Health effects of bushfire smoke in the Australian monsoon tropics

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Thesis submitted for the degree of Doctor of Philosophy
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Declaration

I declare that the work herein is the result of my own investigations and all references to ideas and work of other researchers have been specifically acknowledged. I certify that this work has not already been accepted in substance for any degree, and is not being currently submitted in candidature for any other degree.

Fay Johnston
10 July 2008
Abstract

Introduction

There is increasing scientific consensus about the adverse health effects of outdoor particulate air pollution. Most evidence has come from studies set in large urban centres where airborne particulate matter is primarily derived from fossil fuel combustion by industrial plants and motor vehicles. The impact of ambient pollution originating from biomass combustion, particularly vegetation or forest fires, is less certain. Despite this uncertainty, land managers in Australia and overseas are increasingly required to meet strict air quality targets in their planned fuel reduction burns and address criticism from communities affected by fire disasters about the adequacy of these preventive operations. This thesis contributes to the evidence-base about the health effects of biomass smoke, particularly at the lower levels that occur in Darwin, northern Australia where approximately 95% of outdoor particulate matter comes from low-intensity seasonal vegetation fires.

Methods

I conducted a prospective panel study of the relationship between daily fluctuations in atmospheric particulate matter and daily symptoms, medication use and health care attendances in a cohort of people with asthma. I also conducted case-crossover and time-series studies of daily levels of particulate matter and hospital admissions for respiratory and cardiovascular conditions, and a case-crossover study of emergency department attendances.

Results

Particulate loadings generally remained low with a mean of PM$_{10}$ of 18 µg/m$^3$ over the dry seasons of 2000, 2004 and 2005 and a mean PM$_{2.5}$ of 10 µg/m$^3$ over the dry seasons of 2004 and 2005. In the panel study, both PM$_{10}$ and PM$_{2.5}$ were positively associated with the proportion of people with asthma experiencing symptoms and commencing steroid tablets. In the time-series and case-crossover studies, PM$_{10}$ showed borderline positive associations with respiratory conditions in non-Indigenous people and significant positive associations in
Indigenous people. There were mostly inverse or absent associations for cardiovascular admissions. While no associations were found between either PM$_{10}$ or PM$_{2.5}$ and emergency department attendances, the available data for this outcome were limited and the study lacked adequate statistical power.

**Conclusions**

Adverse health respiratory outcomes were measurable at particulate pollution levels well below Australia’s current air quality standards. Managers of deliberate burns, the primary intervention for preventing severe wildfires, therefore need to manage the health risks associated with smoke from these fires. While these risks might be justifiable when compared with the many harms associated with uncontrolled fires and extreme pollution episodes, it is important that the amount of smoke affecting urban areas is minimised, the impact on air quality in urban areas is evaluated and that advance public advisories are provided to allow people at higher risk from exposure to smoke to take appropriate action.
Acknowledgements

It has been a privilege to have had the guidance of my two supervisors Ross Bailie and Louis Pilotto throughout the four years of this research program. Both willingly gave their time and expertise and were unfailing in their support of my research endeavours. Thanks also to Geoff Morgan who generously provided additional advice on time-series modelling for Chapter Six. It has been a pleasure to have worked with you all.

The epidemiological studies in this thesis contributed to a larger research program on bushfires in Australia’s tropical savannas (The Darwin Smoke Project) and I am grateful to my collaborators from a wide range of disciplines who willingly taught me crucial aspects of their fields. I would particularly like to thank ecologist David Bowman for his inspiration, enthusiasm and leadership of the entire project; David Parry (atmospheric chemist) who with the help of Francoise Foti and Judy Manning overcame innumerable logistical difficulties to keep the air quality and pollen monitors functioning; Mike Foley (meteorologist) who responded to many queries about the atmosphere and regularly extracted data for the project; and palynologists Simon Haberle and Janelle Stephens who identified and counted the pollen and fungal elements we collected. Thanks also to the many other crucial members of the research team especially Rosalind Webby who made major contributions to the Darwin Asthma Study, Anne Myerscough and Ivan Hanigan who were highly organised and good humoured research assistants and Mark Myerscough who kindly helped to collect and identify local plants in Darwin. It was a pleasure to have been part of such a functional and cohesive research team.

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I am grateful to the National Health and Medical Research Council for funding my PhD scholarship for the final two and a half years of my work and to the Australian Research Council and the Northern Territory Government for direct funding of the project.

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This work is dedicated to the memory of my late father Peter Johnston.
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List of Abbreviations

95%CI  95 percent confidence interval
ABS  Australian Bureau of Statistics
ACT  Australian Capital Territory
APHEA  Air Pollution and Health: European Approach
APHENA  Air Pollution and Health: A Combined North American and European Approach
BOM  Bureau of Meteorology
BS  Black smoke
bsp  beta scatter by particles
CDU  Charles Darwin University
CO  Carbon monoxide
CO₂  Carbon dioxide
COAG  Council of Australian Governments
COPD  Chronic obstructive pulmonary disease
CRC  Cooperative Research Centre
CSIRO  Commonwealth Scientific and Industrial Research Organisation
DOW  Day of week
df  Degrees of freedom
ED  Emergency Department
EPA  Environment Protection Agency
EPM  Environment Protection Measure
FEV₁  Forc ed Expiratory Volume in 1 second
GAMs  Generalised additive models
GLMs  Generalised linear models
GP  General Practitioner
ICD  International Classification of Diseases
ICD9  International Classification of Diseases – Version 9
ICD10  International Classification of Diseases – Version 10
IHD  Ischaemic heart disease
ISAAC  International Study of Asthma and Allergies in Childhood
MCAPS  Medicare Air Pollution Study
MSHR  Menzies School of Health Research
NEPC  National Environment Protection Council
NEPM  National Environment Protection Measure
NHMRC  National Health and Medical Research Council
NMMAPS  National Morbidity, Mortality and Air Pollution Study
NT  Northern Territory
OR  Odds ratio
PAHs  Polycyclic aromatic hydrocarbons
PEACE  Pollution Effects on Asthmatic Children in Europe
PEFR  Peak expiratory flow rate
PM  Particulate matter
PMₖ₀  Particulate matter with a diameter less than or equal to 10 micrometers
PM₂₅  Particulate matter with a diameter less than or equal to 2.5 micrometers
RDH  Royal Darwin Hospital
TEOM  Tapered Element Oscillating Microbalance
TSP  Total suspended particles
VOCs  Volatile organic compounds
WHO  World Health Organisation
µm  micrometer (10⁻⁻⁻⁻⁻⁻⁻⁻⁻⁻ micrometers)
UK  United Kingdom
US  United States
USA  United States of America
Introduction to the research program and guide to the thesis.

Fires in the tropical savannas near Darwin
Chapter One

Introduction to the research program and guide to the thesis

This chapter is divided into two sections

- An introduction to the research program
- A description of the structure of the thesis
Introduction to the research program

The purpose of this thesis is to contribute to the knowledge of clinical and public health impacts of particulate air pollution arising from vegetation fires. More information is needed in this area to support fire management and public health interventions in Australia and other fire prone regions of the world. Vegetation fires are an important source of biomass smoke, a term used to describe the emissions generated by the combustion of biodegradable organic material rather than fossil fuels such as coal, oil and natural gas.

There is increasing scientific consensus about the adverse health effects of outdoor particulate air pollution primarily derived from fossil fuel combustion by industrial plants and motor vehicles in large urban centres (1-3). However, the impact of ambient pollution originating from biomass combustion, particularly vegetation or forest fires, is less certain (4). Despite this uncertainty, land managers in Australia and overseas are increasingly required to meet strict air quality targets and address criticism from communities affected by fire disasters about the amount of ‘controlled’ fuel reduction burns and the pollution generated by planned and unplanned fires (5).

Demonstrating a health impact of smoke from bushfires presents numerous practical and technical challenges because of the infrequency of fire events and the large number of potential confounders that make it difficult to detect the epidemiological ‘signal’ from the background ‘noise’. Indeed, there are only very few published studies worldwide that have sought to identify a health impact of wildfire smoke (4), the majority of which have found adverse health associations with measured levels of particulates or episodes of haze arising from fires. However nearly all these studies, by necessity, have used a retrospective design where heath outcomes during an unexpected fire event were compared with ‘equivalent’ periods without wildfire. Clearly, such a design makes it difficult to ensure that important biases, or confounding factors are not introduced (6).

The city of Darwin in the Australian monsoon tropics has many features that are helpful for studies of bushfire smoke. These include: a predictable dry season climate with little
variation in meteorological conditions over several months; a seasonally predictable high frequency of bushfires with corresponding fluctuations in air quality over several months allowing prospective studies to be planned; negligible air pollution from other sources (7) and a single major medical facility with good data collection systems in place. My preliminary work in Darwin (8) demonstrated the strongest link ever reported between asthma and remarkably low levels of particulates arising from bushfire smoke suggesting that current air quality standards set by many nations such as the USA and Australia might be too lax. However, this study was small in scope, did not include information about some important potential confounders and did not explicitly address the issue of serial correlation in the analysis (9). Although subsequent reanalyses addressing these issues (10, 11) did not alter the outcomes, it prompted the development of the more comprehensive epidemiological studies presented in this thesis.

Aims

This research aimed to determine the impact of atmospheric pollution caused by bushfires on ill health in the population of Darwin. Specifically, I examined the relationship between the mean daily atmospheric concentration of particulates with an aerodynamic diameter of 10 microns or less (PM$_{10}$) and 2.5 microns or less (PM$_{2.5}$) per cubic metre with a range of health outcomes including:

- the daily frequency of symptoms, medication use and healthcare attendances in a cohort of people with asthma followed for eight months;
- daily hospital admissions for respiratory and cardiovascular conditions over three years during which particulates were directly measured in Darwin;
- daily hospital admissions for respiratory and cardiovascular conditions over a ten year period during which particulates were estimated from daily visibility records; and
- daily emergency department presentations
Structure of the thesis

This thesis consists of eight chapters. The first is introductory while the next two provide the context and scientific background to the epidemiological studies. These are followed by four chapters, each of which presents original research. The final chapter discusses the results of the epidemiological studies and their relevance for the management of landscape fire. Additional documentation relating to the research program is included as separate appendices.

The chapters include both published and unpublished work. These are identified as follows:

- **Thesis chapter** – this describes unpublished work specifically prepared for this thesis;
- **Published paper** – this describes work that has been published in a peer reviewed journal; and
- **Keynote presentation** – this refers to a keynote paper published in conference proceedings.

Each chapter is preceded by an introductory page that explains the type of chapter, how it fits within the overall thesis and any contributions that have been made by others. Apart from the first and final chapters each also has an abstract summarising the content of the chapter. Some repetition of content is unavoidable with this structure, particularly in the introduction and discussion sections of the published papers.

A guide to the chapters

Chapter One – thesis chapter

An introduction to the research program, description of the structure of the thesis and guide to each chapter.
Chapter Two – keynote presentation

A discussion of some of the evolutionary, ecological, cultural, economic and social aspects of bushfires. The style of writing in this chapter differs from the style of subsequent chapters because this paper was prepared as an oral presentation to a lay audience.


Chapter Three – thesis chapter

A review of the international literature including the health effects of particulate air pollution with a specific focus on ambient biomass smoke. This provides the epidemiological background for the research chapters that follow.

Chapter Four – published paper

A panel study in which 251 people with doctor-diagnosed asthma were followed for eight months. Daily asthma symptoms, medication use and health service attendances were analysed in relation to levels of particulate air pollution.

Citation: Johnston FH, Webby RJ, Pilotto LS, Bailie RS, Parry DL and Halpin SJ. Vegetation fires, particulate air pollution and asthma: a panel study in the Australian monsoon tropics, International Journal of Environmental Health Research. 2006;16(6):391-404

Chapter Five – published paper

This reports a case-crossover analysis of the relationship between ambient particulate air pollution and admissions to Royal Darwin Hospital for cardio-respiratory conditions over a three year period.

Citation: Johnston FH, Bailie RS, Pilotto LS and Hanigan I. Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin Australia. BMC Public Health 2007, 7:240.
Chapter Six – thesis chapter
A time-series analysis of the relationship between estimated daily particulate air pollution and admissions to Royal Darwin Hospital for cardio-respiratory conditions over a ten year period.

Chapter Seven – thesis chapter
A case-crossover analysis of the relationship between ambient particulate air pollution and Emergency Department presentations for cardio-respiratory conditions, over a two year period.

Chapter Eight – thesis chapter
A synthesis and discussion of the preceding work. It considers the implications of the epidemiological findings for fire management in Australia.

Appendices
The appendices include:

- Documentation from the Darwin Asthma Study including the recruitment flyers, questionnaires, protocols, participant information, consent and feedback documents.

- Newsletters from the Darwin Smoke Project, of which the epidemiological work presented in this thesis forms one part.
Fire, landscapes and health.


Detail from Black Thursday, February 6th, 1851 by William Strutt (State Library of Victoria, reproduced with permission).
Chapter Two

Invited keynote paper

Fire, landscapes and health

Fay Johnston and David Bowman


Studies of landscape fire have largely been conducted by disciplines other than environmental epidemiology and understanding these wider perspectives is helpful for interpreting and responding to epidemiological findings. This paper discusses several aspects of bushfires in Australia including the evolutionary, ecological, cultural, economic and social contexts of landscape fires and their management.

This style of this chapter differs from that of subsequent chapters because this paper was prepared as an oral presentation for a lay audience.

Author contributions

I had primary responsibility for preparing and presenting this work. The ecological content is based on the work of my co-author David Bowman who also contributed to the discussion about the practical and ecological implications of current fire management practices in Australia.
Abstract

Functioning natural landscapes are essential for human survival. However, understanding the complex relationships between human health and landscapes beyond the immediate requirements for clean water, food and shelter (classical ‘environmental health’), has only recently taken root in health and ecological research programs. This emerging field is becoming known as ‘ecohealth’. Here we illustrate the opportunities, and challenges to such research collaborations using the example of landscape fire.

Australia is the quintessentially flammable continent. From coastal, tropical, arid and temperate zones fire plays a fundamental role in the functioning of our ecosystems. Following the human colonisation of Australia during the last ice age a balance between fire, land and hunter-gatherers was forged and subsequently maintained for millennia. The breaking of this relationship by pyrophobic European settlers caused dramatic changes to the health of both ‘country’ and the Aborigines who were extirpated of their ancestral lands or denied the right to burn the bush.

Since European colonisation the relationship between fire and people in Australia has increased in complexity. Multiple dilemmas confront those who manage our highly flammable bush and it is essential for current land managers to understand the extraordinary long evolutionary and cultural history of landscape burning. Uncontrolled severe fires destroy property, adversely affect mental and physical health and degrade amenity values. In addition the smoke from bushfires causes severe air pollution that increases cardiovascular and respiratory illnesses in exposed communities. These risks must be balanced against the environmental and social impacts of interventions to reduce fire hazards such as landscape clearing and intentional burning. ‘Controlled fires’ cause frequent episodes of lower levels of air pollution and the public health impact of this pattern of exposure compared with episodic severe pollution is unclear. Further, fuel reduction alone cannot provide a simple answer to preventing severe fires. Global environmental change such as frequent droughts and
increased production of fuels due to the CO$_2$ fertilizer effect may be important contributors to the increased frequency of severe vegetation fires that has been observed around the world.

Clearly the nexus between human and landscape health is complex. Historically there has been intellectual isolation between landscape and health scientists even though these fields are fundamentally related. Nonetheless, as humans adapt to the challenges of global environmental change and the associated changing patterns of human mortality and morbidity, the field of ‘ecohealth’ will become increasingly important.
Introduction

Every summer in Australia we go to considerable lengths to avoid bushfires. We grew up with stories of disasters and learnt very early to fear and respect fire. We have forecasts of fire risk and legislation against deliberate use of fire on certain days or throughout entire seasons. Throughout the country we have internationally respected fire fighting services, and we are committing increasingly more resources to bushfire control strategies and research.

So are we winning the battle? Are fire disasters in Australia becoming less frequent and less severe? And are we coping better when disasters occur? It’s hard to be sure about any of these - the fires in South Australia earlier this year (2005) claimed nine lives - earning the title of ‘the most deadly fires in two decades’. What caused these fires to get out of control so quickly? Had the bush been mismanaged? Had excess fuel been allowed to build up? Were there insufficient firebreaks? Or was this a consequence of the changing global climate causing an increase in the frequency of extreme fire danger days?

Landscape fires make a fascinating case study of the nexus between humans and landscapes, that goes far beyond the simplistic notion of land as a provider of food, water and shelter and beyond the identification, description and quantification of environment hazards. For example, to understand how to live in a ‘land of fire’ we need to understand the interactions between climate, fire and our flora and fauna over vast time scales and how these have interacted with human inhabitants over time. We also need to understand how our management and intervention strategies affect our environment, economy and community. Such transdisciplinary research clearly falls into the domain of the new and growing field of ecohealth (see www.ecohealth.net) which recognises the inherent interdependence of the health of humans with their social and economic environments, wildlife and ecosystems (12, 13). In this paper we examine bushfires from all of these perspectives. First we briefly sketch why Australia is so flammable and why total fire suppression is an impossible and risky dream. Then we will discuss current issues surrounding bushfire disasters and management and finally we reflect on transdisciplinary work for practitioners and researchers alike.
The ecological context of bushfires

In his description of three great ages of fire in Australia: ‘wild, tamed and feral’ Bowman (14) has provided a useful framework for understanding the ecological and evolutionary context of bushfires.

The wild age

This period of wild fires refers to the pre-human period when lightning started massive wild fires and enabled fire-adapted species to eventually subordinate the Gondwanan rainforests that originally clothed the entire continent. As Australia broke from the super-continent Gondwana and drifted northwards the subtropical high-pressure system intensified and also moved northward triggering the progressive aridification of the continent from the south to the north (15). Sometime after the mid-Tertiary (the age of the mammals that followed the demise of the dinosaurs), a monsoonal climate developed in northern Australia (14) in which lightning storms produced fires regularly at the end of each rain free dry season. Occasional storms would have penetrated deep into the continental interior starting fires at the beginning of the drought-prone summer months. Such reliable natural ignitions provided an extraordinary evolutionary theatre allowing for vegetation to adapt to fire and rendering Australia the most fire prone continent on Earth. In this way a ‘coarse-scale’ mosaic of large tracts of burnt vegetation in different stages of recovery was created. (Figure 2.1a)

The tamed age

This age refers to the period during which wildfires were tamed by humans and generations of Indigenous people created a cultural landscape through their skilful use of fire. Sometime towards the end of the ice-age, or about 40-60,000 years ago, humans arrived in Australia. There is little doubt that they brought the technological means to make fire. Over time the colonists gradually 'tamed' wildfire as they used fires to hunt game, a practice often described as ‘fire-stick farming’. The core feature was the reduction in the spatial extent of landscape fires (Figure 2.1b). These changes in fire regimes modified habitats in a way that disadvantaged some species such as *Genyornys newtoni*, the extinct, leaf eating giant bird that required long-unburnt habitats (14), but supported others such as kangaroos that were
better adapted to burnt landscapes. Many smaller animal species, adapted to living at the edges of burnt areas, became dependent upon the fine-grained mosaic created by Indigenous patterns of burning and the more recent cessation of Indigenous fire management has been identified as an important cause of the extinction of smaller mammals and some bird species in Australia’s interior and monsoon tropics (14, 16).

The feral age

The age of feral fire is the age of European colonisation, 1788 to the present. The new colonists clearly could not have understood the fundamental importance of fire in Australia’s evolutionary and cultural history. While early navigators observed fires and commented upon these as evidence of humanity, the underlying reasons the fires were lit were often misunderstood. As Charles Darwin wrote on 19 January 1836:

In the whole country I scarcely saw a place, without the marks of fire; whether these may be more or less recent, whether the stumps are more or less black, is the greatest change, which breaks the universal monotony that wearies the eyes of a traveller.

