extreme weather events will impact on morbidity and mortality. Furthermore air pollution concentrations will likely be affected, with levels of PM, O₃ and aeroallergens of most concern. Population health relies on clean air and drinking water, and adequate shelter and food supplies, all of which may be impacted by a changing climate.
1.6 Methodological issues

Studies investigating the effects of temperature and air pollution on health can be analysed in a number of ways using different study designs and statistical models. The quantitative methods often used, are discussed below.

1.6.1 Study designs

In environmental epidemiology, several different types of study designs have been used to quantify health risks of exposure, and multilevel analysis is often required to address these issues. Panel studies, cohort studies and ecologic studies using time series, case-crossover or case series designs are examples of commonly used methods. Furthermore spatial analysis incorporating geographic information systems (GIS) is becoming a widely used research tool in environmental health studies, enabling analysis of place-based risk and a greater understanding of disease pathogenesis in relation to environmental exposures.

1.6.1.1 Panel studies

Used in several air pollution investigations, panel studies are prospective studies where selected groups of individuals are followed for a predetermined study period. Daily observations of individual respiratory symptoms are recorded and associations investigated with daily air pollution measures. Information gained about potential confounders on an individual level can add significantly to causal interpretation. The small scale of panel studies however, together with dependence on participants' cooperation, may make results less reliable than some other study designs discussed below.

1.6.1.2 Cohort studies

Larger scale cohort studies in which subjects are followed for considerable periods of time can provide the most complete estimates of risk, as they incorporate both long-term and short term effects. The Harvard 'six cities' study, a prospective cohort study involving 8,111 adults followed for 14-16 years, was one of the few studies to
investigate effects of long term exposure to air pollution, revealing a positive association between air pollution and death from lung cancer and cardiopulmonary disease. The American Cancer Society study incorporating data from some 500,000 adults in the United States followed from 1982 to 1998,\textsuperscript{166} found links between combustion-related air pollution and cardiopulmonary disease and lung cancer mortality. Similarly in the Netherlands, an eight year study undertaken to investigate traffic-related pollution,\textsuperscript{119} revealed higher risks of cardiopulmonary mortality for the elderly living near major roads. There have been a limited number of air pollution cohort studies conducted in Australia and these have incorporated much shorter study periods.

\subsection*{1.6.1.3 Ecological studies}

Ecological studies, by their very nature, focus on groups not individuals, and use aggregate measures rather than individual-level data.\textsuperscript{167} These studies do not take into account individual characteristics or exposures, but instead make the assumption that exposures are uniform. Some methodological issues limit causal inference in ecological studies however. In addition to the ‘ecologic fallacy’ issue where generalisations at population level can lead to incorrect inferences at the individual level,\textsuperscript{164} there may be a lack of adequate data, or temporal ambiguity with unknown latent periods between exposure and health outcome.\textsuperscript{167}

The analysis of population level data can be approached in a number of ways including methodologies such as time series, case-crossover design and case series techniques. Commonly used health outcome measures include daily hospital admissions and mortalities, which, being relatively rare events in a population, infer a Poisson distribution\textsuperscript{62,111,168} and hence aggregate counts are often modelled by Poisson regression to assess relative risks.

\section*{Time series}

Time series design involves regressing daily mortality (or morbidity) counts on daily air pollution\textsuperscript{169} or temperature observations ordered in time. Regression models are used to predict the expected number of outcomes as a function of exposure levels.\textsuperscript{170} The benchmark study in air pollution literature was the ‘Air Pollution on Health: a European
Approach’ (APHEA) project incorporating a meta-analysis of data collected in 15 European cities with a total population of 25 million people.\textsuperscript{76,168} One of the objectives of the APHEA study was to formulate a standard methodology for statistically analysing the relationship between air pollution and health effects data.\textsuperscript{168,171} The authors chose an epidemiological time-series design incorporating an autoregressive Poisson regression model to provide relative rate estimates.\textsuperscript{168}

An issue of concern in time-series as in other study designs used in environmental epidemiology is the inability of the model to adequately control for confounders such as trends and seasonality.\textsuperscript{62,90,168} The APHEA authors acknowledged there were some modelling flaws\textsuperscript{76} and in a subsequent larger study (APHEA2) incorporating 29 cities with 43 million inhabitants, addressed the issue of unmeasured confounders such as influenza epidemics, day of the week, holidays, seasonality and long-term trends, using modified methods. These involved a hierarchical approach with regression models to control for confounders in each city, followed by a second stage analysis with univariate or multivariate models to summarise individual city results and investigate possible effect modifiers.\textsuperscript{84} Whilst suitable for ascertaining acute effects of recent exposure, time series studies may underestimate the effect of air pollution on life-span and attributable deaths, due to their inability to adequately quantify chronic effects of long-term exposure.\textsuperscript{68,169}

Risk estimates in these studies are sensitive to subtle differences in statistical techniques. Such is the concern over methodologies that data from some of the major air pollution studies have been re-analysed\textsuperscript{172-175} refining the use of generalised additive models (GAMs) that incorporate non-parametric smooth functions to control for confounders.\textsuperscript{11,93,97,173,176,177} Default settings for GAMs in some statistical packages gave rise to biased standard error estimates\textsuperscript{93,172} and thus inaccurate estimates of risk.\textsuperscript{176} Natural cubic splines, smoothing functions which make no assumptions regarding the relationship between variables, have been used instead in attempts to overcome these problems.\textsuperscript{9,93,172,174}

As well as air pollution studies, investigations using time series analysis have been undertaken to estimate the temperature-health relationship\textsuperscript{2,10} where controlling for
seasonality and potential effect modification by air pollution are important considerations.

**ARIMA models**

Autoregressive integrated moving average (ARIMA) models\textsuperscript{178,179} can be fitted to the data in a time series design. These have been shown to be comparable to Poisson regression models in studies investigating seasonality\textsuperscript{179} and the health effects of heat.\textsuperscript{23,50,180,181} Seasonal ARIMA (SARIMA) models have been used in studies associating the effects of climate variations and infectious diseases.\textsuperscript{182,183}

**Case-crossover design**

Although time-series design was previously the commonly used method of analysis for air pollution-health relationships, recently case-crossover analysis has gained popularity.\textsuperscript{176} The case-crossover method was first introduced by Maclure in 1991\textsuperscript{184} as an analytical method for studying the transient effects of acute exposure where the exposures leading up to a health event (case period) are compared to exposures at one or more periods nearby in time when the event did not occur (control periods).\textsuperscript{93,185,186}

Results can be sensitive to the selection of control periods, particularly if time trends are present in the data.\textsuperscript{185} Unidirectional, bidirectional and time stratified methods have all been employed in the case-crossover approach. The original concept incorporated a unidirectional retrospective design where a single control period preceded the event,\textsuperscript{176,184} but this was found to give inconsistent results due to uncontrolled confounding from time trends in exposure.\textsuperscript{10,186} Bidirectional design incorporates control periods temporally equidistant prior and post event, avoiding bias and controlling better for longer time trends.\textsuperscript{10,93,176} This can be used whenever (a) the health-defining event does not affect subsequent exposure and (b) it is known what the exposure would have been had the outcome not occurred, as is the case in air pollution or temperature studies when measurements are not affected by instances of morbidity or mortality.\textsuperscript{186} The symmetric bidirectional case-crossover design is synonymous to a locally weighted running-mean smoother in time-series analysis, showing that the two methodologies have some inherent similarities.\textsuperscript{170} Finally time stratified case-crossover designs organize data into time periods (strata), nominating all non event times in the
strata as the control period or matching case days to nearby control days. Some match on same days of week within the calendar month (e.g. if the case day is a Tuesday the control days become every other Tuesday within that month and year), others use strata of varying lengths.

A study of temperature related mortality in 20 U.S. cities compared time-series analysis to case-crossover design with varying referent period selection: unidirectional with the control period seven days prior; bidirectional with the control periods seven days before and after the event; and time-stratified with up to four control days selected at seven day intervals during the month (strata) the event occurred. The authors found the bidirectional and time-stratified case-crossover analyses gave quantitatively similar results to the time-series analysis, but unidirectional case-crossover analysis was unreliable giving comparatively overestimated or underestimated odds ratios. Time-stratified designs are preferred as they provide unbiased conditional logistic regression estimates and avoid bias from time trends in the data.

Some authors claim that case-crossover design has lower statistical power than that of time-series models as the standard error of the mean of the regression coefficient is larger and often sample sizes are smaller. Additionally it is only suitable for estimating effects which are acute and transient and will not identify harvesting trends. Nevertheless mathematically the two are very similar and when there is common exposure in the population, the conditional logistic regression estimating equation in case-crossover analysis, equates to the log-linear estimating equation in time series analysis. Case-crossover design is discussed further in Chapter 2.

Case series

Similar to the case-crossover design is the self-controlled case series method designed to analyse acute outcomes of transient exposures. No controls are needed as only cases are used. Within specific observation periods the whole non-exposure period becomes the control period and analysis compares the rate of events in the exposure period to the rate in the non-exposure period. The method is particularly relevant when all members of the population have the same exposure and is thus suitable for use in environmental epidemiology. Further discussion regarding case series methods appear in the following chapter.
1.6.2 Confounding and bias

A multitude of direct and indirect extraneous factors can complicate efforts to accurately quantify population-based cause and effect relationships attributed to environmental exposures. Prevailing climatic conditions for instance, can influence concentrations and mixtures of ambient pollutants. Higher temperatures will increase chemical reactions in the atmosphere influencing levels of photochemical oxidants and precursor gaseous compounds. Wind can act as a dispersion agent for pollutants or cause turbulence, increasing particulate and aeroallergen concentrations. Humidity and precipitation will affect atmospheric water soluble compounds. Seasonal changes affect infection rates and personal activity including time spent indoors. Similarly weekends and holiday periods will affect day-to-day lifestyle. Over time, pollutant loads may be subject to temporal changes attributable to governmental policies limiting industrial or transport emissions. Domestic indoor air quality varies according to sources of heating and cooking, dust, and presence or absence of tobacco smoking. Occupational exposures may also be a confounder. Finally, pollutants that share the same emission sources may have effect modifying or synergistic effects. Well designed epidemiologic studies of the weather-health and air pollution-health associations incorporate into their study designs adequate control for confounders such as co-pollutants, temperature and other weather variables, season, day of the week, school holidays and influenza epidemics.

In both air pollution and temperature studies, the main source of information bias arises from misclassification of exposure when using data from central monitors as a surrogate for personal exposure levels. Pollutant concentrations or temperatures recorded at centrally sited ambient monitors may not accurately reflect personal exposure levels if located some distance away or if individuals spend most time indoors. Using aggregated data on a population basis reduces the risk of this bias.

Zeger et al discuss three components of measurement error as being the difference between:

- an individual’s exposure and their risk-weighted average personal exposure (based on person-specific baseline risks)
- the measured and true ambient levels (includes instrument error)
- average personal exposure and the true ambient level.

The authors claim only the latter is a potential source of substantial bias and that in air pollution studies, the use of ambient particulate concentrations may underestimate pollution effects. Additionally in time series studies the day to day correlation between air pollution measures can lead to autocorrelation in inaccurately proposed models, implying confounding of air pollution associations.\textsuperscript{171}

Finally, in studies investigating disease-specific health outcomes, inaccuracies and bias may occur with the misclassification of ICD codes for disease diagnoses. This may occur at the hospital level, particularly when individuals present with multiple medical conditions, or during data analysis when discrepancies in classifications can occur. Random errors however should not unduly bias findings. As an example, a brief examination of a selection of six Australian and New Zealand studies shows at least four variations in the classification of cardiovascular disease.\textsuperscript{4,11,111,124,125,194} This inconsistency could potentially skew results and make comparisons between study findings problematic.

In studies investigating the effects of heat, underreporting using ICD coding is a major issue\textsuperscript{28} causing inaccuracies in estimates. This stems from the lack of a uniformity in classifications and the fact that the current classification of heat-related death requires the measurement of body temperature at, or just after the time of death,\textsuperscript{195} which clearly precludes heat deaths where a medical practitioner is not in attendance, and those where heat has exacerbated a prior condition. For increased reliability in epidemiological studies, the WHO\textsuperscript{68} recommends using all-cause mortality, rather than cause-specific data when available.

### 1.7 Gaps in current knowledge

This review has shown that current literature confirms the notion that air pollution and extreme temperatures have non-negligible effects on population health, with increased morbidity and mortality rates in the elderly and other at risk sub-groups. Despite the large number of published studies relating to environmental exposures, many
knowledge gaps in the literature exist, highlighting several areas amenable to future research.

Firstly the biological mechanisms underlying the complexity of cardiovascular events triggered by air-pollution are yet to be fully understood, as are the associations with fine and ultra-fine particulates. Additionally very little information exists regarding the chronic health effects of long-term exposure to air pollution, an issue identified by the WHO as being a major research gap. In particular, there is a need for long term Australian cohort studies in this area.

Most of the overseas air pollution-health and temperature-health epidemiological studies reviewed have used mortality as the outcome variable, with relatively few using morbidity. The paucity in information regarding hospital admissions, emergency department visits and ambulance callouts in response to varying environmental conditions is surprising as these may be more representative of recent outdoor exposure than deaths that often occur within climate controlled health care facilities. In Australia population acclimatisation to harsh summer weather results in much lower mortality rates during heatwaves than in countries where extreme heat is rare. Hence morbidity studies may be a more appropriate health indicator. More research is therefore required into characterising non-fatal heat-related illnesses.

With an increase in heatwave frequency expected in the coming decades, it is important that strategies be in place for mitigation of the consequences to population health of climate change. Adaptation will be required as the climate continues to change and consequential health effects accrue. Further research on adaptation and mitigation strategies, particularly in vulnerable populations, is imperative as heat events become more extreme. Furthermore determinants of vulnerability require further investigation. Whilst it is usually the aged, the infirm and the very young who are considered most vulnerable, closer surveillance of temperature-related illnesses during extreme heat may identify other susceptible sub-populations and areas at risk. The health consequences of Adelaide’s rare and extreme temperatures in the summer of 2009 indicate that many aspects of heat-health urgently require investigation to avoid preventable loss of life during heatwaves.
Finally, there is little information about the effect of temperature on the air pollution-health relationship. A substantial body of literature focuses on single and multi-pollutant models whilst controlling for the effects of weather and seasonal trends, however few studies have examined the interaction between air pollution and weather variables such as temperature, humidity and wind speed, on the toxicity of airborne pollutants. Further research is required to identify modifying factors but to date no comprehensive study relating meteorological and air quality data to daily mortality and morbidity has been undertaken. With a warmer year round climate, people may spend more time outdoors and exposures to ambient air pollution may increase. For reasons which include photochemistry effects, increases in air pollution concentrations are likely during summer heatwaves as previously observed during heatwaves in Britain and France. Extreme and/or persistent heat together with predicted extended droughts in Australia may contribute to more days when dusty conditions and/or bushfires prevail, giving rise to high concentrations of airborne particulates. To date correlations between heatwaves and air pollutant levels in South Australia are yet to be defined and further epidemiological research needs to be undertaken to address these gaps in knowledge.

These examples are just a few of the many areas of environmental health research that are amenable to further investigation in order to mitigate the effects of climatic and air quality changes on an ageing and thus more susceptible population. Other possible future research directions are discussed in Chapter 10.
Chapter 2

Study design and methodology

2.1 Introduction

This chapter describes the geography and demographics of the study region, and outlines the overall aims and objectives of the thesis. The research questions are stated and the framework of the study is explained together with an overview of study design and justification of methodologies used in analyses.

2.1.1 Background

As previously mentioned, numerous epidemiological studies over the last two decades have demonstrated adverse health effects associated with urban air pollution and extreme weather conditions. Effects of acute exposure to air pollution can result in conditions ranging from mild irritations of the eyes, nose, or throat to severe exacerbation of respiratory illness and cardiovascular disease, and extreme weather events have been shown to be associated with fluctuations in hospital admission and mortality rates. A predicted rise in average global temperatures combined with occasional decline in air quality could see the health effects of weather and air pollution become a significant public health issue in years to come, particularly in susceptible subgroups of the population such as the elderly.

Most of the major studies investigating the health effects of air pollution and extreme weather have emerged from the Northern Hemisphere, including studies from the United States, Europe, and Asia. Fewer investigations have been carried out in Australia where air quality, population exposures and climatic conditions are likely to differ from those of the U.S. and Europe.
Studies have been undertaken in Australian cities as summarized in Chapter 1, although to date few have taken place in South Australia. Furthermore meta-analyses incorporating health-outcome data from Australian capital cities\textsuperscript{69,83,85,104,125} have been lacking in Adelaide data. The heterogeneity in findings between cities may be a result of variations in local pollutant characteristics and meteorological conditions and it is ill-advised to extrapolate findings from one city and assume they will be valid in another. Indeed with its dry temperate climate, Adelaide may demonstrate notable differences in environmental health responses from other areas in Australia or elsewhere. It is therefore timely that the public health implications of air pollution and weather in Adelaide be available to researchers, public health professionals, policymakers and even the local community, to enable pre-emptive planning for climate change-related variations in population health trends. This thesis addresses these issues in the forthcoming chapters.

It is relevant to note that the time writing of this thesis (2009) coincides with the first year the National Health and Medical Research Council has included climate change in its funding guidelines. In acknowledgement of this important issue, the federal government has allocated $10 million for research into the effects of rising temperatures on human health.\textsuperscript{203} It is anticipated that the findings from this thesis may contribute to that knowledge base.

2.1.2 The study region

Adelaide, the capital city of the state of South Australia, is situated near the coast in central southern Australia (Figure 2.1A). Lying at latitude 34°55’ S, longitude 138°35’ E,\textsuperscript{56} Adelaide is situated wholly within the temperate zone. The Adelaide metropolitan area (Figure 2.1B) lies on a plain with hills to the east and the coast to the west, extending 90 km from Gawler in the north to Sellicks Beach in the south.\textsuperscript{204} The city’s 1.1 million inhabitants in 2006 comprised 73.1% of the state’s total population. South Australia has the oldest populations in Australia, with a median age of 38.8 years compared to 36.6 years for the national average.\textsuperscript{205} A large proportion of the Adelaide metropolitan population (14.8%) is aged 65 years or over, and in accordance with trends elsewhere, the population is ageing.\textsuperscript{206} By 2050 it is predicted that approximately
30% of the population will be at least 65 years of age. Elderly persons (60 years or over) tend to live close to the city with most residing within a 12 km radius of the city centre.

Adelaide’s most common sources of air pollution are from motor vehicles and domestic wood fires in winter. The Adelaide airshed is bounded by Gulf St Vincent to the west, the Mt Lofty Ranges to the east and south, and is open to the north (Figure 2.2). Pollution forming over the city in the morning is moved by easterly winds out to the gulf until late afternoon when afternoon sea breezes recirculate the urban plume back over the city and as far north as Elizabeth and Gawler, bringing with it high concentrations of O₃, PM, and other pollutants. Air pollution in Adelaide is exacerbated by the presence of temperature inversions when pollutant-rich cool air normally in the upper atmosphere sinks to ground level, making conditions ideal for the formation of photochemical smog. Despite greenhouse gas emissions in the state increasing by...
7.8% since 1995, Adelaide’s air quality is reportedly improving, due in part to decreases in CO and SO₂ concentrations over recent years. 

NOTE: This figure is included on page 45 of the print copy of the thesis held in the University of Adelaide Library.

Adelaide has unique and variable climatic conditions with hot, dry summers with an average of 17 days per year of temperatures above 35°C, and mild winters with low annual rainfall, making the state the driest on the continent. Heat and aridity influences infiltrate mostly from the north, whereas moisture and coolness generally come from the south. Climate change is encroaching on Adelaide as evidenced by rising ambient temperatures since the middle of the twentieth century (Figure 2.3). South Australia’s average maximum temperature since 1950 has increased at a faster rate than the national average: 0.17°C per decade compared to 0.13°C per decade nationally. Models predict that heatwaves will become more frequent, and these may be accompanied by an increase in the incidence of heat stress and heat-related mortalities particularly in the elderly.
2.2. Aims and objectives of the study

The aim of the study was to quantify the effect of climate variations and air pollution on short term health outcomes in Adelaide using existing datasets, and to use the findings, together with those of other studies, to formulate an evidence-based risk assessment of the public health impact of changing environmental conditions on Adelaide’s population.

The objectives of the study are:

- To investigate the effect of heatwaves on cause-specific morbidity in Adelaide with a focus on hospital admissions and ambulance callouts.
- To investigate the independent effect of air pollution on acute cause-specific and all-cause morbidity.
- To investigate the interactive effect of weather and air pollution on morbidity and mortality.
- To use current and previous epidemiological evidence to formulate a health risk assessment for environmental stressors in Adelaide.
- To inform public health policy concerning extreme weather and air pollution.
2.3 Research questions

- What are the individual and combined effects of variations in weather and urban air pollution on population health outcomes in Adelaide?
- Based on what we know what effect will changing environmental conditions have on the health of Adelaide’s population?

2.4 Framework of the study

This study broadly addresses the association between exposure to environmental determinants and several health outcomes including all-cause and cause-specific morbidity and mortality.

The study area is restricted to metropolitan Adelaide and the study incorporates health surveillance data obtained from the South Australian Department of Health. Morbidity data in the form of daily hospital admissions, emergency department hospital visits and ambulance callouts are used although it is acknowledged that these are merely a proxy for morbidity, representing only a fraction of total illness in the community. In some studies mortality data are also used.

The studies within this thesis progress in a somewhat hierarchical fashion, beginning with an expansion of findings from a previous study by Nitschke and colleagues.\textsuperscript{31} In this study of morbidity and mortality in Adelaide during heatwaves, an association was found with certain disease-specific health outcomes and ambulance callouts. Hence the first part of this thesis investigates these findings in more detail and, highlights possible heat-sensitive sub-diagnoses and threshold temperatures. Detailed analyses of ambulance callouts and emergency department visits investigate spatial heterogeneity in thermal susceptibility within metropolitan Adelaide.

The second part of the thesis investigates the impact of air pollution on health outcomes. Firstly, the effect of particulate matter with an aerodynamic diameter less than 10 microns (PM\textsubscript{10}) and less than 2.5 microns (PM\textsubscript{2.5}) on cause-specific hospitalisations is quantified, using case cross-over analysis adjusting for ozone, nitrogen dioxide and temperature. The final analyses investigate the interactive effects
of pollutants using two pollutant models and an interactive term, whilst accounting for temperature. Using the evidence from these studies and previously published literature, a risk assessment is undertaken for Adelaide using a climate change scenario. Findings will likely apply to a broader perspective than purely locally. A diagrammatic representation of the overall framework of the study is shown in Figure 2.4.

Figure 2.4: Flowchart outlining the framework of the study
2.5 Methodology used in the study

2.5.1 Data collection and analysis

De-identified morbidity and mortality data sourced from the South Australian Department of Health, Health Statistics section are used in the study, and include cause-specific and all cause hospital admissions, deaths and ambulance callouts. Additionally, climatic data (daily maximum and minimum temperatures and relative humidity) sourced from the Bureau of Meteorology, Kent Town station, centrally situated in metropolitan Adelaide, are used, as well as daily air pollution concentrations accessed from the Environment Protection Authority (South Australia), Netley site.

2.5.2 Heat-health studies

The case series method was chosen for the heatwaves studies. As mentioned in the previous chapter, this method is designed to analyse acute outcomes of transient exposures where the rate of events in an exposure period is compared to the rate of events during a period of non-exposure\(^1\)\(^9\)\(^2\) and exposure levels are the same across the population. Effects of seasonality are modelled non-parametrically whilst time trends are incorporated in the parametric part of the model.\(^2\)\(^1\)\(^0\) This method which is equivalent to a log-linear time series model and incorporates aspects of case-crossover design, avoids the complex data management issues of case-crossover (outlined below). Requiring only cases, it is highly suited to certain types of data including hospital databases\(^2\)\(^1\)\(^0\) and has been used in heat-health studies previously to compare rates of events during heatwaves to rates during non-heatwave periods.\(^3\)\(^1\) Case series analysis incorporating Poisson regression models (or negative binomial regression models if required), was therefore the preferred method of analysis for the heatwave studies in Section II of this thesis.

2.5.3 Air pollution studies

As outlined in the previous chapter, there is a substantial and growing body of evidence pointing to an association between PM and adverse cardiovascular and respiratory health outcomes. With PM being a pollutant of concern in the Adelaide airshed,\(^2\)\(^1\)\(^1\) it is
timely that its effects on the disease burden in Adelaide be investigated within this thesis.

Case-crossover design, widely used in air pollution epidemiology, was the method chosen for use. In case-crossover design the study population is subjects who have experienced a health outcome of interest (e.g. hospital admission or death), and inference is placed on a comparison of exposure distribution. Only subjects with different levels of exposure between hazard and control periods are informative.\(^{212}\) Controls are not required as subjects act as their own controls, and individual susceptibility factors such as age, gender and smoking status can be inherently controlled for by design,\(^{170}\) eliminating the effects of confounding by characteristics both known and unknown that remain unchanged.\(^{10,69,184}\) Whereas time series uses modelling to control for confounders, case-crossover does so by design.\(^{190}\) An effect estimate is calculated by dividing the number of subjects exposed during the hazard period by the number exposed during the control period.\(^{212}\) As mentioned previously, results can be sensitive to the selection of control periods and hence the time-stratified method is preferred as it avoids bias from time trends in the data and provides unbiased regression estimates.\(^{190}\) Conditional logistic regression analysis is typically used to calculate risk estimates.\(^{10,57,69,71,86,103}\)

In summary, as case-crossover eliminates confounding by stable individual characteristics,\(^{212}\) and avoids time trends and biased estimates,\(^{190}\) it was considered a robust method to use in these analyses. Furthermore a respected and experienced user of the method was willing to provide statistical guidance in its use within this thesis. For these reasons it was the methodology chosen for use in the air pollution studies.

The data management required for a case-crossover analysis is however complex, and relevant details are scarce in the literature. Prior to analysis, data need to be assembled in a format where exposures and outcomes (health events) on an ‘index’ day are matched with those on control days. Tables 2.1 and 2.2 give a simple example of the required data formatting using a fictional dataset listing deaths and PM\(_{10}\) exposures. Although each day of the dataset is eventually considered an index day, in this example, day 9 of the 28-day strata is used as the index day (Table 2.1). To reduce correlation
between index days and control days there is a two day exclusion window either side of the index day, leaving days 1-6 and days 12-28 as controls.

Observations for the control days need to be aligned with those of the index day necessitating the merging of an expanded ‘case’ dataset with a ‘control’ dataset for the final dataset as in Table 2.2, which includes a dummy variable identifying cases and controls. Exposures on index days and control days are compared, and an estimate of risk is weighted according to the number of health events on the respective days.

In the air pollution studies presented here, models for PM$_{10}$ and PM$_{2.5}$ were run separately to avoid multicollinearity and were adjusted for potential confounders such as day of the week and public holidays, as well as NO$_2$ and O$_3$ that may be on the same causal pathway as PM. Matching on the level of another pollutant or on temperature was not included in the analyses.

In the final study, interaction between pollutants and between pollutants and temperature was investigated using two pollutant models and by adding an interactive term separately. Confounding by temperature was evaluated by using cool and warm seasons and incorporating temperature into the model. Single and multipollutant models allowed adjustments for possible confounding pollutants.

### 2.5.4 Health risk assessment

A risk assessment for the effects of environmental stressors (high temperatures and air pollution) on population health in Adelaide was undertaken using the enHealth Council of Australia risk assessment paradigm, and incorporated knowledge acquired throughout the thesis. Although designed for Adelaide as per the specifications of this thesis, the implications of the risk assessment and indeed other findings within this thesis can be applied to a much broader context than South Australia, with possible relevance nationally or internationally. It is likely that climatological, air quality and demographic trends occurring locally parallel those occurring in many other cities in Australia and overseas, and it is not unreasonable to assume health risks associated with environmental changes may also be comparable.
Table 2.1: Fictional example data set for case-crossover analysis.

<table>
<thead>
<tr>
<th>Day</th>
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<th>PM$_{10}$</th>
<th>Status</th>
<th>Strata</th>
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<tbody>
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<td>1</td>
<td>21</td>
<td>28</td>
<td>Control</td>
<td>1</td>
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<tr>
<td>2</td>
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<td>Control</td>
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</tr>
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<td>4</td>
<td>15</td>
<td>26</td>
<td>Control</td>
<td>1</td>
</tr>
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<td>5</td>
<td>18</td>
<td>25</td>
<td>Control</td>
<td>1</td>
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<td>Control</td>
<td>1</td>
</tr>
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<td>Excluded</td>
<td>1</td>
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<td>Excluded</td>
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<td>25</td>
<td>55</td>
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<td>21</td>
<td>40</td>
<td>Next strata</td>
<td>2</td>
</tr>
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<td>23</td>
<td>41</td>
<td>Next strata</td>
<td>2</td>
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Table 2.2: Exposure on the index day from Table 2.1, is compared with that of control days.

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<th>Control days</th>
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</thead>
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SECTION II

THE EFFECT OF EXTREME HEAT ON HEALTH IN ADELAIDE
This section focuses on the effect of extreme heat on population health in Adelaide. Each summer, hot spells cause concern for local emergency services and health care providers, particularly when temperatures remain high for several consecutive days. If overnight temperatures also remain high, there is little opportunity for relief from the heat. General health can be affected as a result, and for some, the consequences can be serious.

The saturation of air conditioning is quite high in South Australia, although power outages (unscheduled or scheduled) occasionally occur due to system overload in the heat, cutting power to households across the metropolitan area. Also, for a variety of financial and behavioural reasons, the presence of cooling devices does not guarantee their usage by householders, particularly those who are elderly. During extreme heat, susceptible populations are at higher risk of heat-illnesses as are persons involved in work, school or recreational activities outdoors or in non-air conditioned environments. With a changing climate and an ageing local population, the heat susceptibility of vulnerable subgroups in Adelaide requires investigation in order to formulate targeted mitigation strategies to decrease the public health burden of heat-related illnesses.

The first chapter in this section (Chapter 3) investigates the effect of heatwaves on renal disease admissions whilst Chapter 4 is a study of the association with mental health. Chapters 5 and 6 investigate heat-susceptible populations with a study of ambulance usage and hospital emergency department visits, respectively.
Chapter 3
The effect of heatwaves on renal morbidity

3.1 Introduction

This chapter addresses the effect of heat on renal disease, one of the health outcomes previously identified as being of interest in heat-health investigations. To further explore this association, a new study was conducted with the focus specifically on renal disease. If a causal link is to be considered, an understanding of biological plausibility is necessary to validate potential epidemiological findings. In the case of heat and the onset of renal disease, the role of the kidneys and renal system in the regulation of core body temperature requires clarification.

The role of the renal system in thermophysiology

It is well known that during periods of extremely high ambient temperatures individuals may fall victim to the heat, suffering heat-related conditions including hyperthermia and heat stress. If the body’s thermoreceptors detect a rise in body temperature of less than one degree above 37°C, information is conveyed to the thermoregulatory centre in the hypothalamus which acts as a thermostat, regulating production and loss of heat. In an attempt to maintain homeostasis, peripheral blood vessels dilate, increasing blood flow and warming the skin so that heat can be lost rapidly by radiation, convection and conduction. Sweating is initiated through the cholinergic pathways allowing cooling via evaporation. In susceptible individuals, high ambient temperatures can overwhelm the body’s ability to dissipate heat and hyperthermia can occur if heat gained exceeds heat lost. Sweating without adequate fluid replacement can deplete the body of water
and salt, resulting in dehydration. Defined as a loss of body water of more than 3%, dehydration can compromise the thermoregulatory processes and may be associated with early signs of heat related illnesses. Heat exhaustion for example, results from either water deprivation or salt depletion (hyponatraemia).30

The circulatory adjustments involved in thermoregulation place stress on the renal system.19 The increased blood flow to the skin from 0.2-0.5 litres per minute under normal conditions to 7-8 litres per minute under severe heat stress conditions, leads to a reduced blood flow to other body areas including the kidneys.16 In response to sweating, the production of aldosterone by the adrenal cortex, and antidiuretic hormone by the hypothalamus, limits urine production.44,217 This retention of water and waste products may cause hypernatraemia (high serum sodium levels) and anuria,218 with progression to severe renal impairment and subsequent organ damage.219 Renal insufficiency can also result in other electrolyte derangements including hyponatraemia, hyperkalemia and hypokalaemia, all of which have been reported in heatstroke victims.26

Abnormalities in kidney function can be detected diagnostically by tests including serum urea or creatinine levels, indicative of waste products retained in the bloodstream, and the glomerular filtration rate (GFR), a measure of the volume per minute of blood cleared by the kidneys.16

**Renal disease and heat exposure**

Several studies have reported increases in hospital admissions for renal dysfunction in association with periods of high ambient temperatures.8,19,20,22,220,221 During August 2003, the extreme heatwave across Europe resulted in high numbers of deaths in many countries including France where some 14,729 excess deaths occurred.35 In Paris alone for instance, there were 4,866 excess deaths, 82% of whom were aged over 75 years.7 Many of these elderly decedents reportedly showed evidence of dehydration and renal failure42 and it was observed in one study that heatstroke patients with renal dysfunction had lower survival rates than those without renal dysfunction.26 Similarly in Italy, studies showed that all patients affected by hyperthermia experienced renal failure shortly after admission during the heatwave222 and that severe renal diseases were in fact amongst the prominent causes of excess mortality among the elderly.40 A similar
study in the U.S. investigating the 1995 Chicago heatwave identified that acute rather than chronic renal failure was responsible for the observed increase in renal disease.\textsuperscript{19}

Acute renal failure (ARF), defined diagnostically by abnormal levels of urine output, GFR and serum creatinine, is a serious and often fatal condition where the decline in GFR leads to retention of waste products. Hot weather induced ARF has been reported in several studies\textsuperscript{20,21,23} including an investigation of the recent 2006 Californian heatwave in the U.S., during which emergency department visits were significantly increased for ARF, electrolyte imbalance and nephritis.\textsuperscript{29}

Those at risk of developing renal failure during hot weather include the elderly\textsuperscript{30,42} and those with chronic medical conditions.\textsuperscript{223} Persons with diabetes often have pre-existing kidney conditions\textsuperscript{224,225} making them particularly vulnerable to heat-induced renal dysfunction. In diabetes, less water is resorbed in the kidney - in diabetes mellitus due to elevated glucose concentrations, and in diabetes insipidus due to low concentrations of antidiuretic hormone necessary for water reabsorption.\textsuperscript{217} Severe ARF has been found to occur more often in males, older patients, the critically ill and those with pre-existing comorbidities. Other risk factors for ARF include alcoholism, previous cardiopulmonary disease, connective tissue disorder and cancer.\textsuperscript{225}

Australia regularly experiences extreme heat during summer and predictions suggest climate change will result in an increased frequency of heatwaves.\textsuperscript{157,197} However to date there have been few reports in the literature about heat-aggravated illnesses in Australia and despite the biological plausibility, none addressing heat-triggered renal disorders. It has been reported that during 1999-2000, some 11% of Australian adults aged at least 25 years of age had evidence of reduced kidney function,\textsuperscript{226} a factor that could predispose individuals to heat-associated renal insult. This study therefore aimed to determine if, in a temperate climate, a relationship exists between exposure to extreme heat and renal morbidity. The objectives are to investigate hospital admissions for renal disease and acute renal failure during heatwaves compared to non-heatwave periods, and to evaluate the influence of relevant comorbidities.


3.2 Methods

This study was undertaken using data from Adelaide, South Australia. As seen in Figure 3.1, the warmest months are January and February with mean maximum temperatures of approximately 29°C. Temperatures equalling or exceeding 40°C are recorded on average three days per year \(^{227}\).

\[
\text{NOTE:} \\
\text{This figure is included on page 59 of the print copy of the thesis held in the University of Adelaide Library.}
\]

\textbf{Figure 3.1:} Monthly mean maximum temperatures in Adelaide and days over 40°C. \\

Hospital admission data for the Adelaide metropolitan area were obtained for the period 1 January 1995 to 31 December 2006 from the South Australian Department of Health. Discharge diagnoses were accessed using the Integrated South Australian Activity Collection (ISAAC), an official collection of admitted patient activity in the State’s public and private hospitals.\(^{228}\) Data relating to individuals who were admitted outside of the Adelaide metropolitan area were excluded from the study.

Diseases were classified according to the specifications outlined by the WHO in the International Classification of Diseases 10\(^{th}\) revision\(^{229}\) (ICD-10). For admissions occurring earlier in the study period, coding was reclassified from the earlier revision (ICD-9) to ICD-10. Daily counts of admissions with principal discharge diagnoses of renal disease (N00-N39) were accessed. In an attempt to characterise renal morbidity during heatwaves, ARF (N17) and renal dialysis not otherwise specified (Z49.1) were
included as being representative of acute and chronic disease aetiology respectively. It was expected that renal dialysis admissions would largely be scheduled rather than emergency admissions. Chosen on the basis of biological plausibility and previous studies, specific secondary contributing diagnoses including diabetes (E10-E14) were investigated. Additionally, to investigate possible associations with heat exposure, the classifications ‘exposure to excessive natural heat’ (X30) and ‘effects of heat and light’ (T67) (incorporating hyperthermia, heatstroke and heat exhaustion), were combined and collectively termed ‘effects of heat’. Data were stratified by gender and 5 year age groups initially, and subsequently age groups comprising children (0-14 years), adults (15-64 years), and the elderly (65 years and over).

Weather data for the period 1 January 1995 to 31 December 2006, were obtained from the Australian Bureau of Meteorology. Daily maximum and minimum ambient air temperatures for Adelaide were accessed from a monitoring station situated near the Adelaide central business district, the site considered by the Bureau of Meteorology to best represent conditions across the metropolitan area.

Heatwaves were defined as being three or more consecutive days when daily maximum temperatures reached or exceeded 35.0 degrees Celsius (°C), the 95th percentile of the maximum temperature range for the study period (Table 3.1). Seasons were defined as cool (1 April to 31 October) and warm (1 November to 31 March). As the focus of the study was heat-related renal morbidity, only the warm season was used in analysis with the referent period being non-heatwave days in the warm season.

**Data analyses**

The relation between daily renal admissions and temperature was explored graphically using a lowess (locally weighted regression) smoother with a bandwidth of 0.8, utilizing 80% of the data. Secular trends of admissions over time were identified in a similar manner. Spearman’s correlation was used to examine the association between cause-specific hospital admissions and heatwaves, daily maximum temperature and season. A Poisson distribution was assumed for the count data of hospital admissions. Using a case series approach, conditional fixed effects Poisson regression models were used to quantify the association between daily counts of renal admissions and heatwaves during the warm season. A goodness of fit test was applied to each model.
and if overdispersion was detected, as often occurs with recurrent events such as renal dialysis admissions, a negative binomial regression model was fitted. Seasonality was controlled for by exclusion of the cool season and analysis conducted within years adjusted for long-term trends.

A lag model was used to allow for the latent effects of heat exposure on hospital admissions not merely during the heatwave but on the subsequent days. Indicator variables for lags were created as $X_{t-k}$ representing the covariate for a heatwave $X_t$ with onset $k$ days before the admission. The model assumes:

$$\log[E(Y_{t+k})] = \beta_0 + \beta_1(X_t)$$

where $Y$ is the daily count of admissions, $E(Y)$ is the expected value of that count and $\beta_1$ is the log relative risk of renal admission associated with heatwaves. As previous studies have suggested predominantly short term effects of exposure to heat, the values for $k$ were determined to be 0-10 ranging from the first day of the heatwave to 10 days later.

All statistical analyses were conducted using Stata v9.2. A significance level of 0.05 was adopted for each test. Results for the Poisson models are expressed as incidence rate ratios (IRR) with 95% confidence intervals (CI).

### 3.3 Results

The mean maximum daily temperature in Adelaide during the study period was 22.3°C (Table 3.1), with the corresponding mean maximum temperatures during the cool season, warm season, and during heatwaves being 18.7°C, 27.4°C and 38.0°C respectively. A total of 31 heatwaves were recorded, occurring in ten of the twelve years with a maximum of six heatwaves in one year (2001). The highest recorded temperature was 44.3°C on 14 February 2004 during an eight day heatwave and an extreme heat event occurred in January 2006 with four continuous days over 40°C. The duration of individual heatwaves ranged from 3 to 8 days, with a mean of 3.8 days. Although no heatwaves were recorded outside of the nominated warm season, the daily maximum temperature exceeded 35.0°C on six occasions in the months of April and October.
Table 3.1: Summary statistics of daily temperatures (°C) for Adelaide, 1995-2006.

<table>
<thead>
<tr>
<th>Period</th>
<th>Maximum temperatures</th>
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</thead>
<tbody>
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<td></td>
<td>Mean</td>
<td>5th centile</td>
<td>95th centile</td>
<td>Mean</td>
</tr>
<tr>
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<td>22.3</td>
<td>14.1</td>
<td>35.3</td>
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<td>Heatwaves</td>
<td>38.0</td>
<td>35.2</td>
<td>41.9</td>
<td>23.1</td>
</tr>
</tbody>
</table>

Renal disease and dialysis

Renal admissions totaled 591,917 over the study period, of which 501,197 were admitted for dialysis. Examination of the data showed a secular trend with daily counts of renal (including dialysis) admissions almost doubling over the twelve years of the study. In 1995 there was a mean of 98.7 admissions per day and in 2006 this had increased to a daily mean of 190.1. The denominator population comprised 1,074,700 inhabitants in 1995 and 1.15 million in 2006 representing an average annual population increase of 0.6%.

Overall, the mean daily admissions were 134.5 for the cool season, 135.6 for the warm season and 138.9 during heatwaves in the warm season.

Fixed effects Poisson regression showed that the increase in the number of renal admissions during heatwaves compared to non-heatwave periods in the warm season, was significant at the 0.05 level, with a IRR of 1.024 (95% CI 1.008-1.041). However when corrected for overdispersion, the effect estimate became non-significant.

Renal disease excluding dialysis

By excluding dialysis, investigation focused principally on non-booked admissions. As shown in Table 3.2, there were 90,720 admissions over the study period with the principal diagnosis of renal disease (ICD-10 N00-N39), comprising 45,261 males and 45,459 females. Individuals aged 15-64 comprised 52.3% of all renal admissions, whilst those aged 65 years or over accounted for 42.1%. Daily counts of admissions increased over the twelve years of the study, with a daily mean of 18.9 in 1995 and 22.1 in 2006.
Spearman’s correlation showed significant ($P < 0.005$) correlations of admissions with daily maximum temperature, season and heatwaves (data not shown). Figure 3.2 shows a lowess smoothed curve of daily renal admissions and the association with maximum air temperature, demonstrating the greatest number of renal admissions when the nominated heatwave threshold of $35^\circ$C was reached or exceeded.

![Figure 3.2: Relation between daily maximum ambient air temperature and smoothed daily hospital admissions for renal disease excluding dialysis (N00-N39). The threshold temperature for heatwaves ($35^\circ$C) is indicated. Data were smoothed using a lowess smoother with a bandwidth of 0.8.](image-url)

Compared to non-heatwave periods, a 10% increase in renal admissions was observed during heatwaves (Table 3.2). A higher effect estimate was observed at lag 3 representing a three day latency period. Age-specific regression showed that adults aged 15-64 years had an IRR of 1.130 (95% CI 1.025-1.247). Females of this age group demonstrated a significant increase whilst the effect estimate for males did not reach statistical significance. However in the 45-49 year age bracket, males had an IRR of 1.241 (95% CI 1.037-1.485) whilst those in the 60-64 year age bracket had the highest effect estimate (IRR 1.311 (95% CI 1.096-1.568)). The risk for persons aged 65 years or over was lower than those aged 15-64, however the very elderly aged 85 years and over had the highest risk (IRR 1.196, 95% CI 1.036-1.380) as seen in Table 3.2. Gender-specific analysis in this group showed that the number of females 85+ years (4,171) far exceeded the number of males (2,497) and had the highest overall estimate of effect (IRR 1.218 (95% CI 1.022-1.453)).
Table 3.2: The incidence rate ratio (IRR) of hospital admissions for renal disease during heatwave periods compared to non-heatwave periods in the warm season.*

<table>
<thead>
<tr>
<th>Age</th>
<th>Gender</th>
<th>H/W</th>
<th>Non-H/W</th>
<th>Total</th>
<th>IRR (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td></td>
<td>2796</td>
<td>87 924</td>
<td>90 720</td>
<td>1.100 (1.003-1.206)</td>
<td>0.043</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>1419</td>
<td>43 842</td>
<td>45 261</td>
<td>1.106 (0.981-1.247)</td>
<td>0.098</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1377</td>
<td>44 082</td>
<td>45 459</td>
<td>1.088 (1.029-1.151)</td>
<td>0.003</td>
</tr>
<tr>
<td>15-64 years</td>
<td></td>
<td>1534</td>
<td>45 891</td>
<td>47 425</td>
<td>1.130 (1.025-1.247)</td>
<td>0.014</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>775</td>
<td>22 558</td>
<td>23 333</td>
<td>1.146 (0.986-1.333)</td>
<td>0.075</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>759</td>
<td>23 333</td>
<td>24 092</td>
<td>1.098 (1.018-1.184)</td>
<td>0.015</td>
</tr>
<tr>
<td>65+ years</td>
<td></td>
<td>1137</td>
<td>37 062</td>
<td>38 199</td>
<td>1.086 (0.978-1.205)</td>
<td>0.121</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>583</td>
<td>19 035</td>
<td>19 618</td>
<td>1.051 (0.922-1.199)</td>
<td>0.458</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>554</td>
<td>18 027</td>
<td>18 581</td>
<td>1.085 (0.993-1.186)</td>
<td>0.070</td>
</tr>
<tr>
<td>85+ years</td>
<td></td>
<td>213</td>
<td>6455</td>
<td>6668</td>
<td>1.196 (1.036-1.380)</td>
<td>0.014</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>71</td>
<td>2426</td>
<td>2497</td>
<td>1.046 (0.817-1.340)</td>
<td>0.719</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>142</td>
<td>4029</td>
<td>4171</td>
<td>1.218 (1.022-1.453)</td>
<td>0.028</td>
</tr>
</tbody>
</table>

* The counts of admissions by age group and gender during heatwave (H/W) and non-heatwave periods (Non-H/W) and over the whole study period (Total) are shown.

**Acute renal failure**

Over the study period, admissions for ARF (ICD-10-AM N17) totaled 3,579 representing 3.9% of all N00-N39 diagnoses. Although the small sample size is acknowledged, analysis indicated that the relative risk of admission for ARF was significantly increased during heatwave periods compared to control periods in the warm season. Evidence for an association was strengthened by Spearman’s correlation results which showed a significant ($P < 0.05$) correlation of ARF with both temperature and heatwaves. The overall IRR for admission with ARF during heatwaves compared to non-heatwave periods was 1.255 (95% CI 1.037-1.519). Figure 3.3 compares the point estimates for ARF and renal disease including and excluding dialysis.
Regression results of analysis of admissions during heatwave to non-heatwave periods

![Regression results of analysis of admissions during heatwave to non-heatwave periods](image)

**Figure 3.3**: Plot of IRRs of admissions for renal (including dialysis), renal (excluding dialysis), and acute renal failure (ARF) during heatwave periods compared to non heatwave periods in the warm season.

Figure 3.4 shows ARF admissions and temperatures in Adelaide during the period of February and March 2004 when three heatwaves occurred in the four weeks. It can be seen that trends are often similar, with a short delay between high temperatures and a rise in admissions. Although sample sizes are small, the figure demonstrates ARF admissions peaking during an extreme heat event when the maximum temperature reached 44.3°C.

Gender-specific analysis showed the risk for ARF was greatest in males (IRR 1.350, 95% CI 1.049-1.736); specifically males aged 15-64 years (IRR 1.786, 95% CI 1.169-2.730). Regression analysis yielded the associations presented in Table 3.3 which also shows the number of male admissions overall (1,887) and during heatwaves (70), by strata.

**Dialysis**

There were 501,197 admissions for extracorporeal dialysis (Z49.1) over the study period. Our results showed no association of admissions with heatwaves compared to non-heatwave periods in the warm season (Table 3.4).
Chapter three

Heatwaves and renal disease

Figure 3.4: The relationship between daily hospital admissions for ARF and temperature during February and March 2004 when 3 heatwaves were recorded.

Table 3.3: Age-specific regression results showing the incidence rate ratio of hospital admissions of males with acute renal failure (ARF) during heatwave periods compared to non-heatwave periods during the warm season.

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>N*</th>
<th>n**</th>
<th>IRR</th>
<th>P</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>1887</td>
<td>70</td>
<td>1.350</td>
<td>0.020</td>
<td>1.049-1.736</td>
</tr>
<tr>
<td>15-64</td>
<td>468</td>
<td>35</td>
<td>1.786</td>
<td>0.007</td>
<td>1.169-2.730</td>
</tr>
<tr>
<td>40-44</td>
<td>20</td>
<td>2</td>
<td>6.979</td>
<td>0.031</td>
<td>1.201-40.573</td>
</tr>
<tr>
<td>45-49</td>
<td>54</td>
<td>3</td>
<td>1.451</td>
<td>0.548</td>
<td>0.430-4.894</td>
</tr>
<tr>
<td>50-54</td>
<td>66</td>
<td>2</td>
<td>0.699</td>
<td>0.627</td>
<td>0.164-2.969</td>
</tr>
<tr>
<td>55-59</td>
<td>98</td>
<td>11</td>
<td>3.374</td>
<td>0.001</td>
<td>1.653-6.887</td>
</tr>
<tr>
<td>60-64</td>
<td>118</td>
<td>5</td>
<td>1.839</td>
<td>0.217</td>
<td>0.700-4.833</td>
</tr>
</tbody>
</table>

*N represents the total number of males admitted for ARF in each strata over the study period. **n represents the number admitted during heatwaves.

Comorbidities

Renal admissions for persons with contributing discharge diagnoses of diabetes (ICD-10 E10-E14) showed a small non-significant decrease during heatwaves compared to control periods. As shown in Table 3.3, with diabetes as a comorbidity, the risk of renal admission during heatwaves was estimated as 0.933 (95% CI 0.839-1.037). Only females aged 50-54 years and 85+ years with comorbid diabetes had a notably
increased risk of admission for renal disease during heatwaves. Conversely, renal admissions with comorbid effects of heat (X30, T67) were increased almost 11-fold during heatwaves (Table 3.4). However estimates in this group were compromised due to the small sample size ($N = 9$).

Table 3.4: The effect of heatwaves on hospital admissions for renal disorders and comorbidities, showing incidence rate ratios (IRR), 95% confidence intervals (CI) and $P$-values.

<table>
<thead>
<tr>
<th>Indication</th>
<th>ICD-10 code</th>
<th>IRR (95% CI)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal disease including dialysis</td>
<td>N00-N39; Z49.1</td>
<td>1.029 (0.976-1.084)</td>
<td>0.289</td>
</tr>
<tr>
<td>Renal disease</td>
<td>N00-N39</td>
<td>1.100 (1.003, 1.206)</td>
<td>0.043</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>N17</td>
<td>1.255 (1.037, 1.519)</td>
<td>0.019</td>
</tr>
<tr>
<td>Dialysis</td>
<td>Z49.1</td>
<td>1.013 (0.969, 1.059)</td>
<td>0.564</td>
</tr>
<tr>
<td>Renal disease with co-existing diabetes</td>
<td>E10-E14</td>
<td>0.945 (0.797, 1.121)</td>
<td>0.517</td>
</tr>
<tr>
<td>Renal disease with co-existing effects of heat</td>
<td>T67, X30</td>
<td>10.971 (2.065, 58.292)</td>
<td>0.005</td>
</tr>
</tbody>
</table>

3.4 Discussion

This study is the first to specifically investigate the association between high ambient temperatures and hospital admissions for renal disease in a temperate Australian climatic region. In addition to admissions attributed to diseases of general renal aetiology, ARF and extracorporeal dialysis were specifically investigated, and those with comorbid diabetes or indications of heat exposure. Results showed that during the warm season, the risk of hospital admission for renal disease was increased during heatwaves compared to non-heatwave periods, with highest effect seen in admissions for ARF. These findings are in line with those of others who found that hospital admissions for renal disease$^{22}$ and specifically ARF,$^{223}$ significantly increased in response to high ambient temperatures.

Elsewhere, studies of heatwave morbidity and mortality have also shown evidence of renal impairment attributed to heat exhaustion and heat stroke.$^{20,28,42,220}$ That an association exists between exposure to extreme heat and kidney dysfunction is biologically plausible and has been discussed earlier. Briefly, as a consequence of
hyperthermia and dehydration, the body’s physiological mechanisms attempt to regulate electrolyte and water imbalance. As glomerular filtration rates decrease, renal failure can occur.

The elderly are more vulnerable to the development of heat-related renal disease due to lowered thermotolerance, impaired thirst sensation,40,42 diminished conservation of sodium and water during dehydration, and reduced glomerular filtration rates.30,42 Indeed, findings from this study showed that the very elderly (85+ years) were the age group with the highest risk of renal admission during heatwaves with effect estimates highest for females. Similar findings have been reported in France where elderly females were found to be susceptible during heatwaves. Female mortality ratios were observed to be higher than male mortality ratios for those aged over 65 years, the difference particularly significant for deaths attributed to genitourinary system diseases.230

Findings also showed that individuals in the 15-64 year age group were at risk. Reasons for the susceptibility of persons in this younger age group remain purely speculative, although occupational and recreational activities outdoors in the heat may be behavioural risk factors in this age group, as physical exercise, manual labour and dehydration can compromise thermoregulation by diverting blood flow from the skin to active muscles.16 Indeed a report from Kuwait noted that 28 of 29 patients presenting with volume depletion and anuria due to heat stroke were outdoor construction workers.218 Further research is warranted in this areas as ARF is more commonly associated with exertional heat stroke, common in younger persons,220 than with classic heat stroke typically seen in the elderly.20,223

Many therapeutic drugs such as antihypertensives (including beta-blockers), diuretics, barbiturates and antihistamines20,40,42,43 can be risk factors for heat-related illness and subsequent renal involvement, due to their ability to inhibit thermoregulation via a number of different mechanisms. These may act by increasing heat production, causing dehydration, impairing thirst recognition or inhibiting heat loss. Importantly, in extreme conditions when ambient temperatures exceed skin temperature, body heat can only be lost by the evaporation of sweat216 and drugs that impair sweating via the cholinergic pathway such as anticholinergics, antipsychotics and tricyclics, can increase the risk of
heat-illnesses. A study of 83 heatstroke victims in France reported that 76% were being treated with antihypertensive and/or neuroleptic medications and renal dysfunction occurred in 34.9%. Chapter 4 includes more information about the thermoregulatory-inhibiting effects of drugs, specifically those used to treat psychiatric disorders.

If dehydration occurs, other medications can also cause ill effects. Pharmacokinetics and excretion can be influenced by dehydration, anuria and altered blood flow, either increasing medication toxicity or decreasing efficacy, particularly if the drugs in question have a narrow therapeutic index. Finally, the use of multiple medications, often prescribed to the chronically ill and the elderly, is also a risk factor. Beggs has discussed the influence of climate change on pharmacokinetics and medication-induced heat intolerance, and noted that the elderly are at particular risk being high consumers of medications. Unfortunately in the present study, information regarding medication histories was unavailable due to the ecological nature of the study design.

Acute renal failure (N17) accounted for 3.9% of all N00-N39 admissions in this study. Findings indicated that these admissions were significantly increased by 25.5% during heatwaves. Males appeared to be at greatest risk and those aged in their early forties or late fifties had the highest effect estimates. However results were inconsistent across the 5-year age groups between 40 and 64 years due to the small sample size of this subgroup, dictating cautious interpretation of these results. Further investigations with larger sample sizes are warranted to identify incidence and risk factors for ARF triggered by heatwaves in Australia.

Although renal dysfunction has been reported previously to be associated with heatstroke and heat exhaustion surprisingly few renal admissions with effects of heat as a secondary discharge diagnosis were found, making interpretation problematic. It is possible that our criterion was too specific, or that cases of heat-related illness are simply miscoded or underreported as has been suggested by other authors, possibly yielding imprecise heat-health data. Definitions of heat illnesses are generally inconsistent, with incidences of heat exhaustion, heat stroke and other related conditions often being attributed to more common diagnoses. If indeed the ‘effects of heat’ as a diagnostic category is rarely seen in patients admitted with
renal disease, it is speculated that either there is no association between the two or that other overarching symptoms or diagnoses are recorded instead.

A descriptive survey of emergency hospital presentations in Adelaide during a ten day heatwave just two years prior to the study period (February 1993) revealed, using a case note audit, that a total of 94 patients were diagnosed with heat-related illness, 78% with heat exhaustion and 15% with heat stroke. Plasma creatinine and plasma urea concentrations, biochemical indicators of renal dysfunction, were abnormal in 67% and 64% of these patients respectively. It is possible that discharge summaries may not have recorded renal dysfunction with effects of heat as a contributing factor despite both being contributing clinical factors on admission. This may be an example of why so few instances of effect of heat were listed as secondary diagnoses for renal admissions in the twelve years of our study, thereby highlighting major discrepancies in classification of diagnoses, and potential flaws in a system which specifies coding on discharge not admission.

In persons with diabetes mellitus, the function and structure of the kidney can be altered, potentially increasing susceptibility to heat and heat-related renal dysfunction. In contrast to other studies, results of the present study showed no overall increase during heatwaves of renal admissions with diabetes a comorbidity, although there is a suggestion of increased susceptibility in very elderly females with diabetes. It is possible that the diabetic population in this study may be acclimatized and keep well hydrated during hot weather. It should be noted that the focus for this study was on renal admissions and the investigation of those with a comorbidity of diabetes. Admissions for persons with diabetes as a principal diagnosis were not specifically investigated and thus it cannot be speculated if total admissions for diabetes were increased during heatwaves. A South Australian report states that more than 83,000 persons were diagnosed with diabetes in the State in 2003 with an estimated 15% of cases undiagnosed. With the number of diabetes cases rising rapidly each year, this represents a large subgroup of the population in whom susceptibility to heat-triggered illnesses should be closely monitored.

Consistent with the findings of others this study found that the effects of exposure to heat were prompt, with the greatest effect estimate for total renal disease
admissions occurring on the first day of the heatwave, whereas for renal excluding dialysis, the highest effect occurred after a two day lag. Due to limited statistical power, lag analysis for ARF admissions was inconclusive although Figure 3.3 suggests a similar lag period may apply, in which case a preventative public health approach may be effective.

Day admission for dialysis, a regularly scheduled treatment for sufferers of chronic renal disease or end-stage renal failure, is the leading cause of hospitalisations in Australia, accounting for 12.1% of all hospitalisations in 2005-2006. Not unexpectedly, with admissions unlikely to be affected by climatic conditions, no association was found between dialysis admissions and heatwaves.

**Limitations**

There are several limitations to this study. Firstly, as ICD coding was used to identify primary or secondary discharge diagnoses, the possibility of miscoding cannot be discounted and it is acknowledged that accuracy may be greater using individual case-note audits. These would also provide patient medication histories that may be useful in ascertaining pharmacological risks to renal health during the heat. Additionally using only ICD coding, it was impossible to ascertain those dialysis admissions whose condition became acute on chronic due to the effects of heat exposure. Secondly, several potentially informative renal sub-categories of interest were not investigated in this study including glomerular disease, urolithiasis and other disorders of the kidney, ureter and urinary system. Additionally related diagnoses not classified under diseases of the genitourinary system but which may have indications for heat-induced renal dysfunction, were not investigated. These included rhabdomyolysis (injury of skeletal muscle fibres due to the leakage of extracellular fluid) which is often linked to hyperthermia, hypernatraemia and severe renal involvement. Further research into the association between rhabdomyolysis, renal disease and exposure to extreme heat is warranted.

Thirdly, although the findings are reinforced by those of others, the relatively small number of admissions for ARF in our study dictates cautious interpretation regarding an association with heatwaves. Given the potentially serious consequences
and high mortality of heat-triggered ARF, further research with larger sample sizes is essential to reinforce these findings and identify risk factors. Conversely, the lack of geographical specificity within the data restricts the ability to pinpoint metropolitan areas that could be targeted for facility planning.

Finally, it was considered unnecessary to control for airborne pollution in this study as there is little evidence to suggest a confounding effect of air pollution with the temperature-renal disease association. This is in contrast to similar studies where cardiopulmonary outcomes are of interest and both heat and airborne particulate matter may be on the causal pathway. Furthermore we did not control for day of the week or holidays, factors which may determine daily activities and consequential exposure to the heat. It is acknowledged that the Australian summer vacation between Christmas and mid January may influence exposure levels, population mobility and routine admission bookings. A more accurate indication of non-chronic heat related illness would be gained by investigating details of emergency department visits which were unavailable at the time this study was conducted.

Conclusion

In summary, results of this study suggest that non-chronic renal morbidity and ARF are elevated during heatwave periods. It follows that by using current data to predict future scenarios, it may be speculated that the burden of renal morbidity may increase as periods of extreme heat become more frequent and the population ages. With long term treatment options for renal diseases, particularly ARF, being costly and resource intense, an increase in incidence will have significant public health implications. Health authorities may need to address the availability of facilities and consider strategies for the mitigation of heat-related renal disease such as health promotion programs to encourage adequate hydration during extreme heat. These would require thoughtful formulation to relay appropriate messages, as overzealous fluid intake particularly in exercising persons, can also be risk factor, contributing to hyponatraemia. The WHO recommendation is two to four glasses of cool, non-alcoholic fluid per hour if exercise must be undertaken in the heat. It can be concluded that further investigation is warranted to identify vulnerable sub-populations and other forms of heat-sensitive renal diseases.
Chapter 4
The effect of heatwaves on mental health

4.1 Introduction

Compared with the general population, persons with mental health illnesses often experience poorer overall health with higher rates of morbidity and mortality.\textsuperscript{245} It has been well documented that due to behavioural reasons and medications that interfere with physiological homeostasis, those with mental illnesses are susceptible to the effects of extreme heat.\textsuperscript{44} Affected persons are also likely to fall into other high risk categories for heat intolerance, including the elderly, disadvantaged, socially isolated, alcoholics and those who have cardiopulmonary diseases\textsuperscript{246} or obesity.\textsuperscript{44} Several studies have reported heat-associated increases in hospital admissions\textsuperscript{8,247} and mortalities\textsuperscript{2,21,36,230,248,249} for persons with mental illnesses. However few studies, and none from Australia, have characterised specific mental and behavioural disorders (MBDs) that may be exacerbated by rising ambient temperatures.

Mental health illnesses may range from short-term bouts of depression and anxiety, to long term conditions such as chronic depression, schizophrenia or chronic anxiety disorders.\textsuperscript{250} Accounting for an estimated 13\% of the national disease burden in Australia, mental health rated among the ten leading causes of disease in 2003, and has been declared a National Health Priority Area.\textsuperscript{250} In the State of South Australia, studies have shown that as many as one in five adults has a mental problem.\textsuperscript{251} These figures are similar to those reported in a 1997 National Survey of Mental Health and Wellbeing of Adults showing that one in five Australians will experience a mental illness at some stage in their life and that 18\% had mental disorder in the previous 12 months.\textsuperscript{250}
The aim in this study was to identify mental disorders which contribute to heat-related morbidity and mortality in a temperate climatic region. It is unique in that the literature indicates no similar investigation has previously been undertaken. Moreover, both hospital admissions and mortality data spanning a 13 year period are used in order to establish a comprehensive overview of the temperature-mental health association. With mental disorders causing an already significant burden on the public health system, understanding the relationship between hot weather extremes and psychiatric illness will assist in identifying populations at risk as global warming ensues, and provide valuable information for decision-makers in the mental health and social service sectors.

4.2 Methods

The city of Adelaide, South Australia, has a climate ideal for the study of heat-health effects with heatwaves commonly occurring during the summer months. Health outcome data for the city were obtained for the period 1 July 1993 to 30 June 2006 from the South Australian Department of Health. Data relating to individuals who were admitted outside of the Adelaide metropolitan area were excluded from the study. Principal hospital discharge diagnoses were accessed using the Integrated South Australian Activity Collection (ISAAC), an official collection of admitted patient activity in the State’s public and private hospitals. De-identified mortality data for the period 1 July 1993 to 22 December 2004 were sourced from the Australian Bureau of Statistics (ABS). Administrative delays incurred in accessing mortality data accounted for the shorter timer period. Underlying causes of death were coded by the ABS using computer assisted coding.

Daily counts were collected of admissions and mortalities with principal discharge diagnoses or cause of death respectively, attributed to MBDs, as classified according to the International Classification of Diseases 10th Revision. Any diagnoses originally classified by ICD-9 codes over the study period were converted to ICD-10. Sub classifications of MBDs (F00-F99) included in the study are shown in Table 4.1. Dementia was identified separately although already included within the classification of organic, including symptomatic, mental disorders. Additional to the aforementioned
disorders, Alzheimer’s disease, senile degeneration of the brain, and senility were included to cover age-related cognitive disorders not captured within the F00-F99 sub classifications.

Weather data for the period 1 July 1993 to 30 June 2006 in Adelaide were obtained from the Australian Bureau of Meteorology. Daily maximum and minimum ambient air temperatures for Adelaide were accessed from a central city weather station considered to be representative of conditions across the total metropolitan area as advised by the Bureau of Meteorology.

Data analyses

The relationship between daily admissions or mortalities attributed to MBDs, and daily maximum temperature ($T_{\text{max}}$), was explored graphically using a lowess smoother\textsuperscript{233} which calculates smoothed data point values by performing locally weighted regressions of the $y$-variable on the $x$-variable using 80% of the data (i.e. bandwidth 0.8). Threshold temperatures were estimated visually and confirmed quantitatively using a nonlinear least-squares estimation incorporating the \texttt{n1} function in Stata v9.2.\textsuperscript{233} Based on the assumption of more than one segment in the relationship between two variables, this ‘hockey-stick’ method\textsuperscript{182,252,253} uses a function evaluator program to determine the cutpoint in the curve at which the change in slope occurs, providing this value is common to both functions. Parameters are determined using informative initial estimates of the slope and threshold values in iterative non-linear regression models. Calculated threshold values should approximate visual estimates and have a statistical significance level of 0.05.

The threshold model can be defined as:

\[
E(Y) = \beta_0 \quad \text{for} \quad T_{\text{max}} < x_0 \quad [1]
\]

and

\[
E(Y) = \beta_0 + \beta_1(T_{\text{max}} - x_0) \quad \text{for} \quad T_{\text{max}} \geq x_0 \quad [2]
\]
where, \( E(Y) \) is the expected value of the outcome variable, \( \beta_0 \) is the baseline number of daily admissions and \( x_0 \) is the threshold temperature value to be determined. If a non-zero slope is expected in [1] the model becomes:

\[
E(Y) = \beta_0 + \beta_1(T_{\text{max}}) \quad \text{for } T_{\text{max}} < x_0 \quad [3]
\]

and

\[
E(Y) = \beta_0 + \beta_1(x_0) + \beta_2(T_{\text{max}} - x_0) \quad \text{for } T_{\text{max}} \geq x_0 \quad [4]
\]

Heatwaves were defined as being three or more consecutive days when the \( T_{\text{max}} \) reached or exceeded 35.0 degrees Celsius (°C)\(^3\). Seasons were defined as cool (1 April to 30 September) and warm (1 October to 31 March).

Daily counts of hospital admissions or mortalities were analysed collectively and stratified by age and gender. Conditional fixed effects Poisson regression models accounting for long term trend, were used to quantify the association with heatwaves\(^3\) with the referent period being all non-heatwave days during the warm season. A goodness of fit test was applied to each model and if significant overdispersion was detected, a negative binomial maximum-likelihood regression model was used.\(^\text{234} \) As the focus of the study was heat-related morbidity and mortality, only the warm season was used in the analysis, thereby controlling for season. By performing within year analyses, long term trends were adjusted for.\(^\text{31} \) Results were expressed as Incidence Rate Ratios (IRR) with 95% confidence intervals (CI). All statistical analyses were conducted using Stata V9.2.\(^\text{233} \)

### 4.3 Results

Hospital admission data spanned the period 1 July 1993 to 30 June 2006, and mortality data from 1 July 1993 to 22 December 2004, during which time there were 31 and 29 heatwaves respectively, ranging in duration from three to eight days including an extreme heat event in January 2006 with temperatures over four continuous days exceeding 40°C. The highest recorded maximum temperature was 44.3°C during an eight day heatwave in 2004.
Hospital admissions

Of the 15 categories of mental and cognitive disorders studied, seven showed no significant association of hospital admissions with heatwaves (Table 4.1 and Figure 4.1), however two of these showed age or gender specific associations (Table 4.2), leaving ‘schizophrenia, schizotypal and delusional disorders’ (F20-F29); ‘disorders of adult personality and behaviour’ (F60-F69); ‘mental retardation’ (F70-F79); ‘Alzheimer’s disease’ (G30-G30.9); and ‘senile degeneration of the brain not elsewhere classified’ (G31.1). ‘Behavioural and emotional disorders with onset usually occurring in childhood and adolescence’ (F90-F98) showed a statistically significant decrease in admissions during heatwaves compared to non-heatwave periods. Increases in hospital admissions for the remaining categories were found to occur during heatwave periods as discussed below.

Mental and behavioural disorders (F00-F99)

During the study period there were a total of 171,614 admissions to Adelaide hospitals with the principal discharge diagnoses of MBDs (ICD-10-AM F00-F99). Of these, 4,629 admissions occurred during heatwaves, representing a daily mean of 38.6 admissions compared to 35.8 during the warm season. The exposure-response association between admissions and $T_{max}$ was observed graphically and showed a small rise and fall in admissions in cooler temperatures, then a logarithmic trend as temperatures increased from moderate to extreme (Figure 4.2A). Using a nonlinear least-squares (hockey stick) estimation, a threshold temperature of 26.7°C ($P = 0.001$) was calculated, above which MBD admissions were seen to increase markedly. Using iterative regressions, this threshold value remained robust to several parameter estimations.

Overall, fixed effects Poisson regression analysis accounting for overdispersion, showed a 7.3% increase in admissions during heatwave periods compared to non heatwave periods in the warm season (Table 4.1). Results were also significant for the elderly 75 years and over in this category, with an IRR of 1.183 (95% CI 1.088-1.286) and in male admissions in the 15-64 year age group (Table 4.2).
Table 4.1: Cause-specific mental and behavioural disorder hospital admissions associated with heatwaves in Adelaide 1993-2006 showing incidence rate ratio (IRR) and 95% confidence intervals (CI).

<table>
<thead>
<tr>
<th>ICD-10 code</th>
<th>Details</th>
<th>IRR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>F00-F99</td>
<td>Mental and behavioural disorders</td>
<td>1.073</td>
<td>1.017-1.132</td>
</tr>
<tr>
<td>F00-F09</td>
<td>Organic, including symptomatic, mental disorders</td>
<td>1.213</td>
<td>1.091-1.349</td>
</tr>
<tr>
<td>F00-F03</td>
<td>Dementia</td>
<td>1.174</td>
<td>1.017-1.355</td>
</tr>
<tr>
<td>F10-F19</td>
<td>Mental and behavioural disorders due to psychoactive substance use</td>
<td>1.005</td>
<td>0.913-1.105</td>
</tr>
<tr>
<td>F20-F29</td>
<td>Schizophrenia, schizotypal and delusional disorders</td>
<td>1.034</td>
<td>0.969-1.102</td>
</tr>
<tr>
<td>F30-F39</td>
<td>Mood (affective) disorders</td>
<td>1.091</td>
<td>1.004-1.185</td>
</tr>
<tr>
<td>F40-F48</td>
<td>Neurotic, stress-related and somatoform disorders</td>
<td>1.034</td>
<td>1.017-1.355</td>
</tr>
<tr>
<td>F50-F59</td>
<td>Behaviour syndromes associated with physiological disturbances and physical factors</td>
<td>0.875</td>
<td>0.678-1.130</td>
</tr>
<tr>
<td>F60-F69</td>
<td>Disorders of adult personality and behaviour</td>
<td>1.049</td>
<td>0.905-1.214</td>
</tr>
<tr>
<td>F70-F79</td>
<td>Mental retardation</td>
<td>0.737</td>
<td>0.268-2.026</td>
</tr>
<tr>
<td>F80-F89</td>
<td>Disorders of psychological development</td>
<td>1.641</td>
<td>1.086-2.480</td>
</tr>
<tr>
<td>F90-F98</td>
<td>Behavioural and emotional disorders with onset usually occurring in childhood and adolescence</td>
<td>0.578</td>
<td>0.349-0.955</td>
</tr>
<tr>
<td>G30-G30.9</td>
<td>Alzheimer’s disease</td>
<td>1.154</td>
<td>0.894-1.489</td>
</tr>
<tr>
<td>G31.1</td>
<td>Senile degeneration of brain, not elsewhere classified</td>
<td>7.727</td>
<td>0.701-85.217</td>
</tr>
<tr>
<td>R54</td>
<td>Senility</td>
<td>2.366</td>
<td>1.200-4.667</td>
</tr>
</tbody>
</table>

**Organic, including symptomatic, mental disorders (F00-F09)**

In this subcategory encompassing dementia, cerebral disease, brain injury and other trauma leading to cerebral dysfunction, dementia (F00-F03) accounted for more than half (56.2%) of the admissions. Analysis showed that compared to control periods, hospitalisations for organic, including symptomatic mental disorders were significantly increased by 21.3% during heatwaves (Table 4.1) with increases in both male and female admissions and in the 75+ years age group. Figure 4.2B shows a decline in admissions associated with moderate temperatures and a linear increase as temperatures rise from moderate to high.
Figure 4.1: Point estimates with 95% confidence intervals for the risk during heatwaves compared to non-heatwave periods, of hospital admission for mental, behavioural and cognitive disorders. Abbreviations: MBD: All mental and behavioural disorders; Org: Organic, including symptomatic, mental disorders; Dem: Dementia; Subst: Disorders due to psychoactive substance use; Schiz: Schizophrenia, schizotypal and delusional disorders; Mood: Mood (affective) disorders; Neur: Neurotic, stress-related and somatoform disorders; Phys: Behavioural syndromes associated with physiological disturbances and physical factors; Pers: Disorders of adult personality and behaviour; Ret: Mental retardation; Dev: Disorders of psychological development; Child: Behavioural and emotional disorders with onset usually occurring in childhood and adolescence; Sen: Senility; Alzh: Alzheimer’s disease.

**Dementia (F00-F03)**

The risk of admission for dementia during heatwaves was increased by 17.4% (95% CI 1.7%-35.5%) (Table 4.1) and in females (Table 4.2). Due to dementia accounting for a high proportion of organic, including symptomatic mental disorder admissions, the shape of the relationship with $T_{\text{max}}$ (Figure 4.2C) is almost identical to that seen in Figure 4.2B albeit on a different scale.

**Mental and behavioural disorders due to psychoactive substance use (F10-F19)**

In this category, the elderly (75 years or over) were the only age group to show an increase in hospitalisations during heatwaves (IRR 1.567, 95% CI 1.002-2.450) with female admissions of this age also showing a significant increase (Table 4.2).
Mood (affective) disorders (F30-F39)

Mood (affective) disorders include depression, dysthymia, mania, and bipolar affective disorders. This category accounted for the largest proportion (33.9%) of MBD admissions. During heatwaves compared to referent periods, there was a 9.1% increase in admissions (Table 4.1) with highest estimates in persons aged between 15 and 64 years (IRR 1.102, 95% CI 1.041-1.167).

Neurotic, stress-related and somatoform disorders (F40-F48)

Neurotic, stress-related and somatoform disorders include anxiety disorders, panic disorder, agoraphobia, obsessive compulsive disorder and post-traumatic stress disorder. Results showed an increase in hospitalisations of 9.7% (Table 4.1), and age-specific regression analysis showed those most at risk during heatwaves were the very young and the elderly. Figure 4.2D shows the relationship of admissions with temperature and demonstrates a gradual increase as $T_{\text{max}}$ rises above moderate.

Behavioural syndromes associated with physiological disturbances and physical factors (F50-F59)

This group of syndromes includes eating and sleep disorders, and accounted for just 1.6% of total MBD admissions over the study period. During heatwaves, an increase in admissions of this classification was seen in elderly females only (Table 4.2).
### Table 4.2: Cause-specific hospital admissions stratified by age and sex, showing incidence rate ratio (IRR) and 95% confidence intervals (CI).

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>All ages</th>
<th>15-64 years</th>
<th>65-74 years</th>
<th>75 years or over</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sex</td>
<td>IRR</td>
<td>95% CI</td>
<td>IRR</td>
</tr>
<tr>
<td>Mental and behavioural disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.077</td>
<td>1.030-1.126</td>
<td>1.059</td>
<td>1.008-1.112</td>
</tr>
<tr>
<td>F</td>
<td>1.070</td>
<td>0.997-1.148</td>
<td>1.043</td>
<td>0.966-1.127</td>
</tr>
<tr>
<td>Organic, including symptomatic mental disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.177</td>
<td>1.000-1.385</td>
<td>1.139</td>
<td>0.772-1.681</td>
</tr>
<tr>
<td>F</td>
<td>1.242</td>
<td>1.079-1.429</td>
<td>1.300</td>
<td>0.671-1.941</td>
</tr>
<tr>
<td>Dementia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.118</td>
<td>0.894-1.309</td>
<td>1.282</td>
<td>0.549-2.991</td>
</tr>
<tr>
<td>F</td>
<td>1.215</td>
<td>1.008-1.466</td>
<td>1.396</td>
<td>0.596-3.268</td>
</tr>
<tr>
<td>Disorders due to psychoactive substance use</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.026</td>
<td>0.913-1.153</td>
<td>1.022</td>
<td>0.904-1.155</td>
</tr>
<tr>
<td>F</td>
<td>0.965</td>
<td>0.819-1.137</td>
<td>0.916</td>
<td>0.766-1.094</td>
</tr>
<tr>
<td>Schizophrenia, schizotypal &amp; delusional disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.048</td>
<td>0.966-1.136</td>
<td>1.041</td>
<td>0.958-1.131</td>
</tr>
<tr>
<td>F</td>
<td>1.010</td>
<td>0.880-1.160</td>
<td>0.986</td>
<td>0.842-1.152</td>
</tr>
<tr>
<td>Mood (affective) disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.065</td>
<td>0.955-1.182</td>
<td>1.057</td>
<td>0.960-1.164</td>
</tr>
<tr>
<td>F</td>
<td>1.093</td>
<td>0.973-1.229</td>
<td>1.118</td>
<td>1.001-1.249</td>
</tr>
<tr>
<td>Neurotic, stress-related and somatoform disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.151</td>
<td>1.031-1.285</td>
<td>1.118</td>
<td>0.993-1.258</td>
</tr>
<tr>
<td>F</td>
<td>1.054</td>
<td>0.953-1.166</td>
<td>1.037</td>
<td>0.927-1.160</td>
</tr>
<tr>
<td>Syndromes associated with physiological - disturbances &amp; physical factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>0.777</td>
<td>0.315-1.917</td>
<td>0.840</td>
<td>0.305-2.309</td>
</tr>
<tr>
<td>F</td>
<td>0.885</td>
<td>0.678-1.155</td>
<td>0.825</td>
<td>0.620-1.099</td>
</tr>
<tr>
<td>Disorders of psychological development</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.480</td>
<td>0.877-2.498</td>
<td>3.383</td>
<td>1.247-9.180</td>
</tr>
<tr>
<td>F</td>
<td>1.985</td>
<td>1.013-3.981</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Behavioural &amp; emotional disorders onset childhood &amp; adolescence</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>0.448</td>
<td>0.221-0.908</td>
<td>0.457</td>
<td>0.144-1.453</td>
</tr>
<tr>
<td>F</td>
<td>0.814</td>
<td>0.396-1.670</td>
<td>0.504</td>
<td>0.122-2.092</td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1.009</td>
<td>0.664-1.535</td>
<td>-</td>
<td>1.120</td>
</tr>
<tr>
<td>F</td>
<td>1.258</td>
<td>0.912-1.735</td>
<td>1.133</td>
<td>0.263-4.872</td>
</tr>
<tr>
<td>Senility</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>0.594</td>
<td>0.080-4.404</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>F</td>
<td>3.590</td>
<td>1.666-7.600</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
**Disorders of psychological development (F80-F89)**

Encompassing autism and developmental disorders of speech and language, disorders of psychological development accounted for 0.35% of MBD admissions. A 64% increase in hospitalisations was observed overall (Table 4.1) whereas in the 15-64 year age group, the increase was more than 3-fold (IRR 3.187, 95% CI 1.185-8.571) during heatwaves, possibly linked to varying levels of care depending on age. Figure 4.2E shows admissions plateauing at moderate temperatures and increasing sharply in hot weather.

**Senility (R54)**

Senility includes senescence, asthenia and debility\(^{229}\), relating to mental infirmity and physical deterioration of the aged. Although there were few (\(N = 191\)) admissions for senility over the study period, analysis showed a more than 2-fold increase during heatwaves (Table 4.1), with strongest effect in the elderly (IRR 2.534, 95% CI 1.232 – 5.211), and elderly females. Figure 4.2F reveals an almost U-shaped relationship, showing admissions at their lowest during mild temperatures and highest during the extremes of cold and hot weather.

**Mortalities**

Mortalities attributed to the 15 categories of psychiatric disorders were relatively infrequent in Adelaide and sample sizes during heatwaves were not large. Regression results for the disorders showing significant increases in mortalities are displayed in Table 4.3.

**Mental and behavioural disorders (F00-F99)**

Over the study period there were 2,599 deaths attributed to MBDs, representing less than 1 per day. During heatwaves there were 70 MBD deaths, with 81% (\(N = 57\)) of the decedents aged 65 years or over and regression results showing a 2.4 fold increase in deaths in the 65-74 year age group (Table 4.3).
Figure 4.2: Exposure-response relationships between daily maximum air temperature and hospital admissions for mental disorders: Data were smoothed using a lowess smoother, bandwidth = 0.8. A, all mental and behavioural disorders. B, organic, including symptomatic mental disorders. C, dementia. D, neurotic, stress-related and somatoform disorders. E, disorders of psychological development. F, senility.
Organic, including symptomatic, mental disorders (F00-F09); Dementia (F00-F03)

The majority of MBD deaths (76%, \( N = 1,964 \)) were due to organic, including symptomatic, mental disorders, of which dementia comprised 99% \( (N = 1,953) \). Of the deaths occurring during heatwaves, 67% \( (N = 47) \) were classified as organic, including symptomatic, mental disorders, and all were due to dementia. The risk of death during heatwaves compared to non-heatwave periods was significant for those aged 15-64 years and in males of this age group (Table 4.3).

Mental disorders due to psychoactive substance use (F10-F19)

During heatwaves, deaths in this category were increased more than three-fold in females, specifically those in the 15-64 year age bracket (Table 4.3).

Schizophrenia, schizotypal and delusional disorders (F20-F29)

Analysis of non-stratified data showed deaths due to schizophrenia, schizotypal and delusional disorders were increased more than two-fold during heatwave periods (Table 4.3). This was more pronounced in those aged 75 years or over, and in males of this age group. A J-shaped relationship of mortalities with \( T_{\text{max}} \), was observed with lowest mortality at moderate temperatures and highest when temperatures exceed 35\(^\circ\)C (not shown) although low counts may have compromised the validity of the association.

Table 4.3: Details of mental and behavioural mortalities associated with heatwaves showing incidence rate ratio (IRR), 95% confidence interval (CI) and \( P \) values.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Details</th>
<th>IRR</th>
<th>95% CI</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental and behavioural disorders</td>
<td>65-74 years</td>
<td>2.395</td>
<td>1.165-4.922</td>
<td>0.017</td>
</tr>
<tr>
<td>Dementia</td>
<td>15-64 years</td>
<td>5.058</td>
<td>1.205-21.232</td>
<td>0.027</td>
</tr>
<tr>
<td>Disoders due to psychoactive substance use</td>
<td>Males 15-64 years</td>
<td>12.731</td>
<td>2.064-78.516</td>
<td>0.006</td>
</tr>
<tr>
<td>Schizophrenia, schizotypal &amp; delusional disorders</td>
<td>Females</td>
<td>3.098</td>
<td>1.342-7.155</td>
<td>0.008</td>
</tr>
<tr>
<td></td>
<td>Females 15-64 years</td>
<td>3.211</td>
<td>1.297-7.948</td>
<td>0.012</td>
</tr>
<tr>
<td></td>
<td>All ages</td>
<td>2.079</td>
<td>1.045-4.138</td>
<td>0.037</td>
</tr>
<tr>
<td></td>
<td>75+ years</td>
<td>2.111</td>
<td>1.018-4.380</td>
<td>0.045</td>
</tr>
<tr>
<td></td>
<td>Males</td>
<td>4.051</td>
<td>1.386-11.840</td>
<td>0.011</td>
</tr>
<tr>
<td></td>
<td>Males 75+ years</td>
<td>5.255</td>
<td>1.752-15.758</td>
<td>0.003</td>
</tr>
</tbody>
</table>
4.4 Discussion

This study builds upon previous investigations into the acute health effects of heatwaves in Adelaide where amongst the range of diagnoses examined, mental disorders was the only outcome in which increases were observed in both hospital admissions and (age-specific) mortality.\textsuperscript{31} Other published literature has also identified persons with mental illness as being a heat-susceptible subgroup of the population.\textsuperscript{2,8,21,249} However to the author’s knowledge, this study is the first to endeavour to comprehensively characterise specific disorders which contribute to increased psychiatric morbidity and mortality during heatwaves. Results here suggest several mental illnesses may be sensitive to exposure to high ambient temperatures and show that hospital admissions for MBDs were increased at maximum temperatures above 26.7\degree{}C, and during heatwaves compared to control periods by 7.3\%. Specific nosologic subgroups for which an increase in admissions was evident include:

- organic, including symptomatic mental disorders;
- dementia;
- mood (affective) disorders;
- neurotic, stress-related and somatoform disorders;
- disorders of psychological development

Although classified separately to MBDs, senility was included in this study as a cognitive impairment of the elderly, and admissions were observed to be increased more than two-fold during heatwave periods. Hospitalisations for behavioural syndromes associated with physiological disturbances and physical factors, and MBDs due to psychoactive substance use, were increased for specific age and gender categories.

Having a pre-existing psychiatric illness can more than triple the risk of death during a heatwave.\textsuperscript{254} In this study an increase in deaths classified as MBDs was observed in the 65-74 year age group during heatwaves. Additionally deaths due to schizophrenia, schizotypal and delusional disorders increased more than two-fold, and those due to psychoactive substance use were increased in females and females aged 15-64. Dementia deaths were increased in the 15-64 year age group only, indicating the
susceptibility of those with early onset dementia. As mortality data were limited by small sample sizes in most categories, the association between heatwaves and mental health mortalities warrants cautious interpretation of some results particularly where stratification of data compromised statistical power. More studies need to be conducted using much larger study populations in a range of different climate zones.