The Europeans brought very different knowledge, experience and attitudes towards fire. As Dame Mary Gilmore noted in the 19th Century, Indigenous people managed fires calmly and with familiarity while the whites ‘expended the energy of panic’ (17). While it is true that in some cases squatters, shepherds and cattlemen adopted Indigenous burning practices, this technological transfer ceased with the more intensive husbandry of stock that required investment in infrastructure such as fencing (18). Little thought was given to the consequences of disrupting the system of fire management that had moulded the Australian landscapes for thousands of years. Indigenous people were driven from their lands (19) and every effort was made to suppress fires. Fuel loads built up and frequent small fires were replaced by spatially widespread and intense fires (Figure 2.1c). Fire is, of course, only one part of this story. To this day we are only beginning to come to terms with the many consequences of this cataclysmic disruption for land, ecosystems and people.
Figure 2.1. Schematic representation of the ‘three great ages of fire in Australia’ in a hypothetical tract of tropical *Eucalyptus* savanna (14).

a) ‘Wild’ In the pre-human period, lightning started fires infrequently and burnt large areas, creating a coarse habitat mosaic to which various species of birds and mammals had become adapted.
b) ‘Tamed’ Aboriginal fire management was characterised by a high frequency of fires that burnt much smaller areas, producing a fine-scale habitat mosaic that supported most of the pre-human wildlife assemblage.
c) ‘Feral’ Under European fire management, fires, that had a similar frequency as the Indigenous period, burnt large areas thereby obliterating the pre-existing habitat mosaic created by Indigenous landscape burning.

**Current contexts: coping with ‘feral’ fires**

This transition from tame to feral fire can be considered the root of the current fire management problem. The challenge for modern day land managers is to once again tame fire to both reduce the loss of life and property but also to conserve biodiversity. However, deliberate use of fire now represents a tremendous challenge because of the construction of infrastructure and habitations established in bushland, capricious social values about landscape fire and the very limited practical opportunities to undertake burning. So how much of a problem are bushfires today and what are the practical options for addressing these?
The social, economic and public health impact of bushfires

In the past 40 years major Australian bushfires have been estimated to have caused losses to the order of $2.5 billion (Figure 2.2). This corresponds to an average of about 10 per cent of the cost of all major natural disasters, including droughts, floods, cyclones and earthquakes over the same period (5). These estimates neither include the costs of fighting fires, nor the costs associated with the loss of lives or other adverse health impacts. Bushfires over the same period have directly claimed 259 lives, the greatest loss of life associated with any category of natural disaster in Australia (5). Major bushfires are also well recognised for their long-term health impacts on those directly affected. In South Australia 1526 survivors of the Ash Wednesday fires of 1983 were surveyed one year after the event. A significant increase in stress-related conditions, including hypertension, gastrointestinal disorders, diabetes, and mental illness compared with the general population was recorded (20). Forty-two percent of respondents met the criteria for a psychiatric diagnosis particularly anxiety, depression and post traumatic stress disorder, a much greater proportion than in communities that have not experienced a natural disaster. Another follow up, twenty months after the fires, demonstrated lasting psychiatric morbidity from this event (21).

In addition to these direct effects, major bushfires usually push particulate air pollution concentrations well beyond the threshold National Environment Protection Measures level of 50 µg/m$^3$ for particulate matter measured as PM$_{10}$. During the 1994 Sydney bushfires PM$_{10}$ peaked at 210 µg/m$^3$ (against a background level from non-bushfire sources of 18 µg/m$^3$). During Sydney’s Christmas 2001 bushfires PM$_{10}$ levels above 150 µg/m$^3$ were sustained for 10 days while in Canberra the maximum level on 18 January 2003 was 192 µg/m$^3$ (5). The adverse health effects of particulate pollution are now well accepted. For example, although there were no direct deaths from the Sydney fires in 2001, Australia’s Environment Protection Association (EPA) estimated that particulate pollution generated by the bushfires would have been responsible for 16 deaths and at least 30 admissions to hospital for cardiovascular and respiratory conditions among the population of Sydney (22).
Figure 2.2 Financial losses estimated by the Insurance Council of Australia from major bushfires 1967 - 2003 adjusted to $2001. These estimates do not include the costs of fighting fires, the costs associated with the loss of lives or the costs of adverse health impacts from either the fires or the smoke pollution. Adapted from Aon Re Update 2003 (23).

Mitigation and response: learning to live with fire

If we accept that fire is an essential and recurring part of our landscape, how can we learn to live with it? One response to better equip ourselves to deal with the disasters is to invest more in fire fighting skills, expertise, coordination and sophisticated equipment such as fire bombing aircraft. Indeed, the national inquiry on bushfire mitigation and management commissioned by the Council of Australian Government (COAG) after the 2002/3 fire disasters in South Eastern Australia made many such recommendations in their report (5). In addition, this report stated that the first step needed for the mitigation of future disasters was the education of all sections of the community about the complexities of the problem. The
authors highlighted the importance of overcoming the widespread misplaced attitudes of ‘warfare’ against fire with its implicit, yet ecologically impossible, aim of total fire suppression in our landscapes (5). The need for widespread community education has also been well articulated by other experts in fire management such as Campbell (24) who wrote:

We have to get over the perpetual characterisation of wildfire in Australia as a terrifying aberration, an ineluctable, unpredictable Act of God, and start to see it and manage it as an inherently Australian phenomenon that goes with the territory. Fire is a crucial shaper of many of our landscapes and a valuable resource management tool. Rather than demonising fire with the language of warfare, disaster, destruction and terror, we should have explicit programs that are about learning to live with fire …

Central to all the recommendations of the COAG report was a call for a greater emphasis on further landscape scale research, including the ecological impacts of differing fire regimes in different settings and, coming full circle after 200 years of denial, recognition of the importance of Indigenous fire usage. The authors of this report noted the following:

There is considerable potential for both Indigenous and non-Indigenous Australians to benefit substantially from better understanding of Indigenous Australians’ knowledge and use of fire and of how this might be adapted and implemented in contemporary Australia. Such research—much of which would be action oriented and be conducted in partnership with Indigenous Australians—should include a focus on the process of co-learning between Indigenous and non-Indigenous Australians, and on operationally feasible ways of integrating customary and modern practices and technologies to support bushfire mitigation and management (5).
Recently Bowman et al (25, 26) undertook a major fire ecology project in collaboration with Indigenous land owners in northern Australia. They observed that Indigenous burning created a fine-scale mosaic of burnt and unburnt areas with a lower frequency and scale of burning compared to European fire regimes. In contrast the European systems were characterised by fires of annual or biennial frequencies and the burning of large areas that appear to have triggered a positive feedback cycle between fire frequency and flammable grass fuels. These authors concluded that the preservation of Indigenous fire management regimes should be a high management priority given the difficulty of breaking the grass-fire cycle once it has been initiated. They also noted that current Indigenous landscape burning has a significant impact on vegetation structure, but little effect on either species composition or total tree density apart from a lower biomass of tall grasses and a greater abundance of the fire sensitive tree *Callitris intratropica* (locally known as the Cyprus pine). Their work provides important empirical corroboration of previous descriptive ethnographic and historical analyses that vegetation structure at the time of settlement was strongly influenced by Indigenous peoples’ use of fire. On the basis of this research we have commenced a project to evaluate further Indigenous land management, the nexus with human health and wellbeing and the health of their country (27).

**Never ending reports and the prescribed burning debate.**

The national bushfire inquiry commissioned by COAG in 2003 is part of a long tradition of investigations commissioned to examine events and make recommendations for future practice following devastating fires such as that which occurred in south eastern Australia in 2002/2003. Other recent reports have included the McLeod report from the ACT (28), the Esplin Report from Victoria (29) and a federal parliamentary inquiry (30). The call for greater prescribed burning has been almost unanimous among them. As the House of Representatives Select Committee on the inquiry into the bushfires ‘A Nation Charred’ (30) noted in its foreword: ‘The Committee heard a consistent message right around Australia: there has been grossly inadequate hazard reduction burning on public lands for far too long’
Calls for more deliberate burning are not new. The Royal Commission that followed the Victorian fires of 1939 identified inadequate prescribed burning as an important reason that the 1939 fires in Victoria were so intense and destructive. Justice Stretton put this down to inadequate staffing of land management agencies and the attitudes of foresters who were ‘averse to burning of any sort’, concluding that the Forests Commission must recognise the necessity for protective burning (31). Similarly in 1992 the Chief Fire Officer in Victoria argued for a systemic role for fuel reduction and ecological burns (32). Guidelines and policies for prescribed burning have been developed in some jurisdictions including Western Australia (33) and have been used for many years in the United States (34).

Management agencies have responded to these calls. For example Parks Victoria doubled the amount of prescribed burning before the 2003-2004 fire season compared with previous efforts (35). However, while landscape researchers and management agencies are clearly moving towards more pro-active land management with an increased emphasis on deliberate burning, other sections of the community are not necessarily following.

**Ecological trade-offs with prescribed burning**

Michael Organ included a dissenting report within the COAG Inquiry that expressed caution about increasing prescribed burning. He supported calls by environmental organisations for further research into the role of climate change in producing a greater frequency of extreme fire danger days and the ecological costs and benefits of different fuel reduction regimes (5). This debate has been characterised as a ‘great divide’ between foresters and farmers on one side and environmentalists on the other (36). Indeed, many environment groups have expressed concern that increased prescribed burning is now being promoted as a panacea without sufficient emphasis on research about potential harmful ecological impacts of either inadequate or excessive burning (37). For instance compared to infrequent fires (both planned and unplanned), frequent hazard reduction has potentially negative effects such as disadvantaging plant species that need a substantial fire-free period for seed-set or animals that need unburnt habitat to nest.
These concerns need to be balanced against the potential ecological harms of the current mode of disaster response. For instance, the last resort for fire fighters confronted with high-intensity wildfires in areas of heavy fuel loads is to ‘back-burn’, often from hastily constructed fire-breaks (wide strips of bare earth) that have their own ecological costs such as subsequent weed invasion and soil erosion. Back-burning has the unavoidable effect of sandwiching the resident wildlife between two converging walls of fire which is far worse than a single fire front. Emergency back-burning is particularly destructive in bushland fragments around urban areas. Once wildlife is eliminated from a bushland fragment, natural recolonisation is often impossible because of isolation by lethal ‘rivers’ of speeding motor vehicles (38).

There are other aspects to this debate including the feasibility of safely performing enough burns. For example Mcleod noted that when weather conditions are taken into account as few as 25 to 30 days a year (including weekends) might be assessed as suitable in eastern Australia and only 18 days per annum for Canberra (39). Inevitably some prescribed burns will escape control and turn into wildfires: fire is by nature a risky substrate.

**Health trade-offs with prescribed burning**

The COAG report also noted a new dimension in the prescribed burning discourse that has received little attention in previous reports; that is the trade-off between tolerating reduced air quality and achieving risk reduction by fuel-reduction burning (5). It received submissions from groups such as Advocates for Clean Air who passionately argued against further polluting the air with bushfire smoke. Quoting reputable sources including the American Lung Association and both Australian and US Environment Protection Agencies the Advocates for Clean Air pointed out the high proportion of people who are at risk of illness and death from increases in particulate air pollution (40).

This has been an area that land managers and bushfire researchers have historically been loath to address. In 1992 Incoll (41) bolstered his case for prescribed burning by quoting a
medical review by Streeton that concluded that ‘it would seem unlikely that the general run of particles, the main pollutant in bushfire smoke, represented any real risk to human health’ (42). Smoke was considered a nuisance and an eyesore, but not harmful when compared to industrial smog. What is not well understood by many current fire managers is that there have since been considerable advances in medical research about the adverse health effects of particulate air pollution. For example in his second review of the literature, conducted for Australia’s EPA just five years later, Streeton came to very different conclusions as much new evidence about the health effects of air pollution became available during the 1990’s (43). It is now well accepted that particulate air pollution is associated with short and long-term mortality, particularly among the elderly and those with pre-existing heart and lung disease, neonatal mortality and many lung and heart diseases, including ischemic heart disease. Most studies describe linear relationships with these health outcomes with no apparent lower threshold (3).

However, there is very little information specifically concerning the health impacts of particulates and other pollutants generated by vegetation fires as such studies are difficult to conduct for practical reasons. While there is more health information about pollution generated by wood burning for residential heating, another source of biomass smoke, most research has examined particulate pollution generated by the combustion of fossil fuels by industry and motor vehicles. However, the limited available evidence would suggest that particulates from biomass combustion are no less harmful than particulates originating from the combustion of fossil fuels. Of the dozen published studies of pollution from wildfires, only three have failed to show any association between excessive smoke pollution and adverse health outcomes (44). One large review compared the magnitude of adverse health outcomes of exposure to particulates largely derived from wood smoke with particulates mostly derived from fossil fuel combustion and concluded that particulates derived from wood smoke might be more injurious to human health (45). These results highlight the uncertainty of estimating of the public health risk of bushfire smoke by extrapolating from the results of air pollution studies in urban settings.
Results from studies of air pollution and health exacerbate the difficult trade-offs faced by fire managers who are being urged to burn more and knowingly expose large populations to low levels of poisonous substances. While there are genuine grounds for concern, the actual level of risk this represents is uncertain. However, nearly every capital city in Australia is adjacent to large tracts of bush land and the public is becoming better informed about and more wary of air pollution. For the first time urban dwellers are beginning to realise that they have a direct and immediate stake in how land is managed (40). The issue of air pollution also increases the relevance of land management practices to politicians. If it is accepted that smoke can pose an immediate and potentially serious population health risk, then land and fire management can no longer be simply viewed as longer-term environmental issues.

The Darwin Study

Thus there has been increasing calls for more research into the health effects of bushfire smoke, particularly at levels below the Environmental Protection Measure for ambient air quality maximum threshold of 50µg/m³ for PM₁₀ (46). Our current research program is designed specifically to address this question. Darwin has many advantages for such research: it has a seven-month rain free dry season characterised by continuous fires in the surrounding savanna vegetation providing a continuous background of smoke pollution with peaks and troughs over several months; there is no significant source of atmospheric air pollution other than particulates derived from wildfires; (7) and there is a single major hospital that has systematic data collection systems in place. Our pilot study demonstrated significant increase in asthma presentations to hospital with each 10 µgm/m³ increase particulates measured as PM₁₀. When the PM₁₀ data were aggregated into10 µgm/m³ classes the modelling demonstrated a threshold at 40 µgm/m³ where presentations for asthma increased 2.6 times relative to the baseline category of less than 10 µgm/m³ (8). As part of our current study we are now examining a wide range of both health and ecological outcomes in detail, including the consequences of different land management activities with regard to fire regimes, ecological impacts, levels of pollution and smoke dispersal.
Dealing with complex trade-offs and forging new alliances

Regardless of these ecological and health debates, unplanned wildfires will continue to be a part of life. Every management action from doing nothing, to implementing extensive intervention programs, will involve important health, economic and ecological trade-offs, which will vary from region to region. While Australians are increasingly aware of their evolutionary heritage, beginning to understand the fundamental ecological role of fire in our landscapes and are grappling with the management implications of this, we still have a long way to go. It could be argued that we have reached a fourth age of fire: adaptation. That is, adaptive management based on balancing risk and costs through analysis, research and evaluation of the efficacy of land management programs.

Bushfires are clearly an excellent example of the complexities associated with the quest for sustainable relationships between ecosystems and human welfare. An increasing frequency of severe vegetation fire events throughout the world is just one example of the global change that is confronting human societies today (47). While the problem of wild fires is apparent to all members of society and we are making progress in addressing the issues, many other major ecological problems are less obvious, particularly to decision makers in large urban centres. Jared Diamond, in a great example of transdisciplinary scholarship, has recently described five major factors that contribute to the collapse of established societies (48). These were: climate change, hostile neighbors, few trade partners, environmental problems, and a society’s response to its environmental problems. He argues that while the first four may or may not prove significant in any individual case study the fifth always does.

There is no doubt that society’s response to the world’s many environmental crises will be crucial to our current and future wellbeing. This demands transdisciplinary research that is action orientated, adaptive and built from a constituency in many different communities and perspectives including rural health practitioners and researchers, land managers and ecologists. To be effective such research requires effort and a willingness to venture outside a disciplinary comfort zone which can be both frightening and rewarding. We are all on an adaptive journey.
A review of the literature relating to the health effects of particulate air pollution focusing on biomass burning and vegetation fires.
Chapter Three

Thesis chapter

A review of the literature relating to the health effects of particulate air pollution focussing on biomass burning and vegetation fires.

This chapter provides a review of the literature related to my research program. It is presented under the following main headings:

- The international and local context of biomass smoke and health
- Biomass burning: what’s in the smoke?
- The health effects of particulate air pollution
  - Biological studies
  - Epidemiological studies of individuals
  - Epidemiological studies of populations
- Summary – the health effects of particulate air pollution and ambient biomass smoke

Author contributions
I am the sole author of this work.
Abstract

Introduction

This work aimed to review and summarise the evidence-base concerning the health effects of smoke from vegetation fires.

Methods

I searched English language articles in the Medline, Biological Abstracts and Current Contents databases and citation lists from previously retrieved papers. Due to the large volume of literature concerning ambient particulate air pollution I focussed on large studies, major reviews, methods in air pollution epidemiology and studies of biomass smoke. I reviewed clinical and epidemiological studies of air pollution from vegetation fires in detail.

Results

The bulk of the available literature concerns outdoor particulate air pollution in urban settings where particles predominantly come from the combustion of fossil fuels. Biological and epidemiological studies conducted in these settings have demonstrated associations with many adverse health effects, especially increased respiratory and cardiovascular morbidity and mortality. Biological studies suggest that these effects are most likely to be mediated via pathophysiological mechanisms including increased oxidative stress and inflammation. Studies concerning wildfires and outdoor biomass smoke are limited in number and scope. They demonstrate that vegetation fires are increasing in frequency and severity on a worldwide basis. Their atmospheric emissions contain many toxic air pollutants, the most important of which is carbon-based particulate matter. Most epidemiological studies of outdoor biomass smoke have examined short term respiratory outcomes and their results have generally been consistent with the wider literature on particulate air pollution.
Conclusions

Important gaps in the available evidence include the short-term relationship between biomass smoke pollution and mortality, non-respiratory health outcomes such as cardiovascular diseases, perinatal outcomes and long-term outcomes such as cancer. There is also a lack of information about the effectiveness of public health interventions to reduce the harm associated with severe pollution episodes from wildfires and the relative public health impact of lower versus higher levels of outdoor biomass smoke.
The international and local context of biomass smoke and health

The term *biomass smoke* refers to the emissions generated by combustion of biodegradable organic material originating from plants, animals and micro-organisms and collectively known as biomass. On a global scale, biomass smoke contributes to air pollution in two principal ways. One is through episodic landscape fires and the other is its use as a source of fuel in homes throughout the world, the latter being a source of severe indoor as well as ambient air pollution (4).

It is now well recognised that vegetation fires in Asia, Latin America, Africa, and other parts of the world are an increasingly frequent phenomenon (47). Such fires have the capacity to rapidly convert vast amounts of stored carbon into pollutants such as carbon dioxide, carbon based particulates and many other organic chemicals. The smoke pollution generated by these fires prompted the development of ‘Health Guidelines for Vegetation Fire Events, by the World Health Organisation in collaboration with the United Nations Environment Programme, and World Meteorological Association (47). This document also identified a number of research priorities including the need for further investigation of the health impacts of air pollution due to vegetation fires. Calls for further research in this area have also been made in Australia (5, 46, 49).

Biomass is used as a domestic fuel in several regions of the world. About 2.4 billion people in developing countries, (approximately a third of the world’s population) burn biomass for cooking and heating, often using open indoor fires. This constant burning generates extremely high levels of indoor smoke that has been estimated to cause over 1.6 million deaths each year primarily through acute respiratory infections in children under 5 years of age and chronic lung diseases in adults. Biomass smoke is thus a major contributor to the global burden of disease (50). Biomass combustion is also increasing in its relative importance as a domestic fuel in higher income countries. This has been attributed to its lower cost and community perceptions that wood is a renewable and more benign source of energy than the burning of fossil fuels (4, 51, 52). Adverse public health impacts from the
contribution of domestic wood heaters to outdoor air pollution has been documented in many developed countries around the world including New Zealand, North America and Scandinavia (4). In many Australian cities wood combustion for domestic heating is an important contributor to particulate air pollution and there are localised examples of towns where excessive winter air pollution is principally derived from this source (53). However, I could find no published reports of epidemiological studies conducted in these settings in Australia.