Overall there was a general consistency in findings between hospital admissions and mortalities for persons with mental illnesses, both indicating an increased risk during heatwaves. The nature of the disorders in the two varied somewhat although persons with dementia appeared to be at-risk of both hospitalisation and dying during extreme heat.

**Heat and mental health**

An understanding of the potential relationship between heat and mental health may be useful in causal interpretation. As heat-related illness is very often underdiagnosed it is possible that individuals in this study with a case history of mental illness, may have been suffering the effects of heat rather than (or as well as) the effects of their disorder. The MBD may take precedence for classification purposes, and thus admission would be classified accordingly. Nevertheless, the findings of hospitalisations for MBDs increasing during heatwaves compared to non-heatwaves, suggest the issues concerning heat and psychiatric illness may be two-fold: (1) that having a psychiatric condition can be a risk factor for heat-related morbidity and mortality; and (2) that heat can be a risk factor for the exacerbation or onset of psychiatric morbidity. It is proposed that this can be summarised as behavioural and/or physiological maladaptation, and psychological maladaptation respectively. These concepts are discussed further below. In both cases a rise in ambient temperatures may be influential. The proposed model for the association between heat and mental illness is shown in Figure 4.3.

**1**  *Does having a mental illness confer vulnerability to heat illnesses?*

Mental illness can be a risk factor for heat-related morbidity and mortality for several physiological and behavioural reasons. Many medications used in psychiatry such as antipsychotics (including phenothiazines, thioxanthenes, butyrophenones and atypical antipsychotics), anticholinergics, antidepressants, sedatives and mood stabilizers, can
increase the risk of heat stroke (Table 4.4). The pharmaceutical effects of these drugs can impair sweating and/or alter thermoregulation\textsuperscript{40,42} due to actions affecting the parasympathetic pathway\textsuperscript{43} and the temperature regulating mechanisms of the hypothalamus.\textsuperscript{15}

The inherent nature of these illnesses may also contribute to vulnerability during hot weather. Cognitive awareness of environmental conditions and ability to undertake adaptive behaviours during hot weather such as increased fluid uptake or wearing appropriate clothing\textsuperscript{43,248} are important coping mechanisms which may be compromised in those with disabling mental illnesses such as Alzheimer’s disease, dementia, senility,
psychosis, schizophrenia and developmental disabilities. Additionally, severe or core activity limitations often present in the mentally ill and those with neuropsychiatric conditions, can affect their degree of dependence and be a contributing factor to heat susceptibility. This notion is supported by our findings of increased admissions for individuals with senility, confirming the susceptibility of the frail aged to heat-related illnesses or death.

Table 4.4: Medications that increase the risk of heat stroke in persons with MBDs

<table>
<thead>
<tr>
<th>Medication class</th>
<th>Indication</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antipsychotics (neuroleptic agents)</td>
<td>Psychoses, schizophrenia, Alzheimer’s disease, dementia</td>
<td>Altered sweat production; reduced ability to eliminate heat; weight gain; impaired thirst recognition; set point of temperature regulation centre increased</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Depression</td>
<td>Altered sweat production; weight gain</td>
</tr>
<tr>
<td>Anticholinergics</td>
<td>Alzheimer’s disease, Parkinson’s, depression</td>
<td>Suppression of sweating and vasodilatation</td>
</tr>
<tr>
<td>Selective serotonin reuptake inhibitors (SSRIs)</td>
<td>Depression</td>
<td>Increased heat production, inhibited heat loss</td>
</tr>
<tr>
<td>Lithium</td>
<td>Bipolar disorder</td>
<td>Increased heat production, inhibited heat loss, increased urination</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>Anxiety, hypertension</td>
<td>Dehydration via increased sweating and vasodilatation, impaired cardiac performance</td>
</tr>
<tr>
<td>Anxiolytics (benzodiazepines)</td>
<td>Anxiety disorders</td>
<td>Sedative affect - alters vigilance levels and adaptive behaviour</td>
</tr>
<tr>
<td>Barbiturates</td>
<td>Epilepsy, anxiety disorders</td>
<td>Reflex regulation of body temperature depressed, sedative effect</td>
</tr>
<tr>
<td>Diuretics (includes thiazides)</td>
<td>Hypertension, heart failure, some kidney diseases</td>
<td>Alteration in heat dispersion and heat regulation, increase excretion of water and electrolytes leading to dehydration. Thiazides can reduce the clearance of lithium.</td>
</tr>
</tbody>
</table>

Adapted from: Batscha. Sources: Batscha, Conti et al., Martin-Latry et al., Donaldson et al., Brody et al., Kao & Kelly.

In contrast to the findings of others who reported a correlation of schizophrenia admissions with environmental temperature, in the present study no increase during heatwaves was found for the non-stratified or age- and gender- stratified data. However,
a significant increase in mortalities attributed to schizophrenia, schizotypal and delusional disorders was observed. A coroner’s study of 18 heat-related deaths during the 1999 Cincinnati heatwave also found schizophrenia to be a risk factor for mortality with eight decedents having a mental illness, four of whom had schizophrenia. Assumption as to the nature of the variation in morbidity and mortality rates in this category remains highly speculative. Firstly, as previously mentioned antipsychotic medications are known to impair thermoregulation and may increase the risk of death in psychiatric patients during heatwaves, although studies have shown patients were at risk in the 1950s prior to the introduction of antipsychotic drugs, suggesting that both schizophrenia and its medication increase the risk of heat-related death. This has biological plausibility as the neurotransmitter dopamine plays a role in the hypothalamic thermoregulatory pathway, and the dopamine hypothesis of schizophrenia proposes that the symptoms of schizophrenia result from dopaminergic abnormalities in the limbic area of the brain (where the hypothalamus is located). It is reportedly common for the severely affected to display thermoregulatory inefficiencies as evidenced by the “T-shirt in winter and duffle coat in summer syndrome.”

Secondly, it is possible that for these individuals, death may have occurred rapidly before admission to hospital as reported elsewhere. Furthermore, consequential factors of living with such a debilitating mental illness include social isolation, poor general health and chronic illness, all of which are risk factors for heat-related mortality. Socioeconomic deprivation commonly accompanies chronic severe mental health disorders due to educational and employment limitations and can also increase heat vulnerability. A South Australian report of the geographic distribution in Adelaide of hospital admissions for mental health diagnoses indicates areas with highest rates of hospitalisations for schizophrenia, schizotypal and delusional disorders (Figure 4.4A), are often areas of highest relative socio-economic disadvantage (Figure 4.4B). Where affected individuals reside however, may be determined by many factors including the location of suitable accommodation and access to mental health services.
As well as schizophrenia, the ICD-10-AM category F20-F29 includes delusional disorders (F22.0-F22.9), a subcategory not investigated independently here. Altered mental status can be a symptom of heatstroke, heat stress or hyperthermia, often presenting as delirium or disorientation. Heatstroke is common in the elderly, has a high fatality rate due to multi-organ dysfunction, and often goes either
undiagnosed\textsuperscript{17,28,248} or not recorded as the underlying cause of death.\textsuperscript{2,18,37} Results in this study showed that of the nine patients whose deaths during heatwaves were attributed to schizophrenia, schizotypal and delusional disorders, eight of those were aged 75 years or over. That results indicated more than twice the relative risk of death for these elderly persons during heatwaves compared to control periods, raises the possibility they may have had delusional disorders rather than schizophrenia reported as the underlying cause of death. However to avoid speculation, further investigation would be required at an individual rather than population level of data.

A considerable body of literature has shown that dementia and cognitive impairment confer vulnerability to extreme heat.\textsuperscript{30,40,41,249} Findings showed an overall increase in hospital admissions for dementia during heatwaves, and an increase in mortalities in the 15-64 year age group in Adelaide. Although this latter finding was surprising, it is possible that older persons with dementia are more likely to reside in aged care facilities, thereby lowering their risk of death during heatwaves.\textsuperscript{254} Psychotropic drugs are commonly administered to dementia patients\textsuperscript{257} and pose a risk not only due to their pharmacological effects on thermoregulation\textsuperscript{40,43,248,249} but also their ability to modify vigilance levels\textsuperscript{43} and cognitive awareness of the need for adaptive behaviours to avoid thermal stress. Use of these medications has been shown to be associated with a significantly increased risk of heat-related hospitalization\textsuperscript{43} and death during heatwaves.\textsuperscript{254} Recreational use of drugs in persons with mental disorders can also pose a risk as psychotropic drugs with sympathomimetic properties such as cocaine and amphetamines can elevate body temperature and lead to heat stroke.\textsuperscript{258}

(2) Is heat a risk factor for mental ill-health?

Elevated temperatures may exacerbate psychiatric conditions. Environmental changes can affect mental health\textsuperscript{197} with excessive heat and humidity reported to be major influences affecting mood and behaviour.\textsuperscript{259} Fluctuations in weather have been noted to cause an increase in the incidence of mental stress, depression\textsuperscript{202} and suicide.\textsuperscript{260} As temperatures rise to extreme, stresses of everyday home, social or work life are likely to be exacerbated by lethargy, lack of sleep and the inability to function normally during oppressively hot conditions. For those predisposed to acute or chronic mental problems failure to gain relief from the heat for extended periods of time may trigger irritability,
neurosis and episodic psychological distress accompanied by risk behaviours such as excess alcohol consumption, violence and aggression. For persons living in rural areas, ongoing heat and dry conditions can cause psychological distress in several ways as discussed below in the context of climate change.

(3) The psychological effect of climate change

Climate change may contribute to many types of adversities that can be risk factors for mental ill-health. With heatwaves predicted to occur more frequently, it is possible that an increase in mental problems may be an indirect public health consequence of a warming climate and climate change. The effects of sustained heat and humidity, drought, water restrictions, power outages, and increased ambient air pollution are likely to have marked effects on the mental health of both rural and urban Australia, with possible increases in the incidence of episodic or chronic stress, despair and depression and tragically perhaps, suicide. Additionally there could be for some, a drift towards health-damaging personal behaviours with implications for law enforcement and social cohesion.

Perhaps the most traumatic and potentially devastating consequence of hot dry summers is bushfires. In 1983 a massive bushfire raged across much of south eastern Australia, claiming 28 lives and injuring 1,500 people. Some 385 homes were lost and many more damaged. A study by McFarlane et al investigating the ongoing mental health effect of this ‘Ash Wednesday’ disaster, found a doubling of psychiatric morbidity in victims compared to controls. Twelve months after the fire, 42% of the victims were defined as potential psychiatric cases and at 20 months post-event, 23% were defined as cases with diagnoses such as post-traumatic stress disorder, anxiety, and depression. During the summer of 2009, an exceptional and prolonged heat event occurred in south eastern Australia. Fuelled by high winds and extreme temperatures, fire storms of unprecedented proportions engulfed much of Victoria for weeks, resulting in the deaths of 173 people. Some 2,029 homes were destroyed and more than 500 people were treated for fire-related injuries. Whole communities lost family members, friends, homes, possessions and for many, their source of income. In terms of the mental health impact of bushfire disasters on a community, McFarlane et al state they “may both precipitate new disorders and exacerbate or modify the symptomatology of those who
are already disordered. Mental health services for the thousands of victims of the Victorian disaster will need to be ongoing for years, as it is likely the physical scars will heal long before the psychological ones. Future heatwaves and high fire risk days will no doubt cause stress and anxiety in the affected communities and with the number of days of extreme fire weather expected to increase in Australia some 4-25% by 2020, and possibly 15-70% by 2050, the psychological toll on those directly or indirectly affected by bushfires could be substantial.

The associated 2009 heatwave was reported to be the worst in a century and in Adelaide maximum temperatures remained above 40°C for six consecutive days with little overnight relief. The highest ever overnight minimum temperature for the city (33.9°C) was recorded during this time. Shortly thereafter, it was reported in the press that four patients with mental health disorders died during the heatwave and all were reportedly medicated with the antipsychotic drug Clozapine. As a result the South Australian Health Department advised vigilance for patients on antipsychotics. Clozapine, used in the treatment of schizophrenia, can paralyse thermoregulation due to its dopamine blocking and antimuscarinic properties (i.e. blocks muscarinic cholinergic receptors), and has been shown previously to be associated with heat stroke. As such, recommendations have been made that medicated patients and their carers be educated about heat intolerance and safety during hot weather. Whilst it cannot be claimed that the intense Adelaide heatwaves of 2009 and the preceding year were due to climate change, they may well be an indication of forthcoming summers.

Limitations

This study has several limitations. Firstly, the data assume correct ICD coding for MBDs. Errors in diagnostic classification would influence accuracy of effect estimates, particularly in cases of acute rather than chronic psychiatric illness. Changes in diagnostic criteria with the transition from ICD version 9 to version 10 during the study period may have increased the risk of misclassification. An example of nosologic discrepancy is the classification of ‘Dementia in Alzheimer’s Disease’ (F00-F00.9) being listed in a different chapter to ‘Alzheimer’s Disease’ (G30-G30.9), highlighting the ease with which inaccuracies in coding can occur. Secondly, the small sample size in some categories is acknowledged, limiting statistical power. Further, inflation of
Type 1 error may arise when multiple comparisons are undertaken as in this study. Accordingly cautious interpretation of some results is advised.

The study design prevented examination of possible adaptation over the study period or any potential differences in health impacts between early and late season heatwaves and hence the assumption of non-varying population response to heat over the study period may be one of limitations of the study.

Finally, in an ecological study such as this it is not possible to determine if those whose condition was attributed to a mental or behavioural disorder were admitted or died due to the effects of heat, or an increase in severity of their mental condition. Further research using clinical records would reveal medication history and prognostic outcomes thereby gaining additional insight into the reasons for the susceptibility of the mentally ill to the effects of heat.

Conclusion

This study has shown that heatwaves pose a salient risk to the well-being of those with mental and cognitive disorders. Others have observed that factors associated with prolonged hot dry conditions may exacerbate or initiate psychological illnesses. These points highlight the need for relevant health promotion and health intervention strategies when excessively hot conditions are expected. Government regulations will need to be formulated for action prior to, and during, forthcoming heatwaves, with a coordinated approach across government departments and services. Examples could include counselling by physicians and dispensing pharmacists, and the displaying of posters or information pamphlets in community mental health centres or outpatient clinics which may save lives during heatwaves. Regular monitoring of at risk groups such as those with mental illnesses should also be considered. The need to issue heat alerts to the public and health authorities, and the preparation of heatwave response plans similar to that developed for Queensland is gaining momentum as the occurrence of extreme heat events becomes more frequent.

Statistics indicate an increasing prevalence of mental ill health in Australia with 11% of persons reporting a long-term mental or behavioural problem in 2004-05, up from 5.9% in 1995. Recent figures in Australia show current expenditure on mental health
services totaling $3.0 billion and several factors are likely to impact on future mental health budgets. As a consequence of the demographic shift an increase in age-related mental impairments is imminent. With an estimated 231% rise in new dementia cases per year by 2050, the impact on health care facilities will be considerable. For rural and remote Australia, the psychological costs of drought will be a major ongoing issue.

With heat extremes very likely to occur more frequently in countries around the globe, these findings have relevance on a local and international scale. The extent of the future risk will be largely determined by planning and mitigation. Whilst population acclimatisation will undoubtedly occur to a certain extent, adaptation strategies may, for behavioural and physiologic reasons, be less achievable in persons with mental illnesses. With mental health being declared a National Health Priority Area in Australia, improved prevention and treatment options are imperative to avoid major economic and social costs to society of heat-related psychiatric morbidity and mortality exacerbated by climate change.
Chapter 5

A spatial analysis of ambulance callouts during heatwaves

5.1 Introduction

Numerous studies have reported negative health outcomes associated with exposure to extreme heat using hospital admissions as an indicator of morbidity. However as heat-induced deaths can occur rapidly and often prior to arrival at hospital, the investigation of pre-hospital morbidity during heatwaves may provide an insight into the aetiology of heat-triggered illnesses. Information regarding ambulance usage offers a rarely utilised source of morbidity data, and during heatwaves, may be a more sensitive indicator of the heat adaptability of a population than hospitalisations and deaths. Despite this, there is little literature regarding ambulance usage patterns during extreme heat events.

In addition to the regular suite of emergencies, during very hot weather ambulance services might expect to encounter ailments or incidents attributed to, or exacerbated by the heat. Humans are homeotherms and environmental stresses placed on the body by extreme temperatures can negatively influence health and behaviour. As well as heat-related illnesses such as heat stroke, heat exhaustion, dehydration and hyperthermia, heat exposure can trigger cardiorespiratory and cerebrovascular illnesses in susceptible individuals. Most vulnerable during heatwaves include those with either decreased adaptive capacity or increased exposure. Inability to thermally adapt may be due to age-related, physical, physiological, psychological or financial reasons; whilst increased exposure may occur in those working, exercising or living in hot environments. In addition to illnesses, uncomfortably hot weather has been linked with increased
numbers of work–related accidents\textsuperscript{274} and incidences of aggressive behaviour\textsuperscript{275} that may necessitate ambulance attendance.

An insight into a local population’s health responses to variations in environmental conditions can be gained by investigating spatiotemporal trends in morbidity data. Visualisation of ambulance response data using geospatial techniques can assist health authorities identify susceptible populations and generate hypotheses regarding potential relationships.\textsuperscript{164} In this way, resources and mitigation strategies can be targeted at the neighbourhood level in an attempt to lower the incidence of illnesses occurring during periods of high temperatures.

The city of Adelaide has a population of 1.16 million people,\textsuperscript{276} accounting for 73\% of the state’s total population. As mentioned in previous chapters, the city often swelters through hot spells during the summer months. The South Australian Ambulance Service (SAAS) is the emergency ambulance provider for metropolitan Adelaide and South Australia. The SAAS is an independent fee-for-service organisation, providing pre-hospital medical care and patient transport in cases of accident, illness and injury.\textsuperscript{277,278} Smaller volunteer and private ambulance services operating in South Australia include the St John Ambulance service, Emergency Medical and Trauma Ambulance Service; and WorkCare SA EMR (Paramedic Response).\textsuperscript{279} As well as assisting in medical emergencies, ambulance services in Australia fulfil other roles as listed in Box 5.1.

The aim of this study was to investigate patterns of ambulance callouts during heatwaves in metropolitan Adelaide and, using geospatial techniques, identify heat-susceptible suburbs. Findings will assist emergency services in future planning and resource allocation in the face of climate warming.
Box 5.1: The Ambulance service in Australia fulfils the following roles:

- Providing emergency pre-hospital care and transport in situations of sudden injury or illness
- Retrieving emergency patients
- Assisting in or conducting road and other accident rescue
- Planning and co-ordinating patient services in multi-casualty situations
- Aero-medical services including both fixed and rotary wing services.
- Accessing patients in confined, hazardous and remote places and situations
- Providing inter-hospital transport
- Non-emergency transport of patients

*Source:* Australian Institute for Primary Care.280

### 5.2 Methods

The study period was 1 July 1993 to 30 June 2005. The study area of metropolitan Adelaide was defined *a priori* as those suburbs with postcodes listed numerically between 5000 and 5199 (see Appendix A). Data relating to SAAS callouts/responses in metropolitan Adelaide (excluding between-hospital transfers) were accessed from the South Australian Department of Health and included de-identified data relating to patient demographics. Presenting complaints were categorised into: assault, blunt (non-penetrating) trauma, motor vehicle accidents and other road injuries, sport, work-related incidents and falls, and respiratory, cardiac and neurological conditions. Postcodes of suburbs attended by ambulances were also recorded, and data with missing or miscoded postcodes were excluded from the dataset. Patients may or may not have been transported from the original callout site.

Meteorological data were obtained from the South Australian Bureau of Meteorology Kent Town monitoring station, a central location considered to be representative of conditions across the city. Seasons were defined as warm (October to March) and cool (April to September), and heatwaves were defined as three or more days when daily maximum temperatures ($T_{max}$) reached or exceeded 35°C,31 as in previous studies within
this thesis. As the focus of the study was heat-related, only data for the warm season were used in analyses.

**Data analysis**

SAAS data were stratified into age groups (0-4, 5-14, 15-64, 65-74 years and 75 years or over), presenting condition and postcode. Statistical analysis was undertaken using a case series approach.\(^{210}\) Average daily SAAS callouts during heatwaves compared to non-heatwave periods were analysed using Poisson regression, or if overdispersion was found to be present in the underlying count data, negative binomial regression models were used.\(^{31}\) Results were expressed as Incidence Rate Ratios (IRRs) with 95\% confidence intervals (CIs). Values of \(P < 0.05\) were considered to be statistically significant.

The association of SAAS responses with \(T_{\text{max}}\) was initially investigated using a lowess\(^{233}\) smoothed scatterplot (Figure 5.1). Threshold temperatures were estimated visually and confirmed quantitatively using a nonlinear least-squares estimation (hockey stick method\(^{182,252,253}\)) incorporating the \(\text{nl}\) function in Stata v9.2\(^{233}\) as outlined previously in Chapter 4.

**Spatial visualization of data**

The Adelaide metropolitan area is comprised of 128 Australia Post postcodes, most representing multiple suburbs. Postal areas, an approximation of Australia Post postcode regions,\(^{281}\) were used as the spatial unit. An ESRI shapefile of postal areas within the Adelaide metropolitan area was obtained by utilising the Australian Bureau of Statistics’ (ABS) CDATA2001,\(^{282}\) an information tool that combines ABS census of population and housing data with mapping software. Population figures for 2005 were also sourced from the ABS.

Geospatial analysis was undertaken by merging data sources and using postcodes/postal areas as the spatial unit. Visualisation of the data was achieved by creating choropleth maps using the `spmap` command in Stata v9.2, and by importation of data into the GIS software ArcMap v9.2 (ESRI Corp., Redlands, CA, USA).
## 5.3 Results

During the study period 29 heatwaves occurred in Adelaide ranging from 3 to 8 days in length. The highest maximum temperature was 44.3°C recorded in February 2004. There were a total of 704,517 postcode identified ambulance responses in the Adelaide metropolitan area, of which 18,738 occurred during predefined heatwave periods.

Table 5.1 shows descriptive statistics of callouts by patient age and shows that most calls attended were to persons in the 15-64 year age group followed by those in the 75 years and over age group. Those aged 15-64 years accounted for 41.1% of all callouts but this proportion increased to 42.14% during the warm season and to 44.02% during heatwaves. This trend was not observed for other age groups.

<table>
<thead>
<tr>
<th>Age group</th>
<th>N (h/wave)</th>
<th>% of total</th>
<th>N (warm)</th>
<th>% of total</th>
<th>N (total)</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>839</td>
<td>4.48</td>
<td>16,821</td>
<td>4.92</td>
<td>34,865</td>
<td>4.95</td>
</tr>
<tr>
<td>5-14</td>
<td>563</td>
<td>3.00</td>
<td>10,679</td>
<td>3.12</td>
<td>22,323</td>
<td>3.17</td>
</tr>
<tr>
<td>15-64</td>
<td>8,248</td>
<td>44.02</td>
<td>144,201</td>
<td>42.14</td>
<td>289,537</td>
<td>41.10</td>
</tr>
<tr>
<td>65-74</td>
<td>2,698</td>
<td>14.40</td>
<td>50,444</td>
<td>14.74</td>
<td>104,320</td>
<td>14.81</td>
</tr>
<tr>
<td>75+</td>
<td>6,350</td>
<td>33.89</td>
<td>118,294</td>
<td>34.57</td>
<td>251,071</td>
<td>35.64</td>
</tr>
<tr>
<td>Missing*</td>
<td>40</td>
<td>0.21</td>
<td>1,738</td>
<td>0.51</td>
<td>2,401</td>
<td>0.34</td>
</tr>
<tr>
<td>TOTAL</td>
<td>18,738</td>
<td>100</td>
<td>342,177</td>
<td>100</td>
<td>704,517</td>
<td>100</td>
</tr>
</tbody>
</table>

*Age group not reported

### Threshold maximum temperature

Plotting the relationship of smoothed daily callouts against $T_{max}$ in the warm season (Figure 5.1) showed that after an initial decline in callouts during cooler weather, a
gradual increase was seen above 20°C. An abrupt inflection in the curve occurred around 35°C associated with a strongly positive linear relationship of callouts with increasing temperature. Using non-linear least squares estimation\textsuperscript{233} (hockey stick analysis), the threshold temperature was determined to be 34.6°C (95% CI 30.8-38.4, $P = 0.000$), above which SAAS callouts increased by 2.6 callouts per 1°C rise in $T_{\text{max}}$.

![Figure 5.1: The relationship between SAAS callouts and $T_{\text{max}}$ during the warm season](image)

**Counts of ambulance callouts per postcode**

Over the study period the total number of SAAS callouts per postcode during heatwaves ranged from 1 to 952 with a median of 123. The suburb recording the lowest number ($N = 1$) was Paracombe (postcode 5132) followed by Piccadilly (postcode 5151), Basket Range (5138), Carey Gully (5144) and Inglewood (5133), all of which are small localities near the foothills to the east of the city. By far the most callouts ($N = 952$) occurred within the Adelaide central business district (CBD) (postcode 5000) with the second highest ($N = 641$) being postcode 5112 comprising the northern suburbs of Elizabeth, Elizabeth East, Elizabeth South, Elizabeth Vale, Elizabeth Grove and Hillbank, followed by 5108 (Paralowie, Salisbury, Salisbury Downs and Salisbury North) with 547 responses.

Whilst quantitation is useful, visualisation of the data using geographical mapping tools can more readily allow the identification of spatial patterns.\textsuperscript{283} Figure 5.2A is a choropleth map of ambulance callouts during heatwaves by postcode (postal area)
constructed using Stata, showing high ranking areas extend further to the west and north of the city than to the east and south. Figure 5.2B is an aligned map of the corresponding suburbs.

Categorical examination of presenting conditions revealed heterogeneity between postcodes. In the Adelaide CBD where most callouts occurred, total attendances during heatwaves were highest for blunt trauma followed by neurological and cardiac conditions, and assault, however this pattern varied between postcodes depending on factors such as local demographics, activities and land use. Across the whole
metropolitan area average daily callouts were highest for cardiac and neurological conditions (Figure 5.3). A similar pattern was seen during non-heatwave periods in the warm season (data not shown).

![Classification of Ambulance Usage During Heatwaves](image)

**Classification of Ambulance Usage During Heatwaves**

Adelaide metropolitan area

- Assault
- Work
- Road MVA
- Other road
- Sport
- Fall
- Blunt
- Respiratory
- Neurological

**Figure 5.3:** Pattern of presenting conditions for ambulance callouts across the metropolitan area during heatwaves.

**Counts weighted by population**

In order to determine if areal or population size was responsible for high response rates in some of the larger postal areas, counts were weighted by population. Callouts per 1,000 population ranged from 2.45 to 368.4, with a median of 14.29. Angle Vale to the north of the city, and the foothills communities of Paracombe, Basket Range and Piccadilly had the lowest number of callouts per population. Highest by far was the sparsely populated southern industrial suburb of Lonsdale (postcode 5160) a statistical outlier (see Discussion) with more than three times the rate of the second ranked Adelaide city centre.

To best represent the most susceptible suburbs, Figure 5.4A was constructed in ArcMap v 9.2 using nominated, rather than uniform, categories for counts per population, as indicated by the legend. In a similar trend to Figure 5.2A, the areas with the lowest response rates were located east of the city whilst some suburbs to the north and north-west of the CBD remain higher irrespective of the number of residents. Apart from the
city centre (postcode 5000), industrial suburbs (including 5160) and those with a high proportion of lower income households (postcodes 5015, 5012, 5112) appeared to be high risk localities (Figure 5.4A) in terms of callouts. However other factors including car-ownership, ambulance membership rates, and data relating to residents versus employees in these areas, may need to be considered when inferring vulnerability. Figure 5.4B is an aligned map of industrial areas within the metropolitan region.

Spatial patterns of ambulance callouts during the warm season failed to differ markedly from that during heatwaves (data not shown), giving little indication of areas more
susceptible during heatwaves than at other times. Maps constructed to compare the ratio of total callouts during heatwaves to counts during the warm season, and the ratio of rates per population during heatwaves compared to the warm season as a whole can be seen in Figures 5.5A and 5.5B respectively. Note these maps use colour shadings based on calculations using different denominators, and comparison may be misleading.

Regression analysis

Negative binomial regression showed an increase of 3.6% in the usage of SAAS services in the metropolitan area during heatwaves compared to non-heatwave periods in the warm season (IRR of 1.036, 95% CI 1.006-1.068, $P = 0.020$). In the 15-64 year age group, the IRR of ambulance callouts during heatwaves compared to all non-heatwave periods was 1.075 (95% CI 1.031-1.121, $P = 0.001$). Stratification by gender showed that both males and females of this age were at risk during heatwaves with increases of 7.1% and 7.8% for females and males respectively. These findings are
consistent with the higher proportion of attendances during heatwaves compared to the warm season in this age group as seen in Table 5.1.

With clear patterns of areal heat susceptibility not geospatially evident, regression analysis provided greater statistical rigour in determining postcodes with increased callouts during heatwaves compared to non-heatwave periods. As seen in Table 5.2, the Adelaide CBD showed a non-significant (although 6%) increase in callouts during heatwaves. The highest effect estimates were noted for postcodes 5012 where a large proportion of low-income families reside, followed by 5015. These postcodes both feature as darkly shaded areas of the maps in Figures 5.2A, 5.4A and 5.5A. A satellite image (Figure 5.6) shows the suburbs of Mansfield Park (postcode 5012), located to the centre right and Port Adelaide (5015) on the left, to be highly industrialised areas, as displayed in Figure 5.4B.

Table 5.2: Incidence rate ratios (IRR) and 95% confidence intervals (CI) for postcodes with increases in ambulance attendances during heatwaves 1993-2005.

<table>
<thead>
<tr>
<th>Postcode</th>
<th>Suburba</th>
<th>IRRa</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>5000</td>
<td>Adelaide CBD</td>
<td>1.06</td>
<td>0.99-1.13</td>
</tr>
<tr>
<td>5012</td>
<td>Mansfield Park, Athol Park, Woodville Gardens, Woodville North</td>
<td>1.23</td>
<td>1.08-1.40</td>
</tr>
<tr>
<td>5015</td>
<td>Port Adelaide, Birkenhead, Ethelton, Glenville</td>
<td>1.21</td>
<td>1.04-1.40</td>
</tr>
<tr>
<td>5041</td>
<td>Colonel Light Gardens, Cumberland Park, Daw Park, Panorama, Westbourne Park</td>
<td>1.20</td>
<td>1.05-1.37</td>
</tr>
<tr>
<td>5045</td>
<td>Glenelg, Glenelg South</td>
<td>1.10</td>
<td>1.00-1.21</td>
</tr>
<tr>
<td>5118</td>
<td>Gawler, Gawler Belt, Buchfelde, Hewett, Kalbeeba, Kingsford, Willaston</td>
<td>1.14</td>
<td>1.01-1.29</td>
</tr>
</tbody>
</table>

a IRRs are based on daily mean incidence of ambulance callouts during heatwaves over incidence during non-heatwave periods, with trend and seasonality controlled for.
As indicated previously, patterns of presenting conditions were area-specific. Similarly, regression analysis showed heterogeneity in types of attendances found to be increased during heatwaves. From Table 5.3, postcode 5012 had significant increases in ambulances responses for assault, work and neurological conditions, whilst postcode 5015 showed a significant increase in work related conditions only. Postcode 5118, the township of Gawler and surrounds, 40 km north of Adelaide (Table 5.2 and Figure 5.2B) incorporates a major transport route to the mid north of the state and the Barossa Valley, and showed a significant increase in motor vehicle accidents and blunt trauma during heatwaves.

Table 5.3: Incidence rate ratios (IRR) and 95% confidence intervals (CI) for presenting conditions in selected postcodes during heatwaves compared to non-heatwave periods.

<table>
<thead>
<tr>
<th>Postcode</th>
<th>Presenting condition</th>
<th>IRR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>5012</td>
<td>Neurological</td>
<td>1.61</td>
<td>1.10-2.37</td>
</tr>
<tr>
<td></td>
<td>Work-related</td>
<td>3.27</td>
<td>1.06-10.08</td>
</tr>
<tr>
<td></td>
<td>Assault</td>
<td>1.73</td>
<td>1.13-2.65</td>
</tr>
<tr>
<td>5015</td>
<td>Work-related</td>
<td>3.38</td>
<td>1.47-7.78</td>
</tr>
<tr>
<td>5118</td>
<td>Blunt trauma</td>
<td>1.84</td>
<td>1.14-2.99</td>
</tr>
<tr>
<td></td>
<td>Motor vehicle accidents</td>
<td>2.75</td>
<td>1.73-4.36</td>
</tr>
</tbody>
</table>
5.4 Discussion

This study builds upon the findings of Nitschke et al.\textsuperscript{31} who demonstrated a 4% increase in ambulance transports across metropolitan Adelaide during heatwaves with significant assault-related increases in the 15-64 year age group. The current study is consistent with these findings, revealing a 3.6% increase in ambulance callouts during heatwaves compared to non-heatwave periods, with an associated increase of 2.6 callouts for every 1°C increase in daily maximum temperature above a threshold of 34.6°C. Similar associations between extreme heat and increased ambulance usage have been seen elsewhere in Australia\textsuperscript{270} and overseas,\textsuperscript{6,285,286} validating the appropriateness of ambulance data as an indicator of population morbidity during heat events.

That the available SAAS data includes the real-time location of the patient adds a spatial epidemiological dimension to ambulance studies and can be useful in generating hypotheses regarding association to environmental exposure.\textsuperscript{164} It must be noted that locations attended by ambulances do not necessarily represent the suburb of residence of attendees, a proportion of whom may have been working in, visiting or passing through the area when illness or injury occurred. Notwithstanding this, the present study has highlighted several regions across the city where counts of ambulance callouts were above average and communities may be susceptible to increased morbidity during heatwaves.

The SAAS responded to substantially more calls from the CBD of Adelaide during heatwaves than any other in the metropolitan region, due in part perhaps to the concentration of people working or shopping in the city during the day and socialising during warm evenings. Inference regarding the impact of heat on the resident population is difficult to ascertain. It is recognised that the urban heat island effect and thermal mass in cities leads to higher daytime and nighttime temperatures\textsuperscript{2,24,199} increasing the risk of heat-related illness for both residents\textsuperscript{27} and visitors.

When weighted by population, the Adelaide CBD ranked second behind Lonsdale, identified as a statistical outlier. Lonsdale (postcode 5160) was noted to have more than
25 times the median rate per 1000 population. On closer investigation it was discovered that this mainly industrial southern suburb (Figure 5.4A, 5.4B) had a very low population in 2005, being the site of a recently decommissioned oil refinery and associated housing estate. The number of ambulance callouts was not high; however a drop in population during the study period introduced an information bias and skewed results accordingly. This example highlights the need to exercise caution when using small spatial units with low counts of health data as this can lead to unreliable rates, particularly when enumerating rare events\(^{164}\) such as ambulance responses. This raises the issue of the relevance of population data in this context, particularly in outer suburban areas where there are relatively few residents. As well as Lonsdale appearing to be notable in this respect, similar examples of low population areas showing as darkly shaded areas in Figure 5.5B, include the site of a military base and aerodrome in the north, and a working quarry in the east.

Notwithstanding, the darker shaded areas in Figure 5.4A indicate that suburbs with the highest callout rates include several industrial/residential areas. Postcodes 5012 and 5015 which also feature in Figure 5.5A, were shown by regression analysis to have increases in ambulance callouts during heatwaves of 23% and 21% respectively (Table 5.2). Although sample size was small, the risk of work related illness or injury requiring ambulance attendance increased more than 3-fold in these postcodes. That callouts overall were increased in the 15-64 year age group supports the notion that occupational heat stress may be an underrated factor in studies of heat-related morbidity. These findings are consistent with those of a similar Canadian study investigating the relationship between extreme heat and ambulance responses, which also reported an increase in calls to industrial areas in Toronto.\(^6\) The authors also reported an increase in callouts to some recreational areas, and a more recent but similar study in Toronto found a high rate of heat–related ambulance dispatch calls to recreation areas along the waterfront.\(^{287}\) Similarly in the present study, there was an increase in callouts in postcode 5045 (Table 5.2), location of the beachside suburb of Glenelg. This is a popular recreational and tourist area where a high transient population during summer holiday periods may influence ambulance usage patterns during heatwaves.

In postcode 5012, assault-related callouts increased significantly during heatwaves. A similar increase for assaults in the 15-64 year age bracket was reported in the original
study by Nitschke et al.\textsuperscript{31} Previous research has shown a relationship between hot weather and violence, leading to the generation of the heat-hypothesis that states that uncomfortably hot temperatures increase aggressive motives and aggressive behaviour in humans.\textsuperscript{275} Whether excessive alcohol consumption during the heat was a causal factor in some incidents, injuries or crimes, cannot be determined from the current data. Further research using both qualitative and quantitative approaches should be conducted to investigate this potential association.

Postcode 5041 also showed significant increases in SAAS attendances (Table 5.2), for reasons that were not evident but which may relate to unaccounted risk factors concerning the underlying health status of the population, demographics, housing characteristics, or local heat islands.\textsuperscript{287} This postal area although not labeled specifically, features as a high risk area in Figures 5.2A and 5.5A.

The second and third highest ranking suburbs for counts of ambulance callouts during heatwaves included those with a large proportion of low income households such as postcode 5112 (Elizabeth, Elizabeth East, Elizabeth South, Elizabeth Vale, Elizabeth Grove and Hillbank) and 5108 (Paralowie, Salisbury, Salisbury Downs and Salisbury North), the locations of which can be seen in Figure 5.2A and 5.2B. It has been shown that households with higher incomes are more likely to use air-conditioners.\textsuperscript{213} A recent report on the impact of rising electricity prices on low-income households in South Australia\textsuperscript{288} claims that cooling is often more expensive in many low income dwellings due to energy inefficient housing designs, and that residents often have higher risks of life-threatening thermal-sensitive health conditions when (heating or) cooling cannot be maintained due to financial constraints. These findings are consistent with a U.S. study investigating place-based risk factors associated with heatwaves in St. Louis which identified higher mortality rates in the warmer and more disadvantaged areas of the city.\textsuperscript{289}

It is difficult to draw conclusions regarding the relationship between socioeconomic status and heat-related morbidity using ambulance responses as an indicator. With no free ambulance service available in Adelaide,\textsuperscript{277} the cost of ambulance transport may be prohibitive for those without personal health insurance or SAAS membership, a factor that may influence usage patterns in the urban poor. Alternatively it is possible that
families unable to afford private vehicles may rely more heavily on ambulance services for transport to hospital. Further research to investigate the spatial and socioeconomic characteristics of hospitalisations and emergency department visits during heatwaves may clarify these issues.

With more than 80% of households in South Australia having access to air-conditioning, individuals may be more likely to experience thermal stress outside of the home. It is therefore imperative that heat-awareness messages usually targeted towards the at-risk elderly and the very young, also consider those in other age groups who may be vulnerable to direct heat exposure outdoors due to daily work and travel commitments.

As mentioned in previous chapters, an extreme heat event occurred during the summer of 2009, the data for which was not included in the current analysis. Anecdotal evidence suggests in the first few of the six consecutive 40-plus degree days, there was an increase of between 10 and 15% in patients with heat-related illnesses requiring ambulance assistance, followed by a day when ambulance callouts soared to 900. State Parliamentary transcripts indicate that SAAS attended an average of 580 and 798 emergency and non-emergency incidents respectively per day during the heatwave, an increase of almost 300 more than normal. The extended period of very extreme temperatures led to an increase in callouts well above those seen in the present study. An average of 12 deaths per day were attended by SAAS, compared with a normal average of approximately four per day, however it could not be determined at that stage, if they could be attributed to the heat.

Knowledge of locations at risk can assist in formulating targeted public health initiatives for communities and workplaces. Neighbour-watch programs can be initiated to encourage residents to check on at-risk or elderly neighbours during heatwaves, and local industries could be encouraged to formulate heat-health safety procedures. Some worksites already have well organized protocols in place for employees in heat-prone occupations. As an example, at an Adelaide factory where employees work alongside high temperature furnaces, occupational health and safety measures ensure adequate work breaks and fluid replacement, particularly during hot weather. Hydration guides, using urine colour as an indicator of dehydration, are provided to individuals as well as
being displayed in toilet facilities. With very little research having been conducted on heat stress in indoor and outdoor work places, occupational heat stress is a public health issue warranting further investigation.

**Limitations**

This study has several limitations. First, observations were excluded from the dataset if postcodes were either missing or invalidly recorded; hence the number of ambulance attendances was a slight (1.5%) underestimation of the actual total. Furthermore the study used data from South Australia’s main ambulance service (SAAS) only and did not take into account smaller volunteer and private ambulance services.

Although presenting conditions were categorised as previously mentioned, there is the possibility of ambiguity between classifications and importantly, the proportion of conditions which were specifically heat-related could not be determined. Furthermore, accuracy of estimates may have been compromised by small sample numbers in some instances where data were stratified by presenting conditions and postcode.

Temperature data from a central location were used to represent conditions across the urban sprawl as an indication of personal levels of exposure and may vary considerably from actual exposure levels. It is acknowledged that for geographic and meteorological reasons temperatures vary slightly between Adelaide suburbs, often being higher to the north of the city and cooler in the south, with afternoon seas breezes in the west and gully breezes in the east. However the chosen meteorological monitoring station is located within a 50 km radius of all suburbs as recommended elsewhere.  

Geospatial representation of data may be visually misleading to the reader when spatial units vary in size. Furthermore, ABS postal areas for which population data are available are an aggregation of census collection districts (CDs) and boundaries are not necessarily identical to those of postcode divisions for which health outcome data are available. Areal discrepancies between the two are more likely to occur in less populated outer metropolitan regions as inner city urban areas are usually more highly correlated due to higher population density and hence smaller CDs. Appendix A includes maps of Adelaide metropolitan postcodes (Figure A.1) and postal areas (Figure A.2) for comparative purposes. Furthermore, it is possible that there may be inherent
inaccuracies involved in using aggregated counts of ambulance attendances to calculate rates per spatial unit population,\textsuperscript{283} as the connection between attendance locations and patients’ residential addresses is unknown. Small samples sizes and small population numbers may skew results and lead the reader to draw inaccurate conclusions as discussed earlier. Additionally, data aggregated in spatial units and displayed in choropleth form should not be used to make inferences and generalisations about individuals within a population (ecological fallacy).\textsuperscript{164}

Data in this study did not include ambulance attendances to the Adelaide airport district (postcode 5950), which although in the metropolitan area, fell outside the predefined postcode range of 5000-5199. Additionally, there was no corresponding ABS postal area for the postcode of 5150 comprising the hills suburbs of Leawood Gardens and Eagle on the Hill.

A possible confounding factor not accounted for in this study was air pollution. Ozone concentrations are highest in warm weather\textsuperscript{162,293} and have been shown to have an impact on health during heatwaves.\textsuperscript{200} Ozone is more likely to be a risk factor in suburban areas than in the busy urban centre, due to nitrogen oxide-rich motor vehicle emissions in the city having a scavenging effect on ozone.\textsuperscript{128} In Adelaide it is the coastal western suburbs that are more likely to have higher ozone levels (Mr. R. Mitchell, EPA (SA), pers. comm. 29/11/07)) and hence for biometeorological reasons, ozone as well as temperature may have contributed to morbidity during heatwaves in these suburbs. Day of the week was not controlled for in this study although this would be an informative factor in hypothesis generation concerning heat exposure in occupational settings. Finally, the concept of spatial autocorrelation, where nearby geographic regions are more likely to show clustering than distant ones,\textsuperscript{164} was also not controlled for in this study.