In terms of absolute emissions, the largest source of biomass smoke in Australia is landscape fires (53). This is attributable to our unique biogeography, that has rendered Australia the most fire-prone continent on earth (54). While fires had been successfully used and managed by Indigenous people for millennia, approximately 200 years ago European colonists abruptly imposed a radically different management approach based on attempts to suppress landscape fire (14). Excluding fire from the environment has many ecological consequences including the accumulation of fuels which causes wild fires to become more catastrophic (55). Australia’s history since colonisation has thus been punctuated by a continuing series of major bushfire disasters. Prevention and management of these fires is complicated by increasing numbers of people living in or near bushland, particularly at the interface between urban and rural areas, and by global environmental change that is favouring more frequent and intense landscape fires the world over (56).

In Australia and internationally, regular prescribed burns are now increasingly used to maintain ecosystems and to ameliorate future severe fire events by reducing fuel loads (34). As the harmful effects of low levels of particulate pollution in urban / industrial settings improves, the potential contribution to adverse human health outcomes of pollution generated by wild fires is becoming increasingly recognised. In 1998 the first air quality policy on wildland and prescribed fires was issued in the USA (34). This policy promotes the ‘thoughtful use of fire by all wildland owners and managers while mitigating the impacts of emissions on air quality and visibility’. However, epidemiological evidence to inform such management endeavours is lacking. In Australia there have been calls for increased
research about the health impacts of bushfire smoke (5, 46, 49) to better understand the harms and benefits of frequent small prescribed fires and the less frequent, but much more severe wild fires.

**Biomass burning: what’s in the smoke?**

Smoke from vegetative biomass is composed of hundreds of chemicals in gaseous, liquid, and solid forms. While many are known to have implications for human health, by far the most abundant and important constituent is particulate matter (57) – hence the focus of this review. Box 3.1 contains a brief summary of the toxicity of other pollutants generated by biomass combustion.

**Particulate matter (PM)**

In the context of air pollution, the term *particulate matter* (PM) can refer to any small particle, regardless of composition. Historically, crustal particles such as silica and asbestos became well known for their role in causing dust related respiratory injury (58). However, this review specifically focuses on carbon based particles produced by combustion of either biomass or fossil fuels.

Most air pollution studies classify particulates according to their size. As many particles are not spherical in shape, the aerodynamic diameter is used for these classifications. Aerodynamic diameter is defined as the diameter of a spherical particle with a density of 1 gm/cm³ that has the same inertial properties as the particle of interest. PM₁₀ refers to particles with an aerodynamic diameter of 10 microns or less while PM₂.₅ refers to particles 2.5 microns or less. The size class of particles less than 0.1 micron (or 100 nanometres) is referred to as ultrafine particles (Figure 3.1). Older measures of particles include total suspended particles (TSP), black smoke (BS) and nephelometry. TSP represents the mass of all particles regardless of their size while black smoke (BS) represents the amount of elemental carbon rather than the total mass of particles. BS is assessed by the amount of light reflected or transmitted by particles collected on a filter that has been exposed to
polluted air, compared with that of an unexposed filter (59). Nephelometry is a measure of
the amount of light scattering in a sample of air compared with a known standard. This
measure is reasonably well correlated with the PM$_{2.5}$ size fraction of airborne particulate
matter (60).

The size class of particles 2.5 – 10 microns is known as the ‘coarse’ fraction. Once inhaled,
these are mostly deposited in large and smaller airways (Figure 3.2). PM$_{2.5}$ is known as the
‘fine’ or ‘respirable’ fraction because once inhaled, these enter the alveolar spaces in the
lung. This size fraction is thought to be the more harmful to human health (1).

Figure 3.1 The relative size of particles. Adapted from Donaldson (61)

Figure 3.2 The relation between the size of particles and cellular structures in the
lung. Idealised particles of 10, 1, and 0.1 µm are shown compared with a bronchial
epithelium. Adapted from Donaldson (61)
Fine particles account for 90 -100% of the particulate matter produced by combustion of either fossil or biomass fuels (62). Large and intense forest fires can rapidly convert vast amounts of carbon stored as biomass into CO₂ and airborne particulate matter. For example a forest fire in Carolina that burned over 9000 hectares in its first 20 hours, at times emitted particles into the atmosphere at rates exceeding 0.6 tons per second (63).

Fine particles consist of 60 to 70% organic carbon and also contain many carcinogenic compounds such as benzo[a]pyrene, adsorbed trace elements, condensed toxic elements and free radicals (see Box 3.1). Smoldering releases several times more fine particles than flaming combustion and higher intensity combustion releases more particles than lower intensity fires (57).

Plumes from vegetation fires can travel over 500–1000 kilometers or more in several days and may persist for weeks (64). The size distribution of particles changes during the transport process: particles with dimensions of nanometres to tens of nanometres accumulate and move to sizes of one micrometre, while coarser particles are removed from the air by the deposition process (4).

Detailed studies of emissions from savanna fires near Darwin were conducted during the 1990s. Figure 3.3 illustrates the size distribution of particles emitted from a savanna fire close to the source of the smoke plume. Combustion efficiencies in tropical savannas are slightly higher than southern Australian eucalypt forests. A range of 69% to 74% of the carbon consumed by savanna fires is released by combustion; approximately 87% of this as CO₂ (65). While only 0.42% is released as particulate carbon, this amounts to PM₁₀ emissions of 1,200 kg/km²/year, an amount higher than that in the air sheds of many capital cities in Australia (66).
Box 3.1 Non-particulate constituents of biomass smoke. Adapted from the UNEP, WHO WMO Guidelines for vegetation fire events 2001 (47), Nahear et al. 2007 (4) and Zelikoff et al 2002 (51)

**Inorganic acids**

*Carbon monoxide (CO)*

CO is produced through incomplete combustion of biomass fuels. Human exposure to CO causes carboxyhemoglobin to be produced reducing the capacity of red blood cells to transport oxygen. CO is produced more abundantly from smoldering than flaming combustion.

**Ozone**

Ozone is formed photo-chemically near the top of smoke plumes in sunlight conditions. Concentrations at ground level may not be high enough to be of direct concern to human health. Ozone is a respiratory irritant and associated with exacerbations of respiratory diseases.

**Nitrogen and sulphur-based compounds**

Both nitrogen and sulphur based compounds are produced in proportion to their content in the burning vegetation and the combustion efficiency for the fire. Smoldering combustion produces reduced nitrogen compounds such as \( \text{NH}_3 \) whereas flaming combustion produces oxides of nitrogen. These compounds are respiratory irritants.

**Hydrocarbons**

Produced by incomplete combustion. These may be saturated, unsaturated, monoaromatic or polycyclic aromatic. They are contained in small amounts in the organic fraction of the fine particle matter. Some, such as benzo[a]pyrene, are mutagenic and carcinogenic. Butadiene, an unsaturated hydrocarbon is irritant and neurotoxic.

**Oxygenated organic molecules**

**Aldehydes**

Some aldehydes such as acrolein are extremely irritating to mucous membranes of the human body. Others, such as formaldehyde, are potentially carcinogenic. Some reduce the ability of scavenger cells in the lungs to engulf foreign bacteria, which may accentuate infections of the respiratory system.

**Organic alcohols and acids**

These include methanol and acetic acid, which are irritant and teratogenic

**Phenols**

Examples include catechol and cresol. These are irritant mutagenic, carcinogenic and teratogenic

**Quinones**

Quinones such as hydroxyquinone are irritant, allergenic, cause oxidative stress and inflammation and are possibly carcinogenic.

**Organic acids**

This group includes semi-volatile and volatile organic compounds such as benzene, naphthalene, toluene and phenol compounds. They are abundantly produced from partial oxidation of cellulose fuels and strong respiratory tract irritants.

**Free radicals**

Free radicals, such as semiquinones, are abundantly produced through combustion; most undergo condensation within a few seconds. However, some may persist for up to 20 minutes and some may remain in organic material. They cause oxidative stress, inflammation and are possibly carcinogenic.
Figure 3.3 The concentration of particles according to their size in a sample of air collected 200m from a grass fire in tropical savannas of the Northern Territory. The vast majority of particles are less than 1 micron in diameter. Adapted from Morawska and Thomas (62).

The health effects of particulate air pollution

Here I review the health effects of outdoor particulate pollution with a focus on biomass smoke from vegetation fires. Studies specifically concerning pollution arising from forest or savanna fires are listed in Table 3.2. As these are relatively few in number I also include overviews of research concerning the toxicology and epidemiology of particulate air pollution arising from fossil fuel combustion, domestic wood heaters and, in brief, indoor biomass smoke pollution. This section is structured in the following way:

- Biological studies;
  - Chamber studies of deliberate human exposure to PM
- Epidemiological studies of individuals;
  - Occupational/domestic exposure to high levels of PM
  - Panel studies: short-term associations with ambient PM
  - Cohort studies: long-term associations with ambient PM
- Epidemiological studies of populations;
  - Time-series studies
  - Case-crossover studies
  - Episode and intervention studies
**Biological studies**

The patho-physiological consequences of inhaling particles have been examined in chamber studies in which animals or human volunteers inhale prescribed amounts of particulate matter. The advantages of these studies are that exposures are known and outcomes are reasonably certain. However, they will not reflect the mixtures and temporal variation in the ambient air of human populations (58). Additionally, human studies are usually conducted on a small number of healthy volunteers and outcomes are not easily generalised to wider populations.

Such studies have established that particles are deposited in the lungs by a mixture of inertial impaction, sedimentation and diffusion. The extent of penetration and distribution of particles depends upon ventilation by the subject, the health of the respiratory tract, and the size of the particles. Insoluble particles, such as carbon, are removed from the lungs by two processes, a short-term (24-34 hours) airway phase with active muco-ciliary transport, and a long-term alveolar phase which includes macrophage function, cellular endocytosis, intercellular sieving and removal by lymph and blood (67). Neither process is completely efficient and particles can remain in the alveolar cells and interstitium for years, chronically stimulating the surrounding cells (see Figure 3.4).

Several mechanisms have been postulated to explain the observed epidemiological association of particles with infections, allergic disorders and cancer (68-70). Chamber studies consistently show that exposure to PM causes an increase risk of pulmonary infections, possibly through promotion of inflammatory processes that disrupt the normal immune response (68, 70). In allergic disorders, carbon based particles are thought to act as an adjuvant to allergens. In experimental models, the combination of particulate matters and pollen allergens, causes the induction of allergic disease in the airways (69) and augments the specific immune response to an allergen even when the allergen was introduced several days after the particles (71). The link between PM and cancer might be through oxidative stress which has been shown to cause the formation of DNA adducts, and ultimately mutations (70).
The ultrafine size class can potentially cause harm through a number of additional mechanisms (72-74). Their extremely small size means that there will be a vastly greater number of particles for the same airborne mass as PM\textsubscript{10} providing an extremely large surface area for adsorption of reactive chemicals such as transition metals and polycyclic aromatic hydrocarbons. Ultrafine particles also impair phagocytosis by macrophages. The mechanism for this is uncertain. It may be by causing oxidative stress or through effects on intracellular calcium levels, both of which lead to further inflammation and pro-coagulation.

Figure 3.4 Hypothetical events in an alveolus after exposure to fine particles (left) and ultrafine particles (right). Ultrafine particles are outside as well as inside macrophage, there is additional release of inflammatory mediators due to oxidative stress and transfer of some particles to interstitium. Adapted from Donaldson (61)

Most chamber studies have used concentrated ambient particles, or diesel exhaust as the principal exposure and I could find only one study examining wood smoke inhalation in humans. This study found similar biochemical changes to those produced by other sources of particles, including increased blood markers of inflammation and promoters of coagulation and increased urinary excretion of factors associated with the metabolism of free radicals (75, 76).
In summary, biological studies have identified several plausible biological mechanisms through which inhaled particulate matter might act to impair respiratory and cardiovascular function. These include the promotion of oxidative stress, inflammation and blood coagulation. The single study that specifically examined particulates arising from the combustion of biomass had similar outcomes to studies of non-biomass particulates.

**Epidemiological studies of individuals**

People who have regular exposure to biomass smoke have been studied to examine some of the short-term clinical associations. However, the number of such studies is limited as following individuals and assessing individual exposures is relatively costly and time consuming (77). I consider these in three groups:

- Studies of people exposed to high levels of biomass smoke through their occupation (fire-fighters), chance (severe wildfires) or their domestic environment (unventilated indoor fires).
- Panel studies that examine the short-term impact of ambient levels of particulate pollution on people who are particularly susceptible such as the elderly, or those with pre-existing illnesses. A limited number of these studies have specifically examined particulate pollution arising from biomass combustion.
- Large cohort studies that have been used to study the long-term effects of ambient air pollution, none of which have specifically examined particulate pollution arising from biomass combustion.

**Occupational or domestic exposure to high levels of biomass smoke**

While occupational exposures provide a setting in which pollution exposures can be quantified, the levels of exposure are often far greater than ambient levels experienced by populations. In developed countries, the few available studies have mostly focused on fire fighters. As a group, forest fire fighters are intermittently exposed to extremely high concentrations of particles and other constituents of biomass smoke, well above those experienced by members of the public in adjacent residential areas. Studies of this
occupational group have demonstrated reduced lung function, increased airway hyper-
responsiveness and increases in respiratory symptoms that often extends beyond the fire
fighting season (4). I could find no studies that followed forest fire fighters over time periods
longer than a few months. However, long-term studies of urban fire fighters, who have more
frequent exposures to a different mix of pollutants, have shown increased risk of chronic lung
diseases and of mortality from some cancers (78). One study has found increased excretion
of heavy metals in both fire fighters and members of the general public in association with
smoke exposure from a wildfire (79). Although the clinical or public health relevance of this
finding is not clear, it is consistent with the findings of Karthikeyan et al (80) who found
significantly higher respiratory uptake of metals including zinc, copper and iron from aerosol
samples collected during bushfire haze periods in Singapore. In another study from
Singapore, military recruits provided weekly blood samples during and following the severe
pollution associated with the 1997 forest fires. These demonstrated increased release of
immature immune cells compared with a later period following cessation of fires and the
associated pollution (81). While relevance of these observations to the health status of
individuals is not clear, they demonstrate the influence of exposure to wildfire smoke on
several physiological systems.

In lower income countries, where wood and other forms of biomass are the primary source of
fuel for cooking, studies of biomass smoke pollution have focussed on indoor air. It has been
estimated that women and young children in particular are exposed to very high levels of
biomass smoke in approximately half of all households in the world (4). In these
circumstances the average daily PM$_{10}$ can be in the order of 5000 (and up to 20,000) $\mu$g/m$^{3}$
(82). Biomass smoke exposure in this context is an important contributor to the global
burden of disease through chronic obstructive pulmonary disease, lung and other respiratory
cancers and low birth weight and acute lower respiratory tract infections in children (83).
Ezzati et al have estimated that indoor smoke from solid fuels is responsible for 2.9% of
premature deaths worldwide (84).
In summary, those regularly exposed to very high levels of biomass smoke through their occupation or domestic circumstances are at increased risk of impaired lung function, acute and chronic respiratory diseases, and in the case of indoor domestic exposure, lung cancer and deaths from other respiratory causes. While non-respiratory outcomes in these settings have not been as extensively studied or reported, the message that very high exposure to biomass derived particulates is very harmful to human health is clear.

**Panel studies**

Panel studies (also known as diary studies) are a well-established approach for examining the health effects of exposure to levels of pollution more representative of that experienced by the general population, rather than high risk groups such as fire-fighters. These studies follow a group of individuals and examine how their daily symptoms or other health outcomes are correlated with daily measures of ambient air pollution. This approach was described by Künzli (85) as a ‘semi-individual’ study design as health outcomes are measured for each individual allowing factors such as smoking to be taken into account while the exposure measure used is often ‘ecological’, that is data from a fixed air quality monitoring site is applied to all participants. However, some panel studies attempt to quantify individual exposures through time/activity diaries and the use of personal or home based monitors. Results from panel studies can be more easily generalised to individuals than those from ecological time-series studies which are discussed later in this chapter (85). An early example of the use of this study design in air pollution epidemiology was from the USA where a cohort of 1,800 children completed daily diary studies over 4 years from 1984 to 1988 (86). Such studies have often focused on the association between urban air pollution levels and health outcomes in higher risk groups such as children, the elderly, or those with asthma (87).

Panel studies have identified associations between urban particulate pollution and pulse rate and rate variability in the elderly (88, 89), blood pressure (90), respiratory symptoms in healthy populations (91) and worsening symptoms and lung function in people with asthma (92-95). Ward and Ayes published a systematic review of particulate air pollution and panel
studies in children (96). The majority of the 22 studies they reviewed indicated adverse effects of particulates that were greater for PM$_{2.5}$ than for PM$_{10}$. However, they cautioned that there was considerable heterogeneity between the results, evidence of publication bias and that estimates of effect sizes appeared to be influenced by the method of statistical analysis.

**Panel studies and vegetation fires**

Two previous studies have used panel studies to examine the health effects of bushfire smoke. Jalaludin *et al* were co-incidentally recruiting children with a history of wheeze for an air pollution study in Sydney immediately prior to the city being blanketed by a smoke haze from bushfires in 1994. In the 32 children who had been recruited at the time of the haze, they found no association between peak expiratory flow rates (PEFR) and episodes of excess pollution from fires (97). However, the statistical power of this study was likely to have been limited by the low numbers of participants and the short duration of the pollution episode. Additionally the study relied on PEFR measurements conducted by parents. The ability to obtain reliable PEFR measurements depends on using a consistently correct technique (98) and it was not clear from the report if the planned follow up visit for checking and correcting the parents technique occurred prior to the fire episode or not. Any such measurement error would be unlikely to be associated with ambient PM and would have biased their results towards the null. Sutherland *et al* (99) followed the daily symptom scores of a panel of 21 people with COPD in Denver, USA and used their data to examine the impact of an episode of severe pollution arising from a wild fire. Even though they had fewer participants than the above Sydney study, they found a significant association between symptoms and pollution levels on two separate days in which particulate pollution levels was dominated by smoke from the fires. This study however relied on self reported symptoms and these could have been subjectively influenced by the presence of an obvious smoke haze.

In summary, panel studies have provided good evidence that daily fluctuations in ambient particulate matter in urban settings is associated with respiratory and cardiovascular symptoms, signs and impaired function. Two panel studies coincided with unexpected
excess pollution from wildfires and took the opportunity to examine this source of pollution. However, they were both small in size and had potential for bias in their outcome measures and therefore do not contribute greatly to the wider literature on panel studies in air pollution, or studies of pollution generated by vegetation fires.

**Cohort studies**

While panel studies generally examine the day to day variation in symptoms in association with daily fluctuations in air pollution, cohort studies are designed to examine the longer-term health effects of air pollution. In these studies large cohorts of people with differing exposures to air pollution are followed over years to see if they subsequently experience different rates of particular health outcomes such as mortality or cancer. Such studies are time consuming, costly to run and risk losing participants to follow up over time. For these reasons there are relatively few published studies. However this design provides the best available evidence of temporal associations between the exposures and outcomes and the magnitude of these outcomes following long-term exposure and adjustment for individual factors such as smoking status (77). Several cohort studies examining longer-term mortality attributable to particulate matter have been conducted, the largest of which have been reported from the USA. Some examples of these are given below. All are located in urban industrial settings. I found no long-term studies concerning exposure to outdoor biomass smoke.

The Harvard *Six Cities* study enrolled over 8000 people from six cities with differing ambient levels of particulate pollution. At follow up 14-16 years later, Dockery *et al* (100) found a 26% greater mortality in high compared with low pollution areas, (mortality rate ratio 1.26, 95%CI 1.08, 1.47). These estimates were adjusted for individual characteristics including age, sex, smoking status, education, and body mass index.