**Conclusion**

This study has shown an association between negative health outcomes and heatwaves by observing changes in ambulance usage patterns in an urban area. SAAS callouts increased during heatwaves with a linear increase at maximum temperatures above 34.6°C. Spatial analysis indicated regions including the CBD and some industrial and
disadvantaged suburbs, where the relative risk of ambulance callouts during heatwaves was greatest. Although sample sizes were small, work-related incidents requiring ambulance attendance showed increases of up to 3-fold in some areas.

The results of this study have public health implications for government and local agencies, and highlight the need to prepare for a disproportionate increase in emergency and ambulance services as heatwaves become more intense and frequent. It is timely for health authorities, industrial organisations and local communities to consider adaptation and mitigation strategies and heat-awareness campaigns in the workplace, in order to promote the health and safety of all sectors of the community during periods of extreme heat. Heat response plans and government initiated heat wave warning systems require evidenced based data such as this to determine thresholds for the activation of preventative heat strategies relevant to the health and emergency services sectors. For targeted public health interventions however further research is warranted on the nature of morbidity during extreme heat, using both hospital and pre-hospital data to identify vulnerable populations and occupations, and high risk environments.

The concept of preventative strategies in terms of ambulance usage is not new as seen in Box 5.2 - a light hearted, but insightful poem by Joseph Malins, penned in 1895. Now more than a century later as the planet faces the challenges of a changing climate, policy makers still seem reluctant to ‘build up the fence’ as an alternative to ‘the ambulance down in the valley’.
Box 5.2  The Ambulance Down in the Valley  (Joseph Malins 1895)

Twas a dangerous cliff, as they freely confessed,
Though to walk near its crest was so pleasant;
But over its terrible edge there had slipped
A duke, and full many a peasant.
The people said something would have to be done,
But their projects did not at all tally.
Some said ‘Put a fence ‘round the edge of the cliff,’
Some, ‘An ambulance down in the valley.’

The lament of the crowd was profound and was loud,
As their tears overflowed with their pity;
But the cry for the ambulance carried the day
As it spread through the neighbouring city.
A collection was made, to accumulate aid
And the dwellers in highway and alley
Gave dollars or cents – not to furnish a fence –
But an ambulance down in the valley.

‘For the cliff is all right if you’re careful,’ they said;
‘And if folks ever slip and are dropping,
It isn’t the slipping that hurts them so much
As the shock down below – when they’re stopping.’
So for years (we have heard), as these mishaps occurred
Quick forth would the rescuers sally,
To pick up the victims who fell from the cliff,
With the ambulance down in the valley.

Said one, to his pleas, ‘It’s marvel to me
That you’d give so much greater attention
To repairing results than to curing the cause;
You had much better aim at prevention.
For the mischief, of course, should be stopped at its source;
Come, neighbours and friends, let us rally.
It is far better sense to rely on a fence
Than an ambulance down in the valley.’

‘He is wrong in his head,’ the majority said;
‘He would end all our earnest endeavour.
He’s a man who would shirk this responsible work,
But we will support it forever.
Aren’t we picking up all, just as fast as they fall,
And giving them care liberally?
A superfluous fence is of no consequence,
If the ambulance works in the valley.’

The story looks queer as we’ve written it here,
But things oft occur that are stranger
More humane, we assert, than to succour the hurt
Is the plan of removing the danger.
The best possible course is to safeguard the source
By attending to things rationally.
Yes, build up the fence and let us dispense
With the ambulance down in the valley.

Chapter 6

Heatwaves and hospital emergency department visits

6.1 Introduction

As noted in previous chapters, heatwaves can have major adverse effects on the health of vulnerable sub-populations during summer. Many ecological studies have used hospital admissions or more commonly, mortality, as the outcome of interest in heat-health investigations. These data have limitations as general hospital data generally include routine booked admissions not necessarily representative of acute illness. Furthermore progression or change in clinical status during hospital stays may lead to discrepancies between presentation diagnoses and the discharge diagnoses commonly supplied with hospital data. Mortality data, on the other hand, represent only the most serious of outcomes and thus could under represent the wider impact of extreme heat on population health. This may be particularly so in a community reasonably well adapted to heatwaves such as Adelaide. Hospital emergency department (ED) presentations may be a more appropriate and sensitive indicator of acute onset changes in morbidity during extreme heat in this city.

A limited number of studies elsewhere have used emergency data to assess heatwave morbidity. A small non-significant increase in emergency admissions was observed during a heatwave in London in 1995, and an excess of 1,182 hospitalisations and 16,166 ED visits occurred in association with the Californian heatwave of 2006. In France in 2003, and Australia in 1993 and 2009, ED presentations of patients classified as having a heat-related illness were assessed in relation to single heatwaves. Additionally during the 1995 Chicago heatwave, Dematte et al used ED records to
identify patients admitted to intensive care units with classic heat stroke. However, overall there is little information about the impact of heatwaves on other cause-specific ED visits, especially in the Southern Hemisphere.

High environmental temperatures can have direct and indirect effects on human health. As mentioned previously, the body’s response to heat involves an increase in blood flow to the skin of up to 10-fold and heat transfer to the environment by sweating.\textsuperscript{16,17} The associated loss of salt and body water can lead to dehydration, compromising normal thermoregulation and contributing to the progression from heat stress to heat stroke.\textsuperscript{16,17} Dehydration, together with the alteration to normal blood flow can place stress on the cardiovascular system resulting in haemoconcentration, hence increasing the risk of heart failure or thrombosis.\textsuperscript{16} With warm conditions conducive to microbial proliferation, the incidence of infectious and food-borne diseases may be an example of an indirect association with heat.\textsuperscript{205}

Generally the elderly, the socially isolated, persons with chronic illnesses and mental disorders are considered to be the most vulnerable.\textsuperscript{8,30,34,296} Persons with epilepsy have also been reported by some to be at risk.\textsuperscript{19,41} Demographic indicators of heat susceptibility can include race, gender, and socioeconomic status, along with neighbourhood of residence and standard of housing.\textsuperscript{25,289,297} Hence geospatial techniques involving statistical analysis and visual interpretation of event frequencies can be useful in identifying vulnerable communities and formulating appropriate neighbourhood risk management strategies,\textsuperscript{298} as well as providing useful information to government authorities on relevant resource allocation and infrastructure establishment needs.

Like ambulance callouts, ED visits provide a valuable source of morbidity data that generally represent rapid onset conditions or those that have deteriorated to the extent that urgent medical attention is required. This study uses syndromic surveillance of ED data with a range of outcomes investigated, in an attempt to identify vulnerable populations and their health responses during heatwaves over a five year period in Adelaide.
It should be noted that in order to present a complete picture of the nature of ED visits during heatwaves, this study was undertaken in conjunction with the South Australian Department of Health, and some results included in this chapter are not as a result of my own work. The analyses of heat-related illnesses during heatwaves is however my own, along with spatial analysis of the data.

6.2 Methods

Daily counts of ED visits to Adelaide metropolitan hospitals for the period 1 July 2003 to 31 March 2008 were obtained from the South Australian (SA) Department of Health. Data were restricted to residents of the Adelaide metropolitan area, defined as the region lying within postcodes 5000 to 5199, excluding 5130 to 5134 which are external to the Adelaide Statistical Division (Mr. G. Tucker, SA Health, pers. comm. 2008). Diagnoses were classified according to International Classification of Diseases (ICD) codes. Both ICD version 9 and 10 were specified to ensure all relevant diagnoses were included. The following cause-specific conditions were identified codes: ischaemic heart disease (ICD-9: 410 - 414, ICD-10: I20 - I25); stroke (ICD-9: 430 - 448, ICD-10: I60 - I69), cardiovascular disease (ICD-9: 390 - 459, ICD-10: I00 - I99); respiratory disease (ICD-9: 460 – 519, ICD-10: J00 - J99); mental disorders (ICD-9: 290-294, ICD-10: F00 - F99); epilepsy (ICD-9: 345-345, ICD-10: G40-G41); renal disease (ICD-9: 580 – 599, ICD-10: N00 – N39); food related disorders (ICD-9: 003 or 005, ICD-10: A02 – A05). In an attempt to identify specific heat-related illnesses, a category was included termed ‘effects of heat’ incorporating ‘effects of heat and light’ and ‘exposure to excessive natural heat’ and ‘volume depletion’ (ICD-9: 992, or 276.5 or E900, ICD-10: T67, X30, or E86).

Meteorological data were obtained from the SA Bureau of Meteorology, Kent Town monitoring station. Seasons were defined as warm (October to March) and cool (April to September), and heatwaves were defined as three or more consecutive days when daily maximum temperatures ($T_{max}$) reached or exceeded $35^\circ C$, as in previous studies within this thesis. Only data for the warm season were used in analyses.
Data analysis

Data were stratified into age groups (0-4, 5-14, 15-64, 65-74 years and 75 years or over), and presenting condition. As defined previously, statistical analysis was undertaken using a case series approach, incorporating Poisson regression models. Each model was tested for overdispersion and if found to be present, negative binomial regression models were used. Results were expressed as Incidence Rate Ratios (IRRs) with 95% confidence intervals (CIs). Values of $P < 0.05$ were considered to be statistically significant. Stata v10 was used for data management and analysis.

Spatial visualization of data

Postal areas, formulated by the Australian Bureau of Statistics (ABS), are based on census collection districts, and are an approximation of Australia Post postcode regions. For geospatial analysis, postal areas incorporated in ABS shapefiles of the Adelaide metropolitan area, were used as a proxy for postcodes as outlined in Chapter 5. The few observations with invalid postcodes were excluded from the dataset.

Regression analyses as previously described were performed on postcode stratified data, and regression results entered into a new dataset. Postcodes yielding statistically significant ($P$-value < 0.05) point estimates were identified and percentage deviations calculated. The remaining postcodes were coded as zero to indicate no significant difference between heatwave and non-heatwave periods. Results were displayed geospatially via choropleth maps by linking the attribute data to map coordinate data using ArcView Spatial Analyst software incorporating ArcMap v9.2 (ESRI Corp., Redlands, CA, USA).

6.3 Results

Between 1 July 2003 to 31 March 2008 there were 1,467,688 visits to Adelaide’s public and private hospital emergency departments, 768,221 of which fell during the warm season. Over the study period there were 12 heatwaves ranging in duration from 3 to 15 days, the longest of which occurred in March 2008 with a record-breaking 15 consecutive days of temperatures over 35°C, including 13 days of 37.8°C or above.
Results for all age data showed there was no significant increase in ED visits to metropolitan hospitals during heatwaves. However heatwaves were found to be associated with an increase in all-cause ED visits to metropolitan hospitals for individuals aged 15-64 (IRR 1.05, \( P = 0.039 \)), 65-74 (IRR = 1.06, \( P = 0.048 \)) and 75 years or over (IRR 1.05, \( P = 0.047 \)).

As displayed in Table 6.1, analysis yielded varied results for the 9 cause-specific illnesses investigated. Mental disorders were increased for all ages and for those in the 65-74 year age group. No increase was observed in the number of daily ED presentations for cardiovascular diseases in general, and a decrease was observed in cardiovascular visits in the 15-64 year age bracket. Similarly there was no change in respiratory presentations for all ages and a decrease for those 75 years or over.

Heatwaves appeared to have no effect on ED visits for ischaemic heart disease, stroke or epilepsy. Presentations for renal diseases and food-borne illnesses were statistically unchanged, although there were non-significant increases in renal presentations in the 65-74 year age group and food-borne diseases in children aged 5-14 years.

As shown in Table 6.1, highest increases were observed in presentations for heat-related disorders for all ages (IRR 2.78, \( P < 0.005 \)), and in each age group except children up to 4 years of age. Greatest risk was in the 15-64 year age group (IRR 3.27, 95% CI 2.73 - 3.94, \( P < 0.005 \)) followed by the elderly 75 years or over (IRR 2.77, 95% CI 2.28 - 3.35, \( P < 0.005 \)).

Regression analysis of all ED visits by postcode showed a statistically significant variation in the number of daily ED visits during heatwaves in 11 postcode regions, 10 of which showed an increase (Figure 6.1 and Table 6.2). Among the highest was the Adelaide central business district (CBD), postcode 5000, with ED visits elevated by 21%. Others included several postcodes to the north west of the city with increases ranging from 11% (5014) to 25% (5020). A large postcode region on the southern extremity of the metropolitan area (5172), showed an almost 20% decrease in ED visits during heatwaves.
Table 6.1: Incidence rate ratios (IRR) and $P$-values for regression analyses of ED presentations during heatwaves compared non-heatwave periods.

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>All ages</th>
<th>0-4</th>
<th>5-14</th>
<th>15-64</th>
<th>65-74</th>
<th>≥ 75 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IRR</td>
<td>$P$</td>
<td>IRR</td>
<td>$P$</td>
<td>IRR</td>
<td>$P$</td>
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<tr>
<td>All ED visits</td>
<td>1.04</td>
<td>0.098</td>
<td>0.99</td>
<td>0.801</td>
<td>1.01</td>
<td>0.837</td>
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<td>IHD</td>
<td>0.94</td>
<td>0.143</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Stroke</td>
<td>0.98</td>
<td>0.776</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>0.97</td>
<td>0.171</td>
<td>1.07</td>
<td>0.879</td>
<td>0.94</td>
<td>0.829</td>
</tr>
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<td>Respiratory</td>
<td>0.98</td>
<td>0.648</td>
<td>1.01</td>
<td>0.810</td>
<td>1.16</td>
<td>0.255</td>
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<tr>
<td>Mental</td>
<td>1.06</td>
<td>0.033</td>
<td>2.13</td>
<td>0.093</td>
<td>0.82</td>
<td>0.354</td>
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<td>Epilepsy</td>
<td>1.03</td>
<td>0.696</td>
<td>1.06</td>
<td>0.799</td>
<td>0.85</td>
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<td>Renal</td>
<td>1.06</td>
<td>0.255</td>
<td>1.21</td>
<td>0.107</td>
<td>1.09</td>
<td>0.737</td>
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<td>Food-borne</td>
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<td>0.493</td>
<td>0.54</td>
<td>0.555</td>
<td>2.60</td>
<td>0.129</td>
</tr>
<tr>
<td>Effects of heat</td>
<td>2.78</td>
<td>0.000</td>
<td>1.41</td>
<td>0.214</td>
<td>2.07</td>
<td>0.021</td>
</tr>
</tbody>
</table>
Table 6.2: Incidence rate ratios (IRR) and 95% confidence intervals (CI) for postcodes with statistically significant ($P < 0.05$) increases in ED visits during heatwaves

<table>
<thead>
<tr>
<th>Postcode</th>
<th>Suburb</th>
<th>IRR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>5000</td>
<td>Adelaide CBD</td>
<td>1.21</td>
<td>1.13-1.29</td>
</tr>
<tr>
<td>5013</td>
<td>Gillman, Ottoway, Pennington, Rosewater, Wingfield</td>
<td>1.15</td>
<td>1.06-1.24</td>
</tr>
<tr>
<td>5014</td>
<td>Alberton, Albert Park, Cheltenham, Hendon, Queenstown, Royal Park</td>
<td>1.11</td>
<td>1.02-1.20</td>
</tr>
<tr>
<td>5020</td>
<td>West Lakes Shore</td>
<td>1.25</td>
<td>1.00-1.57</td>
</tr>
<tr>
<td>5033</td>
<td>Cowandilla, Hilton, Marleston, Richmond, West Richmond</td>
<td>1.13</td>
<td>1.12-1.26</td>
</tr>
<tr>
<td>5039</td>
<td>Clarence Gardens, Edwardstown, Melrose Park</td>
<td>1.14</td>
<td>1.03-1.26</td>
</tr>
<tr>
<td>5042</td>
<td>Bedford Park, Clovelly Park, Pasadena, St. Marys</td>
<td>1.14</td>
<td>1.04-1.25</td>
</tr>
<tr>
<td>5044</td>
<td>Glengowrie, Somerton Park</td>
<td>1.12</td>
<td>1.00-1.25</td>
</tr>
<tr>
<td>5046</td>
<td>Oaklands Park, Warradale, Warradale North</td>
<td>1.16</td>
<td>1.03-1.30</td>
</tr>
<tr>
<td>5113</td>
<td>Davoren Park, Elizabeth Downs, Elizabeth North, Elizabeth Park, Elizabeth West</td>
<td>1.06</td>
<td>1.00-1.12</td>
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</tbody>
</table>

Postcodes with the highest number of heat-related ED visits (data not shown) were 5162 (Morphett Vale and Woodcroft) to the south of the city, and the northern areas of 5108 (comprising Paralowie, Salisbury, Salisbury Downs and Salisbury North) and 5113 (comprising Davoren Park, Elizabeth Downs, Elizabeth North, Elizabeth Park and Elizabeth West). Postcode 5113 also showed a 6% increase in total ED visits during heatwaves (Table 6.2).
6.4 Discussion

Studies investigating variations in daily ED visits, hospital admissions and ambulance callouts during heatwaves can assist in monitoring the impact of extreme heat on health, and inform current response policy and planning for future infrastructure establishment and resource allocation. The identification of spatial and demographic trends can further enhance our understanding of the complexities of heat-related morbidity, susceptible subgroups and at risk areas within urban regions.

Unlike many other studies reporting on single heat events, this Adelaide based study spanned a five year period incorporated 12 heatwaves, one of which was extreme in its intensity and duration. Findings revealed all-cause ED visits to metropolitan hospitals were increased during periods of extreme heat for persons aged 15 years and over.

Figure 6.1: Change in emergency department visits across metropolitan Adelaide during heatwave periods compared to non-heatwave periods.
Although the risk was similar in all adult age groups, it was modestly higher in the 65-74 year age group. In particular, heat-related illnesses were markedly increased, both in the all age category, and in four out of the five age groups, the highest being in the 15-64 year age group, where the risk was more than 3 times that observed during non-heatwave periods. This age group was also found to be at risk using ambulance callouts as an indicator of heat-associated morbidity (Chapter 5). With local communities tending to be protective of their elderly and young during the heat, it is likely persons of working age who need to travel and work as normal during heatwaves may have greater exposure to the extreme conditions.

Heat-related illnesses during heatwaves are commonly reported in the literature. During the 1995 Chicago heatwave, excess hospitalisations totaling 11% were reported, most of which were due to heat exhaustion, heat stroke and dehydration. Similarly deaths due to heatstroke, dehydration and hyperthermia contributed markedly to the overall mortalities attributed to the 2003 heatwave in France. Findings in the present study are consistent with those of a similar study investigating the impact of the 2006 California heatwave where the authors reported that heat-related ED visits increased more than 6-fold. Although this demonstrates a greater increase than that observed in the present study, disparities in effect estimates may relate to differences in study design, definition of heat-related illness, or most likely, population acclimatisation to heatwaves.

Increased ED visits for mental and behavioural disorders were observed in this study, concurring with previous studies in Adelaide which have reported increased morbidity and mortality for mental disorders during heatwaves. The heat-susceptibility of persons with mental illnesses has been well documented and may be attributable to risk factors such as maladaptive behaviours, poor underlying health status, socioeconomic disadvantage, or disruption to physiologic thermoregulation caused by medications commonly used in the treatment of psychiatric illnesses. Similarly medications used in the treatment of epilepsy can interfere with thermoregulation, although in the current study, no increase was observed in epilepsy presentations during heatwaves.

Food-borne illnesses such as salmonellosis have often been associated with seasonality with cases most commonly reported during the warm season. That the present
study failed to find a significant increase in food-related ED visits during heatwaves is not unexpected as studies elsewhere have noted delays between periods of warm weather and notifications of food-borne illnesses. Positive associations have been found between salmonella notifications and temperatures during the previous week,\textsuperscript{302} two weeks\textsuperscript{183} or the previous month.\textsuperscript{301} Longer lag periods are suggestive of contamination occurring earlier in the food production process,\textsuperscript{301} possibly during processing, transport or storage rather than during the food handling or preparation stage. Hence with high temperatures reported to be associated with delayed rather than immediate onset of illness, further research is warranted to investigate incidences of food-borne diseases in the weeks following heatwave periods. It should be noted also that ED visits would under represent the true incidence of food borne illness in the community as only the most severe cases would necessitate hospital care.

It is well known that thermal stress with associated circulatory adjustments, dehydration and salt imbalance can place stress on the kidneys that may progress to renal failure.\textsuperscript{17,19,223} Renal hospital admissions during heatwaves have been seen to be increased previously in Adelaide,\textsuperscript{31,303} (Chapter 3) however no significant increase in renal ED visits was noted in the present study. It is possible that heat disorders seen in the ED which progress to renal dysfunction during the period of hospitalisation, may have different ICD codes for presenting diagnoses and primary discharge diagnoses, thus accounting for the discrepancy between findings. It is not unusual that the diagnosis at discharge varies from the initial diagnosis when cause of illness may be presumptive or unknown. A small study in the U.S. reported that of the 77\% of patients with a causative diagnosis in the ED, 16\% had discrepant ED and discharge diagnoses.\textsuperscript{304} Other studies have shown higher discrepancy rates of 26.8\%\textsuperscript{305} and up to 75.6\%,\textsuperscript{306} the latter being from a large study of 5,375 patients over a 12 month period at a large urban medical centre in the U.S. This highlights the need for caution in causal interpretation and the importance of placing epidemiological findings in the context of data sources.

Heat-related illnesses often manifest as cardiorespiratory disorders in vulnerable persons, and previous studies elsewhere have shown increases in heat-exacerbated cardiovascular and respiratory mortality in the elderly.\textsuperscript{2,35,40} The current study failed to detect any such increase and in some age groups significant decreases were observed
(Table 6.1), concurring with a previous study where mortalities attributed to respiratory, cardiovascular and cerebrovascular diseases (stroke) declined in persons aged 75 years or over during heatwaves. Current findings of an overall increase in ED visits in this older age group as well as younger age groups, indicates further research is required to investigate other conditions that may be responsible for the onset of illness during periods of extreme heat.

Geospatial analysis identified some consistencies between areas where ED visits were significantly increased, and those recording high numbers of heat disorders. In particular, the northern postcode area of 5113 was prominent in both. A high proportion of low-income families reside in this area where temperatures are often higher than in the city central. This region and some in the disadvantaged north western areas were shown to be at higher risk of ambulance attendance during heatwaves (Chapter 5). This is analogous to findings in a U.S. study where mortality rates during heatwaves were higher in the warmer and more disadvantaged areas of St. Louis. Postcodes used in the ambulance study however were indicative of callout location and may not have coincided with residential location. Nevertheless there are some geospatial similarities with the present study.

For reasons unknown, a large postcode area on the southern extremity of the metropolitan region showed a marked decrease in ED presentations during heatwaves. Whilst this may be a chance finding, it may also be due to biometeorological reasons as temperatures are often lower to the south of the city, thus reducing individuals’ exposure to heat.

Interestingly, major metropolitan hospitals are located within two of the postcode areas (5000 and 5042) where ED visits were increased during heatwaves. This raises the issue of personal behaviours of individuals seeking medical attention during extreme heat. Are those who reside in close proximity to a hospital more likely to attend an ED rather than travel further in the heat to their local practitioner or medical clinic? Issues regarding demographics, social inequality and access to health care may also influence patient profiles. For instance, with no fee for service charged in EDs, are those in disadvantaged circumstances more likely to visit a hospital than a general practitioner
when requiring medical assistance? These uncertainties highlight the need to consider contextual factors when interpreting findings in spatial epidemiology.

**Limitations**

This study has several limitations. First, air pollution may affect cardiorespiratory morbidity and was not taken into account in this study. However as cardiovascular and respiratory outcomes were not found to be increased during heatwaves in this study, results may not have altered markedly if air pollution concentrations had been adjusted for. Second, as with similar studies elsewhere, the misclassification of ICD codes is a potential source of error in the data although it is assumed that random errors would not bias overall findings.

Third, the ‘effects of heat’ category used in this study included classifications of volume depletion (dehydration) and hyperthermia. Dehydration is a contributing factor in heat-related illness and is the most common cause of hyperthermia. However other conditions including gastroenteritis can cause dehydration. Similarly, a rise in body temperature can be caused by fever as well as hyperthermia, although unlike hyperthermia which is due to thermoregulatory failure, homeostatic responses remain intact with fever. Hence the classification of heat-related illness used in this study may be subject to selection bias with the inclusion of false positives (Type I error). Notwithstanding, omission of any of the chosen symptoms (dehydration, hyperthermia or effects of heat and light) may have led to an underestimation of heat-related illnesses (Type II error).

A more accurate way to identify cases of thermal illness may be via an audit of clinical records. Nevertheless classification of symptoms and case histories is not straightforward and may be subjective. Heat stroke for example, the most severe of heat-illnesses, is defined as occurring when body temperature exceeds 40°C and there is central nervous system involvement. If death due to heat stroke occurs when a medical practitioner is not in attendance to observe changes in mental status and record body temperature, death may be attributed to other causes. Hence heat-related deaths are often underreported, as perhaps are heat-related illnesses.
Finally, spatial analysis in this study was dependent on the accuracy of recorded postcodes, and it was noted that some were invalid. It is possible others may have been listed incorrectly, due to recall limitations of ill persons presenting at the ED. Disease mapping is a useful tool for estimating spatial vulnerability although caution should be used in interpretation as there can be areal discrepancies between postal areas and postcodes, particularly in larger outer suburban areas. Furthermore, interpretation can be complex as the effect of heat on morbidity in a given area may depend on a range of factors including climate variability, population demographics, underlying health status, proportion of elderly residents, socio-economic factors, personal behaviours, adaptive capabilities, standard of housing and access to health care. Importantly, in this study stratification by postcode and classification of heat-related illness resulted in small sample sizes that may have yielded spurious results. Visual presentation of regression results via mapping may therefore be misleading.

**Conclusion**

In conclusion, this study has shown all-cause ED visits during heatwave periods in Adelaide to be elevated in the 15-64, 64-74 and 75 years and over age groups. Specifically, heat-related illnesses were markedly increased for all ages and in each age group except infants to 4 years of age, indicating the health risk for younger adults during Adelaide heatwaves to be comparable to that of the older age groups. Presentations of persons with mental disorders were also increased overall and in those aged 65-74 years.

In addition to the studies investigating hospital admissions (Chapters 3 and 4) and ambulance callouts (Chapter 5), this study has provided another aspect of heatwave-associated morbidity and asserts the burden that high temperatures can place on the health system in South Australia. As targeting purely the elderly with health promotion messages excludes younger persons and older persons who consider themselves not be elderly, broader distribution of messages which specify at-risk groups, may be a more appropriate public health approach to adaptation. With climatologists predicting more intense and frequent heatwaves, policymakers need to be aware of this emerging threat and the need to increase heat-health awareness and resilience across the community to reduce the impact of climate change.
SECTION III

THE IMPACT OF AIR POLLUTION ON HEALTH IN ADELAIDE
Introduction

Section III represents a substantial component of this thesis - an investigation of the effects of air pollution on the health of Adelaide residents. Many studies have associated negative health outcomes with exposure to ambient air pollution, and the World Health Organisation estimates that air pollution is responsible for up to 800,000 premature deaths annually.65 Whilst studies have been undertaken elsewhere in Australia, there have been none previously conducted in South Australia.

The first of the two chapters in this section (Chapter 7) assesses the effect of particulate matter on cardiovascular and respiratory hospital admissions in Adelaide; the second (Chapter 8) is a complex study examining the potential interactive effects of temperature and several air pollutants, on hospital emergency department visits and mortalities. In contrast to the statistical methodologies used elsewhere within this thesis, the current studies incorporate the use of case-crossover analysis, a commonly utilised design in the international air pollution literature, having been adapted from use in clinical medicine to environmental studies at a population level. Dr. Adrian Barnett and Mr. Thomas Sullivan are gratefully acknowledged for their advice and assistance with the methodology.
Chapter 7

Particulate air pollution and cardiorespiratory hospital admissions

7.1 Introduction

Air quality in Adelaide compares favourably with that in other cities in Australia and around the world. Nevertheless, even low levels of air pollution can cause health problems and for some pollutants, such as particulate matter (PM), thresholds below which adverse effects occur, have not been identified. With Adelaide’s unique dry temperate climate, it is of public health interest to determine if outdoor air quality has an effect on population health as no such epidemiological studies have been undertaken to date.

Unlike South Australia’s regional centres where industry impacts considerably on air quality, the main emissions in Adelaide are from motor vehicles and domestic wood-burning heaters. The city’s location between the coast and the Adelaide Hills can restrict air movement and influence air quality, and sea salt in the prevailing sea breezes can inhibit the dispersion of air pollutants. Temperature inversions occur particularly in the winter, when the air layer closest to the Earth is abnormally cooler than the air above, resulting in air pollutants being trapped close to the ground. Other seasonal changes in air pollution levels occur due to prevailing meteorological conditions including temperature and humidity, and factors such as heating sources in the winter; and in the warmer months wind erosion, high fire risk, and photochemical smog formation.
Particulate matter

The major pollutant in the Adelaide airshed is airborne PM,\textsuperscript{211} a heterogeneous mix of solid and liquid particles that can vary in size, composition and source.\textsuperscript{74} Particles with aerodynamic diameters 2.5-10 µm, less than 2.5 µm or less than 0.1 µm are generally classified as coarse, fine or ultrafine particles respectively.\textsuperscript{82,95} The larger particles are generally derived from soil or crustal materials, whereas the smaller particles are derived as a result of combustion processes such as wood burning, fossil fuel use in motor vehicles, and power generation.\textsuperscript{101,112} In the Adelaide airshed solid fuel burning (domestic) contributes to 25.6\% of the total particles with an aerodynamic diameter less than or equal to 10 µm (PM\textsubscript{10}), motor vehicles contribute 20.2\%.\textsuperscript{314} Fine particles (PM\textsubscript{2.5}) are a subset of PM\textsubscript{10},\textsuperscript{101} and the PM\textsubscript{2.5}/PM\textsubscript{10} ratio is typically in the order of 0.5-0.8 in urban areas,\textsuperscript{82} with a tendency for higher ratios in the winter.\textsuperscript{135} The association between exposure to PM and respiratory and cardiovascular outcomes has been well documented\textsuperscript{69,74,85} and can be linked to the inflammatory responses and pulmonary oxidative stress generated when particles are inhaled.\textsuperscript{74} Fine particles derived from combustion sources have been shown to be particularly harmful to health.\textsuperscript{101} (An extensive review of air pollutants, their sources and health impacts, appears in Chapter 1.)

In this state’s often hot, dry conditions, wind erosion occurs due to low soil moisture levels combined with windy conditions. Dust storms can generate elevated levels of PM\textsuperscript{313} and with a high ratio of fine to course particles, dust can travel over large distances from rural areas and impact on urban air quality. Adelaide reports approximately 8.5 “dust in the air” days per year, whereas in other areas such as the far north of the State, there can be approximately 60 dusty days per year.\textsuperscript{315} Dust storms have been reported to be associated with the onset of respiratory conditions and changes in asthma severity in Australia.\textsuperscript{316}

Bushfires can be also be a major source of fine particles in airsheds surrounding Australian capital cities.\textsuperscript{134} Tinder-dry vegetated areas are high risk bushfire zones on days of extreme heat, particularly when winds are strong and unpredictable. Smoke from bushfires occurring in South Australia in areas such as Eyre Peninsula, Kangaroo Island or the Adelaide Hills, or several hundred kilometres away interstate,
can impact on Adelaide’s air quality. Studies have found biomass smoke to be positively associated with respiratory outcomes\textsuperscript{140,142,148} including asthma\textsuperscript{105,317}.

The Australian air quality standard for particles as PM\textsubscript{10} under the Ambient Air Quality National Environment Protection Measure (Air NEPM) is 50 µg/m\textsuperscript{3} maximum concentration measured over a 24-hour period, and although there is no current standard for PM\textsubscript{2.5}, the advisory NEPM is a maximum concentration of 25 µg/m\textsuperscript{3} measured over a 24-hour period\textsuperscript{135}.

**Nitrogen dioxide and ozone**

Among the other pollutants of importance in the Adelaide airshed are nitrogen dioxide (NO\textsubscript{2}) and ozone (O\textsubscript{3}). The major source of NO\textsubscript{2} in Adelaide is motor vehicle emissions and concentrations, although variable in the past, have decreased slightly since 2003\textsuperscript{211}. The effect on health of NO\textsubscript{2} exposure includes eye nose and throat irritations, respiratory outcomes\textsuperscript{124,211} and cardiovascular disease, particularly among the elderly\textsuperscript{69,91,104,125}.

A secondary pollutant, O\textsubscript{3} is formed at ground level by the action of solar radiation on anthropogenically produced nitrogen oxides and reactive hydrocarbons\textsuperscript{74}. Respiratory\textsuperscript{124} and cardiovascular events\textsuperscript{126,318} have been linked with exposure to O\textsubscript{3}. Levels in Adelaide declined from the 1980s to the 1990s but trends now show a gradual increase over time\textsuperscript{211}.

Clearly, air pollution is a complex mixture of gaseous and solid components with effects on human health that are difficult to attribute to a single component. With this in mind, the aim of the current study was to investigate the relationship between cardiorespiratory morbidity in Adelaide and exposure to ambient PM, with adjustment for other pollutants (NO\textsubscript{2} and O\textsubscript{3}). The findings will help fill the existing gap in knowledge concerning the health effects of air pollution in urban Australia and may inform policy decisions regarding air quality in a changing climate.
7.2 Methods

Data relating to hospital admissions for the period 1 September 2001 to 31 October 2007 were sourced from the SA Department of Health. Diseases were classified according to the International Classification of Diseases Codes (ICD-10). As well as all hospital admissions, those specific to diseases of the circulatory system (I00-I99), ischaemic heart disease (IHD) (I20 - I25), stroke (I60-I69), cardiac failure (I50), myocardial infarction (MI) (I21-I22), all respiratory diseases (J00-J99), asthma (J45-J46), chronic obstructive pulmonary disease (COPD) (J40-J44), and influenza (J10-J11) were sourced.

Air quality data for the study period were obtained from the Environment Protection Authority (EPA (SA)) monitoring site located at Netley, a suburb in the Western Region of the Adelaide airshed (Figure 7.1). This site, as well as being centrally located in the airshed, is the only one where PM$_{2.5}$ is monitored. One-day average PM$_{10}$ and PM$_{2.5}$ concentrations, measured using a tapered element oscillating microbalance (TEOM)$^{100}$ were used in the study. One-hour average concentrations of O$_3$ (measured using an ultra violet UV photometric analyser) and NO$_2$ (measured using gas-phase chemiluminescence)$^{100}$ were sourced, and daily maximum 1-hour values calculated. Missing air quality data were rare: 2.82%, 4.58%, 1.42% and 5.82% for PM$_{2.5}$, PM$_{10}$, O$_3$ and NO$_2$ respectively. Missing values were not imputed as the random omissions meant the introduction of selection bias was unlikely.$^{86}$

As in previous studies within this thesis, meteorological data for the study period were sourced from the SA Bureau of Meteorology monitoring site at Kent Town. Data included daily maximum and minimum temperature, from which mean temperature was calculated, and average daily relative humidity. Also included was a daily ‘present weather’ index indicating weather conditions at a manned weather station at the Adelaide Airport located within one kilometre from the EPA monitoring site. The numerically coded World Meteorological Organization index indicates weather at the time of, or in the hour preceding, the observation.$^{319}$ Using this index, days when ‘dust in suspension’, ‘dust raised’ or ‘severe duststorm’ (codes 6, 7 or 34), or smoke (code 4) were present in the air, were identified. It should be noted that the single code of “smoke” does not distinguish the source of smoke, which may be of bushfire,
wood fire, or industrial origin. Corresponding media and EPA reports were useful when identifying days when bushfire smoke prevailed.

NOTE:
This figure is included on page 135 of the print copy of the thesis held in the University of Adelaide Library.

Figure 7.1: Map of the Adelaide Air Quality Index sites and regions, showing the Netley monitoring site. Source: EPA (http://www.epa.sa.gov.au/environmental_info/air_quality/current_air_quality/air_quality_index_sites_and_regions) Accessed 20 December 2009.

Statistical analysis

A time-stratified case-crossover design was used for statistical analysis. Case-crossover design compares exposures at a time prior to a health event (case period) to exposures at other times (control periods). Individuals act as their own controls, eliminating effects of confounding by characteristics that remain unchanged such as tobacco smoking, diet, weight and gender that are well controlled for by design. The time-stratified option has been shown to provide less biased estimates compared to other options for the selection of control periods (eg unidirectional and bidirectional). The methodology used in this study closely followed that of Barnett et al in similar air pollution studies. Data were arranged into consecutive 28-day strata throughout the study period. As described in Chapter 2, for each case (index) day, all other days in the strata (except the index day and the two days either side) were control days. The exclusion of days adjacent to the index day reduced possible correlation between case and control exposure. To allow for a short lag period between exposure and hospitalisation, the pollutant exposure used was the average of
the present and previous day. Included as covariates in the model were: day of the week, public holidays, day after public holidays, daily mean temperature, current minus previous day’s temperature, relative humidity, daily maximum O₃, and daily maximum NO₂. Models investigating PM₁₀ and PM₂.₅ effects were run separately to avoid multicollinearity. To control for influenza outbreaks, admissions for influenza were excluded from total respiratory admissions.¹¹⁸

Analyses were performed on total data and data stratified by season (warm season: October to March; cool season: April to September). A dummy variable was created and coded as 1 if smoke was recorded as present on the current or previous day, or 0 otherwise. Similarly, dust was coded as 1 if dust was recorded on the present or previous day. To test for differences in PM on days of smoke and dust to other days, two-sample $t$-tests assuming unequal variances were performed.

Data management was performed in Stata v10 (StataCorp. College Station, TX) and converted to SAS (SAS Institute Inc. Cary, NC) for case-crossover analysis. Effect estimates determined using conditional logistic regression analysis¹⁰,¹⁸⁶ in SAS were frequently cross checked in Stata using Cox proportional hazard regression, a form of the conditional likelihood model used in stratified case-control investigations.³²⁰ Effect estimates and 95% confidence intervals (CI) were calculated per 10 µg/m³ increase in PM.

### 7.3 Results

The daily mean temperature in Adelaide over the study period 1 September 2001 to 31 October 2007 ranged from 7.4°C to 37.0°C with a mean of 14.2°C, 20.6°C and 17.4°C for the cool season, warm season and the whole study period respectively. Summary statistics for each pollutant and hospital admissions are presented in Table 7.1.

The Air Quality Index (AQI) is a simplified indicator of air quality which takes the measured value of pollutants as a percentage of the Air NEPM standard.¹³⁷ Figure 7.2, reproduced courtesy of the EPA, shows the air quality for the majority of the study period as measured using the AQI. Air quality over this time was good or very good.
91% of the time, however there was a slight decreasing trend from 2002 to 2006 in the percentage of time air quality was regarded as good, and an increase from 2004 in poor or very poor air quality.

NOTE: This figure is included on page 137 of the print copy of the thesis held in the University of Adelaide Library.

Figure 7.2: Air quality indices for Adelaide 2002 to 2006. Source: EPA

Over the study period there were 3 exceedances of PM$_{2.5}$ above the Air NEPM advisory reporting standard of 25 µg/m$^3$ and 36 exceedances of PM$_{10}$ over the Air NEPM standard of 50 µg/m$^3$ (1-day average) in Adelaide. The maximum 1-day average PM$_{2.5}$ measurement of 61.2 µg/m$^3$ occurred on a day when smoke from bushfires on Kangaroo Island (112 kilometres to the south) and in Victoria (460 kilometres to the east) infiltrated the Adelaide airshed. Figure 7.3 shows the rapid elevation in both PM$_{2.5}$ and PM$_{10}$ at that time. On the same day (25 January 2006), the highest daily maximum-1-hour O$_3$ value of 0.105 ppm was recorded, also exceeding the Air NEPM standard.

The maximum PM$_{10}$ concentration of 119.4 µg/m$^3$ was recorded in July 2003 on a day of extreme dust (Figure 7.4). Concentrations of both PM$_{2.5}$ and PM$_{10}$ were higher on dusty days than smoky days (Table 7.1) and were found to be significantly different on these days to other days, with $t$-tests yielding $P$-values of $P \leq 0.001$. Maximum daily NO$_2$ values were also found to be significantly higher on days of smoke than other days.
There was a high degree of correlation between PM$_{10}$ and PM$_{2.5}$ values (Figure 7.2), with a Spearman rank correlation coefficient ($\rho$) of 0.77 over the study period. There were moderate correlations observed between PM$_{2.5}$ and NO$_2$ ($\rho = 0.47$) and between PM$_{10}$ and mean temperature ($\rho = 0.41$). The mean 24-hour average PM$_{2.5}$ and PM$_{10}$ concentrations were highest during the cool and warm seasons, respectively. The PM$_{2.5}$/PM$_{10}$ ratio was higher in the cool season than the warm season and higher on days of smoke than dust (Table 7.1).

**Figure 7.3:** PM$_{2.5}$ concentrations peaked on 25 January 2006 when bushfire smoke blanketed Adelaide.

**Figure 7.4:** PM$_{10}$ concentrations peaked during a dust storm over Adelaide.
Table 7.1: Summary statistics of air pollutants and hospital admissions during the cool and warm seasons, on days of smoke and dust, and across the whole study period.

<table>
<thead>
<tr>
<th></th>
<th>Season</th>
<th>Dust</th>
<th>Smoke</th>
<th>Study Period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cool</td>
<td>Warm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of days</td>
<td>1128</td>
<td>1124</td>
<td>17</td>
<td>82</td>
</tr>
<tr>
<td>PM$_{10}$ (µg/m$^3$), mean</td>
<td>18.5</td>
<td>20.6</td>
<td>61.4</td>
<td>28.2</td>
</tr>
<tr>
<td>(range)</td>
<td>(5.2-119.4)</td>
<td>(5.2-101.4)</td>
<td>(22.9-119.4)</td>
<td>(9.8-101.4)</td>
</tr>
<tr>
<td>PM$_{2.5}$ (µg/m$^3$), mean</td>
<td>8.7</td>
<td>7.9</td>
<td>15.0</td>
<td>12.6</td>
</tr>
<tr>
<td>(range)</td>
<td>(2.9-29.2)</td>
<td>(1.6-61.2)</td>
<td>(9.0-29.2)</td>
<td>(5.6-61.2)</td>
</tr>
<tr>
<td>PM$<em>{2.5}$/PM$</em>{10}$ ratio (%)</td>
<td>46.9</td>
<td>38.5</td>
<td>24.5</td>
<td>44.8</td>
</tr>
<tr>
<td>Maximum 1-hr NO$_2$ (ppm),</td>
<td>0.023</td>
<td>0.017</td>
<td>0.020</td>
<td>0.025</td>
</tr>
<tr>
<td>mean (range)</td>
<td>(0.000-0.054)</td>
<td>(0.000-0.103)</td>
<td>(0.010-0.031)</td>
<td>(0.003-0.054)</td>
</tr>
<tr>
<td>Maximum 1-hr O$_3$ (ppm),</td>
<td>0.027</td>
<td>0.030</td>
<td>0.032</td>
<td>0.032</td>
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<tr>
<td>mean (range)</td>
<td>(0.002-0.049)</td>
<td>(0.013-0.105)</td>
<td>(0.02-0.069)</td>
<td>(0.014-0.105)</td>
</tr>
<tr>
<td>Mean daily respiratory</td>
<td>66.0</td>
<td>51.6</td>
<td>57.0</td>
<td>62.0</td>
</tr>
<tr>
<td>admissions a</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mean daily CV admissions a</td>
<td>75.3</td>
<td>70.1</td>
<td>68.3</td>
<td>79.5</td>
</tr>
<tr>
<td>Mean daily total admissions a</td>
<td>1210.7</td>
<td>1169.3</td>
<td>1113.5</td>
<td>1357.9</td>
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### Table 7.2: Percent increase in risk, with 95% confidence intervals (CI) for all-cause hospital admissions per 10 µg/m³ increase in PM.

<table>
<thead>
<tr>
<th>Age group</th>
<th>PM</th>
<th>Cool season</th>
<th></th>
<th>Warm season</th>
<th></th>
<th>All season</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>% Increase</td>
<td>95% CI</td>
<td>% Increase</td>
<td>95% CI</td>
<td>% Increase</td>
<td>95% CI</td>
</tr>
<tr>
<td>All age</td>
<td>2.5</td>
<td>1.17</td>
<td>0.25, 2.10</td>
<td>1.89</td>
<td>0.97, 2.81</td>
<td>0.84</td>
<td>0.22, 1.47</td>
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<tr>
<td></td>
<td>10</td>
<td>0.68</td>
<td>0.37, 0.99</td>
<td>0.10</td>
<td>-0.19, 0.40</td>
<td>0.39</td>
<td>0.18, 0.61</td>
</tr>
<tr>
<td>0-14</td>
<td>2.5</td>
<td>N/S</td>
<td>N/S</td>
<td>3.37</td>
<td>0.21, 6.62</td>
<td>N/S</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>1.08</td>
<td>0.04, 2.12</td>
<td>N/S</td>
<td></td>
<td>0.96</td>
<td>0.23, 1.69</td>
</tr>
<tr>
<td>15-64</td>
<td>2.5</td>
<td>2.39</td>
<td>1.12, 3.67</td>
<td>1.64</td>
<td>0.39, 2.91</td>
<td>0.89</td>
<td>0.05, 1.75</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>0.91</td>
<td>0.49, 1.33</td>
<td>N/S</td>
<td></td>
<td>N/S</td>
<td></td>
</tr>
<tr>
<td>65-74</td>
<td>2.5</td>
<td>N/S</td>
<td>N/S</td>
<td>N/S</td>
<td></td>
<td>N/S</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>1.21</td>
<td>0.43, 1.99</td>
<td>N/S</td>
<td></td>
<td>0.95</td>
<td>0.40, 1.49</td>
</tr>
<tr>
<td>75 plus</td>
<td>2.5</td>
<td>N/S</td>
<td>N/S</td>
<td>1.97</td>
<td>0.02, 3.95</td>
<td>N/S</td>
<td></td>
</tr>
</tbody>
</table>

* Non-significant (*P < 0.05*) results, except those for all-ages, are represented by N/S.
Table 7.3: Percent increase in risk, with 95% confidence intervals (CI) for respiratory admissions per 10 µg/m³ increase in PM.

<table>
<thead>
<tr>
<th>Outcome and age group</th>
<th>PM</th>
<th>Cool season</th>
<th>Warm season</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>% Increase</td>
<td>95% CI</td>
</tr>
<tr>
<td>Respiratory (all ages)</td>
<td>2.5</td>
<td>-0.86</td>
<td>-4.66, 3.08</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>1.10</td>
<td>-0.22, 2.43</td>
</tr>
<tr>
<td>15-64</td>
<td>10</td>
<td>3.00</td>
<td>0.73, 5.33</td>
</tr>
<tr>
<td>Asthma 0-14</td>
<td>10</td>
<td>N/S</td>
<td>-6.56</td>
</tr>
<tr>
<td>65-74</td>
<td>2.5</td>
<td>-49.84</td>
<td>-73.27, -5.87</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>N/S</td>
<td></td>
</tr>
<tr>
<td>75 plus</td>
<td>2.5</td>
<td>N/S</td>
<td>-70.14</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>N/S</td>
<td>-29.88</td>
</tr>
<tr>
<td>COPD (All ages)</td>
<td>2.5</td>
<td>-10.47</td>
<td>-18.64, -1.48</td>
</tr>
<tr>
<td>15-64</td>
<td>10</td>
<td>N/S</td>
<td></td>
</tr>
</tbody>
</table>

* Non-significant ($P < 0.05$) results, except those for all-ages, are represented by N/S.
Table 7.4: Percent increase in risk with 95% confidence intervals (CI) for cardiovascular admissions/10 µg/m³ increase in PM. (N/S represents non-significant ($P < 0.05$) results)

<table>
<thead>
<tr>
<th>Outcome and age group</th>
<th>PM</th>
<th>Cool season</th>
<th>Warm season</th>
<th>All season</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>% Increase</td>
<td>95% CI</td>
<td>% Increase</td>
</tr>
<tr>
<td>Cardiovascular (all ages)</td>
<td>2.5</td>
<td>4.48</td>
<td>0.74, 8.36</td>
<td>1.49</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>1.48</td>
<td>0.24, 2.74</td>
<td>-0.95</td>
</tr>
<tr>
<td>15-64</td>
<td>2.5</td>
<td>N/S*</td>
<td>N/S</td>
<td>4.32</td>
</tr>
<tr>
<td>65-74</td>
<td>2.5</td>
<td>13.18</td>
<td>4.62, 24.43</td>
<td>N/S</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4.20</td>
<td>1.45, 7.01</td>
<td>N/S</td>
</tr>
<tr>
<td>75 plus</td>
<td>10</td>
<td>N/S</td>
<td>-2.10</td>
<td>-3.97, -0.18</td>
</tr>
<tr>
<td>IHD (all ages)</td>
<td>2.5</td>
<td>9.83</td>
<td>3.02, 17.10</td>
<td>N/S</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>2.63</td>
<td>0.41, 4.91</td>
<td>N/S</td>
</tr>
<tr>
<td>15-64</td>
<td>2.5</td>
<td>15.84</td>
<td>3.98, 29.05</td>
<td>N/S</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4.08</td>
<td>0.36, 7.95</td>
<td>N/S</td>
</tr>
<tr>
<td>65-74</td>
<td>2.5</td>
<td>29.18</td>
<td>13.99, 46.38</td>
<td>N/S</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>7.52</td>
<td>3.06, 12.17</td>
<td>N/S</td>
</tr>
<tr>
<td>75 plus</td>
<td>10</td>
<td>N/S</td>
<td>-4.29</td>
<td>-7.66, 0.79</td>
</tr>
<tr>
<td>MI (all ages)</td>
<td>2.5</td>
<td>13.00</td>
<td>0.32, 27.27</td>
<td>N/S</td>
</tr>
<tr>
<td>65-74</td>
<td>2.5</td>
<td>N/S</td>
<td>59.71</td>
<td>25.52, 103.21</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>N/S</td>
<td>12.41</td>
<td>3.55, 22.03</td>
</tr>
<tr>
<td>75 plus</td>
<td>2.5</td>
<td>N/S</td>
<td>-21.66</td>
<td>-36.34, -3.59</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>N/S</td>
<td>-8.12</td>
<td>-13.79, -2.08</td>
</tr>
<tr>
<td>Heart Failure (15-64)</td>
<td>2.5</td>
<td>N/S</td>
<td>60.40</td>
<td>16.95, 120.00</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>N/S</td>
<td>14.86</td>
<td>2.32, 28.54</td>
</tr>
</tbody>
</table>
Hospital admissions (all cause)

Mean daily hospital admissions were higher in the cool season than the warm season but were highest on days of smoke (Table 7.1). The results derived from the case-crossover analysis of all hospital admissions are shown in Table 7.2. Figures 7.5 and 7.6 display the point estimates and 95% confidence intervals.

A positive association was observed between increases in ambient levels of PM and all-age hospital admissions. For a 10 µg/m³ increase in PM$_{2.5}$, hospital admissions increased by 0.84% (95% CI 0.22-1.47%) whereas for a 10 µg/m³ increase in PM$_{10}$ the increase was 0.39% (95% CI 0.18-0.61 %). During the cool season, both pollutants were positively associated with increased hospitalisations, whereas in the warm season an association was observed with PM$_{2.5}$ only, the effect estimate being higher than that of the cool season. The risk of hospitalisation varied across age groups with the elderly and the young being most at risk of PM$_{2.5}$ exposure during the warm season. For those aged 15-64 the highest risk of hospitalisation was associated with PM$_{2.5}$ exposure in the cool season. PM$_{10}$ exposure was also a risk during the cool season for this age group, as well as those for those aged up to 14 years and those aged 65-74 years.

Respiratory admissions

Compared to the overall study period, mean daily respiratory admissions were highest during the cool season, and on days of smoke, mean daily admissions were higher than on days of dust or during the warm season (Table 7.1). Table 7.3 and Figures 7.5 and 7.6 show the effect estimates yielded from the case-crossover analysis of respiratory admissions. Overall there was no significant positive association between PM and respiratory admissions, except for the 15-64 year age group where there was a 3% (95% CI 0.73-5.33%) increase in respiratory hospitalisations associated with a 10 µg/m³ increase in PM$_{10}$ in the cool season. Analysis of cause-specific respiratory admissions (asthma and COPD) were largely inconsistent, varying from highly negative associations to highly positive, the broad confidence intervals indicative of small sample sizes.
Cardiovascular disease (Diseases of the circulatory system)

Mean daily hospital admissions for cardiovascular disease (CVD) were highest on days of smoke, followed by admissions during the cool season, warm season and days of dust (Table 7.1). The results for CVD admissions are displayed in Table 7.4 where it can be seen that overall hospitalisations were associated with PM$_{2.5}$. All season analysis yielded a 2.70% (95% CI 0.16-5.33%) increase in hospitalisations per 10 µg/m$^3$ increase in PM$_{2.5}$, (Figure 7.5) with greatest effect observed in the cool season (4.48% (95% C.I.0.74-8.36%). PM$_{10}$ also posed a risk in the cool season (Figure 7.6) although this was less marked than that associated with PM$_{2.5}$. Positive associations were seen in each age group except those aged 75 years and over. No positive associations were observed between PM and CVD hospitalisations in the warm season. As with respiratory results, cause–specific CVD effect estimates by age group were varied and questionable, possibly once again attributable to low statistical power. There was however suggestion of a consistent association between PM exposure during the cool season and IHD, as there was for all age MI.

![Figure 7.5](image.png)

**Figure 7.5:** The association between PM$_{2.5}$ and hospital admissions over the study period and by season.
Figure 7.6: Effect estimates for the association between PM$_{10}$ and hospital admissions over the study period and by season.

### 7.4 Discussion

This is the first study to investigate the effects of ambient air pollution on population health in Adelaide. Using health outcome, air quality, visibility and meteorological data, the association of particulate air pollution with cardiorespiratory morbidity was investigated using multivariable pollutant models in a case-crossover analysis. Results show clear seasonal variation in the effects of PM on hospital admissions in Adelaide.

**Adelaide’s air quality**

When compared to other major Australian cities, mean 24-hour PM$_{2.5}$ levels in Adelaide (8.3 µg/m$^3$) for the study period were similar to those in Brisbane, Melbourne, Perth and Sydney with mean levels of 9.7, 8.9, 8.1 and 9.4 µg/m$^3$ respectively, as previously reported.$^{85}$ The mean 24-hour average PM$_{10}$ concentration (19.3 µg/m$^3$) was higher than in these cities$^{85}$ but lower than levels recorded in Darwin during a bushfire season (20.84 µg/ m$^3$).$^{105}$ The small amount of missing monitoring data (no more than 6% for any pollutant) compares favourably with other Australian studies where up to 20%,$^{321}$ 25%$^4$ and 40%$^{104}$ of data were missing.
Health outcomes

In this study, findings show that despite the city’s relatively good air quality, the impact on the health of the population is not insignificant. Over the study period hospital admissions were observed to increase 0.84% and 0.39% in association with 10 µg/m³ increases in PM$_{2.5}$ and PM$_{10}$ respectively when adjusting for NO$_2$ and O$_3$. Whereas PM did not affect respiratory outcomes overall, there was a 3% increase in the risk of respiratory admissions in the 15-64 year age group in association with PM$_{10}$ in the cool season. That this effect was not seen in other age groups may reflect smaller sample sizes or possibly that out-of-hospital care was provided. The most marked finding was the association between PM$_{2.5}$ and CVD as discussed below.

Cardiovascular disease

The well known association between tobacco smoke and heart disease supports the plausibility of the cardiotoxic effects of PM.$^{74}$ Although yet to be clearly defined, the complex biological pathways involved in the PM-CVD association involve inflammatory responses induced by pulmonary and systemic oxidative stress that impair vascular function, alter cardiovascular haemodynamics and promote ischaemic events and plaque instability.$^{71,74}$ PM-induced elevation in blood pressure$^{322}$ increases the risk of adverse cardiovascular health outcomes and evidence suggests the strongest link lies with fine particulates.$^{74,323}$ This has been shown in the present study where an increase in CVD hospitalisations was linked more strongly with PM$_{2.5}$ than with PM$_{10}$.

The link between particulate matter and CVD has been well documented. Morgan et al$^{324}$ found that an increase from the 10$^{th}$ to the 90$^{th}$ percentile in daily mean particulate levels (measured by nephelometry) was associated with an increase of 2.23% in heart disease admissions in Sydney, and a recent Melbourne study found an association between PM$_{2.5}$ and out-of hospital cardiac arrests.$^{325}$ Using Bayesian 2-stage hierarchical models, Dominici et al$^{323}$ found fine particulate pollution to be associated with cardiovascular hospitalisations in the U.S. with a 1.28% increase in the risk of heart failure per 10 µg/m³ increase in same-day PM$_{2.5}$. Similar to the findings of the present study, links between PM$_{2.5}$ and MI have also been established elsewhere,$^{72,73}$ with one study, which also used a case-crossover analysis, demonstrating an 8.65%
increase in emergency hospitalisations per 16.32 μg/m$^3$ increase in PM$_{2.5}$, but not in the cool season as shown here.

The findings in this study are slightly higher than those of Barnett et al$^{69}$ who investigated links between outdoor air pollution and cardiovascular hospital admissions in Australian and New Zealand cities (Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch) and found a 1.3% (95% CI 0.6-2.0%) increase in cardiovascular hospital admissions per 3.8 μg/m$^3$ increase in 24-hr PM$_{2.5}$ in the elderly and a non significant increase in those aged 15-64 years. Difference in disease classification practices, admission criteria, climate, pollution sources, demographics and lifestyles may account for heterogeneity between findings.

**Respiratory disease**

The present study failed to find a clear positive association between PM and respiratory disease whilst controlling for flu epidemics. The exception was a 3% increase in respiratory admissions found to be associated with a 10 μg/m$^3$ increase in PM$_{10}$ (but not PM$_{2.5}$) during the cool season in the 15-64 year old age group. A Melbourne study where cohorts of adults completed questionnaires at different times, also found a lack of significant association between PM$_{2.5}$ exposure and adverse respiratory symptoms.$^{136}$ No consistent associations were seen with COPD or asthma although other studies have linked PM exposure to both COPD$^{103,148,324}$ and asthma.$^{106,317}$ Hospital admissions are a blunt measure of respiratory diseases such as asthma in the community. Only the most serious of asthma cases are hospitalized whereas many asthma sufferers self medicate or seek out of hospital care at the onset of an asthma attack. To capture this information in a quantitative ecological study is problematic and thus many studies investigating the effects of air pollution on asthma do so using a panel study approach where participants record their symptoms daily.$^{143,165,316,317}$ This type of study should be considered as a future research option for the investigation of the effect of particulate pollution on asthma in SA’s unique climate.

**Seasonality and wood smoke**

Local climate may play a role in the susceptibility of the population to diseases of the circulatory system. Being in the temperate zone, Adelaide generally has low humidity
with hot summers, and mild winters. In an Australian and New Zealand meta-analysis, cities with low humidity levels showed greater association between PM$_{2.5}$ and cardiac admissions than those with higher humidity.$^{69}$ Additionally, research by the WHO on cardiovascular health in 21 countries, found coronary events to be more common in cold weather, with locations with mild winters demonstrating greater increases in cold-related cardiovascular events than those with very cold winters.$^{326}$

Perhaps the main reason for the cool season effect of increased PM induced CVD hospitalisations is that mean PM$_{2.5}$ levels were higher in the cool season than the warm season. Similar findings of higher air pollution related risk in the cool season have been reported previously in Australia$^{127}$ although researchers in Melbourne$^{58}$ and Brisbane$^{85}$ found greater effects during the warm season, the authors suggesting photochemical smog may be influential. A source apportionment study carried out on fine and coarse particle samples collected from Australian cities (Melbourne, Sydney, Brisbane and Adelaide), showed that for fine particle samples, contributions of combustion sources and crustal sources were higher in the cooler seasons than the warmer seasons.$^{313}$ The authors speculated this may be due to the decrease in efficiency of motor vehicle engines in the winter, temperature inversions that occur more often in cool weather, and the increase in the use of domestic wood heaters.$^{313}$

Compared to motor vehicles, wood heaters emit 19% more PM, mainly in the form of PM$_{2.5}$$^{134}$ and in Adelaide they contribute to a considerable part of the total PM$_{10}$. Solid fuel burning (domestic) accounted for 25.6% of PM$_{10}$ in the Adelaide air shed in the year 2007-2008 according the National Pollutant Inventory,$^{314}$ and much of the combustion emissions in PM$_{10}$ reside in the PM$_{2.5}$ fraction.$^{327}$ Exposure to wood smoke poses a threat to health not only from particulates but other components including a toxic mix of volatile organic compounds, nitrogen oxides, carbon monoxide and other chemicals.$^{328}$ Indeed the present study showed that maximum daily NO$_2$ values were significantly higher on days of smoke than other days.

A small recent study showed that in some areas of the Adelaide Hills (e.g. Woodside), as many as 72% of people surveyed used wood heaters with 63% using them as their main primary heating source.$^{328}$ Consequently localised levels of particulates can reach up to 100 µg/m$^3$ in some low lying areas on cold still nights.$^{328}$ The effects on health
may be marked. The annual costs of morbidity/mortality linked to wood heaters in Australia has been estimated to exceed $2,000 per wood heater. A potential positive outcome of a warming climate with increased minimum temperatures, may be the need for less wood-fuelled heating sources and thus less domestic exposure to fine particulate pollution.

Smoke and dust

One of the strengths and unique aspects of this study is the identification of days of smoke and dust. Although the limited sample size restricted statistical power, summary statistics showed mean daily admissions (all-cause, respiratory and CVD) to be higher on smoke days than over the study period as a whole. Both smoke and dust contain high levels of particulates of which there is a large fine particle component. With bushfires emitting more PM$_{2.5}$ than any other source, the proportion of PM$_{2.5}$ in the PM$_{10}$ fraction has been reported previously to be higher on smoky days than at other times. This was confirmed in our data with the mean daily 24-hour PM$_{2.5}$ to PM$_{10}$ ratio being 44.9% for smoky days and 25.5% for dust days. Should the number of smoky days increase in a warming climate due to increased bushfire risk, this may have a marked impact on morbidity. A recent NSW survey following an extreme bushfire smoke pollution event, showed that 70% of respondents reported at least one health effect, with increased symptom rates in those with pre-existing respiratory and cardiovascular conditions.

Limitations

This study has several limitations, the first of which is, as in previous studies within this thesis, scheduled hospital admissions were not excluded from the hospital data. Additionally air quality data from only one monitoring site was used in this study. As measurements of coarse particles can be quite variable across distances, PM$_{10}$ measurements may not be representative of the whole metropolitan area. Fine particles on the other hand readily undergo atmospheric mixing and travel long distances and should therefore be evenly distributed across the city. Some days of smoke or dust may not have been recorded as such by the Bureau of Meteorology if for example; the smoke or dust was not evident at the time of the observation but occurred earlier or later.
in the day. Additionally if two weather conditions are present together at the time of observation, only the dominant condition is noted.

The accuracy of TEOM instruments used to measure particle concentrations has been questioned by some authors saying that the true concentrations of particulates may be underestimated. A TEOM is a continuously monitoring unit which draws air through a filter at a constant temperature and flow rate. The mass of PM is determined by measuring a change in oscillation frequency of a tapered quartz element attached to the filter and an impacting mechanism separates particles according to their aerodynamic diameter. As the instruments are incapable of accurate size differentiation, a 50% cutpoint at 2.5 µm and 10 µm is used to measure PM$_{2.5}$ and PM$_{10}$ concentrations respectively. The samplers collecting PM$_{10}$ collect all of the fine particles and part of the coarse particles, whereas the PM$_{2.5}$ samplers collect all of the fine particles and may, particularly in dry conditions, collect a significant although small, fraction of coarse particles. If fine particulates predominate, a PM$_{10}$ sample may contain mostly PM$_{2.5}$. Furthermore as the PM$_{10}$ samplers gather all PM$_{2.5}$ there could be more PM$_{2.5}$ in the PM$_{10}$ sample than that measured by the PM$_{2.5}$ sampler (Mr. R. Mitchell, EPA, pers. comm. 22/8/08). Due to measurement uncertainty, PM$_{2.5}$:PM$_{10}$ ratios greater than 1.0 can even occur. Very few of the vast number of studies quantifying health impacts of PM exposure mention possible limitations in the accuracy of instrument data upon which their findings are based. This issue is expanded upon in Chapter 10.

Adelaide is a relatively small city and statistical power is limited when data for a reasonably short period of time are stratified by season, age group and hospital diagnoses. Some effect estimates for cause-specific respiratory and cardiovascular outcomes were likely to be uninformative whereas those from the all-age categories would have most statistical rigour. A strength of the larger international air pollution studies is their size, some incorporating as many as 100 or more large cities, and up to 204 urban counties. Thus the findings in the present study are in need of replication with a modified methodology for stratification.
Conclusion

In conclusion this study has demonstrated the non-negligible effects of PM on overall and cardiovascular morbidity in Adelaide. Fine particulate pollution was seen to be more toxic than the coarse particles; and PM exposure during the cool season posed a greater risk to cardiovascular health than during the warm season.

These findings have clear policy implications. To date there is no ambient air quality standard for PM$_{2.5}$ in Australia, although an advisory reporting standard ambient Air Quality NEPM is in use. With epidemiological evidence a vital source of information upon which air quality standards are based, this study will be valuable to strategists and policymakers who have thus far been void of any South Australian data regarding the health impacts of PM$_{2.5}$ exposure. Limiting PM$_{2.5}$ exposure would have health benefits for Adelaide residents. Wood heater management in the winter, monitoring of in-service motor vehicle emissions and stronger limits for industry-related fine particle emissions are some of the strategies already under consideration by the National Environment Protection Council.$^{327}$ Research needs to continue to confirm these findings and to refine potential thresholds and associations between air pollution and health outcomes in a changing climate.
Chapter 8

The interactive and confounding effects of temperature and air pollution

8.1 Introduction

Air pollution is a complex combination of assorted airborne gases and solid matter. Toxicity to humans can depend on factors including the physiochemical nature of the coexisting pollutants, their dynamics and concentrations, and on prevailing meteorological conditions. The relationships between individual pollutants, weather and health are scientifically and epidemiologically difficult to unravel. Is the association of an individual pollutant with health outcomes confounded by the presence of other pollutants? Is there effect modification by temperature? Few studies have attempted to answer these challenging questions, perhaps due to the methodological complexity involved in statistical analyses and difficulty in the interpretation of findings.\textsuperscript{334}

In discussing the interrelationships between pollutants, clarification of statistical terminology used in the literature may be useful. Mauderly and Samet\textsuperscript{154} define interaction in statistical terms as “the interdependence of the effects of two or more variables”\textsuperscript{(p.1)}. Interaction can be synergistic (positive interdependence) or antagonistic (negative interdependence).\textsuperscript{128} Synergy can be further defined “as occurring if the effect of the combined exposure is greater than the sum of the effects of the two or more individual components of the mixture”\textsuperscript{154(p.1)}; i.e. greater than additive (Figure 8.1); whereas antagonism occurs if the “effect of the combination is
less than the sum of individual effects”.\textsuperscript{154(p.5)} The epidemiological term for interaction is \textit{effect modification} which occurs if multiple agents are interdependent.\textsuperscript{128,154} This may be more accurately termed \textit{effect measure modification} as it is the measure of effect which changes over values of some other variable.\textsuperscript{335} 

Statistical interaction is often confused with biological interaction, a term which refers to a mechanism where one causal factor is dependent on the presence of another. This can occur when two or more causes co-participate in the occurrence of a disease or other health outcome, and their combination is more potent than either alone.\textsuperscript{335} Biological interaction is thus a property of causality and relates to disease risk factors. Statistical interaction is a measure of association and refers to a statistical model, usually with a product term, that fits the data to predict the joint effects of more than one exposure. Caution should be used when making inferences linking statistical interaction with causation, as it may not reflect true biological interaction.

\textit{Confounding} occurs when two exposures are related and have an effect on the outcome but the confounder is not on the causal pathway of interest. A confounder \((c)\) for the association between an exposure \((a)\) and an outcome \((b)\) occurs if there is an association between \((a)\) and \((c)\), and \((c)\) is an independent cause of \((b)\). Hence a confounding factor must be associated with the exposure as well as the outcome, and controlling for the confounder may lead to a change in the risk estimate.\textsuperscript{335} Tobacco smoke might for instance be a potential confounder of the association between NO\(_2\) and asthma,\textsuperscript{336} or gaseous pollutants a confounder of the association between particulates and mortality.\textsuperscript{187} To add to the complexity, effect modification and confounding can exist separately or together.

Arguably, the air pollutants posing the most risk to health in the present context are PM, O\(_3\) and NO\(_2\). Some studies have demonstrated that the toxicity of PM can depend on its composition and interaction with gases\textsuperscript{128} whereas the association of O\(_3\) with other pollutants varies by location and season although effects can be enhanced by PM.\textsuperscript{128} The interaction of NO\(_2\) with other pollutants has been investigated in some studies but findings are not readily interpretable.\textsuperscript{128}
The present study which was largely exploratory, aimed to attempt to unravel these relationships whilst investigating the interactive and confounding effects of these pollutants on health outcomes in Adelaide.

![Diagrammatic representation of interaction](image)

**Figure 8.1:** Diagrammatic representation of interaction. C, additive effect of the impact of components A and B. D, interaction with a synergistic effect. E, interaction with an antagonistic effect.

### 8.2 Methods

The study period was 1 September 2001 to 31 October 2007. Air pollution monitoring data for particulate matter (PM$_{10}$ and PM$_{2.5}$), ozone ($O_3$) and nitrogen dioxide (NO$_2$) for the period were accessed from the SA Environment Protection Authority (EPA(SA)) Netley site, as outlined in the previous chapter. As the association between air pollutants and hospital admissions had been explored in a previous study (Chapter 7), a decision was made to investigate the association with emergency department visits and mortalities in this study. The relationship with ambulance callouts is yet to be determined.

Data relating to public and private hospital emergency department (ED) visits in Adelaide for the period July 2003 to October 2007 were sourced from the SA Department of Health, as outlined in Chapter 6. Data relating to mortalities (all, cardiovascular (CVD) and respiratory) for the period 1 September 2001 to 31 October 2007 were also sourced from the SA Department of Health using ICD codes to
determine diagnostic categories as previously described. Respiratory diagnoses did not exclude influenza.

The case-crossover over design was chosen as in the previous study in Chapter 7, as this has been validated in many air pollution epidemiology studies.\textsuperscript{72,117,146,152,187,333,337} In preference to the use of different temperature ranges, data were analysed as a whole, as well as stratified by warm and cool season as in previous studies within this thesis.\textsuperscript{85,103} Day of the week, public holidays and day after public holidays were controlled for. An average of the previous and present day’s pollutant levels accounted for possible lag effects. Units for the gaseous pollutants were converted from ppm to ppb. Using a stepwise construction of models, predictor variables were added one at a time. Univariable models included temperature, and multivariable models incorporated maximum temperature and one or two pollutants. To represent potential joint effects, a multivariable model was constructed with a multiplicative term of the two pollutants. These models are represented in equations 8.1, 8.2 and 8.3 respectively.

\begin{equation}
Y = \alpha + \beta_1 t + \varepsilon \quad (8.1)
\end{equation}

\begin{equation}
Y = \alpha + \beta_1 t + \beta_2 x_1 + \beta_3 x_2 + \varepsilon \quad (8.2)
\end{equation}

\begin{equation}
Y = \alpha + \beta_1 t + \beta_2 x_1 + \beta_3 x_2 + \beta_4 x_1 x_2 + \varepsilon \quad (8.3)
\end{equation}

where \(Y\) is the outcome; \(t\) is maximum daily temperature, \(x_1\) and \(x_2\) are the pollutants for which \(\beta_2\) and \(\beta_3\) are the coefficients respectively; \(x_1 x_2\) is the multiplicative interaction term; \(\beta_4\) estimates the joint effect of the pollutants, and \(\varepsilon\) is the error term.\textsuperscript{154} Thus models assessed interaction as well as confounding, the latter by adjusting for confounders in the models.\textsuperscript{335} Effect estimates were determined using conditional logistic regression models in Stata v10.

Separate models were run for PM\textsubscript{10} and PM\textsubscript{2.5} as these have been shown previously to be highly correlated. Analysis was performed by season (warm and cool as previously defined) and for the whole study period. Warm season models incorporated a dichotomous variable for heatwaves (defined as three or more consecutive days when maximum temperatures were \(\geq 35.0^\circ\text{C}\)), or periods when maximum temperature and
O₃ reached or exceeded the 95th percentile (≥ 35.3°C for maximum temperature, ≥ 41 ppb for O₃).

### 8.3 Results

This study was purely exploratory and findings are difficult to interpret. Given the methodological challenges posed, the time and space limitations of this thesis, and the 384 statistical models run during analysis, only an overview of results is presented herein. Table 8.1 shows the all-season effect estimates for the association of temperature and pollutants with ED visits calculated using univariable and multivariable models, and indicates that temperature and pollutants have an effect on ED visits. The associations with pollutants were generally positive in single and multivariable models, the exception being O₃ for which the association was often non-significant or close to unity. There was little evidence of confounding between pollutants.

Table 8.2 shows the effect estimates for high temperature, high O₃ days (N = 57). Estimates from ED visits suggest evidence of positive interaction with the risk being greater in the multivariable models for temperature (OR = 1.399) and O₃ (OR = 1.227) than in the separate univariable models (OR = 1.075 and OR = 1.008 respectively). This was even more evident for mortalities and respiratory mortalities. Odds ratios for the interactive terms in these models did not exceed unity, indicating effects were less than multiplicative.

Table 8.3 shows a non-numerical summary of the findings of the effect of pollutants on ED visits. Overall, analysis showed that temperature, PM₂.₅, PM₁₀, NO₂, and occasionally O₃ have an effect on ED visits. During the cool season PM and NO₂ had an effect, and PM₂.₅ and NO₂ was associated with CVD visits. Respiratory ED visits were positively associated with NO₂ and negatively associated with temperature and O₃. Both PM₁₀ and PM₂.₅ were also associated with respiratory ED visits. The effect estimates associated with PM₂.₅ were generally greater for ED visits (all and CVD) in the cool season than in the warm season (data not shown). During the warm season, there was an association with PM₂.₅ with all-cause as well as respiratory ED visits.
There was a marked increase in visits on high temperature high O₃ days. As in the cool season, CVD and respiratory visits were associated with NO₂.

Table 8.4 shows a non-numerical summary of the findings for mortalities. The overall effect of temperature and pollutants on mortality was less marked compared to the effect on ED visits. There was evidence of an association with NO₂ in all seasons and the warm season. There was also evidence of an association with O₃ in the warm season, the effect being increased on high temperature high O₃ days (Table 8.2). Also in the warm season, CVD deaths were associated with NO₂ (data not shown) and there was evidence of an association between respiratory deaths and O₃. There was no effect of heatwaves on deaths, however on high temperature high O₃ days the effect on respiratory deaths was considerable (Table 8.2).

A three-dimensional exposure-response plot of mortalities relative to the joint association of PM₁₀ and maximum temperature appears in Figure 8.2 and shows little evidence of apparent associations although there appears to be a dip at moderate PM₁₀ and temperatures levels and an increase as maximum temperatures reach their peak.

NOTE:
This figure is included on page 157 of the print copy of the thesis held in the University of Adelaide Library.
### Table 8.1: The effect of temperature and pollutants on emergency department visits in uni-and multi-variable models showing odds ratios (OR) and $P$-values

<table>
<thead>
<tr>
<th>Model</th>
<th>Temp OR</th>
<th>PM$_{2.5}$ OR</th>
<th>PM$_{10}$ OR</th>
<th>NO$_2$ OR</th>
<th>O$_3$ OR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>One variable plus temperature</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temp</td>
<td>1.005</td>
<td>1.004</td>
<td>1.004</td>
<td>1.004</td>
<td>1.005</td>
</tr>
<tr>
<td>PM$_{2.5}$</td>
<td></td>
<td>1.002</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM$_{10}$</td>
<td></td>
<td></td>
<td>1.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO$_2$</td>
<td></td>
<td></td>
<td></td>
<td>0.993</td>
<td>0.000</td>
</tr>
<tr>
<td>O$_3$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Two variables plus temperature</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM$_{2.5}$, NO$_2$</td>
<td>1.004</td>
<td></td>
<td></td>
<td>1.000</td>
<td>0.456</td>
</tr>
<tr>
<td>PM$_{2.5}$, O$_3$</td>
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<td>1.003</td>
<td></td>
<td>0.999</td>
<td>0.000</td>
</tr>
<tr>
<td>PM$_{10}$, NO$_2$</td>
<td>1.003</td>
<td>1.004</td>
<td>1.001</td>
<td>0.999</td>
<td>0.000</td>
</tr>
<tr>
<td>PM$_{10}$, O$_3$</td>
<td>1.004</td>
<td>1.001</td>
<td>0.999</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>O$_3$, NO$_2$</td>
<td>1.005</td>
<td></td>
<td>1.001</td>
<td>0.999</td>
<td>0.000</td>
</tr>
<tr>
<td><strong>Models with interactive term</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM$_{2.5}$, temp</td>
<td>1.006</td>
<td>1.010</td>
<td></td>
<td></td>
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<tr>
<td>PM$_{10}$, temp</td>
<td>1.007</td>
<td>1.004</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO$_2$, temp</td>
<td>1.007</td>
<td></td>
<td>1.004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O$_3$, temp</td>
<td>1.006</td>
<td></td>
<td></td>
<td>1.000</td>
<td>0.770</td>
</tr>
<tr>
<td>PM$_{2.5}$, NO$_2$</td>
<td>1.004</td>
<td>1.006</td>
<td>1.002</td>
<td></td>
<td></td>
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<tr>
<td>O$<em>3$, PM$</em>{2.5}$</td>
<td>1.004</td>
<td>1.006</td>
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<td>1.000</td>
<td>0.379</td>
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<tr>
<td>PM$_{10}$, NO$_2$</td>
<td>1.003</td>
<td></td>
<td>1.002</td>
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<td></td>
</tr>
<tr>
<td>O$<em>3$, PM$</em>{10}$</td>
<td>1.004</td>
<td>1.001</td>
<td>1.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O$_3$, NO$_2$</td>
<td>1.005</td>
<td></td>
<td>1.005</td>
<td>1.002</td>
<td>0.000</td>
</tr>
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</table>
Table 8.2: The effect of temperature and ozone on high temperature (> 35.3°C) and high ozone (≥ 41 ppb) on ED visits in uni- and multi-variable models showing odds ratios (OR) and P-values

<table>
<thead>
<tr>
<th>Model</th>
<th>Temperature</th>
<th></th>
<th>Ozone</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>P</td>
<td>OR</td>
<td>P</td>
</tr>
<tr>
<td><strong>ED visits</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>1.075</td>
<td>0.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>1.077</td>
<td>0.000</td>
<td>1.008</td>
<td>0.000</td>
</tr>
<tr>
<td>O3, temp + interactive term</td>
<td>1.399</td>
<td>0.000</td>
<td>1.227</td>
<td>0.000</td>
</tr>
<tr>
<td><strong>Cardiovascular ED visits</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>1.038</td>
<td>0.120</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>1.043</td>
<td>0.086</td>
<td>1.018</td>
<td>0.006</td>
</tr>
<tr>
<td>O3, temp + interactive term</td>
<td>1.368</td>
<td>0.096</td>
<td>1.249</td>
<td>0.114</td>
</tr>
<tr>
<td><strong>Respiratory ED visits</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>1.058</td>
<td>0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>1.059</td>
<td>0.001</td>
<td>1.009</td>
<td>0.076</td>
</tr>
<tr>
<td>O3, temp + interactive term</td>
<td>1.120</td>
<td>0.421</td>
<td>1.052</td>
<td>0.627</td>
</tr>
<tr>
<td><strong>Mortalities</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>1.065</td>
<td>0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>1.065</td>
<td>0.002</td>
<td>1.005</td>
<td>0.394</td>
</tr>
<tr>
<td>O3, temp + interactive term</td>
<td>2.115</td>
<td>0.000</td>
<td>1.682</td>
<td>0.000</td>
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<tr>
<td><strong>Cardiovascular mortalities</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>1.051</td>
<td>0.167</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>1.035</td>
<td>0.363</td>
<td>0.962</td>
<td>0.007</td>
</tr>
<tr>
<td>O3, temp + interactive term</td>
<td>1.007</td>
<td>0.982</td>
<td>0.942</td>
<td>0.810</td>
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<tr>
<td><strong>Respiratory mortalities</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>1.120</td>
<td>0.110</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>1.111</td>
<td>0.140</td>
<td>0.976</td>
<td>0.354</td>
</tr>
<tr>
<td>O3, temp + interactive term</td>
<td>7.096</td>
<td>0.004</td>
<td>4.085</td>
<td>0.008</td>
</tr>
</tbody>
</table>
Table 8.3: Overall summary of the effect on emergency department (ED) visits of air pollution and temperature determined using interaction models in a case-crossover analysis.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Cool season</th>
<th>Warm season</th>
<th>All seasons</th>
</tr>
</thead>
<tbody>
<tr>
<td>ED visits (all)</td>
<td>Positive associations with temperature. Positive associations with PM$<em>{2.5}$ in all models, PM$</em>{10}$ in most, NO$_2$ in most. Negative association with O$<em>3$ in 2-pollutant models. Significant interaction term in these models: PM$</em>{2.5}$ &amp; NO$<em>2$; PM$</em>{10}$ &amp; NO$_2$; O$<em>3$ &amp; PM$</em>{2.5}$; O$<em>3$ &amp; PM$</em>{10}$.</td>
<td>Positive associations with temperature in all models &amp; with PM$_{2.5}$ in most models. Positive associations with O$_3$ in interaction models. Mixed associations with heatwave days. Significant interaction terms in all models. Markedly increased effect on days of high temperature &amp; O$_3$ with significant interaction term.</td>
<td>Positive associations with temperature &amp; most pollutants in single &amp; multipollutant models. Significant interaction term in these models: PM$_{10}$ &amp; NO$<em>2$; PM$</em>{2.5}$ &amp; NO$_2$; O$<em>3$ &amp; PM$</em>{2.5}$; O$<em>3$ &amp; PM$</em>{10}$.</td>
</tr>
<tr>
<td>Cardiovascular ED visits</td>
<td>Positive associations with temperature in most models. Some evidence of an association with PM$_{2.5}$ &amp; NO$_2$.</td>
<td>Positive associations with NO$<em>2$ in all models. Significant interaction term with PM$</em>{2.5}$ &amp; NO$_2$; O$_3$ &amp; NO$_2$. Positive association with O$_3$ on days of high temperature &amp; O$_3$.</td>
<td>Positive associations with temperature &amp; with NO$_2$ in most models. Significant interaction term with O$_3$ &amp; NO$_2$.</td>
</tr>
<tr>
<td>Respiratory ED visits</td>
<td>Significant negative association with temperature in most models. Negative association with O$<em>3$ in most. Positive associations with NO$<em>2$ in most, PM$</em>{2.5}$ in some. Significant interaction term in these models: PM$</em>{10}$ &amp; NO$<em>2$; PM$</em>{2.5}$ &amp; NO$_2$; O$<em>3$ &amp; PM$</em>{2.5}$.</td>
<td>Mixed associations with temperature. Positive associations with NO$<em>2$ in all models and with O$<em>3$ in interactive models. Some positive associations with PM$</em>{2.5}$ in interactive models, mostly positive associations with PM$</em>{10}$. Significant interaction term in all models except heatwave days.</td>
<td>Positive associations with PM$<em>{10}$ &amp; NO$<em>2$ in all models &amp; PM$</em>{2.5}$ in most models. Significant interaction term in these models: PM$</em>{10}$ &amp; NO$<em>2$; PM$</em>{2.5}$ &amp; NO$_2$; O$<em>3$ &amp; PM$</em>{2.5}$; O$<em>3$ &amp; PM$</em>{10}$.</td>
</tr>
<tr>
<td>Outcome</td>
<td>Cool season</td>
<td>Warm season</td>
<td>All seasons</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Deaths (all)</td>
<td>No significant associations with temperature or pollutants.</td>
<td>Some association with NO(_2); positive associations with O(<em>3) in most models. Protective effect of heatwave days in PM(</em>{2.5}) model with significant and positive interactive term. Significant interactive terms in these models: O(<em>3) &amp; PM(</em>{2.5}); O(<em>3) &amp; PM(</em>{10}). Marked increase in the effect of temperature and O(_3) in interactive model on days of high temperature &amp; O(_3).</td>
<td>Positive association with temperature &amp; PM(_{2.5}). Evidence of an association with NO(_2).</td>
</tr>
<tr>
<td>Cardiovascular deaths</td>
<td>Positive association with O(_3) in some models.</td>
<td>Positive association with NO(_2) in most models. Significant interactive terms in the NO(_2) &amp; heatwave model.</td>
<td>Positive association with temperature &amp; O(_3) with significant interaction term.</td>
</tr>
<tr>
<td>Respiratory deaths</td>
<td>No associations with temperature or pollutants.</td>
<td>No evidence of a positive association with temperature, some negative. Some positive associations with PM(<em>{2.5}) in interactive models. Significant interactive terms in the following models: PM(</em>{2.5}) &amp; NO(_2); O(_3) &amp; NO(_2). Marked increase in the effect of temperature and O(_3) in interactive model on days of high temperature &amp; O(_3) with significant interactive term.</td>
<td>No positive associations with pollutants or temperature.</td>
</tr>
</tbody>
</table>

Table 8.4: Overall summary of the effect on mortalities of air pollution and temperature, determined using interaction models in a case-crossover analysis.
8.4 Discussion

This preliminary study uses a case-crossover approach to estimate the effect of air pollutants on health outcomes. Whilst controlling for confounding, possible interactive effects of air pollutants and temperature were investigated, using single- and multivariable models incorporating at most two pollutants and their interaction term into the models. Findings have shown that the complex connections between pollutants and temperature, and their ultimate effect on human health, can be confusing and difficult to interpret. Overall there was little evidence of confounding or effect modification. The exception was evidence of a synergistic effect on ED visits and mortalities when both temperature and O₃ levels were concomitantly high.