In 1995 the American Cancer Society reported on a study of over 500,000 people recruited during 1982. The report described a 17% increase in all cause mortality when comparing those exposed to the highest tertile with those exposed to the lowest tertile PM$_{10}$. (odds ratio
Further analyses published in 2002 as part of the \textit{Cancer Prevention Study-II} found that 10 $\mu g/m^3$ increases in PM$_{2.5}$ were associated with a 6\% rise in all cause mortality, (adjusted relative risk 1.06 95\%CI 1.02, 1.11), a 9\% rise in cardiopulmonary mortality, (adjusted relative risk 1.09 95\%CI 1.03, 1.16), and a 14\% rise in lung cancer mortality (adjusted relative risk 1.14 95\%CI 1.04, 1.23) (102). These risk estimates were adjusted for age, sex, race, smoking, education, marital status, alcohol consumption, occupational exposure to particulates and diet. In further analysis of the cardiac deaths, long-term PM exposures were most strongly associated with ischemic heart disease, dysrhythmias and heart failure and cardiac arrest with comparable or larger risks being observed in smokers relative to non-smokers (103).

Results from the American Cancer Society data generally had lower mortality estimates than the \textit{Six Cities} study and this has partly been attributed to a higher representation of relatively well educated people, and a lower spatial resolution of the geographic areas that defined each community and their exposure estimates in this study (1).

Other cohort studies have examined the impact of particulate air pollution on the very young. Woodruff \textit{et al} (104) followed a cohort of 12 million newborns in the United States and calculated odds ratios for death by tertiles of PM$_{10}$ exposure in the first 2 months of life. Comparing the tertile with the highest exposure to the group with the lowest they found all cause mortality to be increased by approximately 10\% (OR 1.1 95\%CI 1.04, 1.16), sudden infant death syndrome (SIDS) by approximately 26\% (OR 1.26 95\% CI 1.14, 1.39) and respiratory deaths by 40\% (OR1.4 95\%CI 1.05, 1.85). Kaiser \textit{et al} (105), used the findings of that study to estimate the proportion of post-neonatal infant mortality attributable to exposure to PM$_{10}$ levels greater than a mean value of 12.0 $\mu g/m^3$ during their first 2 months of life. This study was based on 700,000 infants born during the years 1995-1997 in 23 US cities. They concluded that particulate air pollution had accounted for 6\%, (95\%CI 3, 11\%), of all deaths in this group, 16\%, (95\%CI 9, 23\%), of cases of SIDS and 24\% (95\%CI 7, 44\%), of respiratory deaths.
In summary, long-term exposure to outdoor particulate air pollution has been clearly linked with excess mortality from a range of cardiovascular and respiratory diseases including lung cancer. This conclusion is supported by a recent review of the health effects of particulate pollution by Pope and Dockery who argued that the evidence for all cause and cardiopulmonary mortality first presented as part of the Six Cities and American Cancer Society studies remains compelling, and has generally been supported by subsequent cohort studies in other countries and settings (1). There are no reported studies examining the relative contribution of outdoor biomass derived particulates to these outcomes, although the findings are broadly consistent with the literature concerning indoor biomass smoke exposure (83).

### Epidemiological studies of populations

Population based studies have been the primary way in which the short-term health impacts of outdoor air pollution have been examined. The examination of large populations is useful because it enables relatively small effect sizes to be identified. Additionally, the relevant data concerning pollution levels and health outcomes such as deaths or hospital admissions are often routinely collected, making such studies relatively inexpensive to conduct. Population based studies also have inherent limitations. Information about important potential confounders might not be available for inclusion in the analysis (77) and the estimation of air pollution exposure usually relies on data from fixed monitoring sites that are not necessarily representative of any particular individual level of exposure. However, the outdoor concentration of particulate matter is much more closely correlated with personal exposure than that of other air pollutants (106, 107) and day to day fluctuations in pollution are likely to vary in the same direction as personal exposure even if the absolute concentrations are different (107).

The methods used for population based studies have evolved from the study of severely polluting events or episodes such as the London Fog of 1952 (108), to ecological time-series studies and more recently, case-crossover studies. Time-series and case-crossover studies...
now constitute the bulk of studies of the short-term health effects of air pollution in urban industrial settings. However for practical reasons, episode studies have remained the primary way in which pollution generated from vegetation fires has been examined. Finally, there are a few examples of intervention studies that have documented changes in health outcomes in association with reductions in ambient particulate levels. While none of these studies have specifically examined particulates derived from biomass combustion, they have provided useful supporting evidence to the available literature.

**An overview of time-series and case-crossover methods**

Time-series studies are ecological in design. They compare daily fluctuations in air pollution with daily fluctuations in a health outcome such as hospital admissions. Time-series modelling became the predominant approach during the 1990s when computing power, appropriate statistical software and advanced analytical methods were rapidly developed (77). This study design has now become one of the most important tools for examining health associations with ambient air pollution, and has provided the basis for much of the evidence used to establish air quality standards the world over (109). Such studies need to take into account other time varying factors that could confound the relationship between particulate pollution and the outcome being measured. For example, daily meteorological changes and seasonal cycles are well known to influence both hospital admissions and levels of particulate pollution (109). Similarly, long-term secular trends such as the changing demographic structure of the population need to be taken into account as pollution levels can also vary through time. Some factors such as daily meteorology are readily available and can be included in the analysis. However other factors including seasonal cycles and long-term trends can be either unknown or unmeasured and statistical modelling is used to take these into account. Many statistical challenges in time-series analysis have been identified and methodological refinements continue to be made. Issues that have been addressed in the last 10 years have included: the appropriateness of the default convergence criteria used by software packages, the relative merits of using parametric or non-parametric approaches, the accuracy of the estimated standard errors obtained with different approaches and how to select the optimal amount of smoothing that is used to adjust for...
time trends and cycles (110-112). There have now been hundreds of individual time-series studies published, many of which have been re-analysed, reviewed and combined into meta-analyses (113).

The case-crossover design was introduced by Maclure in the 1990s as an alternative approach for examining short-term associations between measures of ambient pollutants and health outcomes (114). In this approach the unit of analysis is an individual person, rather than a date. Pollution levels on the date of an event, such as an admission to hospital, are compared with pollution levels on one or more referent days during which the person did not experience the event. Referent days can be matched on day of week, season and other potential confounders. Similar risk estimates to those derived from time-series analyses are obtained with this approach (115). Lu and Zeger have demonstrated that when there is a common exposure, as is usually the case in air pollution studies, the case-crossover design analysed with conditional logistic regression modelling represents a special case of time-series modelling (116).

**Time-series and case-crossover studies examining short-term health effects of particulates in urban settings**

Below I present results from some of the larger multi-city studies from Europe and the United States of America (USA) and some individual and combined studies from Australia. However these are just a fraction of the studies that have been performed world wide. For example, multi-city studies have also been conducted in Canada, France, Korea and Japan and these have been presented in detailed reviews, such as that by Pope and Dockery of June 2006 (1).

Air Pollution and Health: a European Approach (APHEA) is a collection of studies which incorporated 29 European cities and examined the associations between air pollution and mortality (117) and hospital admissions (118-120). The National Morbidity and Mortality Air Pollution Study (NMMAPS) included a combined analyses of 20, and later 90 cities, in the USA (121, 122). NMMAPS and APHEA now form the basis of a large international
collaboration, Air Pollution and Health: Combined European and North American Approach (APHENA), that is using a common analytical approach across these regions, including exploration of spatial variation in the health effects of air pollution (109). In Australia, time-series studies from Sydney, Melbourne and Brisbane have been combined in a meta-analysis of mortality (123) and further combined with results from two New Zealand cities to examine admissions in children and the elderly (124, 125). Results from some of the above studies are presented in Table 3.1.

In summary, the most commonly reported short-term mortality outcomes associated with ambient particulates are increased deaths, particularly from respiratory and cardiovascular causes, with effect size estimates ranging from 0.6 to 4.1% increase per 10 unit rise in PM$_{10}$ (126). While the relative risk is greater for respiratory deaths, the absolute number of cardiovascular deaths due to particulates exceeds that of respiratory deaths because of the greater population at risk in western urban settings (127). Several analyses have demonstrated that these effects are not due to a ‘harvesting effect’ or the precipitation of death in people who were already about to die (3). It has also been estimated that the effect size estimates more than double if longer term effects are considered (128).

Ambient particulate matter has been clearly associated with respiratory hospital admissions, especially for asthma and COPD and, more recently, cardiovascular hospital admissions (see Table 3.1) (3, 58). Other cardiovascular outcomes that have now been clearly associated with airborne particulate matter include blood markers of cardiovascular risk, sub-clinical atherosclerosis, arrhythmias, ischaemic heart diseases and ischaemic stroke (1, 129).

**Time-series and case-crossover studies examining biomass smoke**

While there is now a vast body of literature concerning the health impacts of particulate air pollution, the evidence specifically relating to outdoor biomass smoke, or particulates derived from this source remains sparse (4). Understanding the relative toxicology of different types of particulates (e.g. due to differing sources, metal content or size class) has been identified as an important gap in the available evidence (130, 131). Part of the reason for this gap in
the evidence is the difficulty in discriminating biomass combustion from other sources of particulate air pollution particularly in settings dominated by fossil fuel combustion. One approach to resolving this difficulty has been to take advantage of local settings such as Christchurch, New Zealand where the majority (approx 80%) of particulates have been estimated to arise from biomass combustion (132). Here a time-series study of hospital admissions found a 3.37% (95%CI 2.34, 4.40) increase in respiratory admissions and a 1.26% (95%CI 0.31, 2.21) rise in cardiac admissions for each interquartile rise (14.8 µg/m³) in PM₁₀ (132). The equivalent point estimates for a 10 µg/m³ rise in PM₁₀ in this study would have been 2.27% for respiratory admissions and 0.85% for cardiovascular admissions. In a separate study Hales et al examined deaths in Christchurch where an increase in PM₁₀ of 10 µg/m³ after a lag of one day 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 (133). In Darwin, where 95% of particulates originate from bushfires, an earlier ecological time-series study observed a 20% (95%CI 9-34) rise in daily presentations for asthma per 10 unit rise in PM₁₀ (8).

However, there are relatively few settings in which biomass smoke is the principal source of ambient particulates. Another approach has therefore been to try to differentiate the contribution of different sources so they can be independently examined. This has been done in some settings by using chemical markers of biomass combustion. However the methods for this approach have yet to be sufficiently refined for widespread use (134). In a study from Spokane, Washington, Schreuder et al used total carbon as a marker of particulates derived from vegetative burning, and zinc and silica as markers of motor vehicle and soil derived particles respectively. They found respiratory emergency department visits increased 2% (1.023, 95% CI 1.009–1.038) for a 3.0 µg/m³ interquartile range change at a lag of one day of total carbon, while no associations with other specific sources of particulate pollution nor PM₂.₅ overall (135).

In Australia, bushfire smoke intermittently affects the air quality of large cities where air pollution is usually dominated by fossil fuel combustion. Research teams in both Sydney and Brisbane have combined bushfire records with routine air quality measurements to estimate
Table 3.1 Estimates of mortality and morbidity due to particulate air pollution from multi-city studies in Europe and the United States and single and multi-city studies in Australia.

<table>
<thead>
<tr>
<th>Study</th>
<th>Exposure measure and increment</th>
<th>Outcome</th>
<th>Percent change (95% Confidence Intervals)</th>
</tr>
</thead>
<tbody>
<tr>
<td>APHEA 2 - 29 cities (117)</td>
<td>10μg/m³ PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Mortality - all causes</td>
<td>0.6 (0.4, 0.6)</td>
</tr>
<tr>
<td>NMMAPS - 20 cities (121)</td>
<td>10μg/m³ PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Mortality:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>All causes</td>
<td>0.5 (0.1, 0.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined cardiovascular respiratory</td>
<td>0.7 (0.2, 1.2)</td>
</tr>
<tr>
<td>NMMAPS - 88 cities (136)</td>
<td>10μg/m³ PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Mortality:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>All causes</td>
<td>0.2 (0.1, 0.4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Combined cardiovascular respiratory</td>
<td>0.3 (0.2, 0.5)</td>
</tr>
<tr>
<td>Australia - 4 cities (123)</td>
<td>10μg/m³ PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Mortality – all causes</td>
<td>0.9 (-0.7, 2.5)</td>
</tr>
<tr>
<td>Sydney (137)</td>
<td>Rise from 10&lt;sup&gt;th&lt;/sup&gt; to 90&lt;sup&gt;th&lt;/sup&gt; centile by nephelometry (size 0.01 – 2.0 μg)</td>
<td>Mortality:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>All causes</td>
<td>2.6 (0.9, 4.4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiovascular</td>
<td>2.7 (0.3, 5.2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Respiratory</td>
<td>3.3 (-2.1, 9.1)</td>
</tr>
<tr>
<td>APHEA 2 - 29 cities (119)</td>
<td>10μg/m³ PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Admissions:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiac all ages</td>
<td>0.5 (0.2, 0.8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiac - 65+yrs</td>
<td>0.7 (0.4, 1.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ischaemic heart disease - 65+yrs</td>
<td>0.8 (0.3, 1.2)</td>
</tr>
<tr>
<td>USA national database</td>
<td>10μg/m³ PM&lt;sub&gt;2.5&lt;/sub&gt;</td>
<td>Admissions:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Injury (control group)</td>
<td>-0.4 (-1.0, 0.2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cerebrovascular disease</td>
<td>0.8 (0.3, 1.3)</td>
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<tr>
<td></td>
<td></td>
<td>Peripheral vascular disease</td>
<td>0.9 (-0.1, 1.8)</td>
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<tr>
<td></td>
<td></td>
<td>Ischemic heart disease</td>
<td>0.4 (0.02, 0.9)</td>
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<td></td>
<td></td>
<td>Heart rhythm</td>
<td>1.3 (0.8, 1.8)</td>
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<td></td>
<td></td>
<td>Heart failure</td>
<td>0.9 (0.2, 1.6)</td>
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<td></td>
<td></td>
<td>COPD</td>
<td>0.9 (0.4, 1.4)</td>
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<tr>
<td></td>
<td></td>
<td>Respiratory tract infection</td>
<td></td>
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<tr>
<td>NMMAPS - 14 cities (139)</td>
<td>10μg/m³ PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Admissions:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiovascular disease</td>
<td>1.1 (0.9, 1.2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>COPD</td>
<td>1.4 (1.0, 1.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pneumonia</td>
<td>1.6 (1.3, 1.9)</td>
</tr>
<tr>
<td>Australia and New Zealand -5 cities (125)</td>
<td>Interquartile range rise PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>Admissions:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>respiratory 1-4 yrs</td>
<td>1.7 (0.5, 2.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>respiratory 5-14 yrs</td>
<td>1.9 (0.1, 3.8)</td>
</tr>
<tr>
<td>Australia and New Zealand -5 cities (124)</td>
<td>Interquartile range rise PM&lt;sub&gt;2.5&lt;/sub&gt;</td>
<td>Admissions:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiovascular 15-64 yrs</td>
<td>0.2 (-0.5, 0.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiovascular 65+ yrs</td>
<td>1.3 (0.6, 2.0)</td>
</tr>
<tr>
<td>Sydney (140)</td>
<td>Rise from 10&lt;sup&gt;th&lt;/sup&gt; to 90&lt;sup&gt;th&lt;/sup&gt; centile PM&lt;sub&gt;10&lt;/sub&gt; by nephelometry (size 0.01 – 2.0 μg)</td>
<td>Admissions:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiac</td>
<td>2.2 (0.6, 3.9)</td>
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<tr>
<td></td>
<td></td>
<td>COPD</td>
<td>2.4 (-0.9 5.8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Asthma 15-64 yrs</td>
<td>1.3 (-2.3, 5.1)</td>
</tr>
</tbody>
</table>
the proportion of particulates arising from bushfires. In Brisbane, Chen et al compared days with PM$_{10}$ > 20 µg/m$^3$ with those <15 µg/m$^3$ and found that daily respiratory hospital admissions were consistently increased with increasing levels of PM$_{10}$. The relationship was greater during bushfire periods with an adjusted relative risk of 1.19 (95%CI 1.09, 1.30) compared with 1.13 (95%CI 1.06, 1.23) during non-bushfire periods (141).

Similarly, Morgan et al reported a larger association between all respiratory admissions in association with a 10 µg/m$^3$ rise in bushfire PM$_{10}$ of 2.33% (95%CI 0.83, 3.87) compared with 1.04% (95%CI 0.02, 2.07) in association non-bushfire PM$_{10}$ in Sydney (142). When examining specific respiratory diagnoses this study found a 3.29% (95%CI 0.86, 5.7) increase in COPD admissions and a 5.02% (95%CI 1.77, 8.37) increase in adult asthma admissions in association with bushfire derived PM$_{10}$ but no associations between these outcomes and particulate matter from all other sources. Cardiovascular admissions had the opposite pattern in this study as they did not find an association between this outcome and bushfire PM$_{10}$. In contrast to this a 10 µg/m$^3$ rise in non-bushfire PM$_{10}$ was associated with a 1.21% increase (95%CI 0.41, 2.03) in all cardiovascular admissions, a result in keeping with the wider international literature and previous studies in Sydney (140). The results from this study by Morgan et al also suggested a weak same day association between all cause mortality and bushfire derived PM with an increase of 0.80% per 10 µg/m$^3$ rise in bushfire PM$_{10}$ (95%CI -0.24, 1.86), in contrast with a larger association with non-bushfire derived PM of 1.35%, (95%CI 0.38, 2.32) at a lag of one day and 1.07%, (95%CI 0.14, 2.00) at a lag of two days.

In summary, ecological time-series and case-crossover studies have clearly demonstrated short-term associations between urban particulate air pollution and many adverse cardiovascular and respiratory health outcomes, including health care attendances, admissions to hospital and deaths. While the sizes of these associations are generally smaller than those associated with long-term exposure, they have a considerable public health impact because of the large populations affected. Only a tiny fraction of the available studies have specifically attempted to discriminate the health impacts of particulates derived
from biomass smoke from either wood heaters or vegetation fires. Biomass smoke studies have generally found that respiratory outcomes, such as asthma and COPD hospital admissions, have greater effect sizes than those reported in the wider literature. However the available studies are few in number and generally set in single cities with relatively small populations in contrast to large international multi-city air pollution studies. In conclusion, available evidence concerning the health effects of ambient particulate air pollution suggest that adverse respiratory effects of biomass derived particulates are no less, and potentially greater, than that of particulates from other sources. However if population exposure to smoke is brief, as often the case with vegetation fires, the number of attributable admissions would be relatively small. Further evidence is needed to confirm and quantify any differences in respiratory toxicity. It is not possible to draw any conclusions about biomass smoke and short-term exacerbations of cardiovascular diseases as there are insufficient studies available and possibly because the associations are weak.

**Episode studies in air pollution epidemiology**

The study of air pollution episodes such as the 1952 smog of London (108), or the 1997 forest fires in South East Asia, are those in which health outcomes during the episode are contrasted with those at a comparable time period. There are many inherent limitations with this study design, which are inevitably retrospective. Relying on the examination of routinely collected data limits the ability to adjust for important confounders such as weather, pollen levels and community incidence of respiratory viral illnesses (6). Additionally, the brief duration of some episodes limits the statistical power for the analysis (4). For these reasons the use of this approach in the study of air pollution in urban settings has largely been superseded by other approaches, such as time-series modelling. However, for logistical reasons, episode studies remain the primary way in which the health impacts of vegetation fire smoke have been studied. Of the 23 published reports examining the health effects of biomass smoke from wildfires (Table 3.2), 19 fall into this category.
Table 3.2 Summary of epidemiologic studies of wildfire smoke and human health

<table>
<thead>
<tr>
<th>Year and location of study</th>
<th>Exposure metric</th>
<th>Max $PM_{10}$ ($\mu g/m^3$)</th>
<th>Outcome measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>North America</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2002 Denver, USA (99)</td>
<td>Episode of smoke haze (2 days)</td>
<td>90</td>
<td>Symptoms in patients with COPD</td>
<td>Significant increase in symptom index relative to control periods</td>
</tr>
<tr>
<td>1987 California, USA (144)</td>
<td>Episode of smoke haze (2 1/2 week period, compared with periods before and after)</td>
<td>Not stated</td>
<td>Hospital attendances for asthma and COPD</td>
<td>30% increased for asthma, 40% increase COPD</td>
</tr>
<tr>
<td>1999 California, USA (145)</td>
<td>Episode of smoke haze (6 weeks)</td>
<td>600</td>
<td>Symptoms and health care attendances</td>
<td>52% increase in clinic attendances, 63% increase in respiratory symptoms, greater in those with preexisting heart or lung conditions</td>
</tr>
<tr>
<td>1991 California, USA (146)</td>
<td>Episode of smoke haze (1 week)</td>
<td>Not stated</td>
<td>Hospital attendances for respiratory conditions</td>
<td>Many hospital attendances on the days of the fire, no comparison period reported</td>
</tr>
<tr>
<td>1998 Florida, USA (147)</td>
<td>Episode of smoke haze (1 week) compared with the same week one year previously</td>
<td>Not stated</td>
<td>Hospital/clinic attendances</td>
<td>91% increase for asthma, 132% increase for COPD, 37% increase for chest pain</td>
</tr>
<tr>
<td>2003 California, USA (148)</td>
<td>Number of days of smoke haze</td>
<td>337</td>
<td>Respiratory and eye symptoms, medication use and physician visits in children</td>
<td>All outcomes monotonically increased in association with exposure to smoke haze.</td>
</tr>
<tr>
<td>2003 British Columbia, Canada (149)</td>
<td>Episode of smoke haze (4 weeks) compared with 10 year mean</td>
<td>250</td>
<td>Weekly physician billing data for respiratory cardiovascular and mental illness in two regions</td>
<td>46%-78% increase in attendances for respiratory illness above the 10 year mean rates in one only No association with cardiovascular or mental illnesses in either region.</td>
</tr>
<tr>
<td>2002 Denver, USA (143)</td>
<td>Episodes of smoke haze (2 separate days) Compared the entire month and the same month one year previously</td>
<td>90</td>
<td>Mortality</td>
<td>No association</td>
</tr>
<tr>
<td><strong>Europe</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2002 Vilnius, Lithuania (150)</td>
<td>Episode of smoke haze (6 weeks)</td>
<td>250</td>
<td>Health centre attendance for asthma, Health centre attendance for all respiratory diseases</td>
<td>Three fold increase in both outcomes</td>
</tr>
<tr>
<td>Year and location of study</td>
<td>Exposure metric</td>
<td>Max $PM_{10}$ ($\mu g /m^3$)</td>
<td>Outcome measures</td>
<td>Results</td>
</tr>
<tr>
<td>----------------------------</td>
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</tr>
<tr>
<td><strong>South East Asia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1997 Singapore (81)</td>
<td>$PM_{10}$</td>
<td>216</td>
<td>Weekly white blood cell count in healthy volunteers</td>
<td>Positive association between $PM_{10}$ and banded neutrophils</td>
</tr>
<tr>
<td>1997 Malaysia, (152)</td>
<td>Episode of smoke haze (3 months)</td>
<td>Not stated</td>
<td>Hospital attendances for ARI asthma and conjunctivitis</td>
<td>10 to 20 fold increases all outcomes</td>
</tr>
<tr>
<td>1997 Indonesia (153)</td>
<td>Episode of smoke haze. Survey data compared with 1993 surveys of the same households</td>
<td>Not stated</td>
<td>Self report of 1) ability to conduct strenuous tasks 2) coughing in the previous month 3) general health score</td>
<td>Deceased respiratory health and ability to function associated with the haze</td>
</tr>
<tr>
<td>1997 Indonesia (154)</td>
<td>Episode of smoke haze</td>
<td>3000</td>
<td>Monthly birth cohort data examined by region to estimate peri-natal and infant mortality</td>
<td>1% decrease in expected cohort size (16,400 infant deaths) across Indonesia</td>
</tr>
<tr>
<td>1997 Sarawak, Malaysia (155)</td>
<td>Episode of smoke haze (3 months)</td>
<td>930</td>
<td>Hospital admissions</td>
<td>20–80% increase in admissions for asthma and COPD</td>
</tr>
<tr>
<td>1997 Malaysia (157)</td>
<td>Days $PM_{10} &gt; 210 \mu g / m^3$ compared with less than 210 $\mu g / m^3$</td>
<td>423</td>
<td>Mortality</td>
<td>19% increase in all-cause mortality</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Effect greater in ages &lt;1 and &gt;65 y</td>
</tr>
<tr>
<td>1997 Singapore (158)</td>
<td>Episode of smoke haze</td>
<td>150</td>
<td>Outpatient attendances, Hospital admissions Mortality</td>
<td>19% increase in attendances for asthma</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>38% increase in attendances for upper respiratory conditions. No association with hospital admissions or mortality</td>
</tr>
<tr>
<td>1994 Singapore (159)</td>
<td>$PM_{10}$</td>
<td>60</td>
<td>Hospital attendances for asthma</td>
<td>Positive correlation with $PM_{10}$ (not quantified)</td>
</tr>
<tr>
<td><strong>Australia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000 Darwin (8)</td>
<td>Rise of 10 $\mu g / m^3 PM_{10}$</td>
<td>70</td>
<td>Hospital attendances for asthma</td>
<td>20% increase</td>
</tr>
<tr>
<td>1994 Sydney (97)</td>
<td>Inter-quartile range rise of $PM_{10}$</td>
<td>210</td>
<td>Peak Expiratory Flow Rates (PEFR) in children with wheeze</td>
<td>No association</td>
</tr>
<tr>
<td>1997-2000 Brisbane (141)</td>
<td>Days in which bushfire derived $PM_{10}&gt;20$ compared with days $PM_{10}&lt;15$</td>
<td>60</td>
<td>Daily respiratory hospital admissions</td>
<td>19% increase respiratory admissions during bushfire periods</td>
</tr>
<tr>
<td>1994 Sydney (160)</td>
<td>Episode of smoke haze (1 week)</td>
<td>250</td>
<td>Hospital attendances for asthma</td>
<td>No association</td>
</tr>
<tr>
<td>1994 Sydney (161)</td>
<td>Episode of smoke haze (1 week) Daily nephelometry</td>
<td>250</td>
<td>Hospital attendances for asthma</td>
<td>No association with haze period or daily nephelometry</td>
</tr>
<tr>
<td>1991 Sydney (162)</td>
<td>Episode of smoke haze Daily nephelometry</td>
<td>Not stated</td>
<td>Hospital attendances for asthma</td>
<td>30% increase during smoke haze period No association with nephelometry</td>
</tr>
</tbody>
</table>
Episode studies of outdoor biomass smoke and health

The health impacts associated with episodes of smoke pollution from wildfires have been studied in North America (99, 143-149), Europe (150), South East Asia (80, 151-159) and Australia (97, 160-162). In spite of their inherent technical limitations, most of these have demonstrated associations between higher than usual concentrations of atmospheric particles arising from vegetation fires and adverse health effects.

On a global scale, the most severe and most protracted pollution event occurred in association with uncontrolled forest fires in Kalimantan and Sumatra in late 1997 and early 1998. The associated smoke pollution affected many surrounding countries (163). PM$_{10}$ in Singapore peaked at approximately 230 µ/m$^3$, while in Sarawak, Malaysia, PM$_{10}$ peaked at approximately 930 µ/m$^3$ – over 15 times their usual background levels (156). The first studies of associated health outcomes associated with this episode were reported from Singapore and Malaysia. In Singapore, Emmanuel documented a 30% increase in outpatient attendances for haze-related conditions (defined as asthma, rhinitis and upper respiratory symptoms) but did not find an association with mortality (158). However, in Kuala Lumpur Malaysia, Sastry did find a significant linear relationship between PM$_{10}$ and mortality during the pollution episode (157). A large study of hospital admissions by Mott et al in Sarawak found clear associations with admissions for respiratory conditions, especially among those with previous heart or lung conditions (155).

Early reports of the health impacts in Indonesia from the 1997-8 smoke haze were largely descriptive, documenting increases in visits to health facilities across the country, without quantifying the association with indices of the pollution (151, 164). For example, in a description of long-term trends of air pollution in Malaysia, Awang et al presented data from Malaysian hospitals indicating 10 to 20 fold increases in acute respiratory infections, asthma and conjunctivitis coinciding with haze periods (152). Researchers have since used more creative ways to quantify the health impacts in Indonesia where national health outcome data are less readily available than in some other countries. For example Jayachandran (154) used satellite data to identify the geographic areas affected by the haze and matched
this with national census data to estimate perinatal and infant deaths based on ‘missing children’ in the 2000 census. By analyzing sub-district monthly birth cohorts and exploiting the sharp timing and spatial patterns of the pollution he calculated that pre and post-natal exposure to pollution were both inversely associated with survival. He calculated that, overall in Indonesia, the fire-induced pollution caused a 1.0% decrease in cohort size, or over 16,400 ‘missing’ children.

Using similar methods to estimate exposure, Frankenberg et al used an existing cohort study assessing self reported health to examine the impact of the smoke haze (153). They concluded that between 1993 and 1997, individuals who were exposed to haze experienced greater increases in difficulty with respiratory function and activities of daily living than did their counterparts in non-haze areas.

In North America one reported study has examined mortality in relationship to a wildfire episode. Vedal et al compared mortality on two separate days affected by smoke pollution in Denver June 2002 and compared these with mortality for the entire month of June and with June 2001 for both Denver and a control city of similar size (143). Their descriptive comparison could find no association with smoke pollution and mortality. However, even for a city with 2 million people, an exposure period of only 2 days limits the power of the study to find an association.

Six other North American studies have examined wildfire events and reported increases ranging from 30 to 60 percent in patients presenting to health services for respiratory conditions during the episodes of smoke pollution compared with time periods during which it was absent (144-147, 149). The Canadian study (149) used physician billing data rather than hospital or health centre records as their outcome measure and only documented an association with increased attendances for respiratory conditions in one of two regions studied. The study by Mott et al (145) included one of the first evaluations of public health interventions in relation to pollution from wildfires. In a native American township in California, they were able to demonstrate that (a) the use of high-efficiency particulate air
cleaners in private homes and (b) the recollection of public service announcements were both associated with a reduced odds of reporting adverse respiratory health effects.

The largest and most recent report from North America involved 5,551 children participating in the University of Southern California Children's Health Study who were exposed to smoke from several regional wildfires in 2003. Künzli et al (148) took the opportunity to examine the impact of this event by administering a questionnaire to the children that collected information about days of exposure to smoke haze (assessed as the number of days that smoke had been smelled indoors) and respiratory symptoms. Symptoms of nose, eyes, and throat irritations; cough; bronchitis; cold; wheezing; asthma attacks, medication usage and physician visits were all associated with individually reported exposure differences within communities and these risks increased monotonically with the number of reported smoky days. They also examined these outcomes in relation to fire-related PM$_{10}$ for which data were available at a community level and found the reporting rates between communities for most outcomes were also associated measured PM$_{10}$. The main strength of this study was its large size that included several cohorts of children already participating population based study across a wide geographic area. The nesting of the study within the larger cohort study also allowed for later follow up and the attainment of relatively good response rates. The main limitation was that individual objective measurements of exposure were not possible and the impact of reporting and bias remains unknown (165, 166). However the dose response relationship in the analyses that were based self reported exposure and the general consistency of theses results with the findings of the community level studies which did have objective PM measurements both increase confidence in their findings (167).

Contrary to these reasonably consistent results from international studies, only one of three Australian event studies has provided support for an association between smoke from bushfires and adverse health outcomes. Churches and Corbett (162) documented an association between days of smoke haze and hospital attendances for asthma in Sydney in 1991 while Cooper et al (160) and Smith et al (161) were unable to find any relationship between particulate levels and asthma attendances during bushfires in 1994. The study by
Cooper et al was limited by the relatively small number of attendances analysed (less than 100), as they only examined three inner city hospitals. The study by Smith was more comprehensive, although only a single reference period was used for comparison.

In summary, episode studies have provided the majority of evidence concerning the health impacts of exposure to smoke from vegetation fires. It is not possible to directly compare results from these studies because the level and duration of exposure to smoke haze varied widely and was quantified in a number of different ways and different time periods and methods were used to compare the episode with non-episode conditions. It is also not possible to quantify the extent to which an obvious smoke haze might have influenced health seeking behaviour or reporting of symptoms in these studies. Apart from the two negative Australian studies (160, 161), there is general coherence in the findings relating to respiratory outcomes including increasing symptoms, health care attendances, and respiratory hospital admissions in association with episodes of smoke pollution from wildfires. Two studies have also found associations with perinatal and total mortality. All of these outcomes are consistent with current evidence concerning particulate air pollution regardless of source. However, studies specifically concerning wildfire smoke are few in number and it is well recognised that negative studies are less likely to be published (168).

**Intervention studies**

Intervention studies are the corollary of episode studies as they examine the population health benefits of reduced air pollution. There are not many published examples and none specifically concerning ambient biomass smoke. However, documented reductions in morbidity and mortality following improvements in air quality further strengthen the evidence for causality in the associations between particulate pollution and ill-health that have been documented in the wider air pollution literature. For example in the USA, reductions in respiratory and cardiovascular morbidity were noted in association with reduced ambient particulates following the closure of a steel mill, both of which returned to previous levels when the mill was reopened (169). In Germany, air quality improvements (particularly in airborne concentrations of particulates and sulphates) following the re-unification in the early
The 1990s were associated with fewer non-allergic respiratory illness such as bronchitis and sinusitis in children (170). Finally, when the sale of coal was banned in 1990 in Dublin, there was an immediate and sustained reduction in the mean concentration of black smoke by 35.6µg/m³. This was associated with a reduction in cardiovascular deaths by 10% and respiratory deaths by 14%, equating to about 116 fewer respiratory and 243 fewer cardiovascular deaths per year. These improvements were greater than what would have been predicted from available evidence about the short-term relationships between air pollution and mortality (171).

**Summary: the health effects of particulate air pollution and ambient biomass smoke**

There is no doubt that carbon based combustion-derived particles are a major cause of illness and death (84). A recent analysis by Cohen *et al* estimated that outdoor air pollution measured as PM$_{2.5}$, is responsible for about 0.8 million (1.2%) premature deaths each year worldwide (172). Thus, while the magnitude of health effects on individuals is often small, when applied to entire populations the public health impact is considerable (173, 174). Subpopulations most at risk are elderly, those with pre-existing illnesses and children (175-177). The biological mechanisms for the observed adverse health effects appear to include the promotion of allergic responses, inflammation, coagulation and oxidative stress all of which can lead to, or exacerbate, many cardiovascular and respiratory diseases (129, 178-180).

The vast majority of studies have been conducted in urban settings where airborne particulate matter predominantly arises from the combustion of fossil fuels by motor vehicles and industrial plants and far fewer studies have specifically examined outdoor biomass smoke. However, it has been well established that particulate pollution arising from biomass combustion is associated with a distinctive suite of toxic co-pollutants (4, 181). While some authors have suggested that biomass derived particles might be associated with greater respiratory morbidity than particulates from urban and industrial sources, more evidence
about the relative toxicology of this source, and indeed particulates derived from other specific sources, is needed (1, 4, 45).

As vegetation fires increase in frequency and severity around the world, the need for information about the public health impacts of the air pollution they generate is becoming more important (47). The global evidence base, although small, is growing. For example, of the 23 studies presented in Table 3.2, just over half were published in the last two years (2005-7). Apart from the three studies of the same wildfire event near Sydney in 1994, all have documented associations with respiratory symptoms, health care attendances or admissions to hospital. Additionally, two of four studies have found associations with mortality. The magnitude of the reported adverse health effects from biomass smoke has generally been higher, especially for respiratory outcomes, than that reported for urban sources of particulate air pollution. However it is difficult to compare these effects directly as methodological approaches and study size in the study of biomass smoke pollution are usually quite different from other air pollution studies. Important exceptions to this are the two Australian studies that directly compared bushfire derived with other sources of PM$_{10}$ in cities with large populations (141, 142).

Important gaps in the available epidemiological evidence include the short-term relationship between biomass smoke pollution and mortality, non-respiratory health outcomes such as cardiovascular diseases and long-term heath outcomes such as cancer (4). Additionally, further information is needed about the public health impact of lower levels of biomass smoke pollution generated by deliberate burns conducted to reduce fuel loads and prevent catastrophic fires (5) and about the effectiveness of public health interventions to reduce the harm associated with pollution episodes from wildfires (4, 46, 47).
Vegetation fires, particulate air pollution and asthma: a panel study in the Australian monsoon tropics.
Chapter Four

Published paper

Vegetation fires, particulate air pollution and asthma: a panel study in the Australian monsoon tropics


International Journal of Environmental Health Research. 2006 Nov/Dec;16(6):391-404

This paper presents results from the Darwin Asthma Study, the first major research project I conducted as part of this PhD. In this study 251 people with doctor-diagnosed asthma were followed for seven months for daily asthma symptoms, medication use and health service attendances in relation to levels of particulate air pollution.

Author contributions
I was responsible for the study design, questionnaire development, spirometry protocol, recruitment of participants, sub-contracting a company for telephone data collection, data cleaning, data analysis and preparation of the paper for publication. I was guided and supported by my two supervisors, Ross Bailie and Louis Pilotto throughout these stages, and received statistical advice from Stephen Halpin. David Parry supervised aerosol data collections. I supervised a research assistant Anne Myerscough who conducted the majority of the spirometry assessments, collected botanical specimens for the pollen library and coordinated production of the project newsletter for stakeholders. In the early part of the project I also supervised a masters student, Rosalind Webby, who contributed to the asthma study design and conducted some early analyses of the panel data under my supervision.
Abstract

We examined the relationship between particulate matter <10 and <2.5 microns in diameter (PM$_{10}$ and PM$_{2.5}$) generated by vegetation fires and daily health outcomes in 251 adults and children with asthma over a seven month period. Data were analysed using generalised estimating equations adjusted for potential environmental confounders, autocorrelation, weekends and holidays. PM$_{10}$ ranged from 2.6-43.3 µg m$^{-3}$ and was significantly associated with onset of asthma symptoms, commencing oral steroid medication, the mean daily symptom count and the mean daily dose of reliever medication. Similar results were found for PM$_{2.5}$. No associations were found with the more severe outcomes of asthma attacks, increased health care attendances or missed school/work days. These results help fill a gap in the evidence about the population health impacts of lower levels of pollution characteristic of deliberate landscape burning to control fuel loads versus the better documented risks of more intense and severely polluting wildfires.
Introduction

The increasing frequency of severe vegetation fire events throughout the world is just one example of global environmental change that is affecting human well-being (47). For example, fire prone regions of the world such as Australia have documented major and increasing economic and human costs from loss of infrastructure, fire-fighting, deaths and the precipitation of physical and mental illnesses, often with long lasting morbidity (20, 21). In addition to these direct effects, major vegetation fires usually push particulate air pollution concentrations well beyond background levels and can affect population centres far from the fires themselves with measurable impacts on morbidity and mortality (64, 157). This has created an increasing dilemma for park and property managers who recognise that the management of highly fire adapted and fire prone vegetation requires deliberate burning (5). While nearly every major inquiry into fire disasters in Australia has called for much more prescribed burning to reduce the risk of disasters, there has been falling community tolerance of the smoke pollution these fires generate, even though this is usually far less than that generated by wildfires (40).

There is currently insufficient evidence to determine if there is a safe or acceptable level for deliberate population exposure to vegetation smoke or to assess the population health trade-offs between more frequent exposure to low levels of smoke, compared with the less frequent occurrence of severely polluting and dangerous wildfires. Large multi-centre studies from around the world have clearly established a relationship between air borne particulate matter (PM) and several indices of morbidity and mortality with no apparent lower threshold (118, 121, 182, 183). However, most studies have been conducted in urban settings in which PM largely arises from industrial and motor vehicle emissions rather than biomass combustion. The tropical Australian city of Darwin provides a useful setting to examine the health impacts of low levels of vegetation fire smoke. Deliberate landscape burning is a well established management practice and approximately half of the surrounding savanna is burnt during the six month dry season each year (184). Other sources of air pollution are minimal. During the dry season it has been calculated that 95% of atmospheric PM is
derived from vegetation fire smoke while industrial and motor vehicle emissions together contribute just 5% (7).