Univariable models

A negative association was observed between respiratory ED visits and temperature particularly in the cool season. This is plausible as respiratory disease is most often associated with the winter months. There was little evidence of a consistent positive association between mortality and maximum temperature. The lack of association between heat and mortality in Adelaide has been shown previously in a study of mortality and morbidity during heatwaves in Adelaide.³¹

Controlling for temperature, analysis showed the air pollutants NO₂, O₃, PM₁₀ and PM₂·₅ were linked with poor health outcomes. An association was found between NO₂ and ED visits for CVD and respiratory disease (Table 8.3). These findings concur with other studies which have found links between NO₂ exposure and CVD and respiratory diseases.⁵⁷,⁸⁵,¹⁰⁴,¹¹¹,¹⁵⁵

Associations with O₃ were mixed overall and in the cool season, although there was some suggestion of an association between O₃ and CVD deaths (Table 8.4). Other studies have found an association between O₃ and mortality¹⁵³,¹⁹⁴ which cannot be readily explained as confounding by temperature or secondary particles.¹²⁶,¹⁵³ As observed by others¹³⁹ the O₃ effects on all-cause deaths were higher in the warm season than the cool season or for year-round data (data not shown). Some researchers have found O₃ effects to be restricted to the warm season only.¹⁵³
It was interesting to find an association between both PM$_{10}$ and PM$_{2.5}$ on respiratory ED visits (Table 8.3) which was not observed with hospital admissions (Chapter 7). This supports the notion that only the most serious of respiratory events will be hospitalised, and that a true effect of PM exposure on respiratory disease in Adelaide may not be captured with the analysis of hospital admissions alone. That the effect of PM$_{2.5}$ was observed to be greater for ED visits (all and CVD) in the cool season than in the warm season, is in keeping with the previously observed effect of PM$_{2.5}$ on CVD hospital admissions in the cool season (Chapter 7).

**Confounding**

The addition of a second pollutant to the models showed little evidence of confounding as noted in other studies.$^{341}$ Perhaps more likely confounders of the effects of air pollution could be factors such as seasonality,$^{168}$ cigarette smoking or occupational exposure.$^{81}$

**Interaction**

The only evidence of interaction was observed on very hot days when O$_3$ levels were high. The association with all-cause deaths and respiratory deaths was much greater on these days indicating a synergistic effect (Table 8.2, 8.4). This correlates with a European study which found evidence for a synergistic effect of O$_3$ and high temperatures on respiratory and total mortality.$^{342}$

It should be noted that there appears to be some discrepancies in the literature regarding the use of the terms ‘interaction’ and ‘effect-modification’ and whether these are always consistent with the true statistical and biological meanings stated earlier is unknown. Notwithstanding, several studies have reported significant positive interactions between PM, NO$_2$ and O$_3$ with examples including enhanced (synergistic) adverse temperature effects found at high particulate levels,$^{11}$ higher PM$_{10}$ effects found on warmer days,$^{152}$ and a modification of temperature effects by O$_3$.$^{343}$ A three-way interaction between O$_3$, particles and temperature has also been suggested due to the oxidation-reduction activity of particles being enhanced by O$_3$.$^{344}$
Interestingly most of the published studies have investigated interactive effects of PM using only PM$_{10}$. Some studies suggest maximum temperature modifies the effect of PM$_{10}$ and that high temperatures together with high PM concentrations may have a synergistic effect on cardiorespiratory health outcomes. Staffogia et al\textsuperscript{152} also noted an increased effect on mortality of PM$_{10}$ by temperature level in Italy, suggesting a synergism between the two exposures. The authors hypothesised this effect modification may be due to greater exposure to air pollution in the warmer months.

Other studies suggest that PM$_{10}$ modifies the effect of temperature\textsuperscript{11} and that O$_3$ positively modifies the association between temperature and CVD mortality.\textsuperscript{343} A Sydney study found that PM$_{10}$ and O$_3$ confounded the association between temperature and mortality but no significant interaction was detected using multiplicative terms in the models.\textsuperscript{47} Yet others have found no evidence of interaction between the effects of temperature and particulates on mortality\textsuperscript{4} and some claim that no air pollutant is an effect modifier or confounder of the temperature-mortality association.\textsuperscript{346,347} The heterogeneity of study findings in the literature may reflect local pollution mixtures, exposure levels, population demographics and spatial settings.

**Heatwaves and air pollution in Adelaide**

Analitis et al\textsuperscript{348} have claimed that O$_3$ and PM concentrations have an active role in the heatwave-mortality relationship. Indeed this may have been the case in Adelaide during a major heatwave which occurred during the summer of 2009 with six consecutive days over 40°C.\textsuperscript{51} The heatwave also extended to Victoria, where the Black Saturday bushfires occurred. Figure 8.3 displays the Adelaide air quality during the three week period beginning 27 January 2009. This example of the interrelation between weather and air pollution shows that during the very hot days in the first week, air quality was mainly ‘fair’ and occasionally ‘poor’ (Figure 8.3A). Although the relevant data from air pollution monitors are yet to be sourced, the poor air quality during this period as indicated by the EPA’s air quality indices, was possibly due to high O$_3$ and PM levels which have been claimed to play an active role in heatwave mortality.\textsuperscript{348} Indeed, elevated O$_3$ levels occurred during the French heatwave of 2003 and it was ascertained that O$_3$ exposure contributed to the huge death toll.\textsuperscript{300}
Conditions in Adelaide on the day the bushfires occurred interstate (7 February 2009) were extremely hot with blustery north winds. The air quality index soared, indicating ‘very poor’ conditions. With the cool change shortly thereafter, air quality returned to ‘good’ (Figure 8.3B). The following week the incursion of bushfire smoke into the Adelaide airshed once again caused air quality to decline to ‘poor’ and at times ‘very poor’ (Figure 8.3C). As this period was outside the limits of the study period, the impact on air pollution-related and heat-related morbidity and mortality is yet to be evaluated, although media reports claim that numerous excess mortalities occurred during the extreme heat event, unlike previous heatwaves in Adelaide.

Limitations

As with many of the studies within this thesis, the relatively small city population poses statistical challenges and some stratified data may have yielded uninformative estimates. Moreover, the study period for ED visits was shorter than that for mortality. This has implications for investigating potential synergistic and antagonistic interactions between pollutants, this usually requiring large sample sizes to establish relationships with confidence.\textsuperscript{128}

Interpretation of confounding and interactive effects is often difficult, and it would be unwise to attempt to draw definitive statistical conclusions from the vast number of models fitted. Given the time limitation of the candidature, only a brief overview is presented here. The study design was based on best available evidence. Prior discussions were undertaken with experts locally, nationally and internationally (e.g. Dr Bart Ostro, Chief of the Air Pollution Epidemiology Unit in the Office of Environmental Health Hazard Assessment within the California Environmental Protection Agency). The WHO however suggests that using multivariate models may mis-specify the true interrelationships between pollutants, particularly in the case of NO\textsubscript{2} which is a precursor to other pollutants including nitric acid and O\textsubscript{3}.\textsuperscript{128} The study design thus may not be the most appropriate for this type of analysis although the fact that few researchers have tackled the questions relating to interaction underlines the difficulties associated with defining a suitable methodology and interpreting findings.
Figure 8.3: The relationship between weather and air pollution, classified as very good, good, fair, poor or very poor using an Air Quality Index (AQI), during a 3 week period in January-February 2009. A, 6 days over 40°C. B, air quality declined markedly on 7/2/09, a day of extreme heat and blustery hot winds, when the Victorian bushfires began; C, Adelaide’s air quality affected by the incursion of smoke from the Victorian bushfires.

*Source:* EPA (URL: http://www.epa.sa.gov.au/)
Conclusion

Whilst highlighting the complexity of investigating inter-relationships between environmental exposures, the findings of this study have reinforced those of the previous study (Chapter 7) which demonstrated that air pollution in Adelaide has a measurable effect on the health of the population. Caution however should be exercised in the attribution of specific causal relationships. Although exploratory, this study has yielded some interesting results including the possible synergistic effect of temperature and O₃ on very hot days with high O₃ concentrations. The influence on health of airborne pollutants during extreme heat events requires further investigation in light of predictions these events will become more frequent. There is also a need for source apportionment studies to determine the components of PM in Adelaide that interact with, or form, other pollutants that are toxic to human health under changing environmental conditions. Nevertheless it is clear that in the interests of public health, harmful emissions need to be identified and technological and behavioural adaptation needs to take place to lower emissions and exposure levels.
SECTION IV

USING THE EVIDENCE
Section IV draws together the knowledge gained from a large body of literature relating to the health effects of environmental stressors, and findings from studies undertaken within this thesis.

Chapter 9 uses currently available information to formulate an evidence-based environmental health risk assessment for Adelaide under a climate change scenario. Complex computational simulations for meteorological and health assessment modelling are beyond the scope of this thesis, rather predictions are drawn from existing models formulated by renowned authorities such as Australia’s Commonwealth Scientific and Industrial Research Organisation (CSIRO) and the Intergovernmental Panel on Climate Change (IPCC). The studies and literature review undertaken within this thesis focus on Adelaide specific information. The chosen risk assessment paradigm is based on that used by the Australian enHealth Council in their “Environmental Health Risk Assessment Guidelines for Assessing Human Health Risks from Environmental Hazards” report.\textsuperscript{55}

Chapter 10 concludes the thesis with a comprehensive discussion of the key findings, strengths and limitations of the studies undertaken, challenges faced, significance of the studies, policy implications, and a range of options for future research aimed at investigating temperature-health and air pollution-health relationships.
Chapter 9

Health risk assessment for environmental stressors in Adelaide

9.1 Introduction

Risk is the product of the probability of an event occurring and the consequence of that event, and increases as the consequences and/or the likelihood of a consequence, grows (Figure 9.1).\(^{350}\) Low risk results when consequences are of marginal impact and unlikely to occur. Conversely, high risk results when catastrophic consequences are very likely to occur.\(^{350}\) Risk assessments are key tools for summarising risks to health from exposure to environmental agents\(^{67}\) and are increasingly being used in the formulation of environmental health policies and regulations.\(^{351}\) Here a risk assessment framework is used to predict the effect on human health of environmental variations to weather/climate and air quality in Adelaide, that may occur as a result of climate change.

Although detailed risk assessments are complex and generally require purpose-built climate projections,\(^{352}\) the nature of the assessment presented here is determined by its context within this thesis and thus is more simplistic in its approach. Risk assessments can range from those that are purely qualitative to those that are quantitative,\(^{201}\) the latter being subject to assumptions of exposure levels, choice of appropriate dose-response relationships and other decisions.\(^{67}\) Both quantitative and qualitative approaches are incorporated herein.
The risk assessment process is generally quite prescriptive although there are a range of approaches that can be used. Nevertheless there are similarities between different frameworks and several common themes. A health risk assessment has two basic components - health hazard characterisation and health impact assessment, and generally these comprise the key elements of Hazard Identification, Hazard Assessment and Risk Characterisation. The Hazard Assessment step often involves Dose-response Assessment and Exposure Assessment. To ensure the assessment is informative, stakeholder involvement and management strategies should be considered, in combination with an adequate risk communication strategy.

Climate change specific risk assessments which estimate climate-sensitive disease burdens may involve a comparative risk assessment approach embedded within a broader health impact assessment framework. Figure 9.2 is a diagrammatic representation of the comparative risk assessment model showing how the climate change-attributable disease burden is calculated. McMichael et al used a similar approach to estimate attributable climate-change disease burden for 2020 and 2050 in Australia, using different greenhouse gas emissions scenarios and future population projections. Basically, a comparative risk assessment comprises four steps: “(a) identifying climate-sensitive health outcomes, (b) quantifying dose-response relationships for baseline climate, (c) selecting future climate scenarios, and (d) estimating the climate change-attributable burden of disease and the burden that is avoidable by plausible reductions in the risk factor.” Finally, some researchers
have used complex computational climate modelling to predict heat-related mortality under a climate change scenario, a methodology beyond the scope of this study.

**Figure 9.2:** Comparative risk assessment model in the context of climate change, with definitions of attributable and avoidable disease burden. *Source:* Reproduced with permission from Environmental Health Perspectives (Campbell-Lendrum et al.355)

Given that the present assessment broadly incorporates the effects of air pollution as well as weather on health outcomes, the chosen framework in this thesis is based on that used by the Australian enHealth Council,55,357 and the National Environment Protection Council (NEPC) in their investigation of a need for a national PM$_{2.5}$ air pollution standard.312,327 The following steps are incorporated in the framework: Issue Identification, Hazard Assessment (comprising Hazard Identification and Dose-response Assessment), Exposure Assessment, Risk Characterisation and Risk Management as shown in Figure 9.3. Invariably there is a degree of overlap between the steps. The present example also incorporates Risk Communication and public risk perception components considered to be important in climate change discussions.
9.2. Issue Identification

For several decades there has been concern amongst scientific observers that human activities involving the burning of fossil fuels have the ability to interfere with the balance of atmospheric gases and radiative forcing (the influence of natural and human factors on warming and cooling of the planet).\textsuperscript{157} Incoming solar energy that warms the Earth’s surface is partially re-radiated back into the atmosphere where some is trapped by greenhouse gases such as CO\textsubscript{2} and methane. This natural process keeps the planet at an inhabitable temperature.\textsuperscript{45} Mounting evidence over time has confirmed that anthropogenic activities during the past century have caused marked increases in greenhouse gases, with consequential effects on solar absorption, reflection and radiation. The effect has been a build up of heat retention in the lower atmosphere and
changes in weather patterns. The phenomenon has contributed to a change in the
global climate and a gradual increase in global average surface temperatures of
approximately 0.74°C in the period 1906-2005, compared to 0.6°C for 1901-2000. The
warming trend has been greatest in the second half of the century, and eleven of the
twelve years from 1995 to 2006 have been amongst the warmest on record.

In addition:

- the average temperature of the global ocean has increased
- sea levels have risen
- mountain glaciers and snow levels have declined
- there have been notable changes in precipitation and evaporation rates
- more intense and longer droughts have occurred
- mid-latitude westerly winds have strengthened
- cold days and cold nights have become less frequent
- hot days, hot nights and heatwaves have occurred more frequently.

These worldwide climate changes have been unequivocally observed in Australia. Temperatures in Adelaide and across South Australia (SA) have risen in the past half century as discussed in Chapter 2. Although they cannot with certainty be attributed to climate change, extreme and/or unusual heatwaves have occurred in Adelaide in 2008 and 2009, and winter in 2009 was the warmest on record for SA, Victoria and New South Wales. Additionally, in southern Australia rainfall has declined, a phenomenon linked to a dramatic shift in climate systems over the past 30 years.

Certain changes in air quality may be linked to climate change. Photochemical reactions between atmospheric components can be influenced by changes in temperature, humidity and solar radiation. The association between heat and air quality is complex, with the main air pollutants associated with hot conditions being O₃ and PM. This has been demonstrated in a Sydney study of synoptic weather patterns where it was found that warm air masses contained the highest O₃ and PM₁₀ concentrations. Levels of PM₁₀, PM₂.₅ and O₃ on occasions peak at concentrations at or above NEPM standards and there is no evidence of a consistent downward trend over time, unlike other criteria pollutants. Extreme pollution events in recent years have been linked to drought-related dust storms, particularly following dry periods, and bushfires. Since the
1980s, major bushfires have occurred in SA and/or Victoria (with smoke affecting SA’s air quality) in 1983, 2003, 2005, 2006, and 2009, indicating a recent trend of increasing frequency.

With greenhouse gas emissions being the drivers of anthropocentric climate change, it is of concern that levels are not abating. Current evidence shows that CO\textsubscript{2} emissions are tracking above the IPCC’s worst case scenarios and that Australia continues to increase, not decrease, its greenhouse gas emissions at a rate of 2\% per year.\textsuperscript{361} Furthermore indications are that climate change in Australia is progressing faster than expected.\textsuperscript{361,362} The impact on Australia and SA are predicted to be wide ranging, encompassing many areas including agriculture, finance, water and food security, forest ecology, settlements, and health. The effect of the latter on the local population were the focus of this risk assessment, and both heat and air pollution are addressed in the process.

### 9.3 Hazard Assessment

This step investigates why a variation in climate can have negative health outcomes, with recognition that biological plausibility and validation against historical data are important factors in the hazard assessment process.\textsuperscript{295} Here the specific health hazards are identified, exposure-response relationships discussed, and likely exposure scenarios considered.

#### 9.3.1 Hazard Identification

It was recently claimed that “climate change is the biggest global health threat of the 21\textsuperscript{st} century.”\textsuperscript{363(p. 1693)} Worldwide in 2000, climate change was responsible for 5.5 million disability adjusted life years lost\textsuperscript{65} and 154,000 deaths.\textsuperscript{65} A substantial amount of evidence suggests future impacts will be wide and varied with direct and indirect effects on population health. This includes increases in illnesses and injuries from severe weather events, increases in cardiorespiratory and infectious diseases (food-borne, vector-borne and water-borne), threats to water and food supplies, and effects on renal\textsuperscript{303} and mental health.\textsuperscript{364-366} For the purposes of this risk assessment the specific climate-change associated environmental health hazards to be investigated are periods
of extreme hot weather, and air pollutants (specifically O$_3$ and PM), exposures to which may increase under a climate-change scenario.

### 9.3.1.1 Effects of heat on human health

The epidemiological literature on heat-associated illness and death is comprehensive and is gathering public health importance as global temperatures increase. Policymakers aim to formulate effective intervention strategies to prevent often catastrophic health consequences of extreme heatwaves, particularly when heat is of an intensity not previously experienced by the local population. Much of this thesis has covered the negative impacts exposure to heat may have on health and wellbeing. The literature review in Chapter 1 discusses this issue in general, and the effects of heatwaves on population health in Adelaide are covered in Section II where Chapter 3 investigates the effects on renal health, Chapter 4 mental health, Chapter 5 ambulance usage and Chapter 6 emergency department visits.

In brief, mortality may be attributable to heat-specific illnesses such as heat stroke, or to cardiovascular and cerebrovascular causes due to the increased cardiac output necessary for physiological heat loss.\textsuperscript{17} Due to diminished physical and cardiovascular reserve, these effects are more often seen in the elderly, up to 1,100 of whom are estimated to die annually in Australian cities due to the effects of heat.\textsuperscript{3,197} Other sub-groups of the population susceptible to thermal stress, as well as contextual factors influencing vulnerability, have been discussed in Chapter 1 and Section II. Generally, the effect of high temperatures on mortality in Adelaide has not been observed to the same extent as in many international cities where heatwaves rarely occur.\textsuperscript{31} Anecdotal evidence however suggests the extreme 2009 heatwave may have been exceptional in this regard, and indeed the same heatwave in Victoria reportedly claimed some 374 lives.\textsuperscript{265} Heat-associated morbidity studies within this thesis, including those published during the candidature,\textsuperscript{300,303} have shown notable impacts of heat on renal and mental morbidity, ambulance callouts and emergency department visits in Adelaide.
9.3.1.2 Effect of air pollution on human health

As previously outlined in Chapters 1 and 7, numerous reports of the adverse effects that ambient air pollution can have on health have been published in the epidemiological literature. These have been supported by toxicological data, occupational studies, chamber studies and animal studies. In short, exposure to xenobiotics and their absorption into the body can lead to physiological responses which may be detrimental to health, with strongest effects on the respiratory and cardiovascular systems. In more serious cases, acute exposure may result in death as can long term chronic exposure.

The pollutants of interest in the context of climate change are ground level O₃ and PM as previously mentioned, and studies have shown these to be contributors to heatwave deaths. As levels of O₃ increase with heat and sunlight, elevated concentrations during heatwaves have been associated with increased mortality and morbidity due to both respiratory and cardiovascular disease. As well as these direct effects, high temperatures can have indirect effects on air quality via airborne dust, bushfire smoke or pollen-bearing particulates. Worldwide, increases in mortality, hospital admissions and ED visits have been linked with daily changes in ambient PM levels, and a recent study from Korea has shown that the effect of PM₁₀ on mortality and morbidity increases during the summer months. Considerable evidence suggests that fine particulates (PM₂.₅) may be even more detrimental to health than PM₁₀ as confirmed by studies within this thesis. Reasons include their smaller size allowing inhalation deeper into the respiratory tract, easier penetration indoors, longer airborne suspension and transportation over vast distances; and their toxicity, as fine particles can be formed from gases and may be comprised of sulphates, nitrates, metals, acids and other chemicals that can be hazardous to health.

Both dust storms and bushfire smoke have been associated with the onset of respiratory conditions and changes in asthma severity in Australia. Additionally dust has been shown to carry airborne microorganisms including pathogens over long distances and studies have shown there to be a ten-fold increase in airborne microorganisms on dusty days. Bushfire smoke also contains high levels of PM and the 2009 bushfire storms in Victoria following the extreme heatwave, caused a serious and extended decline in air quality for much of the state. Although to the
author’s knowledge health effects of the exposure are yet to be evaluated, a similar situation occurred in 2003 in Victoria and was associated with a marked increase in respiratory ED visits. To date no such study has been carried in South Australia.

9.3.2 Dose-response Assessment

This step, often referred to as ‘exposure-response assessment’ in air pollution health impact assessments concerns quantifying relationships between exposure and health outcomes. Exposure estimates for temperature, heatwaves and air pollutants can be obtained from numerous published epidemiological studies and for the latter, controlled exposure chamber studies using single pollutants at various durations and concentrations have also been used to determine human exposure data.

9.3.2.1 The heat-health exposure-response relationship

The relationship between mortality and temperature is often described as a U-shaped pattern with lowest counts of deaths at moderate temperatures, increasing in cold or hot weather. The shape of the curves causes some difficulty in estimating accurate exposure-response data and as a result often temperature thresholds (or change-points) are used, above which the association becomes more linear. In a study in Christchurch, New Zealand, Hales et al determined that above 20.5°C, an increase of 1°C was associated with a 1% (95% CI 0.4-2.1%) increase in all-cause mortality and a 3% (95% CI 0.1-6.0%) increase in respiratory mortality. McMichael et al also used Christchurch data in a climate change risk assessment involving cities including Adelaide, but instead used a threshold of 28°C above which mortality was estimated to increase by 3% per degree. These estimates are similar to that used in a European study where apparent temperature thresholds (and increases in natural mortality per degree above that) for Mediterranean and north-continental cities were 29.4°C (3.12%) and 23.3°C (1.84%) respectively.

Heatwave studies undertaken within this thesis using a hockey-stick regression method to estimate thresholds, showed that hospitalisations in Adelaide for mental and behavioural disorders increased, with a threshold temperature of 26.7°C identified (Figure 4.2A). For ambulance callouts in Adelaide, the threshold temperature was
higher, at 34.6°C (Figure 5.1) with a demonstrated increase of 2.6 callouts per 1°C degree rise in maximum temperature. Research is underway to determine mortality thresholds. There is a high level of uncertainty in extrapolating any of these heat-health estimates to circumstances of unprecedented heat as the shape of the exposure-response curve may change when very extreme conditions prevail.

9.3.2.2 The air pollution-health exposure-response relationship

Unlike temperature-health relationships, air pollution exposure-response relationships often appear to be linear with no clear threshold above which poor health outcomes occur. This appears to be case the for PM, although the evidence is less clear for O₃.

The exposure-response relationship between PM and mortality is often reported to be linear although it may actually be curvilinear as small changes in exposure at the low end have been observed to have similar effects to large changes at higher concentrations. Other researchers feel the association can adequately be estimated using a linear model. In the Christchurch study, an increase of 10 µg/m³ in PM₁₀ was associated with an increase in all-cause mortality of 1% (95% CI 0.5-2.2%) and a 4% (95% CI 1.5-5.9%) increase in respiratory mortality, incorporating a lag of one day. In Europe the effect estimate for the same increase in PM₁₀ was found to be 0.6% (95% CI 0.4-0.8%) increase in mortality whereas in the U.S. it was 0.5% (0.1-0.9%). In Australia similar although non-significant increases in mortality have been reported for both PM₁₀ and PM₂.₅. It has been observed elsewhere that locations with higher PM₂.₅/PM₁₀ ratios have stronger associations, suggesting PM₂.₅ may the more toxic of the two pollutants. Figure 9.4 displays relative risks and 95% confidence intervals from O₃ and PM studies undertaken in Europe and the U.S. Table 1.1 (page 26) contains details of findings from recent Australian air pollution studies.

Ozone has been associated with increased mortality in Australia and in a meta-analysis of 43 individual studies from around the world where the combined random effects estimate was reported to be 1.6% excess mortality (95% CI 1.1-2.0%) per 20 ppb increase in 24 hr average O₃ with generally larger effects observed in the summer. With PM in the model the effect estimates were slightly reduced. The shape of the
exposure response curve is reportedly linear or near linear even at low exposure levels, nevertheless uncertainties exist at widely differing O3 levels.

Exposure to PM and O3 also have an effect on acute non-fatal health outcomes, particularly cardiovascular and respiratory morbidity as discussed in the preceding chapters. Using local data, it has been shown that this is also the case in Adelaide. As reported in Chapter 7, a 10 µg/m³ increase in and PM2.5 and PM10 were associated with an increase in all year all-cause hospital admissions of 0.84% (95% CI 0.22-1.47%) and 0.39% (95% C.I. 0.18-0.61%) respectively, and PM2.5 was associated with a 2.70% (95% CI 0.16, 5.33%) increase in admissions for diseases of the circulatory system. This estimate was higher in the cool season.

NOTE:
This figure is included on page 180 of the print copy of the thesis held in the University of Adelaide Library.

Figure 9.4: Point estimates and 95% confidence intervals for mortality related to a 10 µg/m³ increase in PM10, black smoke (BS) and PM2.5 from European and U.S. studies. Source: Reproduced with permission from WHO.

Exposure to biomass smoke can occur during times of extreme heat, with effects on health including exacerbation of respiratory conditions. Table 9.1 from the EPA (Victoria) shows 24-hour and 1-hour PM levels and visibility, together with corresponding health categories in relation to bushfire smoke. This shows that air quality is considered to be ‘good’ when concentrations of 24-hour PM10 are less than 50
µg/m³, whereas levels above 310 µg/m³ are considered hazardous. Although pertaining to bushfire smoke, these PM levels are also likely to be relevant for dust conditions.

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### 9.3.2.3 Sensitive subpopulations

Table 9.1 indicates sensitive and unhealthy individuals are more likely to experience symptoms of ill health at lower exposure levels. This is an important point of recognition in risk assessments relating to air pollution, as sub-populations such as infants, children, and persons with chronic lung disease or heart diseases may be more sensitive to certain air pollutants, as will some who have inherent genetic susceptibilities. In terms of heat extremes, the sub groups likely to have increased sensitivity are those with thermoregulatory or cardiovascular impairment due to age, disease or use of certain medications.

It is relevant at this point to distinguish between sensitivity and vulnerability – terms often used interchangeably in discussions on exposure-health response relationships. Figure 9.5 clarifies this issue and shows the potential health impact of a stressor depends on exposure levels and the sensitivity of the individual to the exposure. This together with the individual’s (or community’s) capacity to adapt to the situation will ultimately determine their vulnerability. An added component is their level of resilience which can affect adaptive capacity. This aspect will be discussed in more detail in Section 9.5.1.
NOTE:
This figure is included on page 182 of the print copy of the thesis held in the University of Adelaide Library.

9.3.3 Exposure assessment

Individuals have little control over the weather, the ambient air they breathe, or the extent to which they are exposed environmentally. Indoor temperatures and air quality can often be influenced to a certain degree depending on factors such as standard of housing, number of open doors and windows, air movement, and use of air conditioning/fans. Furthermore, O\textsubscript{3} can react with materials and form toxic secondary products indoors, and PM can readily penetrate indoors depending on particle size and home characteristics. As it is almost impossible to determine the numbers of people exposed, duration of exposure or exposure levels (now and in the future), exposure assessment is the source of most uncertainty in the environmental risk assessment process. It is thus an assumption that the whole or at least most of the population will be exposed although in reality exposure levels, duration of exposure and response to exposure will vary greatly between individuals.

Factors such as occupation (outdoor/indoor workers), exercise, mobility, social context, place and socioeconomic status may have a bearing on exposure levels. Populations living in cities for example are generally exposed to higher temperatures than rural populations. This is due in part to the urban heat island effect whereby by the heat-retaining properties of the thermal mass in cities causes higher daytime and night time temperatures. Heat is also generated from transport and industry associated
energy production in cities,\textsuperscript{24} which also leads to higher levels of air pollution in comparison to rural areas. Additionally there are higher concentrations of susceptible people living in cities\textsuperscript{310} due in part perhaps for the need to have ready access to health care. Spatial analyses within this thesis have demonstrated several instances where the Adelaide CBD poses a higher heat risk than surrounding areas.

An increase in exposure may have different effects on some sectors of the population (sensitive subpopulations have been discussed in Section 9.3.2.3). The most highly sensitive persons may respond adversely for example, to pollution exposures that are close to or at, natural background levels.\textsuperscript{67} Figure 9.6 illustrates that a population’s distribution of exposure may shift to a higher level resulting in unacceptable risk for a sub-population who are more sensitive than others (Figure 9.6A), whereas the same shift in exposure in another population may result in no one falling outside of the acceptable risk zone (Figure 9.6B). This becomes an important aspect to consider when defining thresholds and setting standards.

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{figure96.png}
\caption{An increase in exposure may pose an unacceptable risk in a population in which A, a proportion is sensitive, or B, no unacceptable risk in a less sensitive population. Source: Adapted from American Thoracic Society.\textsuperscript{67}}
\end{figure}

\subsection*{9.3.3.1 Heat exposure under climate change scenarios}

Using a range of different greenhouse gas and sulphate aerosol emission scenarios for the 21\textsuperscript{st} century, the IPCC predicts global temperatures will rise with climate change.
Each of the Special Report on Emission Scenarios (SRES) scenarios represents a variation within one of four storylines. Briefly, the A1 storyline describes a situation of rapid economic growth, efficient technologies and population numbers peaking in 2050. Subgroups A1F1, A1T and A1B represent fossil fuel intensive, non-fossil fuel, and a balance across all energy sources respectively. The A2 storyline describes a situation where local identity is preserved, there is regional self-reliance, population steadily increases, and economic growth and technology change are slower than A1. This scenario is characterised by weak environmental concerns, high CO2 emissions and a population rapidly increasing to 15 billion worldwide by 2100. The B1 storyline has the same population as A1 but there are global solutions to economic, social and environmental sustainability, and efficient technologies. The fourth and final storyline, B2, has an emphasis on local solutions and efficient technologies and the population increases at a slower rate than in A2. Some reports have used A1F1 to represent a high climate change scenario, A1B to represent the mid scenario and B1 to represent the low climate change scenario; whereas others have used A2 and B2 to represent high and low greenhouse gas projections respectively.

Using these scenarios, the IPCC predict there will be warming of about 0.2°C per decade over the next two decades. The best estimates of global temperatures by the end of the century range from an increase of 1.8°C (1.1-2.9°C) compared to 1980-1999 temperatures under a B1 (low) scenario, to 4.0°C (2.4-6.4°C) under the A1F1 (high) scenario. This increase in temperatures will likely lead to an increase in more hot weather, heatwaves and temperature extremes (Figure 9.7).

![Figure 9.7: The effect of increasing mean temperatures on temperature extremes. Source: Reproduced with permission from IPCC.](image-url)
Heat exposure in Australia

In Australia, there is little doubt weather patterns are changing. Best estimates for 2070 suggest a temperature rise of between 1.8°C (1.0-2.5°C) for a low emissions scenario, to 3.4°C (2.2-5.0°C) for a high emissions scenario. A mean surface temperature increase of 2°C above pre-industrial times will be damaging to many small Australian ecosystems and effects will extend to agricultural yields, food and water supplies, sea level rise and infectious disease risks. Figure 9.8 shows the probability of annual temperature rises above 1-4°C in Australia based on modelled levels of greenhouse gas emissions, and shows that even with low emissions it is likely the 2°C threshold will be exceeded in inland areas later in the century.

NOTE:
This figure is included on page 185 of the print copy of the thesis held in the University of Adelaide Library.

It follows that the average number of days per year of temperatures exceeding 35°C in Australian cities is expected to rise considerably in some areas, more so in the north than the south of the nation. Climatologists predict that in Hobart (Tasmania), there will
be little change, with the average 1.4 days currently above 35°C per year increasing to a maximum of 3.4 by 2070 under a worst case scenario. By contrast, in Darwin (Northern Territory) the change may be drastic, with the current annual average of 11 days above 35°C increasing as much as 28-fold by 2070, to 141-308 days under a worst case scenario.352

Heat exposure in Adelaide

In Adelaide as with elsewhere in Australia and globally, temperatures are expected to increase with climate change. Figure 9.9 shows a range of CSIRO projections for Adelaide and the Mt. Lofty region, showing an increase by 2100 ranging from approximately 1.5°C to more than 3.5°C. Warming for Adelaide will not be as great as for areas inland or the northern regions of the State.350 Currently there are on average, 17 days per year above 35°C, the majority of which occur in January and February (Figure 9.10) making these the months of greatest risk for heat exposure. Projections suggest there will be 21-26 days above 35°C annually by 2030, and 24-47 by 2070, depending on the rate of greenhouse gas emissions.352

Figure 9.9: A range of CSIRO simulated annual temperature anomalies for Adelaide and the Mt. Lofty Ranges regions smoothed by an 11-year running mean. Source: Reproduced with permission from Suppiah et al.350

Heatwaves in Adelaide (defined here as three or more consecutive days of maximum temperatures ≥ 35°C)31,300 occur with regularity each summer. In one study within this
thesis 31 heatwaves were recorded in the study period January 1995 to December 2006,\textsuperscript{303} yielding an average of almost 2.6 heatwaves per year. According to CSIRO, projections suggest this may increase to as many as 3.64 by 2030 under the A1B scenario; and up to 3.23-4.54 by 2070 under the B1 scenario or 4.17-7.63 under the A1F1 scenario (Dr. S. Williams, University of Adelaide, pers. comm. 24/11/09). When evaluating risk, it should be recognised that heatwaves of duration and intensity not previously experienced are likely to occur in the future, increasing uncertainty in projections of health responses.\textsuperscript{295,355} This has been the case in the previous two years when unprecedented heatwaves have occurred in Adelaide.

**Figure 9.10:** Graph showing the mean number of days per month in Adelaide ≥ 35°C and the monthly mean maximum temperatures. *Source:* Bureau of Meteorology (URL: http://www.bom.gov.au/).

Finally, the influence of minimum temperature on heat exposure can be important as cooler overnight temperatures enable residents to ventilate their homes and gain relief from stifling heat. Coastal suburbs of Adelaide regularly experience sea breezes in the afternoons which can offer relief from the heat. As this air mass moves inland, eastern suburbs near the foot of the Mt Lofty Ranges benefit from nighttime downslope gully breezes in the months from November to March.\textsuperscript{381} Adelaide experienced its warmest night on record during the 2009 heatwave, when temperatures dropped to only 33.9°C.\textsuperscript{51} Anecdotal evidence suggests ambulance services reported a reduced number of calls.
only when overnight minimum temperatures started to fall, even though the daily maxima still exceeded 38°C. In Melbourne it has been found that excess deaths occur when minimum temperatures exceed 24°C and hence a heat alert system is based on a combination of present day maximum and minimum temperatures. Warmer overnight temperatures expected with climate change, particularly in cities where the urban heat island effect occurs, will lead to increased heat exposure.

9.3.3.2 Air pollution exposure under climate change scenarios

Humans are generally exposed to air pollution via nasal or oral inhalation into the respiratory tract. Inhalable PM (< 10-15 µm in aerodynamic diameter) may be trapped in the nasopharyngeal region, whereas smaller particles can travel further into the tracheobronchial region. The very small (< 1µm) particles can reach and biologically interact with the surface of the 300 million alveoli present in the adult human lungs. At rest, approximately 6 litres of air per minute is moved in and out of the lungs, increasing to 60 litres per minute with exertion. For risk assessment purposes, the assumed volume is 22 m³/day for adults, 15 m³/day for children. The degree of exposure to air pollutants however may depend on current concentrations, prevailing meteorological conditions, temporal and spatial variations, and personal factors such as mobility, rate of exercise, and overall level of respiratory health.

Climate change may affect atmospheric chemistry dynamics, vertical mixing of pollutants, synoptic airflow patterns, and height of the atmospheric layer near the ground that is affected by moisture and heat transfer to and from the Earth’s surface. Whereas the IPCC and CSIRO temperature projections can be used as a basis for an exposure assessment for future heat, there are few similar quantitative projections for air pollution. Some air modelling data are available in Australia however they tend to focus on dispersion modelling of point and diffuse sources of industrial contamination. One relevant study undertaken in the U.S. on the effect of future climate change on air pollution has shown that the severity and duration of summertime regional pollution episodes will increase, accompanied by a 5-10% increase in pollutant concentrations by 2045-2052. It is unknown if these findings would be readily transferable to Australian conditions.
**Ozone**

An assumption can be made with some certainty that tropospheric O$_3$ levels will increase in a warming climate. Being a photochemical oxidant, O$_3$ formation relies mainly on the presence of heat, sunlight and anthropogenic precursor pollutants, all of which are likely to increase in parallel with temperature and population increases. Additionally higher temperatures can elevate emissions of isoprene, an O$_3$ precursor emitted by woody plants.$^{383}$ A modelling study in the U.S. simulated five summers in the 2050s using the IPCC A2 scenario and estimated summer averaged daily 1-hour maximum O$_3$ concentrations would increase from 0.3 to 4.3 ppb.$^{385}$ Several other studies have predicted greater increases using this scenario, together with increases in the number of high O$_3$ days.$^{383}$

In particular, O$_3$ levels in southern Australia may be elevated in conjunction with summer heatwaves which occur as a result of an interruption to the easterly progression of a high pressure system (or anticyclone) causing a change in wind direction to northerly or north easterly.$^{386}$ Airflow along the flanks of anticyclonic systems can transport O$_3$ precursors,$^{383}$ and if the anticyclone is slow moving or stationary successive days of extremely high temperatures may be accompanied by high O$_3$ levels. Although Australian urban airsheds regularly experience short-term episodes of high O$_3$ concentrations, longer episodes can occur and it has been shown that up to 20% of elevated O$_3$ levels remain so for a duration of five hours or longer.$^{375}$

In Adelaide, greatest exposure potential occurs during summer afternoons as confirmed by aircraft observations showing traffic-based emissions moving off shore late morning and the O$_3$-rich plume being transported back into the Adelaide airshed by the afternoon sea breezes$^{56}$ (Figure 9.11). The NEPM goal for O$_3$ is rarely exceeded in Adelaide at present$^{387}$ however this may alter due to climate change and the likelihood of increased photochemical smog production.
**Figure 9.11:** Model of photochemical smog formation over the Adelaide airshed at different times (11.50, 12.50, 14.00 and 15.00 hrs) during a summer day. Model shows morning emissions moved out to the Gulf encroach back over the city and northern areas as O₃ by mid afternoon. Source: Reproduced from EPA. ⁵⁶

**Particulate matter**

A warmer drier climate in areas of Australia will likely lead to an increase in PM as a result of elevated levels of airborne spores, pollens, windblown dust and biomass smoke. ³⁸⁸ Both dust and smoke can travel across large distances, and hence for risk assessment purposes, the focus should not be restricted to local areas. South Australia is the driest state in the nation and there is predicted to be a greater warming trend inland. ³⁵⁰ Most simulation models show future decreases or strong decreases, in rainfall mainly in winter and spring. In the Adelaide Mt Lofty region rainfall is expected to decline by 1-30% by 2030 and by 3-30% by 2070 ³⁵⁰ (Figure 9.12).
These projected changes in precipitation together with temperature-associated increases in evaporation rates, will likely result in a decline in soil moisture content and long-term drying trends over much of southern Australia, and an extension of drought periods. Simulation models show up to 20% more drought months over most of Australia by 2030. Airborne dust resulting from aridity inland travels via northerly winds into the Adelaide airshed on average 8.5 days per year. This number may be expected to increase resulting in elevated exposure to dust-related particulate pollution for Adelaide residents.

A drying trend, together with more storms and very hot days, render vegetated areas and surrounds at risk of bushfires. As well as a predicted lengthening of the bushfire season in Australia, CSIRO predicts the number of days of very high and extreme fire weather risk will increase 4-25% by 2020 and 15-70% by 2050. Estimates by Lucas et al (2007) are higher suggesting increases up to 30% for Australia by 2020 and up to 100% by 2050 for very high fire weather days; whereas extreme fire weather days are predicted to increase up to 65% by 2020 and up to 300% by 2050 depending on the rate of global warming (Table 9.2). Adelaide figures also displayed in Table 9.2 correspond to an estimated 19.2-22.3 days per year of ‘very high or greater’ fire danger rating in 2020 (compared to 18.3 at present), and 19.9-30.2 by 2050; whereas days of extreme fire weather (currently 1.2 days per year) are predicted to increase to 1.4-1.8 by 2020, and 1.4-3.8 by 2050. (Note, in 2009 a new fire danger rating of ‘Catastrophic’
was introduced, which specifies conditions conducive to fires of an unpredictable, uncontrollable, and fast moving nature.\footnote{390}

Table 9.2: Percent changes in the number of days of very high and extreme fire weather using different global warming scenarios (Low and High) for 2020 and 2050, relative to 1990 for Australia and Adelaide. Source: Adapted from Lucas et al.\footnote{389}

NOTE:
This table is included on page 192 of the print copy of the thesis held in the University of Adelaide Library.

The 2009/2010 bushfire season is projected to have above normal bushfire potential for much of south eastern Australia including the Adelaide region and other highly populated areas of SA, as seen in the 2009-10 fire potential map (Figure 9.13) produced by the Bushfire Cooperative Research Centre.\footnote{391} With the higher bushfire potential for the future, residents and local authorities are encouraged to reduce fuel loads around built up areas, and thus days of prescribed burn offs will likely also increase. It is important to note too that the risk of exposure is not limited to the number of days when bushfires are burning locally. Under still conditions poor air quality due to smoke haze may persist for days or weeks after the main fires are extinguished whereas air movement can extend the plume across vast areas. Figure 9.14 (A, B) show smoke trails extending into SA from the fire source hundreds of kilometres away in Victoria. Finally, the National Pollutant Inventory (see Section 9.5.1.4) reports that compared to the previous year, PM$_{10}$ emissions in Australia increased by 15\% in the year 2006-2007. Sources included industrial and diffuse (non-industrial) sources, with the major diffuse source of PM$_{10}$ being burning, comprising 38\% of the total.\footnote{392}

The already considerable contribution that motor vehicles and electricity generation make to the particulate load in Adelaide\footnote{314} will increase with an increasing population.
This and the above projections clearly highlight the increased risk of exposure to PM for Adelaide residents in the coming years.

**Figure 9.13:** Fire potential map 2009-10. *Source:* Reproduced from the Bushfire CRC.\(^{391}\)

**Figure 9.14:** Smoke trails (arrowed) extending into SA, from Victorian bushfires. *A,* February 2009 (*Source:* Earth Snapshot. [http://www.eosnap.com/?tag=victoria](http://www.eosnap.com/?tag=victoria)); *B,* January 2006. *Source:* Reproduced with permission from the Bushfire CRC.\(^{393}\)

**Uncertainties**

This Exposure Assessment has outlined the likely increases in warm weather-associated \(O_3\) and PM air pollution but consideration has not been given thus far to cool season air
pollution. Studies within this thesis have shown higher fine particulate air pollution in the cool season (Chapter 7), and it was hypothesised that this may be due to the abundance of domestic wood fuelled heating sources throughout the city and the Adelaide Hills regions. It is possible that woodsmoke-driven air pollution may decrease in the winter if there are fewer very cold days and nights, but whether this will be outweighed by the emerging threat of increased exposure to bushfire smoke and dust, is uncertain. It is also uncertain if cool season O$_3$ levels in Adelaide will increase, although this is likely in a year-round warming climate.

Another uncertainty is that of individuals’ behaviour change which may affect exposure levels. Exposures to air pollution may increase as warm seasons are extended and individuals open their windows more often at home, or if they spend more time outdoors undertaking leisure or recreational activities. However less time may be spent outdoors during days and nights of extreme heat reducing exposure. Thus uncertainties abound with these exposure assessments, particularly with the lack of accuracy in climate change related air pollution predictions.

**Summary**

The extent to which climate change affects levels of O$_3$ and PM depend in part on ongoing regulatory control by authorities.$^{383}$ Nevertheless this risk assessment predicts there may be more annual exceedances of the NEPM goals and standards for O$_3$ and PM in Adelaide under a climate change scenario, and that the number of these exceedances will increase as global temperatures increase. Furthermore,

- Levels of O$_3$ are likely to increase in line with an increase in anthropogenic precursor pollutants and elevated daytime temperatures.
  - Exceedances are likely to occur on days of extreme heat and for episodes of longer duration than at present.

- Compared to present times, day to day PM levels in summer may increase moderately in the warm season due to a larger population, and power plant emissions related to increased use of air conditioning.
  - These may be offset in winter by consequent reductions in woodsmoke derived PM and reduced heating-associated power-plant emissions due to warmer days and nights.
• There will likely be considerable increases in the number of severe pollution events arising from dust storms and bushfires, with air quality exceeding NEPM targets.
• In the absence of sustainable technologies, PM from traffic-based PM emissions will increase as the population increases.

### 9.4 Risk Characterisation

The Risk Characterisation step uses information from the preceding Hazard Assessment to describe the nature, extent, severity and likelihood of potential adverse health effects for the conditions specified in the Exposure Assessment step. The process can involve a qualitative or quantitative assessment. The latter may give a misleading implication of accuracy and the enHealth Council of Australia states that current risk methods do not enable accurate estimates of risk for low levels of exposure to environmental hazards, due to variability in the population, exposure data, and the environmental agent.

#### 9.4.1 Population projections

The median age of SA’s population in 2007 was 38.9 years, the second highest of any state or territory in Australia. This is expected to increase to 43.8-46.6 years by 2056. Figure 9.15 displays the population pyramids for 2006 and 2056 and in line with national trends, shows a marked increase in the proportion of the very elderly by 2056, particularly very elderly females. The projected figures show each of the younger age categories comprise a smaller proportion of the population compared to 2006 whereas from age category 60-64 and above, all comprise a larger proportion of the population compared to 2006. This increases by far the pool of people vulnerable to environmental stressors in SA. By 2056 the state’s population is projected to increase by 39% to 2.2 million. For Adelaide, models show the population increase to be approximately 38% from the 1.16 million in 2007 to 1.6 million in 2050 (Figure 9.16).
Figure 9.15: The age structure for SA 2006 and 2056. Source: Reproduced from ABS.

Figure 9.16: Adelaide’s projected population to 2056 using 5 different models. Source: Reproduced from ABS.

9.4.2 Temperature-attributable health outcomes

It has been reported that some 1,100 elderly Australians die each year due to the effects of high temperatures. Another report states that up to 1,500 die from extreme heat in Australian cities, a figure which is predicted could double or triple by 2050 as heatwaves increase in frequency, intensity and duration. The temperate Australian cities of Adelaide and Perth which already experience high summer temperatures are likely to experience a considerable impact. Several different approaches have been used by authors to assess disease burdens attributable to climate change. Patz et al use a
comparative risk assessment model as shown in Figure 9.2, where estimates are calculated by multiplying the calculated relative risk by the total burden of disease that would have been expected to occur in the absence of climate change. Woodruff et al\textsuperscript{24} estimated future temperature–related mortality in Australia using the following equation:

\[
A = [\text{TM}_{\text{Hot}} \times \text{Thresh}_{\text{Hot}}] \times M \times P
\]

Where,

- \(A\) = average number of deaths attributable to temperature each year
- \(\text{TM}_{\text{Hot}}\) = percentage increase in deaths for each degree C increase above the hot threshold
- \(\text{Thresh}_{\text{Hot}}\) = frequency of days in the future when the daily maximum temperature is projected to exceed the threshold
- \(M\) = mean annual all-cause mortality during the baseline period
- \(P\) = estimated proportional change in population size, relative to baseline

The authors identified a temperature threshold above which heat-related mortality occurred, the dose-response relationship, future projected maximum temperatures, and predicted change in population size. The possibility of population acclimatisation and adaptation was not accounted for in the model. Calculations for Adelaide were made using a baseline figure of 200\textsuperscript{24} annual temperature-attributable deaths for those aged 65 years and older (based on 1997-1999 figures) and a projected increase in annual maximum temperatures of 1.4-3.9°C. The study estimated temperature-attributable deaths in the city would increase to 265-417 in 2100, depending on the climate scenario and model used.\textsuperscript{24} The key factors in these estimations are clearly the frequency of days above the threshold and increase in population size.

Previously McMichael et al\textsuperscript{3} investigated heat-related deaths using a similar methodology and projected higher estimates (500-700 deaths) by 2050 in the 65 years and over age group. Mid-range estimates from this work are illustrated in Figure 9.17.
Temperature-related deaths will vary considerably between states as seen in recent projections in the Garnaut Climate Change Review\textsuperscript{161} where it was noted that in cooler regions of the nation figures may fall initially due to fewer cold-related deaths, but by the second half of the century these will be exceeded by the increase in heat-related deaths. Figures for SA suggest a baseline figure of 806 and 811 temperature-related deaths in 2030 and 2100 respectively decreasing to 758 and 740 respectively under a hot, dry extreme climate change scenario.\textsuperscript{161} These estimates may not be taking into consideration extreme heat events with unprecedented intensity and duration as seen in recent local heatwaves. Nevertheless it is clear there is a considerable amount of variation and uncertainty in climate change mortality estimates.

9.4.2.1 Heat-attributable ambulance callouts in Adelaide

Within this thesis, several aspects of heatwave-related morbidity in Adelaide have been investigated. Chapter 3 described a heat-associated increase in renal disease, Chapter 4 mental health disorders, Chapter 5 ambulance callouts, and Chapter 6 Emergency Department visits. Whilst time and space limitations do not permit the quantitative calculation of comprehensive morbidity estimates for Adelaide, an example of a suitable methodology that could be used is outlined below. Modelled with variation on that used by Woodruff et al.,\textsuperscript{24} the annual numbers of heat-attributable ambulance
callouts for the present and in 2050 are estimated based on findings from within this thesis:

The annual number of ambulance callouts currently attributable to heat is calculated to be 150 using the following formula:

\[
A = [C \times (T_{\text{Max}} - T_{\text{Hot}})] \times D_t \times P \\
= [2.6 \times (38.0 - 34.6)] \times 17 \times 1 = 150
\]

*Where,*
- \( A \) = average number of ambulance heat-attributable callouts per year
- \( C \) = increase in ambulance callouts per degree C above threshold temperature (see Chapter 5)
- \( T_{\text{Hot}} \) = threshold temperature (see Chapter 5)
- \( T_{\text{Max}} \) = average maximum temperature above threshold (see Chapter 3)
- \( D_t \) = number of days of maximum temperature 35°C or above per year at time \( t \)
- \( P \) = estimated proportional change in population size, relative to the present

From information presented in the Hazard Assessment, Dose-response Assessment and Exposure Assessment steps above, we can make some broad assumptions that by 2050:
- Adelaide’s population will increase by 38%[^394];
- Temperatures will increase 0.2°C per decade over the next two decades.[^157] Conservatively estimating this rate will continue to mid century, \( T_{\text{Max}} \) will equate to 38.8 °C;
- The number of days above 35°C (taking the midrange projections from the averages of the 2030 and 2070 projections)[^352] will approximate 29.5.

*Therefore,*

\[
A = [2.6 \times (38.8 - 34.6)] \times 29.5 \times 1.38 = 445
\]

We can thus make a crude projection that the average number of heat-attributable ambulance callouts will increase from 150 per year at present to approximately 445 per year by 2050.
9.4.3 Air pollution-attributable health outcomes

In a warming climate, hot days with high O₃ concentrations would be expected to occur more frequently, and it follows that occurrences of O₃-associated adverse health effects may also increase. A study in the New York metropolitan region estimated an increase of 4.5% in the number of summer O₃-related deaths in the 2050s due to a climate change associated increase in 1-hour maximum O₃ concentrations of 0.3 to 4.3 ppb.³⁸⁵ It is noteworthy when assessing future impacts that even though it is likely there will be many more days with mildly elevated O₃ concentrations rather than very high concentrations, this will still place an added burden on public health.⁷⁷ Nevertheless the high O₃ days will cause greatest health impacts, and furthermore a synergistic effect may occur with the effect of heat on very high temperature days (see Chapter 8).

There have been very few studies conducted to assess the impact of climate change on PM³⁸³ and its consequent health effects. In fact only one (very recent) publication has been identified thus far. This study, using complex modelling systems predicted using the IPCC A1B scenario, that by 2050 two thirds of the U.S. would experience adverse health effects due to climate-change driven changes to air quality.³⁹⁵ The authors claimed that although there would be spatial variations with both positive and negative changes in annual mean PM₂.₅ concentrations, overall annual premature mortality due to PM₂.₅ would be 15 times higher than that due to O₃. In comparison to 2001, the study predicted 4,000 additional annual premature deaths related to climate change-driven changes in PM₂.₅ and 300 related to O₃, not accounting for population increases.³⁹⁵

9.4.3.1 Bushfire-attributable hospital admissions in Adelaide

Performing a complex quantitative risk assessment for the health impacts of climate change induced changes to air quality in Adelaide is beyond the scope of this study. Nevertheless some projections can be made about bushfire related cardiorespiratory hospital admissions using information ascertained thus far, together with findings from the air pollution studies conducted within this thesis. Making the assumption that on days of extreme fire weather, the increases in respiratory and cardiovascular admissions observed on smoke days compared to the study period overall (Chapter 7) relate to
bushfire smoke-associated health effects, the annual attributable burden of disease for the present and in 2050 can be calculated using the following formula:

$$ A = [H_S - H_{SP}] \times D_t \times P $$

Where,

- $A$ = average number of bushfire smoke-attributable respiratory (or cardiovascular) hospital admissions per year
- $H_S$ = average number of daily respiratory (or cardiovascular) hospital admissions on days of smoke (see Chapter 7)
- $H_{SP}$ = average number of daily respiratory (or cardiovascular) hospital admissions over the study period (see Chapter 7)
- $D_t$ = number of days of extreme fire weather days per year at time $t$
- $P$ = estimated proportional change in population size, relative to the present

Therefore at present,

$$ A_R = [62.0 - 58.8] \times 1.2 \times 1 = 3.8 \text{ annual respiratory admissions} $$
$$ A_C = [79.5 - 72.7] \times 1.2 \times 1 = 8.2 \text{ annual cardiovascular admissions} $$

From information presented in the Hazard Assessment, Dose-response Assessment and Exposure Assessment steps above, we can make some broad assumptions that by 2050:

- Adelaide’s population will increase by 38%\(^{394}\)
- The average number of “extreme” fire weather days per year for Adelaide (currently 1.2) will approximate 2.6 (taking the midpoint of the low and high scenarios).\(^{389}\)

Therefore by 2050,

$$ A_R = [62.0 - 58.8] \times 2.6 \times 1.38 = 11.5 $$
$$ A_C = [79.5 - 72.7] \times 2.6 \times 1.38 = 24.4 $$

We can thus make a crude prediction that by 2050 average annual bushfire-smoke-attributable respiratory and cardiovascular hospital admissions will increase to 11.5 and 24.4 respectively. This conservatively assumes bushfire smoke is only present on days of extreme fire weather, and not those days with fire danger ratings of very high or greater (currently 18.2 increasing to 19.9-30.2 by 2050).\(^{389}\) Furthermore it does not take...
into account Victoria’s increase in potential bushfire days (approximately double that of Adelaide’s) and the possible impact on air quality and cardiorespiratory health in SA.

### 9.4.4 Qualitative approach

Given the lack of precision and numerous uncertainties linked to quantitative climate-change estimates, qualitative assessments may be just as informative. Based on research undertaken within this thesis, Table 9.3 summarises the likely health responses due to a climate change-driven increase in heatwaves and air pollution in Adelaide. Table 9.4 rates the current level of evidence for an increase in indicators of morbidity and mortality associated with more frequent heatwaves, and possible elevated levels of air pollution.

**Table 9.3**: Likely health responses to an increase in heatwaves and air pollution in Adelaide.

<table>
<thead>
<tr>
<th>Health outcomes</th>
<th>Increased heatwaves</th>
<th>Increased air pollution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambulance callouts</td>
<td>Increase in callouts when temperature above 34.6°C. Age 15-64 years most at risk.</td>
<td>Not assessed but the effects on CVD would indicate CVD-related ambulance callouts would increase</td>
</tr>
<tr>
<td>ED visits</td>
<td>Increase overall and in heat related visits for age over 15.</td>
<td>Increase in respiratory and CVD visits</td>
</tr>
<tr>
<td>Hospitals admissions</td>
<td>Increase overall and in heat related visits, renal including ARF, and mental/behavioural.</td>
<td>Increase overall and in CVD visits, especially in cool season</td>
</tr>
<tr>
<td>Mortalities</td>
<td>Increase in heat related, certain mental health conditions. ARF has high mortality rate. Increase in respiratory deaths on high air pollution high temperature days</td>
<td>Increase in CVD and respiratory deaths, particularly on high air pollution high temperature days</td>
</tr>
</tbody>
</table>
Table 9.4: Evidence that an increase in adverse health outcomes in Adelaide will occur in association with an increase in exposure to heatwaves or air pollution.

<table>
<thead>
<tr>
<th>Health outcome</th>
<th>Increased heatwaves</th>
<th>Increased air pollution</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Amb</td>
<td>ED</td>
</tr>
<tr>
<td>All-cause</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>Heat-related</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>Renal</td>
<td>**</td>
<td>***</td>
</tr>
<tr>
<td>Mental</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>**</td>
<td>**</td>
</tr>
<tr>
<td>Respiratory</td>
<td>*</td>
<td>*</td>
</tr>
</tbody>
</table>

* some evidence  ** good evidence  *** very good evidence

* Limited data are available for ambulance usage in this context, however an assumption is made from the strong evidence associating air pollution to cardiorespiratory ill health, that a link with ambulance transports is likely.

Amb: ambulance callouts; ED: emergency department visits; Hospital: hospital admissions

9.4.5 Uncertainties and limitations

An assessment process which extrapolates previous environment-health associations to future climate change scenarios lies within a framework of uncertainty, as climate variation and its impacts may vary considerably from that predicted.\(^\text{295}\) Indications are that CO\(_2\) emissions are increasing at a rate faster than expected\(^\text{396}\) and that IPCC worst case scenarios are currently being realised or exceeded.\(^\text{158}\) Some risk assessments undertaken previously may thus prove conservative in their estimates of health effects. The extent of behavioural, technological and demographic change and speed of mitigation activities in the future are unknown as is the extent to which these changes may affect CO\(_2\) concentrations.\(^\text{24}\)

This section has focussed on the likely acute health effects of environmental stressors on the population of Adelaide assuming causal pathways, and although climate change health risk assessments often incorporate several different projection scenarios\(^\text{3}\) only one is presented here. Longer term health effects which have a much greater effect on population health\(^\text{397}\) have not been taken into consideration although it is recognised that systemic toxicity and carcinogenesis have been linked with long term exposures to some pollutants.\(^\text{358}\) Pollen and aeroallergen specific health effects have also not been
considered here. Also unaccounted for is the degree to which societal infrastructure may change or the degree to which the population will adapt\textsuperscript{295,310} or acclimatise to a variation in climate. A further limitation of this risk assessment is the omission of a discussion regarding potential costs to the health care system.

A final uncertainty relates to temporal changes to the baseline health of Adelaide’s population. It is known that those who are ill or who have chronic conditions are more likely to experience adverse health outcomes as a result of exposure to environmental stressors. With the ageing population chronic disease rates and thus overall population susceptibility is likely to increase to an unknown degree.

\textbf{9.4.6 Summary}

Heat- and air pollution-related rates of cardiorespiratory morbidity and mortality may thus increase, decrease, or change little over time. Models have shown increases in heat-related deaths in Adelaide, but an overall decline in temperature-attributable deaths, possibly due to a fall in cold-related deaths. Furthermore in the absence of climatological and epidemiological modelling expertise, this risk assessment has been unable to characterise with confidence the extent to which PM and O\textsubscript{3} levels will increase with warmer, drier conditions, and the consequent effects on vulnerable populations. It is known however that extreme heat and air pollution adversely affect the health of the vulnerable including the elderly, the disadvantaged and the chronically ill and that this proportion of the population is increasing annually.\textsuperscript{388}

Climate change offers many challenges to public health in Australia and the complex possible causal pathways to increased morbidity and mortality in southern Australia are diagrammatically represented in Figure 9.18. This shows that an increased frequency of heatwaves or heat extremes can directly affect health via heat illnesses, mental health issues, bushfires or poor air quality; or indirectly via drought and its influence on bushfire risk, ongoing mental health, water quality and insecurity, and air pollution.
9.5 Risk Management, Risk Communication and Public Perception

This the final step of the risk assessment process, defines and evaluates the options, makes and acts upon informed decisions and monitors their effectiveness. Communication to the public plays a vital role in managing risks associated with environmental health, and this is outlined with some recent examples. Public perceptions of climate change are also discussed.
9.5.1 Risk Management

Climate change risks can be addressed in a number of ways including mitigation strategies to curb the increase in greenhouse gas emissions. In 2004, Australia’s per capita CO₂ emissions were 4.5 times that of the global average indicating the need for reductionist strategies. The SA Government’s Strategic Plan 2007 aims as a major priority to reduce the state’s greenhouse gas emissions by 60% by 2050 and to have 20% of the state’s electricity in the form of renewable energy by 2014.

Adaptation will be enable populations, communities and individuals to deal with the health challenges of a changing climate. This will involve increasing resilience, defined as the capacity to cope with or recover from climate change induced events. Vulnerability to a hazard involves both reducing susceptibility and increasing human resilience. Ways of increasing resilience include reducing exposure using primary prevention methods, increasing public health preparedness, and formulating response activities encompassed within a risk management framework. Adaptive capacities can be strengthened in this way to reduce the health impacts of, and vulnerability to, climate change events such as heatwaves, drought, and bushfires. Adaptation is generally not such an effective risk management strategy in terms of air pollution however, due to physiologic mechanisms limiting the ability to decrease susceptibility to poor air quality, although technological adaptations to limit emissions should be pursued.

9.5.1.1 Behaviour change

Adaptation and mitigation at an individual level requires a degree of behaviour change. The psychological processes necessary for change to occur have been outlined in the “transtheoretical model of behavior change”, the six steps of which include:

- pre-contemplation (*action not intended in the foreseeable future*)
- contemplation (*action intended in the next 6 months*)
- preparation (*individual intends to take action within the next month*)
- action (*modifications made in behaviour within the preceding 6 months*)
- maintenance (*individual is working to prevent relapse and sustain changes*)
- termination (*no temptation to revert to previous behaviour*)
Undertaking voluntary mitigation behaviours involves these processes as discussed by Semenza et al., who incorporated the transtheoretical model in their analysis. It was found those who engaged in behaviour change were more likely to be younger rather than older, have a high level of education, and an increased level of concern about climate change. The authors suggested appropriate education messages may be required to overcome social and structural barriers to behaviour change, thus encouraging wider engagement with adaptation and mitigation interventions. An understanding of the psychosocial aspects underlying health behaviour change may be useful when formulating and communicating risk management strategies.

### 9.5.1.2 The Precautionary Principle

The Precautionary Principle has been emerging in recent years in the context of environmental policy and is particularly relevant in climate change risk assessments. First raised at the 1998 Wingspread Conference, the precautionary principle states that:

> “Where an activity raises threats of harm to the environment or human health, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.”

The premise of the Precautionary Principle is that uncertainty is not a reason to postpone or avoid action. The relevance arises when there is scientific and evidential uncertainty as there is with predicted exposures and health responses to climate change. In this instance these emerging threats to public health cannot await full scientific certainty and for risk management, it is important to take steps and make predictions which err on the side of caution.

### 9.5.1.3 Risk management for heat extremes

A warming climate in combination with an ageing population heightens the need for risk management to limit heat exposures, increase resilience in the community and thus decrease vulnerability. Much was learnt from the experience of the 2009 Adelaide heatwave including the need to act promptly, as there was a delay of several days before emergency management was underway. In recognition of the emerging threat to health that unprecedented heat poses, a cross-agency coordinated, structured ‘Extreme Heat Arrangements’ plan has recently being formulated by policymakers and the State
Emergency Service (SES) that allows government departments, hospitals and emergency services (e.g. fire, ambulance, SES) to prepare for an increased workload when prolonged and extreme heat is imminent. A controversy during the aforementioned heatwave centred around the failure of the power grid resulting in load shedding and unforced power outages. The Extreme Weather Hazard Plan should enable authorities to better prepare for surges in power demands and if load shedding is unavoidable, residents hopefully could be informed of times and locations to enable alternative cooling arrangements. Ideally residents could prepare in advance for forthcoming days when activities and behaviours may need to be modified to limit heat exposure.

Several intervention activities used in heatwaves overseas (including media announcements, promotion of a ‘buddy system’ to encourage friends, neighbours and relatives to be mindful of susceptible individuals, a telephone help line for health advice, home visits, and increased emergency medical service staffing) were used in Adelaide 2009. Additionally a telephone service operated by Australian Red Cross volunteers in Adelaide, called registered frail, elderly or isolated people up to three times per day to check on their well being during the heat. Despite these interventions, increased community engagement, and coordination between government and non-government organisations during the crisis, there were numerous preventable deaths caused by this heatwave (Nitschke et al, unpublished report) indicating a shortfall in public health or health awareness campaigns at some level.

Long term risk management strategies should avoid sole reliance on air conditioner usage which increases greenhouse gas emissions via electricity production, and generates waste heat. Changes to building design will need to be considered; as well as support for preventive measures such as enhancing community resilience and long term adaptive capacity. Whilst increasing green space has been found to decrease the risk of heat illness in a community, this is compromised in Australia and particularly in SA, by climate change–induced water insecurity.
9.5.1.4 Risk management for air pollution

Setting air pollution standards and goals to protect human health and the environment requires a systematic risk assessment process to establish individual NEPMs for each pollutant. Measuring daily concentrations of pollutants allows surveillance of air quality, and the identification of exceedances of NEPM standards. The EPA has been conducting monitoring in this state since the 1970s\textsuperscript{211} and comparisons of air quality measures are made with the NEPM standards and goals that are outlined in Table 9.5. Note that for PM\textsubscript{2.5} an advisory reporting standard is currently in place.

The National Pollutant Inventory (NPI) is a pollutant release register which records in each airshed, pollutant emissions from industrial facilities, and non-industrial activities such as transportation, bushfires and households.\textsuperscript{392} Part of the purpose of the NPI is to “promote the need for cleaner production and waste minimisation technologies in programs in industry, government and the community”.\textsuperscript{392} The 74 substances listed for Adelaide in the 2007-2008 reporting year, by substance, total emissions and emission source, include PM\textsubscript{10} and PM\textsubscript{2.5}\textsuperscript{314} but exclude O\textsubscript{3}, a secondary pollutant.

\begin{table}[h]
\centering
\caption{NEPM standards and goals for O\textsubscript{3} and PM\textsubscript{10} and advisory reporting standard for PM\textsubscript{2.5}. Source: Dougherty.\textsuperscript{100}}
\end{table}

In terms of strategies to minimise O\textsubscript{3} production, efforts to restrict precursor emissions particularly those from motor vehicles, are useful. Innovative technologies, less reliance on fossil fuels and promoting greater use of public transport will be important future
considerations. Regarding PM exposure, little can be done to curb hot dry, windy days that generate dust and present high fire risk, and these days are likely to become more frequent. Nevertheless reducing fuel loads pre-season will lower the risk of major bushfires and increasing penalties for arsonists may prove a deterrent for offenders who deliberately light fires. A ‘polluter-pays’ approach may help to lower emissions from sources such as domestic wood heaters and diesel engines, encouraging users to seek cleaner alternatives. The State Government’s goals to improve energy efficiency and increase development of renewable energy sources as outlined in SA's Strategic Plan 2007 will assist in reducing Adelaide’s PM$_{2.5}$ load, 49.7% of which is due to electricity generation. Finally, limiting personal exposure can involve restricting outdoor activities when air quality is poor as recommended by the NSW Health website which provides useful advice for the public and groups sensitive to O$_3$, PM and other pollutants.

9.5.2 Risk Communication

9.5.2.1 Risk Communication for heat extremes

Risk communication for heat-health awareness is crucial during times of extended extreme heat and has the potential to save lives. It is possible Adelaide residents are somewhat complacent in this regard as heatwaves occur with regularity each summer. In a warming climate however where heat extremes are likely to reach previously unrecorded levels, the population may be as much if not more at risk than those where heat is a rare occurrence. With heat-related illnesses being preventable, it is imperative that the public are aware than heat can kill and protective strategies are necessary. For the vulnerable, minimising barriers to adaptation is important. These barriers may include concerns about air conditioner related energy costs, not opening doors and windows due to security concerns or not drinking adequate fluids due to mobility restrictions.

The 2009 Adelaide heatwave became an emergency situation after the first few days of unabated daytime and nighttime heat. The State Premier called emergency meetings daily, and risk communication strategies were actioned. These took the form of a public
health alert with information for doctors and a heat-health warning for the public (Figure 9.19) which was distributed via the media, internet, and a mobile telephone network.

Heat-health warnings are in place in many other cities around the world, particularly since the 2003 European heatwave disaster. In Philadelphia, Pennsylvania, a Hot Weather Health Watch Warning System was first reported in 1996 and takes the form of a three-tier structure. A health watch is issued if an oppressive air mass is imminent; a health alert and a subsequent health warning is issued as the heatwave intensifies. In September 2009, the SA Government and the State Emergency Service finalised a four-stage “SA Government Extreme Heat Communications Plan” incorporating an Extreme Heat Advice, Heat Health Watch and Heat Health Warning. The plan was implemented just two months later with the onset of a pre-summer heatwave in November.

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**SA SES Heat Health Warning**

The State Emergency Service (SES) and SA Health are warning South Australians that the current heatwave is a threat to public safety. There have been significant SA Ambulance call outs and an increase in people presenting to Adelaide metropolitan hospitals, some suffering from heat associated illness. Heat related illness can result in severe health issues and can be fatal.

The SES and SA Health urge the public to exercise extreme care during the heatwave and to take the following precautions:

- Make contact with elderly relatives, friends and neighbours.
- Drink plenty of water and avoid alcoholic and caffeinated drinks.
- Be aware of the symptoms of heat stress.
- Stay indoors and close curtains during the day. Open up your home at night if cooler winds occur.
- Use air-conditioners and fans wherever possible or visit public facilities such as shopping centres, cinemas and libraries.
- Take cold baths or showers and use ice packs or wet towels to cool down.
- Remember that cordless telephones do not work during power outages. Make sure you have alternative means of communication.
- Limit outdoor activities to mornings and evenings.
- Consider the safety of your pets and animals. Wet them down and ensure they have adequate shade and water.

Stay tuned to this radio station on a battery powered radio for more information.

For up to date warnings and further information on the weather forecast please visit the Bureau of Meteorology’s website at [www.bom.gov.au](http://www.bom.gov.au).

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9.5.2.2 Risk Communication for air pollution

From a national perspective, the NPI gathers information on a geographical basis about emissions of polluting substances in airsheds around Australia (including Adelaide) and makes this information available to the community to meet their “community right-to-know obligations”.392

At a state level, risk communication relating to air quality appears to be less well established in SA than in other Australian states, the exception being the Asthma Foundation of SA’s daily pollen count available on their website and broadcast via the media. Whilst details of current air quality are available on the EPA website, health alerts are not. It was suggested previously as part of a 2005 review of SA’s Ambient Air Quality Monitoring Program, that hospitals and persons with respiratory diseases would benefit from having next day AQI forecasts available for Adelaide; that explanations of air quality could be offered to the public on days when there are significant levels of haze; that email alerts of pollution events could be issued to local authorities; and that the media could broadcast alerts, particularly in association with winter woodsmoke.387 However few of these initiatives seem to have been implemented. Interstate examples of risk communication include email alerts of pollution events in NSW and Victoria,387 ‘Don’t light tonight’ alerts relating to woodsmoke pollution in NSW,411 and NSW Health’s online advice to sensitive persons and the general public for each pollutant and air quality category.409 In Victoria, the EPA website lists cautionary advice for the public including sensitive subgroups, relating to bushfire smoke at low and high level bushfire smoke alerts (Table 9.6). This risk assessment recommends similar initiatives be implemented in SA.

9.5.3 Public perceptions

Australians are understandably concerned about the uncertainty of future environmental issues associated with climate change. In 2008, a survey of 1,000 people conducted by the Climate Institute showed that 92% were concerned about more frequent and intense droughts, and 86% were concerned about more frequent and intense bushfires.412 A larger survey of 15,800 dwellings across Australia conducted by the ABS in 2007-2008 found 73% of respondents were concerned about climate change, the proportion varying between Australian states.413 Figure 9.20 illustrates that the proportion of respondents showing concern in SA ranked second highest in Australia. This concern may manifest as psychological stress in some individuals, adding to the mental health burden of climate change (see Section 10.7.1.11).
9.5.3.1 Public perceptions of climate change induced health impacts

A public opinion poll conducted in 2007 found 45% of Australians believed environmental problems such as climate change had affected their health.\textsuperscript{159} With the elderly being particularly vulnerable to the health effects of a global warming, it is of interest that only 30% of Australians aged 65 years and over expressed concern about climate change.\textsuperscript{413} A recent study in England conducted to ascertain perceptions of heatwave risks, found that few of the elderly respondents aged 72-94 years, perceived themselves to be either old or susceptible to the effects of heat.\textsuperscript{311} The study questioned the notion of targeted public health messages to the elderly, suggesting instead a whole population approach would be inclusive of those who consider themselves not to be at risk. That the elderly of Australia may have similar views could be a consideration when formulating age-appropriate public health messages.

9.6 Summary

Although well based on epidemiological evidence, predictions of the public health impacts of climate change in Adelaide and elsewhere are largely speculative, and there is a pressing need for ongoing systematic research in this area. It is unknown to what extent individuals will adapt or acclimatise to changing environmental conditions, or
the efficacy of greenhouse gas mitigation strategies. Nevertheless based on considerable international and local evidence ascertained thus far, it is highly likely:

- Average temperatures will rise in accordance with climate change
- Annually there will be more days with temperatures of 35°C or above
- The number of heatwaves will increase
- The annual number of days with high O₃ concentrations will increase
- The annual number of days with bushfire smoke in the air will increase
- Population growth will continue
- The average age of the population will increase

Furthermore it is very likely:

- The annual number of days with dust in the air will increase
- Annual average PM concentrations will increase
- The proportion of the population in Adelaide susceptible to the negative health effects of environmental stressors will increase

For the health sector (assuming valid causal pathways), these projections will more than likely lead to disproportionate increases in:

- The incidence of heat-related illnesses
- Ambulance callouts during heatwaves and days of poor air quality
- Hospital admissions during heatwaves and days of poor air quality, particularly during the bushfire season
- ED visits during heatwaves and days of poor air quality
- Renal disease hospital admissions during heatwaves
- Mental health morbidity and mortality during heatwaves
- Cardiorespiratory disease on days of poor air quality

and,

- A reduction in PM-induced cardiovascular disease during the cool season

Mortality due to the following is likely to:

- Increase for heat-related causes (including heat-stroke and its complications such as acute renal failure)
• Increase for heat-exacerbated conditions (such as heart disease)
• Decrease for cold-attributable causes (including cardiorespiratory diseases)
• Increase for air pollution-triggered cardiorespiratory disease

This risk assessment has identified several areas where these emerging threats can be addressed. Recommendations are therefore that:

• Mitigation strategies be upscaled to reduce the state’s greenhouse gas emissions
• There be adequate communication to the public of the risks posed by heat and air pollution prior and during days of high risk
• A full-scale Heat Alert system involving government departments, non-government organisations, the health and emergency services sectors, and industry be implemented
• Efforts continue to maintain and improve ambient air quality
• Fuel-reduction measures be undertaken prior to the commencement of the bushfire season
• Age- and culturally-appropriate health promotion messages be conveyed to vulnerable sub-groups promoting adaptive and preventive strategies that may involve relevant behaviour change
• Means of enhancing community and individual resilience be pursued to reduce vulnerability to environmental stressors
• Horizon scanning for new and emerging health threats as a result of climate change be undertaken
• There be appropriate resource allocation for personnel, infrastructure and resources to cope with increased demands associated with the consequences of climate change.
Chapter 10

General Discussion

10.1 Introduction

This chapter concludes the thesis with a general discussion concerning the study as a whole. Key findings are addressed as well as strengths and limitations of the study, challenges faced along the way and significance of the study. Policy implications and recommendations are discussed and several options for further research into the health impacts of extreme heat and air pollution are suggested. The chapter closes with a brief conclusion.

10.2 Key findings of the study

The findings in this study indicate that exposures to high temperatures and air pollution have a measurable effect on population health in Adelaide. Although the city’s inhabitants may be acclimatised to hot dry summers, morbidity nevertheless increases during heatwaves. Mortality increases appear to be restricted to periods of very extreme heat as occurred during the 2009 heatwave (Nitschke et al, unpublished report). Despite Adelaide’s comparatively good air quality, air pollution in the form of PM is detrimental to cardiovascular health, particularly in the cool season. The fine component of PM was found to pose the greatest risk due to its ability to penetrate further into the human respiratory tract.
10.2.1 Heat-health studies

Much of the work presented here has stemmed from initial studies undertaken by Nitschke et al.\textsuperscript{31} concerning the effect of heatwaves on morbidity and mortality in Adelaide. This benchmark study, often referred to in this thesis, paved the way for the subsequent heat-health studies.

Renal hospitalisations were investigated (see Chapter 3) and it was found that increased admissions during extreme heat were due in part to acute renal failure, possibly heat-related. An investigation of comorbidities associated with renal admissions indicated a link with reported coexisting ‘effects of heat and light’, indicating a possible causal pathway between heat exposure and kidney dysfunction. Similarly, findings in the initial study\textsuperscript{31} suggesting extreme heat posed a risk to persons with mental ill health, were confirmed in a subsequent study within this thesis. Subcategories of mental and behavioural disorders were investigated and several were identified for which hospitalisations and mortality increased during heatwaves. Discussed in Chapter 4 was the possibility that either these individuals were more susceptible to heat due to their psychiatric/cognitive condition affecting behavioural thermoregulation, their prescribed medications affecting thermophysiology, or that heat was linked to the onset of a (new or pre-existing) mental condition.

An interest in spatial epidemiology prompted an investigation of place-based risk associated with ambulance usage during heatwaves. Findings showed certain areas of Adelaide including the CBD and certain industrial and lower socio-economic areas appeared to be more at risk of ambulance attendance during heatwaves. There was spatial heterogeneity in cause-specific effects across the metropolitan area although it was noted that in some industrial suburbs work-related callouts increased, prompting the need for further research into occupational heat stress (to be discussed below). A spatial investigation of ED visits during heatwaves showed some areas of increased heat susceptibility in common with the ambulance study although once again considerable heterogeneity existed across the metropolitan region. A noteworthy difference in study design was that ED visits were mapped by residential postcode whereas postcode of ambulance attendance was used in the latter study.
10.2.2 Air pollution studies

Air quality in Adelaide compares favourably with that in other Australian and international cities (Figure 10.1). Nevertheless adverse health effects appear to be triggered by increases in even low levels of air pollution, lending support to the notion of a no threshold approach to the association of air pollution with health. Findings in this study (Chapter 7) showed a rise in airborne fine particulates may be linked to an increase in cardiovascular hospitalisations, particularly in the cool season. Whilst some of the estimates were higher than those of other studies in Australia and elsewhere, there are no other Adelaide studies with which to compare findings. Respiratory hospitalisations did not increase significantly overall in this study, perhaps as these are indicative of severe illness only and thus may not be the best indicator of respiratory morbidity. The final study showed that all pollutants investigated had an effect on health, and days of high temperatures and high O$_3$ concentrations could be particularly hazardous with evidence of synergistic interaction.

10.2.3 Risk assessment

A comprehensive environmental health risk assessment for heat and air pollution exposure in Adelaide under a changing climate was undertaken as detailed in Chapter 9. Findings showed that in the absence of acclimatisation and/or adaptation, by 2050 climate change driven heat-related morbidity is likely to increase with noticeable impacts on the health sector. Heat-triggered hospitalisations for mental ill-health and renal disease are likely to disproportionately increase as will ambulance callouts during the increasing number of very hot days. Much uncertainty surrounds future levels of air pollution and consequent health effects, although it is likely severe pollution events (e.g. days with high O$_3$ levels, airborne smoke or dust) may increase due to the effects of a warmer climate on aridity and fire risk. This may be offset in the cooler months by lower levels of particulate pollution and an improvement in overall cardiovascular health.

Risk communication to the public in the form of heat-health warnings and air pollution alerts was seen to be increasingly important for days when heat or air pollution
exposure may jeopardise health. The need for mitigation strategies to substantially lower greenhouse gas emissions was reinforced. Finally, it was noted that an increase in adaptive strategies (such as changes in behaviour, building design, etc.) and community resilience may reduce vulnerability and help to minimise health impacts. Whilst Adelaide focussed, the risk assessment has implications on a much broader scale.

**Figure 10.1:** How Adelaide’s A, ozone and B, PM$_{2.5}$ levels compare with other cities around the world. *Source:* Reproduced with permission from Ontario Ministry of the Environment.$^{414}$
10.3 Strengths and limitations of the study

10.3.1 Strengths

A major strength of this study is that it is unique in several aspects. Firstly, no previous study has investigated in detail heat-associated renal disease and its comorbidities, nor undertaken syndromic surveillance of heat-triggered mental and behavioural disorders. As a consequence, manuscripts of these studies were accepted for publication in prestigious international journals. The spatial analyses of ambulance callouts and emergency department visits during heatwaves in Adelaide are also unique having not previously been undertaken in this state. These studies may make a valuable contribution to local and international heat-health knowledge and have implications for policy development and practical interventions.

Until now, there has been a knowledge deficit regarding the effect of air pollution on population health in Adelaide, despite studies having been undertaken in most other Australian capital cities. This study may therefore be of great interest to policymakers, strategists and public health professionals. The long awaited findings may also be useful in formulating a PM$_{2.5}$ Air NEPM for Australia to replace the current advisory reporting standard. With investigations into potential interactions between air pollutants and temperature rarely attempted elsewhere, the techniques and findings presented herein may prove useful groundwork upon which to base further interaction studies. These issues are expanded upon in Section 10.5.

10.3.2 Limitations

10.3.2.1 Statistical limitations

In the studies undertaken within this thesis, sample sizes were often small. This occurs as a result of investigating rare health events in a population the size of Adelaide’s (1.16 million people as at June 2007). Statistical rigour is decreased further when, to ascertain relevant subpopulations at risk, data are stratified into gender, age or postcode categories. Many studies use a single category for the elderly, being those aged 65 years
and over, whereas in many analyses in this theses, the categories of 65-74 years and 75 years and over were used. In retrospect, combining the age groups together in some studies may have proved a better strategy to avoid statistical dilemmas and spurious results.

Another statistical limitation is that in several of the studies within this thesis, many statistical models were run from which a relatively small proportion yielded statistically significant results at the 5% level. In this situation the possibility of spurious associations exists and raises the issue of the ‘multiple comparison fallacy’ where there is a greater than 5% risk of a Type I error occurring.415 However by not correcting the significance level for multiple comparisons, the likelihood of missing important associations was avoided.85

Whilst spatial analysis can be undertaken using complex methodologies, the approach used in this study was quite basic and could be seen as a limitation. As well as the previously discussed issue of discrepancies between postcodes and postal areas (Chapter 4), spatial autocorrelation (i.e. that near things will likely share more similar values than distant ones)164 was not taken into account and thus maps could possibly be visually misleading to the reader. Some studies elsewhere have employed Bayesian frameworks in disease-mapping studies, where models based on conditional autoregressions are implemented using software packages such as WinBUGS.416,417 Although this was an option here, the required level of complexity involved was considered unnecessary within the scope of this study.

10.3.2.2 Heatwave studies

In the study on renal hospitalisations during heatwaves, the chosen method of identifying comorbidities (i.e. the secondary diagnosis of those admitted with renal disease) was possibly too restrictive to adequately identify persons at risk. A preferred method may be one used by Knowlton et al29 who combined the primary and first nine secondary diagnoses to identify comorbidities. In the present study if any of the comorbidities under investigation were not listed as the primary diagnosis, the data would not have been captured. Additionally it may have been advisable, given the biological plausibility and clinical relationship with heat-related renal disease, to
include ‘volume depletion’ (dehydration) as one of the comorbidities, or include it in the classification of ‘effects of heat’ as in later study (Chapter 6). Options for further research into thermal impacts on kidney function are discussed in Section 10.7.1.7.

Disease diagnosis in itself can also be a limitation of these studies as miscoding is not unusual. It has been reported elsewhere that up to 15-20% of diagnoses are inaccurately coded. Hospital protocols and methods of disease coding vary across the world and between cities, as can the reporting and retrieval of hospital data. Admissions may be influenced by certain political agendas, and temporal trends in hospital funding and availability of beds. Levels of access to health care will also vary across and within populations as will the underlying health status of different populations.

Another limitation is that personal exposure levels are unknown and, as in most such ecological studies, an assumption is made that during heatwaves the whole population is exposed to heat, although this may not be the case for individuals who spend their time in a cooled environment. A final limitation of the heat-health studies is that lag periods were not adequately investigated and therefore admissions in the days following heatwaves due to late onset heat-related conditions or subsequent health deterioration were not identified as being associated with heatwaves.

10.3.2.3 Air pollution studies

Limitations in the air pollution studies are mainly twofold and relate to possible instrument error and (as in the heatwave studies) misclassification of exposure. The limitations in accuracy of the TEOM samplers used for measuring PM concentrations have been discussed briefly in Chapter 7. In short, the inlet air stream in a TEOM is heated to 35°C to 50°C and volatilisation of semi-volatile organic material in the inlet can cause low readings. Additionally, particles which adhere to the side of the cyclonic inlet are not measured (Mr. R. Mitchell, EPA, pers. comm. 22/8/08) and particles cannot be accurately differentiated by size. A CSIRO study found a discrepancy of 30% between PM$_{2.5}$ measurements using two types of samplers. Hence concentrations are approximations only. The EPA state that measurements using TEOM samplers should not be compared to advisory reporting standards until sampling methods are verified and formally included in the Air NEPM.
For practical reasons, only one centrally-sited monitoring station was used to source air quality information. This was the only site where all the required pollutants were consistently measured and was close to the Adelaide airport where visibility was measured. It is recognised there are spatial and temporal variations in air pollution concentrations across the urban sprawl, although there would be less intra-urban variation expected in fine particle concentrations as these tend to be more evenly distributed than PM$_{10}$. Furthermore central monitoring data may have uncertain relationships with exposures in the study community. Personal exposures depend on time spent outdoors and ambient pollutant concentrations during that time, as well as time spent indoors and indoor concentrations, which cannot be accounted for in ecological studies. Misclassification of exposure is therefore a well recognised limitation in air pollution studies and the most likely source of substantial bias.