Our study aimed to gather evidence concerning the safety of exposure to vegetation fire smoke for people with asthma. We examined the relationship between PM and asthma symptoms and medication use in a panel of 251 adults and children over a seven month period in 2004. The study was approved by the human research ethics committees of Charles Darwin University and the joint committee of the Menzies School of Health Research and the Northern Territory Department of Health and Community Services.

**Methods**

**Participants and study setting**

Those eligible for inclusion were adults and children with all of the following: asthma diagnosed by a doctor, presence of asthma symptoms or use of medication for asthma in the last year, resident in the Darwin urban region and access to a telephone. Several methods were used for recruitment. All 44 primary and secondary schools, all 16 pharmacies and 16 of the 29 general practices located in the study region agreed to distribute leaflets inviting their clients with asthma to participate in the study. In addition we contracted a market research company to undertake telephone recruitment using their pre-existing database of residents in the study region supplemented by random dialling from the telephone directory. Participants were informed that many environmental factors, not specifically vegetation fire smoke, were being examined for their effects on asthma.

**Exposure measures**

We measured the daily mean mass of particulates per cubic meter of air with diameters of <10 microns (PM$_{10}$) and <2.5 microns (PM$_{2.5}$). Our primary monitoring site was located close to the main residential areas of the study region. We used a Rupprecht and Patashnick Partisol plus Model 2025 sequential air sampler which provided 24 hour gravimetric
measures of PM$_{10}$ and PM$_{2.5}$. These data were validated by inter-laboratory comparison gravimetric analyses conducted with the Marine and Atmospheric Research division of Australia’s Commonwealth Scientific and Industrial Research Organisation (CSIRO). A secondary monitoring site was established in another major residential area approximately 22 kms from the primary site to confirm the regional nature of air pollution in Darwin and provide a back up for equipment failure at the primary site. Here we used a Rupprecht and Patashnick Tapered Element Oscillating Microbalance (TEOM) series 1400a which provided continuous PM$_{10}$ loadings with 30 minute time resolution. Data from this site were validated by testing in parallel with a co-located Partisol sampler. All equipment was calibrated and maintained according to the manufacturer’s specifications.

The daily concentration of pollen and spores in air was measured using a 7-day electronic spore counter, Burkard Sporewatch® (Burkard Scientific Ltd, Uxbridge, UK) co-located with the air quality monitoring equipment at the primary monitoring site. The spore counter collects airborne particles as they settle onto an adhesive tape on a slowly rotating drum. The seven day tapes were cut into daily lengths, stained and mounted on glass microscope slides. Each slide was viewed by Zeiss light microscope at a magnification of x400 and pollen counted along 4 full lengthwise transects. Due to the much greater abundance of spores the spore counts were completed on 2 separate full lengthwise transects. Counts were then converted to correspond to grains per cubic meter of air as a daily mean value.

Records of the maximum and minimum air temperature, relative humidity, dew point and rainfall measured at Darwin Airport were provided by the Bureau of Meteorology. Weekly consultation rates for influenza-like illness from 14 sentinel general practitioners (GPs) located in Darwin and Palmerston were provided by the NT Department of Health and Community Services. The sentinel GPs use a standard case definition for influenza and record their total weekly numbers of patients. The rate is reported as weekly number of influenza cases per 1000 consultations. This information was used as a marker of respiratory viral activity in the community.
Outcome measures

Demographic, clinical, social and environmental data were collected using a questionnaire completed at the time of enrolment. Clinical questions were adapted from the protocol of the International Study on Asthma and Allergy in Children (185, 186). The questionnaire also included a validated asthma severity scale (reproduced in the appendix) which identifies participants at high risk of hospital attendance over a 12 month period (187). Spirometry was performed on adults and children greater than 8 years of age following the guidelines of the National Asthma Council of Australia (188, 189) using a Welch Allyn Pneumocheck 61000 spirometer. Reversible airflow obstruction was defined as an increase of 15% or more in forced expiratory volume in 1 second (FEV$_1$) following inhaled bronchodilator. Salivary samples were collected, centrifuged, frozen and forwarded to the toxicology department of the Adelaide Women and Children’s Hospital for cotinine estimation by micro-plate enzyme immunoassay to test for tobacco smoke exposure.

Participants used a daily diary to record their asthma symptoms, medication use, missed school or work days and health care visits for asthma. These data were collected and entered in an electronic database via a fortnightly telephone call. We recorded presence or absence of asthma attacks and individual symptoms of cough, breathlessness, wheezing and chest tightness for both day and night. Asthma attacks were defined as any asthma episode involving breathlessness and/or wheezing and/or chest tightness and/or coughing that interrupts ongoing activities or requires some procedures, such as resting or using a nebuliser to resume normal and comfortable breathing. Each medication, strength, dose and number of applications taken each day was recorded individually and classified as follows: (1) short acting inhaled bronchodilator (reliever), (2) inhaled steroids, cromoglycates and oral montelukast (preventers), (3) long acting bronchodilators (symptom controllers) and (4) oral steroids. The main outcome measures examined were the daily proportion of the group that: (1) had any symptoms attributable to asthma, (2) became symptomatic after being symptom free for at least 7 days, (3) used reliever medication, (4) commenced a reliever after at least 7 days without any reliever, (5) commenced a course of oral steroids, (6) experienced exercise induced asthma, (7) experienced an asthma attack, (8) missed school or work due
to asthma and (9) saw a health profession about their asthma. We also examined the mean number of symptoms present and the mean number of times a reliever medication was used each day.

**Statistical analysis**

Data were analysed using Stata8 statistical software package (190). We used generalised estimating equations to generate population averaged models adjusted for minimum daily air temperature, relative humidity, pollen and spore counts, the weekly rate of consultations to general practitioners for influenza-like illness, temporal autocorrelation of outcomes, weekends and holiday periods. We examined the relationship between same day exposures and lags of up to 5 days to both PM$_{10}$ and PM$_{2.5}$. Logistic regression models were used to calculate odds ratios (OR) for dichotomous outcomes and negative binomial regression models were used to calculate incidence rate ratios (IRR) for count outcomes. Subgroup analyses were conducted on adults and children separately and on participants who reported either moderate or severe asthma, using a preventer, or meeting criteria for at being at high risk of hospital attendance. Subgroup analyses were further adjusted for age (adult vs child), self reported severity, use of a preventer, smoking status (smoker vs non-smoker) Indigenous status, household crowding (ratio of people to bedrooms) and education level (highest qualification of any adult in the household).

**Missing PM data**

For single missing values of just one day, data were replaced by the average of the readings before and after the missing value. For gaps of two or more days PM$_{10}$ data from the secondary site were used to estimate values for the primary site as results from the two sites were correlated. The nine missing values from the primary site between 7th April and 7th June were calculated by multiplying the values recorded at the secondary site by 2.1, the ratio of the mean values at the two sites for that period. For the remainder of the study the mean PM$_{10}$ readings were very similar (ratio=1.03) and missing data at the primary site were directly replaced with the readings from the secondary site. Missing PM$_{2.5}$ data were
calculated by multiplying the PM$_{10}$ value for that day by 0.56 as this ratio remained consistent throughout the study period.

We examined the sensitivity of our results to the imputed values by conducting additional analyses using imputed data values set 25% higher and 25% lower than in our original estimation. We also examined outcomes against PM$_{10}$ data from the secondary site only.

**Results**

**Participants**

Two hundred and fifty one people started the study of whom 235 (94%) contributed data for the full duration of the study. An average of 210 people contributed data each day. Of the 16 who dropped out 14 relocated away from the study site and 2 relocated and became un-contactable. Their data were included for the time they participated. An average of 5% (range 2-14%) were absent from the study region each day. The proportion of absent participants was higher during school holidays (mean = 8%) and peaked at 14% over the Easter break. Individual data were excluded from the analysis for the days in which participants were absent. The mean number of days contributed per participant was 186 out of a maximum possible of 214 (87%).

Approximately half the group were less than 18 years of age (Figure 4.1) and 57% identified vegetation fire smoke as a trigger of their asthma symptoms. 74% classified their asthma as being mild and 26% met criteria for being at high risk of a hospital attendance within 12 months. At enrolment 63% said they used regular inhaled steroids, however the average daily proportion of participants that actually used an inhaled preventer during the study period was 39% (range 36-43%). The demographic and clinical characteristics reported by participants at the time of enrolment are summarised in Table 4.1. Our group had similar characteristics to people with asthma in the general Australian population reported by the National Health Survey in 2001 (191). We had a higher proportion who initially reported regular use of inhaled steroids (63% vs 39%), a slightly lower proportion of adult
smokers (22% vs 26%), a greater predominance of males among children (63% vs. 56%) and females among adults (72% vs. 58%). We also had a higher proportion of Indigenous participants compared with the 2001 census count for Darwin (13% vs. 9%) and a high proportion of participants living in a house in which any adult had a tertiary or trade qualification (72%). In the 2001 census 37% of adults in Darwin and 35% adult Australians reported holding a tertiary qualification (192, 193).

**PM data quality and availability**

PM10 and PM2.5 data measured at the primary site were found to be valid and reliable on independent quality control checks. Data at this site were available for 166 of the 215 days of the study period (77%) with gaps ranging from 1 to 12 days in length. Data from the TEOM also met all quality control checks and were more complete (87%).
Table 4.1  Characteristics of participants in the Darwin Asthma Study at baseline, Darwin, February/March 2004.

<table>
<thead>
<tr>
<th>Participant characteristics</th>
<th>Adults N=130</th>
<th>Children N=121</th>
<th>Total N =251</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio/demographic indicators</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>63</td>
<td>45</td>
</tr>
<tr>
<td>Female</td>
<td>72</td>
<td>37</td>
<td>55</td>
</tr>
<tr>
<td>Indigenous</td>
<td>7</td>
<td>19</td>
<td>13</td>
</tr>
<tr>
<td>Smoker</td>
<td>22</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>Exposure to environmental tobacco smoke at home</td>
<td>26</td>
<td>35</td>
<td>30</td>
</tr>
<tr>
<td>Ratio of people:bedrooms &lt;1</td>
<td>46</td>
<td>7</td>
<td>28</td>
</tr>
<tr>
<td>Ratio of people:bedrooms =1</td>
<td>25</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>Ratio of people:bedrooms &gt;1</td>
<td>29</td>
<td>26</td>
<td>27</td>
</tr>
<tr>
<td>Adult in house with a tertiary qualification</td>
<td>72</td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td><strong>Clinical features</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Using regular inhaled steroids</td>
<td>68</td>
<td>57</td>
<td>63</td>
</tr>
<tr>
<td>Mild asthma in the last month</td>
<td>76</td>
<td>70</td>
<td>73</td>
</tr>
<tr>
<td>Moderate asthma in the last month</td>
<td>19</td>
<td>27</td>
<td>23</td>
</tr>
<tr>
<td>Severe asthma in the last month</td>
<td>5</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Used oral steroids in the last month</td>
<td>12</td>
<td>17</td>
<td>14</td>
</tr>
<tr>
<td>Woken from sleep with asthma in the last month</td>
<td>34</td>
<td>40</td>
<td>37</td>
</tr>
<tr>
<td>Admission to hospital for asthma in last 12 months</td>
<td>5</td>
<td>16</td>
<td>10</td>
</tr>
<tr>
<td>Saw more than 1 GP for asthma in last 12 months</td>
<td>22</td>
<td>31</td>
<td>26</td>
</tr>
<tr>
<td>High risk of hospital attendance*</td>
<td>19</td>
<td>33</td>
<td>26</td>
</tr>
<tr>
<td>History of eczema or hay fever</td>
<td>79</td>
<td>51</td>
<td>65</td>
</tr>
<tr>
<td>Positive cotinine in non-smoker</td>
<td>5</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Reversible airflow obstruction** (of 109 adults and 42 children able to perform spirometry)</td>
<td>11</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td><strong>Reported triggers of asthma</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cold or respiratory viral infection</td>
<td>58</td>
<td>59</td>
<td>59</td>
</tr>
<tr>
<td>Change in weather</td>
<td>68</td>
<td>63</td>
<td>65</td>
</tr>
<tr>
<td>Bushfire smoke</td>
<td>62</td>
<td>52</td>
<td>57</td>
</tr>
<tr>
<td>Exercise</td>
<td>57</td>
<td>48</td>
<td>53</td>
</tr>
<tr>
<td>Dust</td>
<td>65</td>
<td>38</td>
<td>52</td>
</tr>
<tr>
<td>Pollen</td>
<td>56</td>
<td>31</td>
<td>43</td>
</tr>
<tr>
<td>Cigarette smoke</td>
<td>58</td>
<td>24</td>
<td>41</td>
</tr>
<tr>
<td>Animal hair/fur</td>
<td>41</td>
<td>20</td>
<td>31</td>
</tr>
<tr>
<td>Foods</td>
<td>27</td>
<td>11</td>
<td>19</td>
</tr>
<tr>
<td>Swimming pools</td>
<td>3</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Drugs</td>
<td>10</td>
<td>2</td>
<td>6</td>
</tr>
</tbody>
</table>

*according to the severity scale developed by Wakefield et al (187)

**FEV1 increase of 15% following inhaled bronchodilator

Environmental conditions during the study

Metrological conditions were unusual during the study period. Rainfall, usually a rare event in the tropical dry season, persisted well into June and delayed the curing of the savanna grasses. This resulted in lower than usual fire hazard conditions and lower than expected air pollution during the study. Environmental data are summarised in Table 4.2.
Table 4.2 Description of measured environmental variables, 7 April - 7 November, Darwin 2004.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Mean</th>
<th>Standard deviation</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM$_{10}$ at primary monitoring site (µg/m$^3$)</td>
<td>20.0</td>
<td>6.4</td>
<td>2.6 – 43.3</td>
</tr>
<tr>
<td>PM$_{2.5}$ at primary monitoring site (µg/m$^3$)</td>
<td>11.1</td>
<td>5.4</td>
<td>2.2 – 36.5</td>
</tr>
<tr>
<td>PM$_{10}$ at secondary monitoring site (µg/m$^3$)</td>
<td>18.0</td>
<td>8.4</td>
<td>2.9 – 53.7</td>
</tr>
<tr>
<td>Total pollen count (grains/m$^3$)</td>
<td>16.4</td>
<td>9.4</td>
<td>0.5 – 60.5</td>
</tr>
<tr>
<td>Grass pollen count (grains/m$^3$)</td>
<td>3.5</td>
<td>5.0</td>
<td>0 – 22.2</td>
</tr>
<tr>
<td>Fungal spore count (spores/m$^3$)</td>
<td>1843</td>
<td>1043</td>
<td>0 – 6049</td>
</tr>
<tr>
<td>Daily maximum temperature (degrees C)</td>
<td>31.8</td>
<td>2.1</td>
<td>25.7 – 36.1</td>
</tr>
<tr>
<td>Daily minimum temperature (degrees C)</td>
<td>21.3</td>
<td>3.2</td>
<td>13.3 – 27.5</td>
</tr>
<tr>
<td>Relative humidity at 9.00am (percent)</td>
<td>64.8</td>
<td>14.8</td>
<td>9 – 96</td>
</tr>
<tr>
<td>Weekly GP consultation rate for influenza-like illness (rate/1000 consultations)</td>
<td>12.2</td>
<td>6.8</td>
<td>3.3 – 27.3</td>
</tr>
</tbody>
</table>

Particulate levels fluctuated with several peaks and troughs throughout the study period. The PM$_{10}$ often climbed or fell by 15-20 µg/m$^3$ over periods of just a few days. Australia’s national air quality target for PM$_{10}$ of 50 µg/m$^3$ was exceeded once at the secondary monitoring site only. PM$_{2.5}$ exceeded the national advisory reporting standard of 25 µg/m$^3$ on 5 days. PM$_{2.5}$ was highly correlated with PM$_{10}$ at the primary site ($r=0.9$) with a mean value of 56% of PM$_{10}$.

For the first 3 months of operation PM$_{10}$ data from the TEOM at the secondary site was consistently half that recorded by the Partisol sampler at the primary site. There was no obvious explanation for this difference. Thereafter readings from the two sites remained similar apart from two episodes in which PM$_{10}$ peaked at a higher level at the secondary site. Both these episodes could be attributed to local fires closer to the secondary site. Measurements from the two sites were correlated ($r=0.7$).

Pollen and fungal counts were low compared with other Australian capital cities (194). The weekly proportion of GP consultations for influenza-like illnesses fluctuated with two small peaks during March and June.
**Health outcomes**

The frequency of health outcomes among participants are shown in Table 4.3. Significant first order autocorrelation was found for all outcomes except for the less frequent events of commencing reliever, commencing oral steroids, missing school or work due to asthma, and seeing a health professional about asthma.

**Relationship between particulate matter and health outcomes**

We found small associations between PM$_{10}$ and PM$_{2.5}$ and the proportion of participants starting a course of oral steroids and becoming symptomatic following a symptom free period. Additionally there were associations between PM$_{2.5}$ and starting to use reliever medication and between PM$_{10}$ and the average number of asthma symptoms and the mean number of times reliever medication was used by participants each day. Most associations were of slightly greater magnitude in adults compared with children and in those using a preventer (Table 4.4). We did not find any associations between pollution levels and exercise induced asthma, asthma attacks, missed work or school due to asthma or health care attendances for asthma in the whole group. However an inverse association was observed between asthma attacks and PM$_{2.5}$ in children. When we examined outcomes for time lags of up to 5 days associations were identified at lags of one day between PM$_{10}$ and onset of symptoms and PM$_{2.5}$ with both onset of symptoms and commencing a reliever (data not shown). Lags of 2 – 5 days were not associated with any of the health outcomes measured. These outcomes were not substantially altered by using the 25% higher or lower estimations for the imputed PM data. When we examined outcomes against PM$_{10}$ recorded from the secondary monitoring site we also found very similar results. The main differences with the latter analysis were that the association between PM$_{10}$ and commencement of reliever medication achieved statistical significance while the association between PM$_{10}$ and commencement of oral steroids lost statistical significance.
Table 4.3 Summary of outcomes measures from the Darwin Asthma Study, Darwin, April – November, 2004.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Mean daily percentage</th>
<th>Standard deviation</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheeze</td>
<td>9.7</td>
<td>2.5</td>
<td>3.4-18.4</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>10.6</td>
<td>2.4</td>
<td>6.0-17.4</td>
</tr>
<tr>
<td>Cough</td>
<td>20.0</td>
<td>3.9</td>
<td>11.1-31.6</td>
</tr>
<tr>
<td>Chest tightness</td>
<td>9.8</td>
<td>2.4</td>
<td>4.7-16.1</td>
</tr>
<tr>
<td>Any symptom(s) attributable to asthma</td>
<td>25.7</td>
<td>4.5</td>
<td>15.8-37.7</td>
</tr>
<tr>
<td>Become symptomatic (after at least one symptom free week)</td>
<td>1.8</td>
<td>1.2</td>
<td>0-5.7</td>
</tr>
<tr>
<td>Exercise induced asthma (of those who exercised)</td>
<td>6.8</td>
<td>2.0</td>
<td>2.1-13.0</td>
</tr>
<tr>
<td>Asthma attack</td>
<td>3.0</td>
<td>1.2</td>
<td>0.9-8.4</td>
</tr>
<tr>
<td>Used reliever</td>
<td>23.2</td>
<td>3.0</td>
<td>16.1-31.2</td>
</tr>
<tr>
<td>Commenced reliever (after at least one week without using reliever)</td>
<td>1.45</td>
<td>1.1</td>
<td>0-5.45</td>
</tr>
<tr>
<td>Used any preventer (including inhaled steroids, cromolyn and montelukast)</td>
<td>44.7</td>
<td>1.9</td>
<td>39.8-48.9</td>
</tr>
<tr>
<td>Used inhaled steroids</td>
<td>39.5</td>
<td>1.6</td>
<td>36-42.8</td>
</tr>
<tr>
<td>Used combination preventer/symptom controller</td>
<td>25.7</td>
<td>1.4</td>
<td>22.7-29.5</td>
</tr>
<tr>
<td>Used leukotriene antagonists</td>
<td>4.8</td>
<td>0.6</td>
<td>3.2-6.0</td>
</tr>
<tr>
<td>Used inhaled cromoglycates</td>
<td>2.3</td>
<td>0.5</td>
<td>1.4-3.9</td>
</tr>
<tr>
<td>Used symptom controller</td>
<td>2.6</td>
<td>0.4</td>
<td>1.8-4.1</td>
</tr>
<tr>
<td>Used oral steroids</td>
<td>1.1</td>
<td>0.6</td>
<td>0-3.0</td>
</tr>
<tr>
<td>Commenced oral steroids (after at least one week without oral steroids)</td>
<td>0.12</td>
<td>0.2</td>
<td>0-0.9</td>
</tr>
<tr>
<td>Saw a health professional about asthma</td>
<td>0.6</td>
<td>0.5</td>
<td>0-2.3</td>
</tr>
<tr>
<td>Missed school or work due to asthma</td>
<td>0.8</td>
<td>0.9</td>
<td>0.3-9.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Counted outcomes</th>
<th>Mean daily count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily number of asthma symptoms (maximum of 8)</td>
<td>0.8</td>
</tr>
<tr>
<td>Daily number of times reliever was used</td>
<td>0.6</td>
</tr>
</tbody>
</table>
Relationship between other environmental exposures and health outcomes

We found small associations between the following environmental exposures and health outcomes: (1) minimum air temperature and the prevalence of asthma symptoms (an inverse association, OR 0.98, 95%CI 0.97, 0.99 p=0.03); (2) GP consultation rates for influenza and the prevalence of asthma symptoms (OR 1.06, 95%CI 1.01, 1.12 p=0.025); and (3) the fungal spore count that was associated with both onset of asthma symptoms (OR 1.0001, 95%CI 1.00002, 1.0002 p=0.011) and commencement of reliever medication (OR 1.0002, 95%CI 1.000014, 1.0007 p=0.020). We did not find any health outcomes associated with pollen levels, rainfall, relative humidity, dew point or maximum air temperature.