Additionally, as suggested elsewhere, the shape of the exposure response curve in the air pollution–health relationship may not be strictly linear as the slope may be greater at the lower concentrations. Thus small increases in pollutant levels in an environment of relatively good air quality may be just as harmful as large increases in a more polluted environment. Finally, individual levels of exposure that cannot be measured in ecological studies will vary geographically according to local meteorological conditions, as well as human lifestyle, behaviour and prevalence of tobacco smoking. Researchers nevertheless continue to compare health outcome findings in environmental health studies, with little reference to these factors.

### 10.4 Challenges faced in the study

Many challenges were faced during the course of this candidature, mainly concerned with data issues and statistical methodologies. Generally these were overcome with extensive research and consultations with experts.
10.4.1 Data

10.4.1.1 Definition and acquisition of data

Prior to the commencement of any of the studies within this thesis, many decisions had to be made regarding the data required for analysis. These included defining study periods and ICD codes (versions 9 and 10) for differential diagnoses. The latter was not straightforward as on close examination there is a great deal of discrepancy in the definition of disease classifications within the literature, an important issue rarely raised when findings across studies are compared. Contact was therefore made with the Director of the National Centre for Health Information Research & Training (Dr. Sue Walker) at the Queensland University of Technology for clarification and advice.

Most of the data for the heatwave studies were obtained through the Health Statistics Unit at the SA Department of Health, but another source was initially used for the air pollution studies and several attempts were made to get the correct dataset. The first dataset received was dated by separation date, not admission date, a major detail fortunately noticed by accident. The results using this dataset would have been meaningless as the focus was on exposure prior to admission, not discharge. Second was the realisation that data were from across the state not just Adelaide. Third, it was realised that data should be restricted to residents of, and hospitals within, the Adelaide metropolitan area. It was decided to exclude individuals who resided outside of the city limits and were admitted to metropolitan hospitals, and city residents who were admitted to hospitals outside of the metropolitan area. There were also several ways of defining the ‘metropolitan area’ which included by postcode, local government areas, statistical local areas, or statistical division, each with some discrepancies.

Decisions also had to be made about the most appropriate air monitoring site(s) to best represent the conditions across the urban area, and the air pollutants to use in the analysis. On advice, it was eventually decided to exclude the pollutants for which Adelaide measures are low and vary little (e.g. carbon monoxide and sulphur dioxide) even though they feature in some other Australian studies. Once chosen, much research was undertaken together with discussions with experts, to determine the most informative pollutant measures to use (e.g. 1-hour, 4-hour, 8-hour or 24-hour averages...
or maxima) as these vary considerably in the literature. An additional problem was that reliable air monitoring data were available unavailable for some years, thus limiting the study period.

Temperature data were obtained from the Bureau of Meteorology (SA), although temperatures were also measured at the air monitoring site, with values in the two sources differing somewhat. With missing values in the latter dataset, it was decided to use the Bureau of Meteorology data. Furthermore there were several different temperature metrics which could have been used - maximum daily temperature, mean temperature, or apparent temperature (which factors in humidity), with no consensus on this in the literature. Daily maximum temperature was used in the heat studies as it is indicative of extremes, whereas mean temperature was used in the air pollution studies.85

10.4.1.2 Quality of data

This thesis has raised awareness of the limitations in data collection, data analysis and reporting of results, particularly with morbidity data. Statistical analysis will be totally misleading if the original source data are inadvertently flawed. External factors can influence the coding of diseases, including current clinical, political or funding trends, as well as human error, introducing the opportunity for selection bias. Diagnoses on admission may be quite different to discharge diagnoses, yet it is the latter which is generally used in analysis of hospital data with inference regarding acute health effects of pre-admission exposure. The use of a single principal diagnosis (for cause of death or discharge) is probably therefore unwise and secondary diagnoses should be considered when undertaking epidemiological surveillance studies. Additionally, total hospital admissions include booked and emergency admissions, but the use of the latter only may be more appropriate when investigating acute responses to environmental exposure. Finally, the importance of ensuring data are clean before use (no matter how credible the source), has been realised.
10.4.2 Methodology

Learning the relevant statistical techniques and software packages involved in this study was a particular challenge. Complex data management and analysis using Stata, and spatial techniques involving ArcView (ArcMap) were difficult to master.

10.4.2.1 Overdispersion

As explained in Section 2.5.2, case series design was chosen as the most appropriate methodology for the heat-health studies. Overdispersion was not taken into account in the original method and upon this realisation, analyses from the early studies had to be redone. All Poisson regression models were re-run and a goodness of fit test applied to each. Negative binomial regression models were fitted when overdispersion was detected, a process requiring many extra hours of work.

10.4.2.2 Case crossover analysis

Considerable thought was given to the most appropriate methodology for the pollution studies. The trend in recent times has been to use case crossover analysis in favour of time series analysis traditionally used in these studies, and following discussions with Dr. Adrian Barnett, (Queensland University of Technology) author of several Australian air pollution studies, this became the preferred approach. A time-stratified case-crossover design was decided upon incorporating a 28-day strata period with a two day exclusion window either side of the index day. The programming code for the complex data set up required for this methodology, was made available with permission, via Dr. Barnett’s website. As this was written for SAS not Stata software, Mr. Thomas Sullivan (Data Management and Analysis Centre, University of Adelaide) kindly offered to run the program in SAS, and convert the final dataset into Stata format for analysis.

Understanding the data management required for a case-crossover analysis was difficult as details are scarce in the literature and the terminology confusing. The terms “case” (index) and “control” for instance, refer to days in the strata, not individuals as in case-control studies. Details of the required data formatting appear in Tables 2.1 and 2.2.
(Chapter 2), and to the author’s knowledge this level of detail has not been published previously.

Defining the statistical model was challenging as the chosen covariates of other air pollutants, temperature, day of the week, school holidays, public holidays, and day after public holidays, each required substantial data management prior to inclusion in the models. Re-analysis was also required as it was realised that due to autocorrelation PM$_{10}$ and PM$_{2.5}$ could not be included in the same model, just as maximum and minimum temperatures could not. Furthermore coefficients required conversion to represent increments of 10 µg/m$^3$ in PM in accordance with other published studies.

10.4.2.3 Risk of errors

With methodologies sensitive to even minor changes, the realisation of the number of steps and potential errors that can be made along the statistical journey from study conceptualisation to publication of findings has enhanced the author’s ability to critically evaluate and question other published studies. The possibility of errors in the original datasets also exists as clerical and other errors can be introduced at every stage of data collection and processing.$^{47}$

The higher effects estimates obtained in the air pollution study compared to similar studies were initially cause for concern. However there could be several possible reasons for this, the first of which was the fact that multipollutant models were used as opposed to single pollutant models that yield different effect estimates. Other reasons are the small sample sizes or that results may be driven by the high values recorded during dust and bushfire events. It was also pointed out that hospitalisation rates in Adelaide may differ to elsewhere, or be more readily coded as a cardiovascular disease. Alternatively, different exposures and lifestyles may occur in Adelaide whereby people spend more time outdoors or with windows or doors open (Dr. A. Barnett, QUT, pers. comm. 14/7/09).
10.5 Significance of the study

The progressive nature of this study has been somewhat remarkable. At its commencement in 2006, the concept of climate change was still a contentious issue, but by 2007 the topic was in the media almost daily. With the change in government at the 2007 federal election, the Department of Climate Change was instigated and a Minister for Climate Change and Water appointed. As the drought became more severe in SA issues with the River Murray and the drying of the Lower Lakes were constantly in the press. The onset of the extended 2008 heatwave and in 2009, an intense heatwave together with the warmest winter on record, and an extraordinary November heatwave, have left less room for scepticism about climate change. Consequently this work now takes on a high level of relevance and significance and has already informed government policy. The new “South Australia State Emergency Service Extreme Heat Arrangements” plan formulated in October 2009 cites publications from this thesis, proving the value of this work.

The studies presented herein are unique and significant in several aspects. The investigation of the effect of heatwaves on renal disease is the first of its kind in Australia to discuss, in depth, renal involvement in heatwave morbidity. According to Google Scholar, the publication from this work has been cited seven times. Validating its importance and significance, was a commentary by Pascal in the International Journal of Epidemiology, which states:

“Following the example of Hansen et al...“additional studies on the impact of the 2003 and 2006 heatwaves on morbidity and hospital admissions are needed in France, to assess the efficiency of the preventive measures implemented and to help identifying at risk people.” (p. 1366)

Although there have been some recent reports concerning the impact of climate change on mental health, the study in this thesis on the impact of heatwaves on mental ill-health in Adelaide is significant in that, as discussed in Chapter 4, it may be the first to propose a model for the bimodal association between heat and mental disorders. The model postulates that firstly there can be behavioural/physiological maladaptations to
heat in persons with mental disorders, resulting in heat-related illness, and secondly there can be psychological maladaptations to heat resulting from direct (heatwave) or indirect (drought, bushfires) effects whereby heat can either trigger new, or exacerbate pre-existing conditions. During the 2009 heatwave, the Australian Red Cross in Adelaide operated a telephone assistance program (Telecross REDi Service)\(^407\) operated by volunteers who contacted vulnerable persons regularly to check on their wellbeing. The Australian Red Cross website reports an instance of direct heatwave-exacerbated anxiety:

“A woman who suffers from anxiety became stressed on the phone during her contact call from a Telecross volunteer. She felt the heat was worsening her condition and she was fearful. ....”\(^420\)

As mentioned previously, the air pollution study is significant in that it is the first to systematically investigate the health impact of ambient air pollution on the Adelaide population. The findings will be of great interest to government and non-government agencies, policy makers and strategists. The study is also unique in that it may the first to incorporate present weather condition visibility codes\(^319\) to indicate days of dust and smoke. To the author’s knowledge this has not been reported elsewhere. Finally, a considerable contribution has been made towards understanding the potential interactions between pollutants, together with the added consideration of heat on their effects. This work may help reduce the knowledge deficit in this complex area. Options for further research are outlined in Section 10.7.2.

### 10.6 Policy implications and recommendations

#### 10.6.1 Government policy and guidelines

The recent increased awareness and interest in heat-health in Adelaide has seen what was to be a recommendation for the formulation of public health alerts and heat health warnings, become a reality. Finalised in September 2009, the “SA Government Extreme Heat Communications Plan”\(^410\) required implementation during the extraordinary pre-summer heatwave just two months later.\(^359\) As previously mentioned this work helped inform part of this plan.\(^404\)
The role of certain medications in increasing susceptibility to heat illnesses has been discussed several times during this thesis. It is a recommendation that there be more awareness amongst the medical profession, pharmacists and the public, of the risks associated with taking some prescribed (and illicit) drugs during hot weather. Persons on medications for mental health disorders are one of several groups at risk. In reference to neuroleptic drugs it has been claimed that “the same mechanism that treats psychosis may create heat illness”.258(p. 801) With mental health separations in SA increasing by 3.1% in the period between 2002-03 and 2006-07,240 the safe use of psychiatric medications during periods of heat is rapidly gaining importance. See Section 10.7.1.5 for future research directions in this area.

For air pollution, the policy implications and recommendations are twofold: that the PM$_{2.5}$ advisory reporting standard become an official Air NEPM; and as mentioned in Chapter 9, wider use of air pollution health alerts and ‘Don’t Light Tonight’ alerts similar to those in NSW$^{411}$ where air quality reports are also available via a freecall line, in the press and online.$^{421}$ The inferred association between particulate pollution and a serious and potentially fatal health outcome (CVD) requires that preventive policies be formulated to protect public health when air quality presents a risk.

10.6.2 Infrastructure

Infrastructure changes may be a necessary means of adaptation to climate change. Instituting infrastructural improvements in retirement villages and aged care facilities may prove an effective preventive strategy. Older style window- or wall-mounted air conditioners may no longer be adequate for the elderly in a warming climate, and perhaps should be replaced with ducted or split system units. Furthermore insulation and ceiling fans are recommended in dwellings occupied by the elderly. Pull down canvas outdoor blinds which are cumbersome for the elderly to adjust and are subject to wind damage, could be replaced with motorised roller shutters that are easily operated from inside the home. The designers of new facilities might consider maximising shade areas and having only north or south facing windows in bedroom and living areas.
10.6.3 Resource allocation

It is highly recommended that resources be available for further environmental health research including air pollution and heat-health research. With Adelaide’s worst heatwaves occurring only recently, these were not included within the heatwave studies in Section II. Additional human and financial resources may be required for the health and emergency services sectors to accommodate the potential increased burden associated with climate change. In order to address barriers to adaptive capacity in the elderly, governments could consider allocating resources to subsidise power costs for the very elderly during the summer months. Costs may potentially be offset by savings to the health sector.

10.6.4 Organisational practice

Several studies within this thesis have shown those in the 15-64 age category to be a sub-population at risk during the heat. A recommendation for this group that may become more feasible over time involves a change of working hours to avoid the hottest part of the day. The introduction of siestas, a popular phenomenon in continental Europe where shops, businesses, factories and labourers cease work to rest during part of the afternoon, may be an option to consider in the future.

A loss of body water of just 3% of body weight constitutes clinical dehydration, the most common cause of hyperthermia. For those occupations where there is a risk of dehydration during hot weather, the use of hydration guides could be more widely promoted. These simple, cheap, easy to use resources are an aid in the self-diagnosis of volume depletion that can be used in the workplace. Often used in sports physiology, hydration charts (Figure 10.2) use urine colour as an indication of hydration status and could avoid progression to more severe life-threatening heat illnesses such as heat stroke. This preventive strategy could be used not only in workplaces with employees susceptible to heat exposure, but wherever there may be persons at risk of heat-related illnesses. The charts have easy to understand instructions and suggestions to drink more fluids if urine colour is indicative of dehydration, and to seek medical attention if
serious dehydration is indicated. See Section10.7.1.4 for research options regarding heat exposure and occupational health.

To enable appropriate epidemiological assessment of heat risk, there is a need for a universal consensus on the definition of heat-related illness and/or heat-exacerbated illnesses within health organisations. With the recognised definition of a heat-related death relying on body temperature at time of death, there can be an underestimation of the impact of heat on mortality as often deaths are attributed to pre-existing, easily identifiable conditions.\(^{195}\) As many heat-related deaths occur prior to medical assistance being summoned,\(^{23}\) the temperature based definition of heat-related death may be inadequate. Nevertheless some may argue that in the interest of accurate health statistics, the definition of heat-related death is already too broad and more specific diagnoses are preferred (e.g. cardiovascular, renal causes etc). An alternative option may be to have more than one cause of death or illness listed that includes heat-related death/illness as a co-morbidity/co-mortality where appropriate. In this way, the health impacts of heatwaves can be evaluated more readily and would take into account those deaths/illnesses due to indirect causes that would not have occurred in the absence of heat exposure.

NOTE:
This figure is included on page 233 of the print copy of the thesis held in the University of Adelaide Library.

**Figure 10.2:** Example of a hydration chart. Source: URL: [http://nats.us/images/health/urine_color_chart_320x240.jpg](http://nats.us/images/health/urine_color_chart_320x240.jpg) Accessed 17 August 2009
A preferred definition of a heat illness or death could be:

The presence of hyperthermia and/or volume depletion and/or central nervous system (CNS) involvement and/or electrolyte imbalance if symptoms could most likely be linked to behavioural, psychological or physiological maladaptation as a result of exposure to ‘warm to hot’ ambient (or environmental) temperatures.

A heat-exacerbated illness or death could be defined as:

The onset of a pre-existing (or otherwise) mental, cardiovascular, respiratory, neurological or renal condition (not necessarily including hyperthermia and/or volume depletion and/or central nervous system (CNS) involvement and/or electrolyte imbalance) if symptoms could most likely be linked to behavioural, psychological or physiological maladaptation as a result of exposure to ‘warm to hot’ ambient (or environmental) temperatures.

10.6.5 Community participation and engagement

The media may play a considerable role in risk communication and as well as broadcasting heat alerts, air quality alerts could be issued for certain sections of the metropolitan area and hills townships when air quality is expected to be poor, or when a combination of meteorological factors and air pollution are expected to result in conditions potentially damaging to health. Studies have shown interstate that public health advisories are effective in supporting behaviour change in extreme pollution events such as bushfires.330

Local councils and non-government organisations could be encouraged to play a more participatory role in building community resilience, promoting adaptive strategies and identifying persons at risk. Studies have shown that enhancing social capital and engaging communities in adaptation plays a role in increasing resilience to climate change and other community stressors.400 For maximum efficiency and community acceptance however, outreach groups should avoid replication of services and assistance programs. ‘Linking social capital’400 is important to connect neighbourhood
groups with government officials in order to implement appropriate and coordinated interventions to promote community adaptation.

10.7 Further research

With Adelaide gaining national and international notoriety for the intensity and duration of local heatwaves, there are several avenues of investigation which could be followed to continue with the important research undertaken within this thesis.

10.7.1 Heat-health relationship and climate change

10.7.1.1 Threshold temperatures

A study similar to that undertaken elsewhere in Australia\textsuperscript{382} and overseas\textsuperscript{232} will determine threshold maximum (and minimum) temperatures for heat-health in Adelaide. An analysis of morbidity and mortality using a combination of minimum and maximum temperatures and durations, as well as humidity (of more importance in subtropical cities than in Adelaide) will inform public health policy on heat alerts and heatwave warning systems. This study commenced recently in Adelaide.

Although the studies presented here used the warm season as the risk period, occasionally days with maximum temperatures 35°C or above occur during the months outside of the designated period. It remains to be investigated if adverse health effects occur in susceptible populations as a result of the onset of above average temperatures during the cooler months when persons are less acclimatised to summer conditions, or if heatwaves occurring early in the season have more effect on health than later in the season. These issues will be of interest if warm seasons become extended.

10.7.1.2 Adaptive capacity during heatwaves

A study incorporating both qualitative and quantitative aspects needs to be undertaken to investigate the adaptive capacities and resilience of the elderly during extreme heat. Barriers to adaptation may include a hesitation or unwillingness to use air conditioning due to consideration of energy costs, diminished physical or cognitive health, social
isolation, or security issues limiting the opening of doors and windows. Identifying and hopefully removing these barriers may increase resilience in the elderly and hence decrease their vulnerability to heat illness. Similar studies have been undertaken in the U.S. and Europe\textsuperscript{311,422,423} and could be carried out in an Australian environment where demographics and climatic conditions differ from locations overseas. This study will proceed shortly.

10.7.1.3 The effect of race on heat-susceptibility

Several international studies have identified race as a predictor of heat-related mortality,\textsuperscript{9,39,239} however little research has been undertaken in Australia on this issue. Studies within this thesis have indicated that having a mental illness or a predisposition to renal disease can be risk factors for heat-associated morbidity. Indigenous persons in Australia suffer more prominently from these health issues than the non-Indigenous population. Nationally in 2006-07, Aboriginal and Torres Strait Islander peoples were hospitalised for chronic kidney disease at seven times the rate of other Australians.\textsuperscript{226} In SA, the crude hospitalisation rates for renal disease and mental health conditions for Aboriginal peoples in 2006-07 were 8.0 times and 3.5 times higher respectively than for other South Australians\textsuperscript{240} (Figure 10.3). These indicators suggest that heat-susceptibility may be increased in Aboriginal populations.

Additionally the health impacts of overbearing heat on new migrants and persons from non-English speaking backgrounds who may hesitate to call for medical assistance, are yet to be determined. It is therefore timely that research be undertaken to investigate the effect of race on heat-susceptibility thus providing an evidence base for culturally appropriate intervention strategies. A qualitative study design could be considered if sample sizes were a limiting factor for a quantitative study.
10.7.1.4 Heat stress in the workplace

The study of ambulance usage (Chapter 5) identified certain industrial areas of Adelaide as having increased rates of ambulance callouts during heatwaves, particularly those of a work-related nature. This highlights the fact that little is known about heat stress in the Australian workplace or the effect of high temperatures on workplace injuries and productivity rates.
A study of risk factors during the French heatwave of 2003 found the risk of heat-related death was 2.75 times higher for manual workers compared to managers.\(^{424}\) Workers undertaking manual labour in hot environments are subject to certain occupational exposure limits,\(^{16}\) but these do not necessarily apply to outdoor workers with a high risk of heat stroke.\(^{218,310}\) A warming climate may pose an under recognised occupational health and safety risk to these employees and research needs to be done in this area. Contact has been made with the SafeWork SA, the state’s Occupational Health and Safety Agency, to formalise a study approach.

Furthermore an investigation into exertional heat illness in the U.S. military has shown the incidence of heat stroke in soldiers is increasing.\(^{425}\) A similar study could be conducted in conjunction with the Australian armed forces.

**10.7.1.5 The 2009 Adelaide heatwave**

As mentioned often throughout this thesis, Adelaide experienced an extraordinary heatwave in January-February 2009, with nine consecutive days above 35°C including six above 40°C, and the warmest Adelaide night on record (33.9 °C).\(^{51}\) Morbidity and mortality were reportedly increased markedly as a result. To establish the determinants of heat-related illness and death during this period, an investigation could be undertaken to examine the clinical details of those who presented at hospitals or died during that period. A study protocol may involve an audit of hospital case notes and coroner’s reports. These could provide details of patient demographics, comorbidities, medications, thermal responses and location at time of illness onset.

An expansion of this protocol could be a semi-qualitative case control study similar to that reported by Semenza\(^{34}\) with several controls per case matched by age, gender and residential location, who may be neighbours, friends and/or family members of cases. Here additional information could be sourced such as use of air conditioning, social isolation, and power outages.

**10.7.1.6 The effect of medications on the risk of heat illnesses**

With their ability to disrupt normal thermoregulatory pathways, some medications can increase thermal impacts in a hot environment. The list may include, but is not limited
to: ACE inhibitors/AR (angiotension II receptor) blockers, diuretics, anti-inflammatory drugs, certain antibiotics (sulfonamides), certain antivirals (such as indinavir), neuroleptics, sympathomimetics, antipsychotics, anticholinergics, antihistamines, tricyclic antidepressants, lithium, sedatives, opioids, beta-blockers, amphetamines and analgesics.

There is scant pharmacoepidemiological research reported on this issue in the literature and an investigation could be undertaken to establish the medications that may compromise heat-health in the ageing population of Adelaide. A recent study revealed that 43% of persons 65 years and over in South Australia take four or more prescribed medications daily and 14.8% of persons aged 55 years and over were taking six or more. It is likely these include some of the aforementioned pharmaceuticals. It is therefore important that with heatwaves very likely increasing, a study be undertaken to identify drugs associated with thermal intolerance. The findings could formulate the basis for a health promotion campaign to raise awareness amongst physicians, pharmacists and users about at-risk drugs, and the possible need to alter prescribing regimes during periods of extreme heat.

Similarly, a clinical study could be designed to determine the effects of heat on the pharmacokinetics of certain medications. Dehydration can affect metabolism, concentration, action, excretion and toxicity of medications, especially those with a narrow therapeutic index. The influence of increasing environmental temperatures on the efficacy and shelf life of manufactured drugs stored or transported at room temperature could also be investigated.

10.7.1.7 Heatwaves and infectious diseases

A possible link between heatwaves and the onset or exacerbation of infectious diseases is worthy of exploration. Infectious diseases have been shown to give rise to high mortality ratios during heatwaves in France, due partly to septicaemia, perhaps as a complication of other infections.

The study of ED visits at Adelaide hospitals (Chapter 6) revealed a non-significant increase of foodborne diseases in children during heatwaves. This requires further investigation factoring in lag periods of up to 28 days in light of evidence which shows
that foodborne diseases have been shown to increase one, two or four weeks following periods of high ambient temperatures. The critical heat-sensitive steps in the food production, processing and handling stages need to be identified along with potential changes in the food consumption behaviour of individuals during heatwaves.

With warmer temperatures favouring the growth of microbiological agents, climate change is likely to affect rates of some other types of infectious diseases including waterborne and vectorborne diseases, however one rarely discussed is legionellosis, the causal agent of which is the water-inhabiting bacteria *Legionella pneumophila*. With most cases reported during summer or autumn in the U.S. and increased incidence in England following warm periods followed by cool rainy weather, it is thought heat and humidity associated with a warmer, wetter climate may result in a rise in legionellosis cases. Legionellosis is a notifiable disease in Australia and a simple retrospective study in conjunction with state and territory Health Departments could be undertaken to investigate if links exist between incidence and certain climatic factors. Interestingly, media reports suggest there has been an increase in the number of reported cases of legionnaires’ disease in SA in 2009.

### 10.7.1.8 Extension of study investigating thermal impacts and renal outcomes

Chapter 3 within this thesis detailed a study investigating the impact of heatwaves on renal hospitalisations, paving the way for more detailed research in this area. Further investigation is warranted to identify other specific renal diagnoses, as well as ARF, that may be triggered by extreme heat. These may include hypertensive renal disease, rhabdomyolysis, hyperkalaemia, hyernatraemia, hyponatraemia, nephritis, nephritic syndrome, acute tubular necrosis, urolithiasis, anuria, hyperuricaemia, and other disorders of the kidney and ureter. Additionally, an investigation would be useful to investigate trends in renal-related admissions to intensive care units (ICU) during heatwaves.

Not previously identified is the possibility of a link between heat-related mental and renal morbidity. It is possible that persons with diagnosed mental disorders could be hospitalised with heat illnesses and subsequently develop renal complications. A possible causal pathway is shown in Figure 10.4. Indeed several such cases have been
reported in the literature. For example: during a London heatwave an individual with schizophrenia suffered heat stroke involving acute tubular necrosis in the kidney;\textsuperscript{246} in Canada a heat-stroke victim with schizophrenia and bipolar disease, suffered renal failure before death;\textsuperscript{15} in Hong Kong an individual with schizophrenia (and diabetes) suffered heat stroke with associated renal impairment;\textsuperscript{433} and finally from Brisbane, an individual with a childhood brain injury developed severe dehydration and hypernatraemia during a period of extreme heat, subsequently requiring renal replacement therapy.\textsuperscript{242} A study to quantify heat associated renal disease in persons with a history of mental disorders may provide a valuable epidemiological contribution to heat-health knowledge.

10.7.1.9 Do persons with diabetes have increased susceptibility to heat?

Several authors have observed increases in the heat susceptibility of persons with diabetes.\textsuperscript{19,39,218,239,408} Chapter 3 detailed the investigation of diabetes as a comorbidity to renal disease hospitalisations during heatwaves, however diabetes as a principal diagnosis was not considered. Diabetes is often associated with peripheral vascular disease,\textsuperscript{216} impairment of autonomic control, endothelial function\textsuperscript{239} and renal function,\textsuperscript{218,224} and low concentrations of antidiuretic hormone necessary for the re-absorption of water in the kidney.\textsuperscript{217} These factors may imply an increased risk of heat-illness including heat-triggered renal disease. As a considerable proportion of the population in SA have been diagnosed with diabetes (83,000 persons in 2003),\textsuperscript{240} it is important that thermal risk in this group be investigated.

10.7.1.10 The effect of climate change on rural and remote communities

The potential effects of climate change on the physical and mental health of communities in rural and remote regions across the state and the nation requires investigation. Climate change-health studies locally and abroad have focussed on urban populations, whereas those in rural and remote regions have largely been overlooked, due in part to lack of data. Rural areas in Australia often experience harsher climatic conditions than urban areas and are becoming more vulnerable to climate-related natural disasters\textsuperscript{434} such as dust storms, droughts, bushfires, high winds and flash flooding, all of which can have wide-ranging effects on physical and mental health. Remote Aboriginal and Torres Strait Islander communities may be particularly at risk
of climate change related health impacts\textsuperscript{435,436} due to factors such as poverty, low employment, high rate of incarceration, poor housing and poor sanitation\textsuperscript{32} with inequality in terms of access to health care\textsuperscript{434} adding to the city/country health divide. As studies within this thesis concentrated on Adelaide only, further research would establish the likely effect of a warmer climate on the health of rural South Australians so that future trends can be pre-empted and met with appropriate interventions.

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure10.4}
\caption{The possible association between mental conditions and the onset of renal disease during periods of extreme heat.}
\end{figure}
10.7.1.11 Psychological aspects of, and responses to, heat and climate change

A knowledge gap has been identified regarding the psychological aspects of climate change. Scant information exists regarding relevant personal attitudes, opinions, responses to health messages, and willingness to change behaviours. A recognition and understanding of the underlying psychology regarding decision making in terms of adaptation and mitigation to climate change will assist in the formulation of prevention strategies.

Concern about environmental degradation and climate change may manifest as mental health issues such as stress, anxiety, depression, or more serious disorders in certain individuals. One such case has recently been reported in Australia where a young man presented with ‘climate change delusion,’ a psychotic condition where the patient imagined that his consumption of water would lead to exhaustion of water supplies and deaths of millions. The prevalence of similar climate change related psychiatric disorders is unknown.

Heat can be associated with an increase in certain crimes including aggressive behaviour and assault, however little research has been undertaken on this issue in Australia. A simple qualitative/quantitative study could be conducted in conjunction with law enforcement authorities and correctional institutions to examine trends in criminal activities and possible associations with weather conditions.

10.7.1.12 Climate change impacts on the criminal justice system

The prison population may be susceptible to heat stress for several reasons. First, while many correctional facilities in Australia are air conditioned, there remain several in Western Australia and the Northern Territory that are not (Dr. E. Grant, University of Adelaide, pers. comm. 16/11/09). Conditions in one prison in the Pilbara region of WA have been described as “oppressive” with temperatures at one stage reaching up to 53°C during the day and almost 40°C at night. Second, being confined and unable to access a cool environment restricts behavioural thermoregulation as discussed previously, thereby increasing the risk of heat-related illness. Third, the criminal justice system is increasingly accommodating people with mental health problems. A recent Australian study showed the 12-month prevalence of any psychiatric illness in the previous year
was 80% in prisoners compared with 31% in the community. A cross-sectional survey of the NSW prison population found that 25% of female and 13% of male prisoners were currently taking psychiatric medication. Findings within this thesis have shown persons with mental disorders are highly susceptible to heat illness, due in part perhaps to their pharmacotherapy. Fourth, Indigenous persons are overrepresented in the prison population as some 24% of the total prisoner population incarcerated in Australia are Indigenous. With higher rates of renal disease, diabetes and mental and behavioural disorders than non-Indigenous people, heat susceptibility may be increased disproportionately in Indigenous prisoners.

There have been reports of heat-related deaths of mentally ill inmates in U.S. correctional facilities prompting recommendations for thermal conditions within some jurisdictions and in others, administrative regulations aimed to reduce heat risks in medicated prisoners. No such thermal regulations have been enforced in Australian correctional facilities. As prisoner health is an important part of public health, research could be conducted to investigate the incidence of heat-related illness in persons in custody, as well as the effect of heat on their physical and psychological health. A discussion needs to begin about the implications of climate change-induced warmer temperatures on the health of prison populations, and possible adaptation strategies including modifications to future prison design.

10.7.2 Air Pollution and health

10.7.2.1 More Adelaide based studies required

With levels of O₃ and PM failing to decline in accordance with other air pollutants in Australia, continued investigation into the association between ambient air pollution and poor health outcomes in Australia is warranted. In Adelaide further studies need to be undertaken to confirm the findings within this thesis. The findings of a potential association between cool weather and adverse cardiovascular effects prompts the need for further investigations to classify specific cardiovascular conditions that may be triggered by air pollution. Furthermore, discussions with the Bureau of Meteorology
may assist in identifying when temperature inversions occurred, allowing an assessment of air pollution and health outcomes on those days.

The association between ambulance callouts and air pollution has emerged as a knowledge gap in the prolific air pollution literature. Data are readily available for such an analysis in Adelaide and may provide useful information to policymakers. Finally, a research gap worldwide as identified by the WHO, concerns the chronic health effects of exposure to air pollution. This would require a cohort study similar to that by Dockery et al which would be difficult and expensive to undertake. Nevertheless if resources were available, such a study could be instigated in Adelaide, and could possibly incorporate other Australian cities.

10.7.2.2 Spatial analysis of air pollution-related health effects

A spatial analysis of air pollution related health effects and place-based risk in Adelaide could be valuable for city planners and local governments. At any one time there can be considerable intra-urban variations in pollutant concentrations within the airshed. This is demonstrated in Table 10.1 which shows pollutant concentrations at four different monitoring stations (shown in Figure 7.1) on a day during a heatwave in February 2009. Previously, monitoring during the period December 2003-January 2005 in a western industrial suburb revealed PM$_{10}$ levels exceeded NEPM guidelines 16 times during the study period, the report stating it is “the most affected area in metropolitan Adelaide that has been monitored” An investigation into the health of nearby residents is warranted. This could be done using stratification by postcode as in other studies within this thesis and results mapped to give a visual indication of air pollution-associated health effects.

Furthermore, it would also be of interest to identify possible health risks of living near main roads. International studies have identified persons residing near major traffic thoroughfares as being at almost twice the risk of cardiopulmonary mortality due to air pollution exposure, however few studies of this nature have been undertaken in Australia. Whilst some roadside monitoring of criteria air pollutants and air toxics have been carried out in Adelaide, the association with health outcomes remains to be investigated.
The spatial relationship during winter between poor air quality in the Adelaide Hills due to domestic wood burning, and air quality in the city would be interesting to ascertain. Preliminary information sent from the EPA (courtesy Mr. Timothy Lubke, EPA (SA)) indicates a definite, albeit weak, correlation between PM$_{10}$ levels at Woodside in the Adelaide Hills and the Netley monitoring site, the relationship being strongest at night. The geospatial relationship between air quality at the two sites is possibly due to a tunnelling effect of air movement through the hills onto the Adelaide plains but requires further investigation, as does the association with health impacts across the regions.

Finally, the EPA monitors air quality in several regional centres in SA (Whyalla, Port Pirie, Port Augusta) however to the author’s knowledge, associated cardiorespiratory health effects have not been assessed. Whilst quantitative studies are restricted by low statistical power in small populations, qualitative studies, panel studies or cohort studies in these industrial communities may provide valuable information to public health practitioners.

10.7.2.3 The heatwave–air quality association

With the predicted increasing intensity and duration of heatwaves, more research could be undertaken to investigate the health effects of air pollution during extreme heat when high concentrations of O$_3$ and/or PM prevail. Findings within this thesis suggest heat in combination with poor air quality may be highly detrimental to health and anecdotal evidence suggests an increase in asthma presentations in children during the 2009
heatwave. It is unknown if this was linked to the heat or high O₃ concentrations, or a combination of the two. The effects of O₃ during heatwaves could be quantified with a comprehensive study incorporating details of asthma medication usage and general practitioner visits as well as data on ED visits, hospital admissions and mortality.

10.7.2.4 Identification of subgroups susceptible to air pollution

Relatively little is known concerning the susceptibility of subgroups of the population to air pollution, except for those with chronic heart or lung conditions.⁷⁴ Several overseas studies have suggested that having diabetes is a risk factor for air pollution induced health effects.⁸⁷,⁴¹⁸,⁴⁴⁹ This diagnosis was not included in the present studies but warrants further investigation.

The effect of race on susceptibility could also be an option for future research as studies in Darwin have shown the effects of vegetation smoke on cardiorespiratory hospital admissions were greater for Indigenous than non-Indigenous persons.¹⁴²,¹⁴⁸ Whether a similar situation exists elsewhere in Australia is yet to be determined.

10.7.2.5 The cardiovascular effects of PM

Although the association between PM and cardiovascular disease is well documented, the physiological pathways are poorly understood. Further epidemiological studies in combination with animal studies and in vitro laboratory studies may clarify the toxicokinetics of air pollutants.

The strongest evidence for an association of PM with cardiovascular effects lies with fine particles. Here a deficit in the literature has been identified as there are few comprehensive reviews of the acute cardiovascular and pulmonary health impacts of fine and ultrafine particles, perhaps due to the fact that worldwide PM_{2.5} monitoring has been undertaken on a regular basis only relatively recently.

10.7.2.6 The acute health effects of exposure to bushfire smoke

The CSIRO predict an increase in fire weather risk as a result of climate change induced warmer drier conditions.²⁶⁸ Increased human exposure to biomass smoke may result from not only bushfires but prescribed burn offs pre-season to reduce fuel loads.
Study findings here indicate acute health effects of smoke exposure require further attention incorporating a range of indicators of respiratory and cardiovascular morbidity.

In 1983, the Ash Wednesday bushfire destroyed vast areas of the highly vegetated Adelaide Hills and another may occur near Adelaide or in fire-prone areas such as Kangaroo Island or Eyre Peninsula at any time. A qualitative analysis of the wider health impact of bushfires on rural and urban residents, similar to that undertaken in country NSW, would be useful. A survey instrument could be prepared and ethics approval sought, in readiness for the following summer to ensure a timely response.

10.7.2.7 The acute health effects of exposure to dust

The significance of dust to the health and economy in this state is notable. Some ten years ago the CSIRO estimated the cost of dust and wind erosion to SA, to be between $11 million and $23 million per year; only $1 million to $3 million if health costs were excluded. With the potential for rural dust to be transported vast distances across the country, many persons could be exposed to extreme levels of PM during dust storms, as occurred very recently (September 2009) when an extreme and unprecedented dust storm travelled across much of Australia. Dust originating near Alice Springs, NT, (Figure 10.5A) moved across SA’s far north (Figure 10.5B) and two days later blanketed Sydney (Figure 10.5C) where media reports suggested up to 75,000 tonnes of dust per hour were blown across the city. There was a spike in ambulance callouts to people with breathing difficulties, and particle pollution was at times over 15,000 µg/m³, the highest on record (in comparison, levels in bushfire smoke can be up to 500 µg/m³). From Sydney, the dust moved northward into Queensland, blanketing Brisbane the following day (Figure 10.5D). A climate change-induced increase in aridity may see these events become more frequent.

This could be of particular health significance to Adelaide, as a source apportionment study of PM collected over 12 months in four Australian cities (Melbourne, Sydney, Brisbane, Adelaide) showed the Adelaide samples to have the highest contribution of crustal matter, reflecting the impact of more frequent windblown dust events. Hence a comprehensive study of the effect of dust on cardiovascular and respiratory health in
rural and urban regions of SA could be a valuable addition to public health knowledge in this state.

NOTE:
This figure is included on page 249 of the print copy of the thesis held in the University of Adelaide Library.


10.7.3 Summary

Several options for further research have been presented here highlighting the importance of continuing with the enormous amount of work still to be done in the area of environmental epidemiology. With heat and air pollution extant in the environment and likely to increase with climate change, impacts may be minimised with a more complete knowledge and understanding of the associations with human health.
10.8 Conclusion

Work towards this thesis began in mid 2006. A comprehensive literature review of the health impacts of heat, air pollution and climate change in (Chapter 1, published in *Environmental Health*, 2009) preceded the first heat health studies. These investigated the effect of heatwaves on renal disease (Chapter 3, published in the *International Journal of Epidemiology*, 2008), mental and behavioural disorders (Chapter 4, published in *Environmental Health Perspectives*, 2008), ambulance callouts (Chapter 5) and ED visits (Chapter 6). In each study it was evident that exposure to extreme heat increases morbidity and the burden of disease in Adelaide. The public health significance of this work has escalated during the course of this candidature with three record-breaking heatwaves occurring within 20 months.

In Section III the focus shifted to air pollution and its effects on cardiorespiratory hospitalisations (Chapter 7). It was found that fine particulates were linked with cardiovascular admissions with greatest effect in the cool months. An attempt at an air pollution-temperature interaction study demonstrated difficulties in methodological issues and interpretation of findings (Chapter 8).

Using the knowledge base acquired throughout the study, a comprehensive risk assessment for Adelaide of the public health effects of these environmental stressors on population health was undertaken (Chapter 9). This final chapter has concluded the thesis by summarising the key findings, public health significance, policy implications, recommendations and a range of future research possibilities.

This work, with implications that extend further than South Australia, has highlighted the non-negligible effect of extreme heat and air pollution on population health, and stressed the need for mitigation strategies to lower greenhouse gas emissions, and adaptation strategies embedded in risk communication messages to minimise the negative health impacts of a changing and warming climate.
APPENDICES
Appendix A

Differences between postcodes and postal areas

There may be inherent differences between geographic regions defined by postcodes and postal areas used in spatial analytical techniques. Australia Post-derived postcodes were never intended for mapping purposes but rather for the efficient processing and delivery of mail. Postcode boundaries are determined by local councils and government land administration authorities.452 In metropolitan areas, several suburbs are usually included within the one postcode.

Postal areas, the spatial entity used in mapping, are defined by the Australian Bureau of Statistics (ABS) and are based on census collection districts. The ABS aggregates collection districts into postal areas roughly approximating postcodes and the two are given the same identifier number,281 causing some confusion for spatial epidemiologists. As dwelling density is high in the inner suburban core, postcodes and postal areas coincide well, however as population density declines in outer areas, collections districts become larger and areal disparities can be considerable. A Victorian study has shown discrepancies ranging from 38 and 201% in some outer Melbourne areas.281

In this Appendix, maps show the similarities and differences between postcodes (Figure A.1) and postal areas (Figure A.2) for the Adelaide metropolitan area.
Figure A.1: Map of (Australia Post) postcodes for metropolitan Adelaide. For the purposes of the study, the metropolitan area was approximated to the area encompassing postcodes 5000-5199. Source: Government of South Australia. Department of Environment and Heritage. (URL: http://www.deh.sa.gov.au/mapland/pdfs/lm_metro_postcodes_a3.pdf Accessed 3 April 2009)

NOTE:
This figure is included on page 253 of the print copy of the thesis held in the University of Adelaide Library.
ABS postal areas in the Adelaide metropolitan area

Figure A.2: Map showing the metropolitan Adelaide ABS-derived postal areas used in this thesis for the spatial analysis of data
Appendix B

Publications during candidature

Published:


Under review:


Technical reports:


NOTE:
This publication is included on page 256 in the print copy of the thesis held in the University of Adelaide Library.

It is also available online to authorised users at:

http://dx.doi.org/10.1093/ije/dyn165

NOTE:
This publication is included on page 257 in the print copy of the thesis held in the University of Adelaide Library.

It is also available online to authorised users at:

http://dx.doi.org/10.1289/ehp.11339

NOTE:
This publication is included on page 258 in the print copy of the thesis held in the University of Adelaide Library.
An Investigation of Potential Health Benefits from Increasing Energy Efficiency Stringency Requirements

Building Code of Australia Volumes One & Two

Associate Professor Terry Williamson
Dr Elizabeth Grant
Alana Hansen
Associate Professor Dino Pisaniello
Dr Myla Andamon

The University of Adelaide

SEPTEMBER 2009

Adelaide Research and Innovation Pty Ltd
The University of Adelaide, AUSTRALIA 5005
References


References


References


References


129. Monn C, Fuchs A, Högger D, Junker M, Kogelschatz D, Roth N, et al. Particulate matter less than 10 µm (PM$_{10}$) and fine particles less than 2.5 µm
References


References


References


265. Hannan E. January heatwave 'killed more than Black Saturday bushfires'. The Australian. 6 April 2009.


349. Edwards V, Walker J. *Increase in sudden deaths of elderly in Adelaide appear to be due to the heatwave.* The Australian. 7 February 2009.


References


References


References


References