In absolute terms most of the associations we identified were due to response variation in just 5% of the group or 10-11 participants.

Discussion

In this study we identified associations between relatively low levels of PM\textsubscript{10} and PM\textsubscript{2.5} derived from vegetation fire smoke and asthma symptoms and medication use, but not health care attendances or missed school or work due to asthma.

These findings are consistent with many epidemiological studies that have described linear relationships with no apparent lower threshold between particulate air pollution and a wide range of adverse health outcomes including all cause mortality and exacerbations of respiratory and cardiovascular diseases (3, 96, 195-197). Toxicological studies have also shown that exposure to particulate matter promotes inflammation, an important pathophysiological mechanism in exacerbations of asthma (47, 178, 179).

Our results are largely consistent with the few previously published epidemiological studies specifically examining the health impacts of particulate pollution from vegetation fires. Of the
Table 4.4 Relationships between particulate matter and health outcomes. Darwin, April-November 2004.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>PM$_{10}$ (rise of 10 µg/m$^3$)</th>
<th>p</th>
<th>PM$_{2.5}$ (rise of 5 µg/m$^3$)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms attributable to asthma</td>
<td>OR 95%CI</td>
<td>p</td>
<td>OR 95%CI</td>
<td>p</td>
</tr>
<tr>
<td>Overall</td>
<td>1.010 0.98-1.04 0.401</td>
<td>1.000 0.98-1.01 0.848</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.027 0.98-1.068 0.183</td>
<td>1.000 0.976-1.026 0.944</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>0.930 0.96-1.057 0.641</td>
<td>1.008 0.980-1.037 0.558</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.022 0.985-1.060 0.243</td>
<td>1.013 0.990-1.037 0.249</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Became symptomatic</td>
<td>Overall 1.240 1.106-1.39 0.000</td>
<td>1.150 1.071-1.23 0.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.277 1.084-1.504 0.003</td>
<td>1.165 1.058-1.284 0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>1.247 1.058-1.468 0.008</td>
<td>1.148 1.042-1.264 0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.317 1.124-1.543 0.001</td>
<td>1.181 1.076-1.296 0.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Used reliever</td>
<td>Overall 1.010 0.99-1.04 0.264</td>
<td>1.000 0.98-1.02 0.788</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.026 0.990-1.063 0.147</td>
<td>1.007 0.980-1.035 0.573</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>1.006 0.960-1.055 0.779</td>
<td>1.002 0.972-1.034 0.861</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.035 1.004-1.06 0.024</td>
<td>1.020 1.000-1.042 0.050</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Became symptomatic</td>
<td>Overall 1.132 0.99-1.29 0.065</td>
<td>1.120 1.031-1.210 0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.199 0.994-1.446 0.57</td>
<td>1.141 1.021-1.275 0.019</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>1.093 0.906-1.319 0.93</td>
<td>1.112 0.994-1.243 0.061</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.194 0.996-1.432 0.55</td>
<td>1.129 1.013-1.257 0.028</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Commenced reliever</td>
<td>Overall 1.540 1.01-2.34 0.046</td>
<td>1.310 1.031-1.66 0.023</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.752 1.008-3.045 0.047</td>
<td>1.601 1.192-2.150 0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>1.292 0.682-2.448 0.431</td>
<td>0.995 0.625-1.459 0.832</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.430 0.888-2.304 0.141</td>
<td>1.350 1.040-1.752 0.024</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Commenced oral steroids</td>
<td>Overall 1.030 0.95-1.12 0.434</td>
<td>0.980 0.94-1.04 0.639</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.08 0.976-1.202 0.129</td>
<td>1.026 0.962-1.095 0.424</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>0.861 0.710-1.044 0.129</td>
<td>0.832 0.731-0.946 0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.051 0.939-1.175 0.382</td>
<td>1.002 0.934-1.075 0.937</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma attack</td>
<td>Overall 0.980 0.92-1.05 0.667</td>
<td>0.990 0.95-1.03 0.741</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>0.988 0.902-1.081 0.793</td>
<td>0.998 0.943-1.065 0.95</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>0.972 0.844-1.119 0.696</td>
<td>0.982 0.899-1.071 0.684</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.026 0.928-1.134 0.608</td>
<td>1.002 0.942-1.067 0.929</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise induced asthma</td>
<td>Overall 1.030 0.85-1.26 0.742</td>
<td>1.030 0.911-1.16 0.652</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.064 0.794-1.424 0.676</td>
<td>1.079 0.899-1.296 0.412</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>0.998 0.749-1.328 0.989</td>
<td>1.003 0.841-1.195 0.973</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>0.924 0.731-1.169 0.513</td>
<td>0.980 0.847-1.133 0.803</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saw a health professional for asthma</td>
<td>Overall 1.102 0.941-1.290 0.226</td>
<td>1.025 0.928-1.310 0.628</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.135 0.897-1.435 0.290</td>
<td>1.077 0.923-1.247 0.323</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>1.073 0.862-1.333 0.527</td>
<td>1.000 0.873-1.458 0.998</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.025 0.857-1.228 0.783</td>
<td>1.005 0.897-1.124 0.936</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missed school or work due to asthma</td>
<td>Overall 1.020 1.001-1.031 0.014</td>
<td>1.003 0.99-1.01 0.594</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Adults</td>
<td>1.027 1.005-1.049 0.017</td>
<td>0.998 0.984-1.012 0.799</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Children</td>
<td>1.016 0.986-1.047 0.304</td>
<td>1.004 0.985-1.023 0.669</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Using preventer</td>
<td>1.034 1.011-1.058 0.004</td>
<td>1.013 0.999-1.028 0.067</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

All analyses adjusted for minimum air temperature, humidity, rainfall, pollen count, spore count, influenza rates, weekends, holiday periods and temporal autocorrelation. OR=Odds ratio IRR=Interval rate ratio CI=confidence interval Bold=p<0.05.

*Subgroup analyses additionally adjusted for age, smoking, education status, Indigenous status, household crowding, self reported severity and current use of preventer.
twelve published studies we identified, nine found associations with a range of health outcomes including hospital or clinic attendances for respiratory conditions (8, 144, 145, 147, 158, 162), exacerbation of symptoms in a cohort of people with chronic obstructive pulmonary disease (99) and hospital admissions and all cause-mortality (155, 157). The remaining three, all of which studied fires near Sydney in 1994, were negative. (97, 160, 161). Jalaludin et al.(97) were recruiting children for a longitudinal study of lung function when the fires occurred. Their analysis was based on data from just 32 participants and might have lacked sufficient statistical power. The other two studies, which examined hospital presentation data for asthma, were subject to the inherent limitations of retrospective studies. Relying on the examination of routinely collected data limits the ability to adjust for important confounders such as weather, pollen levels and community activity of respiratory viral illnesses (6). Moreover, pollution from fires is often subject to considerable media attention which may influence the medication use and health seeking behaviour of people with diseases such as asthma.

Our study was able to overcome many of these limitations because it was located where a prolonged period of fluctuating smoke haze is a predictable annual phenomenon and other sources of air pollution are minimal (7). Data were collected prospectively over a 7 month period during which the day to day variations in pollution were not obvious to the general public and received little media attention. We were also able to control for pollen counts, fungal spores, dew point, humidity, temperature, rainfall, influenza rates, days of the week, public holidays and school holidays all of which could confound the association between PM and health outcomes.

There are several potential explanations why we did not find any associations between particulate levels and the more severe outcomes of asthma attacks, missed school or work, and attending a health service for asthma. The impacts of air pollution might be less severe at relatively lower levels of pollution, we might not have had sufficient power to detect associations with the outcomes that were infrequent in this study, or this could have partly been due to the particular characteristics of our participants. Although their age and gender
distributions were broadly similar to people with asthma in the general Australian population (191), we may have had a greater proportion of people competent in managing their (or their children’s) asthma as evidenced by the higher proportion of households containing a person with a tertiary qualification and the higher proportion of participants using regular preventers. The latter observation might also reflect a group with more severe disease. Subgroup analysis by use of preventers had very similar outcomes to the overall findings with just a slightly greater magnitude in the association of PM with some symptoms and medication use. The inverse relationship we observed between asthma attacks and PM$_{2.5}$ in children could have been a chance finding. It is biologically implausible outcome and did not fit with the overall pattern of findings in this study.

An important contribution of our study is that we were able to examine PM derived from vegetation fire smoke at levels considerably lower than those examined in previously published epidemiological studies and well below Australia’s air quality standards for PM$_{10}$ and PM$_{2.5}$ (198). This helps to fill an important gap in the evidence concerning the public health consequences of lower levels of pollution generated by smaller fires deliberately lit for land management purposes (34, 46). The risks of this practice need to be put in context with the risks of failing to manage fuel loads, particularly the economic, health and ecological costs of uncontrolled wildfires (5, 22). Pollution generated by major fires is usually considerably greater than that produced by prescribed burns and important public health impacts can be expected if large populations are exposed. For example, although there were no direct deaths recorded from the Sydney fires of 2001 in which the PM$_{10}$ remained above 150 µg/m$^3$ for 10 days, Australia’s National Environment Protection Council (NEPC) estimated the excess pollution would have been responsible for 16 deaths and at least 30 hospital admissions (22).

**Conclusion**

We have identified several clinically important associations between symptoms and medication use for asthma and particulate air pollution (PM$_{10}$ and PM$_{2.5}$) at levels below current national air quality targets. Our findings add to the growing literature about the
adverse health impacts of population exposure to smoke from vegetation fires. The public health risks of deliberate burning for fuel management should be examined in relation to the better documented, more severe risks of large uncontrolled fires. While deliberate burning may be justifiable in this context, minimisation of population exposure to particulate pollution should remain a high priority.

Acknowledgements and funding

Thanks are due to our research assistants Anne Myerscough, Judy Manning and Francoise Foti, and collaborators Michael Foley, Simon Haberle, David Bowman and John Gras. This study was funded by a linkage grant from the Australian Research Council with cash and in kind support from the Northern Territory Government, Bureau of Meteorology and Asthma NT.
Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin Australia.

Left: Partisol air sampler, Darwin 2005. Right: Filter papers from the air sampler showing grey discoloration from accumulated particulate matter.
Chapter five

Published paper

Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin Australia.

Fay H Johnston, Ross S Bailie, Louis S Pilotto and Ivan C Hanigan

BMC, Public Health 2007, 7:240

This chapter reports the second study I conducted as part of this thesis. For this study I examined the relationship between particulate air pollution and hospital admissions for cardio-respiratory conditions over a three year period using a case-crossover analysis.

Author contributions

I was responsible for the study design, statistical analyses and writing the paper and was guided and supported by my two supervisors, Ross Bailie and Louis Pilotto throughout the study. I supervised a research assistant, Ivan Hanigan who assisted with data cleaning and management and final preparation of the paper.
Abstract

Background

Increasing severe vegetation fires worldwide have been attributed to both global environmental change and land management practices. However, there is little evidence concerning the population health effects of outdoor air pollution derived from biomass fires. Frequent seasonal bushfires near Darwin, Australia provide an opportunity to examine this issue. We examined the relationship between atmospheric particle loadings <10 microns in diameter (PM$_{10}$), and emergency hospital admissions for cardio-respiratory conditions over the three fire seasons of 2000, 2004 and 2005. In addition we examined the differential impacts on Indigenous Australians, a high risk population subgroup.

Methods

We conducted a case-crossover analysis of emergency hospital admissions with principal ICD10 diagnosis codes J00-J99 and I00-I99. Conditional logistic regression models were used to calculate odds ratios for admission with 10µg/m$^3$ rises in PM$_{10}$. These were adjusted for weekly influenza rates, same day mean temperature and humidity, the mean temperature and humidity of the previous three days, days with rainfall > 5mm, public holidays and holiday periods.

Results

PM$_{10}$ ranged from 6.4 - 70.0 µg/m$^3$ (mean 19.1). 2466 admissions were examined of which 23% were for Indigenous people. There was a positive relationship between PM$_{10}$ and admissions for all respiratory conditions (OR 1.08 95%CI 0.98, 1.18) with a larger magnitude in the Indigenous subpopulation (OR1.17 95%CI 0.98, 1.40). While there was no relationship between PM$_{10}$ and cardiovascular admissions overall, there was a positive association with ischaemic heart disease in Indigenous people, greatest at a lag of 3 days (OR 1.71 95%CI 1.14, 2.55).
Conclusions

PM$_{10}$ derived from vegetation fires was predominantly associated with respiratory rather than cardiovascular admissions. This outcome is consistent with the few available studies of ambient biomass smoke pollution. Indigenous people appear to be at higher risk of cardio-respiratory hospital admissions associated with exposure to PM$_{10}$. 
Background

Short-term associations between cardiovascular and respiratory hospital admissions and particulate air pollution have been demonstrated in many settings around the world, including multi-city studies in Europe, North America and Australia (118, 139, 199-201). In most of these settings the main source of particles is fossil fuel combustion by industrial plants and transport, although dust and biomass combustion can also make important contributions. An important gap in the currently available evidence concerns the roles of different sources of particles in contributing to ill health (130). Although indoor biomass smoke is well recognised as a major cause of death and illness in developing countries (83), there is little evidence about the relative population health impacts of biomass combustion compared with other sources of airborne particulates (4). However biomass combustion is becoming increasingly important as a source of ambient air pollution. The use of wood and other biomass fuels increased faster than overall energy demand in North America during the 1990s (202). This has been attributed to its lower cost, renewable nature and a perception that wood smoke may be less harmful than exhaust from fossil fuel combustion (4). Additionally there is a world wide increase in severe vegetation fire events associated with climate change and shifts in population settlement patterns (47, 56). The smoke from such fires has the potential to travel vast distances and affect major population centres far from the fires (64). The increase in wild fires has prompted an increase in deliberate landscape burning to reduce fuel loads and avert major disasters but this practice has become increasingly controversial as the adverse health effects of particulate air pollution become more widely known (5). While it has been argued that deliberate population exposure to smoke from management fires is justifiable to prevent large wild fires, there is an immediate need for evidence concerning the public health risks or potential benefits of various burning regimes (11).

The city of Darwin, northern Australia, provides a useful setting to examine the population health impacts of outdoor air pollution from biomass combustion. Here, approximately 95% of particulate pollution is derived from fires in the surrounding savanna, which cause a smoke haze of variable severity over the city for up to eight months each year (7). Of the
110,000 residents of the city, 11,500 (approximately 11%) are Indigenous and 25,000 less than 15 years of age (192). The relatively high proportion of indigenous Australians provides an opportunity to examine the differential impact of ambient air pollution in this population subgroup. Indigenous Australians have a disproportionate burden of social disadvantage, diabetes, and chronic heart and lung conditions, all of which modify the effect of air pollution on health (203-209). Examination of the magnitude of the likely disproportionate impact of air pollution in this group has been identified as an area of research priority by Australia’s Environment Protection and Heritage Council (210).

We examined the relationship between atmospheric particle loadings 10 microns or less in diameter (PM$_{10}$) and hospital admissions for respiratory and cardiovascular conditions for the three fire seasons (April to November) of 2000, 2004 and 2005. Air quality was not measured during 2001-2003.

**Methods**

**Study Design**

We used a case-crossover design, in which each case is their own control (115). Comparison of environmental data is made between the days that each case was admitted to hospital, and several referent days on which they were not admitted. Measured and unmeasured individual variables such as age and smoking status are controlled by this design. The referent days were selected from the same month and year and matched by day of week of the admission. This time-stratified method of selecting comparison days has been recommended as it ensures unbiased conditional logistic regression estimates and avoids bias resulting from time trends in the environmental exposures being examined (211).

**Exposure Measures**

During 2000 PM$_{10}$ was measured using a Rupprecht and Patashnick Tapered Element Oscillating Microbalance (TEOM) series 1400a. These data were validated by comparison with filter collections using an Ecotech MicroVol aerosol sampler with a 10 micron size.
selective inlet (7). During 2004 and 2005 we used a Rupprecht and Patashnick Partisol plus model 2025 air sampler which provided 24 hour filter collections of PM$_{10}$ ($\mu g/m^3$) that were subsequently weighed on a mass balance. These data were validated by inter-laboratory comparison of gravimetric analyses conducted with the Marine and Atmospheric Research Division of Australia’s Commonwealth Scientific and Industrial Research Organisation (CSIRO). Our monitoring site was located close to the main residential areas. Particulate air pollution in Darwin has been demonstrated to be regional with high correlations between monitors located up to 25km apart (212).

Daily meteorological data were provided by the Bureau of Meteorology and weekly consultation rates for influenza-like illness were provided by the Northern Territory (NT) Department of Health and Community Services from data routinely gathered from sentinel general practitioners.

**Outcome Measures**

Hospital admission data were collected by the Royal Darwin Hospital (RDH), the single major public hospital and referral centre for the northern half of the Northern Territory. Data were not available from the only other facility, a smaller private hospital that did not have an emergency department operating during the study periods. At discharge from hospital, separation diagnoses are assigned according to the International Classification of Diseases version 10 (ICD10)(213). De-identified emergency admissions data for 2000, 2004 and 2005 with a principal ICD10 diagnosis code for respiratory and circulatory conditions were extracted from the Northern Territory Government database (Table 5.1). These included patients admitted through the hospital’s emergency department or by direct arrangement with private doctors. Each admission included details of date of birth, gender, ethnicity, ICD principal diagnosis code, occupation, place of residence, dates of attendance, admission and discharge and a unique identifier. These data were cleaned by identifying gaps and errors in the data extraction process and by finding and eliminating duplicate records. Those whose primary residential address was not in Darwin were excluded. The excluded group comprised residents of rural communities adjacent to Darwin, remote towns and Indigenous
communities within the NT and interstate travellers. We included the first admission only for each episode of illness by excluding readmissions within 4 weeks of discharge.

Table 5.1 Clinical conditions and ICD codes examined for admissions to Royal Darwin Hospital. Darwin, April – November in 2000, 2004 and 2005 (N=724 days).

<table>
<thead>
<tr>
<th>Clinical group</th>
<th>ICD10 codes</th>
<th>Number of Admissions</th>
<th>Number Indigenous</th>
<th>Number &lt;15 years old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory conditions - all</td>
<td>J00-J99</td>
<td>1474</td>
<td>384</td>
<td>548</td>
</tr>
<tr>
<td>Asthma</td>
<td>J45-46</td>
<td>253</td>
<td>65</td>
<td>149</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>J40 – J44</td>
<td>305</td>
<td>76</td>
<td>0</td>
</tr>
<tr>
<td>Respiratory infections</td>
<td>J00-J22</td>
<td>778</td>
<td>218</td>
<td>378</td>
</tr>
<tr>
<td>Cardiovascular conditions - all</td>
<td>I00 – I99</td>
<td>992</td>
<td>186</td>
<td>12</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>I20 – I25</td>
<td>422</td>
<td>81</td>
<td>0</td>
</tr>
</tbody>
</table>

**Data Analysis**

Respiratory and cardiovascular admissions were described according to Indigenous status and specific diagnostic groups for which associations with particulate air pollution have been previously documented. We used conditional logistic regression models to calculate odds ratios (OR) and 95% confidence intervals (CI) for hospital admission in relation to variation in \( \text{PM}_{10} \) adjusted for weekly influenza rate, days with rainfall > 5mm, same day mean temperature and humidity, the mean temperature and humidity of the previous three days and public holidays. These potential confounders were chosen *a priori* and included in all models. School holidays were additionally included as a dummy variable for all respiratory conditions, asthma, and respiratory infections as these groups included a substantial proportion of children (see Table 5.1).
Results

Environmental conditions and weekly influenza rates during the study period are summarised in Table 5.2. PM$_{10}$ levels fluctuated throughout each dry season and often climbed or fell by 15-20 µg/m$^3$ over periods of just a few days. Australia’s national air quality target for PM$_{10}$ of 50 µg/m$^3$ was exceeded a total of seven times throughout the study period.

Table 5.2 Summary of meteorology data, PM$_{10}$ and influenza consultation rates in Darwin, April – November in 2000, 2004 and 2005 (N=724 days).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Min</th>
<th>10th percentile</th>
<th>25th percentile</th>
<th>50th percentile</th>
<th>75th percentile</th>
<th>90th percentile</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily temperature ($^\circ$C)</td>
<td>19</td>
<td>24.1</td>
<td>25.6</td>
<td>27.5</td>
<td>28.9</td>
<td>29.9</td>
<td>31.9</td>
</tr>
<tr>
<td>Daily relative humidity (%)</td>
<td>22</td>
<td>49.9</td>
<td>61.4</td>
<td>67.4</td>
<td>72.1</td>
<td>76.5</td>
<td>91.4</td>
</tr>
<tr>
<td>Daily precipitation (mm)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1.2</td>
<td>123.4</td>
</tr>
<tr>
<td>PM10 (µg/m3)</td>
<td>1.1</td>
<td>10.3</td>
<td>13.6</td>
<td>17.4</td>
<td>22.3</td>
<td>27.7</td>
<td>70.0</td>
</tr>
<tr>
<td>Influenza (weekly GP diagnoses per 1000 consultations)</td>
<td>0</td>
<td>4.3</td>
<td>6.9</td>
<td>10.9</td>
<td>18.2</td>
<td>25.7</td>
<td>61.9</td>
</tr>
</tbody>
</table>

There were 2466 emergency admissions examined of which 23% were for Indigenous people. The number of admissions by Indigenous status and clinical grouping are summarised in Table 5.1.

The relationship between hospital admissions and PM$_{10}$ for the same day and lags up to three days are presented in Figures 5.1 and 5.2. Bordering on statistical significance, admissions for all respiratory conditions were positively associated with 10 µg/m$^3$ increases in PM$_{10}$ from bushfires (OR 1.08 95%CI 0.98, 1.18) with a larger magnitude in Indigenous people alone (OR 1.17 95% CI 0.98, 1.40). Conditions with the greatest positive associations were chronic obstructive pulmonary disease (COPD) (OR 1.21 95%CI 1.0, 1.47), asthma (OR 1.14 95%CI 0.90, 1.44) and asthma and COPD combined (OR 1.19 95%CI 1.03, 1.38). The effect sizes were greater in Indigenous people, particularly those
admitted for COPD whose odds of admission approximately doubled with each rise of 10 µg/m³ in ambient PM₁₀. (OR 1.98  95%CI 1.10, 3.59). No association was observed between PM₁₀ and respiratory infections.

There was no association between cardiovascular admissions in total and same day PM₁₀, or lags of up to 3 days. However among Indigenous people there was a positive non-significant association at lags of 1 to 3 days. For ischaemic heart disease (IHD), there was a significant same day negative association (OR 0.82  95%CI 0.68, 0.98) overall and in non-Indigenous people (OR 0.75 95%CI 0.61, 0.93). In contrast Indigenous people had a positive association that reached statistical significance at a lag of 3 days (OR 1.71 95%CI 1.14, 2.55).

Discussion

We have described positive associations between particulate air pollution derived from vegetation fires and admissions to hospital for several respiratory conditions. Effect estimates were greatest for chronic lower respiratory conditions with Indigenous people being at highest risk. However, for cardiovascular conditions the associations were either negative or absent except in the Indigenous subpopulation, for which associations tended to be positive.

Our findings of predominantly respiratory impacts of air pollution from bushfires are consistent with the comparatively few available studies concerning attendances to health facilities associated with ambient biomass smoke. Of these, five out of seven studies have documented rises of 30 -160% in attendances for respiratory conditions during episodes of poor air quality due to wildfires (8, 144, 145, 158, 160, 161). Additionally, hospital admissions for both respiratory and cardiovascular outcomes in associations with bushfire smoke have been examined two time-series studies. Morgan et al compared the impact of PM₁₀ attributable to bushfires with PM₁₀ attributable to all other sources in a hospital admissions series in Sydney from 1997 – 2001. While they found associations between
Figure 5.1 Adjusted odds ratios and 95% confidence intervals for admission to hospital for respiratory conditions per 10µg/m³ rise in PM$_{10}$ for the same day and lags up to 3 days. Darwin, April-November 2000, 2004 and 2005.
cardiovascular and respiratory admissions and PM$_{10}$ from all sources, the principal associations observed in relation to bushfire generated PM$_{10}$ was with respiratory outcomes (214). Mott et al. examined cardio-respiratory admissions in Malaysia during severe forest fires in 1997 and also noted that admissions for respiratory, particularly asthma and COPD, rather than cardiovascular admissions were primarily affected by particulate levels (155). This contrasts with many studies and meta-analyses of urban air pollution which have consistently demonstrated small positive associations with a range of both respiratory and cardiovascular outcomes including cerebrovascular diseases, IHD, heart failure and cardiovascular admissions overall (120, 215). Morgan et al. postulated that association with cardiovascular morbidity from biomass smoke could be driven by PM$_{2.5}$, a smaller size fraction of particulates, rather than PM$_{10}$ the principal exposure measure for vegetation fire smoke reported in the above studies. This was based on their observation of an association between IHD admissions and bushfire derived particulates as measured by BSP, a measure
of light scatter which is better correlated with PM$_{2.5}$ than PM$_{10}$. However, this is a less likely explanation in our region where previous studies have demonstrated a very high correlation ($r^2 = 0.81$) between PM$_{10}$ and PM$_{2.5}$ (216).

Similarly, our findings of relatively large associations with respiratory outcomes are consistent with other studies of pollution from wildfires and biomass derived particulates. We observed increases of approximately 8% for all respiratory admissions, 20% for COPD and 13% for asthma admissions with incremental rises of 10µg/m$^3$ of PM$_{10}$. These were of similar magnitude to estimates reported by Mott et al for the South East Asian fires of 1997 and also to an earlier study of bushfire smoke and asthma attendances conducted in Darwin (8, 155). These findings are also supported by a recent study from Brisbane, Australia, which directly compared the association between bushfire and non-bushfire derived particulates on total respiratory hospital admissions excluding influenza (217). That study analysed the PM$_{10}$ distribution as a three-level factor with levels defined as low (<15 µg/m$^3$), medium (15-20 µg/m$^3$) and high (>20 µg/m$^3$). They found that for an increase in same-day PM$_{10}$ from low to high there was an increase in the relative risk for total respiratory hospital admissions of 19% (95%CI: 9%, 30%) whereas on non-bushfire days the associated increase was 13% (95%CI: 6%, 23%). Similarly Morgan et al reported 3.8-5% increases in association with bushfire derived particulate matter (PM) for COPD and asthma in Sydney, while not finding any association between these outcomes and PM from other sources (214). All these estimated associations for biomass derived PM are well above those reported from meta-analyses of studies conducted in large cities of Europe and the USA that are of the order of 1-2% for all respiratory admissions and 1.5% for COPD and asthma in association with 10µg/m$^3$ increases in PM$_{10}$ (118, 139, 199). The findings from the studies discussed above fit the pattern of a recent review of studies of PM$_{10}$ and asthma that found greater relative risks documented in studies for which wood combustion was considered to be a major source of particulate matter (45).

While there appears to an emerging pattern of relatively greater respiratory and lesser cardiovascular adverse effects associated with particulates derived from vegetation fires
compared with non-biomass sources, the available evidence is limited and further research is required to investigate this hypothesis. The relatively big effect sizes we observed could have other explanations. Darwin has relatively uniform population exposure from source-specific particulates, a single major hospital and excellent data collection systems in place, all of which reduce the risk of misclassification bias in the exposure and outcome measures compared with large cities with considerable regional variation in pollution levels and multiple health services (218). However, our relatively small population inevitably limits the precision of our point estimates as evidenced by wide confidence intervals.

The high proportion of Indigenous admissions is unlikely to have contributed to our higher effect estimates as the results for non-Indigenous admissions were very similar to the overall findings. However, it is notable that the size of the associations between PM$_{10}$ and admissions for all respiratory conditions, COPD and asthma were all more than double in Indigenous people. Additionally, and in contrast to non-Indigenous people, we observed a positive association with cardiovascular admissions in this group. While admission numbers were relatively small, and associations did not achieve statistical significance, it is clear from our findings that Indigenous people are at greater risk from ambient air pollution. This cannot be explained by differences in exposure, or individual factors such as smoking or socio-economic status as we controlled for these factors in the design of the study. It is more likely to reflect the greater burden of chronic cardio-respiratory diseases among Indigenous people, placing them at higher risk from environmental hazards such as air pollution (208, 210). Understanding the differential effects of air pollution in more vulnerable groups of people is important for determining public health policy such as the setting of air quality guidelines.

This study also highlights the public health implications of land management practices in countries with fire prone vegetation. Studies of severe air pollution generated by intense, uncontrolled fires in the USA and South East Asia have clearly demonstrated an association with serious outcomes such as hospital admissions and deaths (11). Indeed, a recent economic analysis of a Canadian forest fire that burned for just 5 days, estimated that the
health cost of the fire amounted to approximately $12 million, largely due to the premature mortality caused by air pollution. These costs were similar to the estimated cost of timber losses, and greatly exceeded the costs of containing the fire (219). There is no doubt that the prevention of such large fires is of high priority and the public health risks from smaller fuel reduction burns should be evaluated in this context. Our findings suggest that the health impacts of lower levels of pollution as observed during our study and frequently generated by deliberate burns also require serious consideration. For example, prescribed fires should be explicitly managed to minimize pollution over urban areas and be accompanied by public health advisories to reduce the impact on people at higher risk.

Conclusions

PM$_{10}$ was predominantly associated with respiratory rather than cardiovascular admissions in this setting where the vast majority of particulates are derived from vegetation fires. This pattern of results is in keeping with findings from the few other similar studies that have been reported. Adverse health associations were identified at relatively low levels of pollution, a result that has particular relevance for land and fire management practices worldwide. Indigenous Australians are at greater risk of harm from particulate air pollution.

Ethical approval

The study was approved by the human research ethics committees of Charles Darwin University, the NT Department of Health and Community Services and the Menzies School of Health Research.

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Authors' contributions

FHJ conceived of the study, conducted the analysis and drafted the manuscript. RSB and LSP provided guidance on epidemiological methods and helped draft the manuscript. IH assisted with data analysis and helped draft the manuscript. All authors read and approved the final paper.

Competing interests

There are no competing interests


A savanna fire near Darwin, July 2005.
Chapter six

Thesis chapter


This chapter presents a time-series analysis of 10 years of hospital admissions data in relation to an estimated daily measure of PM$_{10}$.

I presented this work at the 14th World Congress of the International Union of Air Pollution and Environmental Protection Agencies (IUAPPA), Brisbane, Australia. 10-14 September 2007 as: Fay Johnston, Ivan Hanigan and Geoff Morgan. Vegetation fire smoke, indigenous status and cardio-respiratory hospital admissions in Darwin, Australia, 1996-2005.

Author contributions

For this chapter I acknowledge the support of Dr Geoff Morgan for explaining the different approaches to non-linear, time-series modelling in air pollution epidemiology and Ivan Hanigan who assisted with data cleaning and management and in using the R statistical software package for this analysis.
Abstract

Introduction

In this study the relationships between daily PM$_{10}$ and admissions to Royal Darwin Hospital for respiratory and cardiovascular conditions were examined. While the case-crossover study described in Chapter Five also examined these outcomes, its statistical power was limited by Darwin’s relatively small population and the short time periods for which air quality data were available. This study addressed this limitation by examining a 10 year rather than 3 year time period.

Methods

As atmospheric particulate loadings were not directly measured for all of this time, daily PM$_{10}$ was predicted from daily visibility and other meteorological data using a previously validated model. We analysed the association between modelled PM$_{10}$ on the same day and at lags of 1 to 3 days on daily emergency hospital admissions for cardio-respiratory diseases for residents of Darwin during each dry season between 1996 and 2005. The number of daily admissions were stratified by Indigenous status and grouped within diagnostic categories. The effects of PM$_{10}$ at each of the lags were investigated in turn using an over-dispersed Poisson generalized linear model. Interactions between Indigenous / non-Indigenous status and PM$_{10}$ were also investigated.

Results

There was a borderline positive association between PM$_{10}$ from vegetation fires and same day admissions for respiratory conditions with a 4.81% (95%CI -1.04%, 11.01%) increase per 10µg/m$^3$ rise in PM$_{10}$. The associations were greater in Indigenous people (9.40% 95%CI 1.04, 18.46) and smaller and not significant in non-Indigenous people (3.14% 95CI% -2.99, 9.66). There was a tendency towards inverse associations between PM$_{10}$ and same day cardiovascular conditions (-3.49% 95%CI: -8.79%, 2.13%) with similar results for Indigenous and non-Indigenous people.
Conclusions

$PM_{10}$ from vegetation fires was associated with respiratory hospital admissions particularly in Indigenous people. The overall results were similar to those from the case-crossover study reported in Chapter Five, although the effect estimates were generally of smaller magnitude and the 95% confidence intervals were narrower.
Introduction

The setting of Darwin is useful for examining the health effects of particulate air pollution arising from biomass combustion as 50-70 percent of the surrounding savanna burns annually during the 8 month dry season (April-November) (184) and the smoke from these fires is the source of 95 percent of measured PM$_{10}$ in the city (7). However, Darwin has a relatively small population of approximately 110,000 people limiting the power of epidemiological studies to detect adverse health impacts from poor air quality which are of small magnitude. In the previous chapter I presented a case-crossover study that identified some associations between PM$_{10}$ and hospital admissions in Darwin. However, the precision of the estimated magnitude of association for all outcomes examined in that study was low. Here I present another approach to the investigation of the associations between vegetation fire smoke and hospital admissions where I attempt to address the statistical limitations of the previous study by using hospital admissions data over a 10 year, rather than a three year period. I also used the analytical approach of time-series modelling which in direct comparisons with case crossover over analyses has been shown to provide slightly narrower confidence intervals (220-222). As directly measured particulate pollution data was not available over this time period, daily PM$_{10}$ was estimated using a previously validated predictive model based on daily airport visibility data (212).

The aims of this study were the same as those of the case-crossover analysis. That is to examine the relationship between daily PM$_{10}$ and hospital admissions for respiratory and cardiovascular conditions in Darwin, and to examine the differential impact on Indigenous people. Examination of the same outcomes using different exposure estimates and different analytical methods provides the opportunity to examine the consistency of findings with two different approaches.
Methods

Outcome measures

The same outcome measures were used in this study as in the previous case-crossover study (see Chapter Five). De-identified unit record data were obtained for emergency admissions to the Royal Darwin Hospital, the only major public hospital and referral centre for the region. Data were not available from the smaller private hospital that does not have an emergency department and admits few emergency medical patients. Time-series of daily counts were constructed for all respiratory disease; asthma; chronic obstructive pulmonary disease (COPD); respiratory infections; all circulatory diseases and ischaemic heart disease (IHD) for each 8-month dry season between 1996 and 2005. Data were extracted by their assigned principal diagnosis codes classified according to the International Classification of Diseases (ICD) codebooks, versions 9 and 10 (see Table 6.1). As this study included the changeover in the diagnostic coding of hospital separation data from version 9 to version 10 in 1999 the New Zealand Health Information Section ICD concordance was used to map the diagnosis codes across these two classification systems (223). Principal diagnosis, Indigenous status and primary residence were recorded on discharge from the hospital. Patients whose primary residence was not in Darwin were excluded. The estimated resident population of Darwin was obtained from the Australian Bureau of Statistics for each year of the study (224). The proportion of Indigenous people in the population of Darwin in 2001 was used to calculate the Indigenous population for the other years (225).

Exposure measures

Daily estimates of ambient PM$_{10}$ were derived from a predictive model constructed using the relationship between more recent monitored air quality data with temperature, humidity and visibility (212). The model was constructed using observations from the years 2000 and 2004, and the predicted estimates were validated against observations from 2005. The predicted PM$_{10}$ values correlated well with the observations during 2005 with an $r^2$ of 0.68 and a slope of 0.9. (Figure 6.1). On average the deviation between the predicted and
Table 6.1  Hospital admissions for respiratory and cardiovascular conditions. Darwin, April-November 1996-2005.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>ICD9 codes</th>
<th>ICD10 codes</th>
<th>Indigenous</th>
<th>Non-Indigenous</th>
<th>Total</th>
<th>Proportion &lt;15 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>390-459</td>
<td>I00-I99</td>
<td>589</td>
<td>2854</td>
<td>3443</td>
<td>1%</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>410-414</td>
<td>I20-I25</td>
<td>246</td>
<td>1287</td>
<td>1533</td>
<td>0%</td>
</tr>
<tr>
<td>Other Circulatory</td>
<td>-</td>
<td>-</td>
<td>343</td>
<td>1567</td>
<td>1910</td>
<td>2%</td>
</tr>
<tr>
<td>Respiratory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>460-519</td>
<td>J00-J99</td>
<td>1285</td>
<td>3551</td>
<td>4836</td>
<td>40%</td>
</tr>
<tr>
<td>Asthma</td>
<td>493</td>
<td>J45-J46</td>
<td>232</td>
<td>776</td>
<td>1008</td>
<td>58%</td>
</tr>
<tr>
<td>Chronic Obstructive Pulmonary</td>
<td>490-492</td>
<td>J40-J44, J47, J67</td>
<td>242</td>
<td>753</td>
<td>995</td>
<td>1%</td>
</tr>
<tr>
<td>Disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory infections</td>
<td>461-466</td>
<td>J00-J22</td>
<td>728</td>
<td>1681</td>
<td>2409</td>
<td>53%</td>
</tr>
<tr>
<td>Other Respiratory</td>
<td>-</td>
<td>-</td>
<td>83</td>
<td>341</td>
<td>424</td>
<td>16%</td>
</tr>
</tbody>
</table>

Figure 6.1 - Validation of the model daily predictions of PM\textsubscript{10} (µg/m\textsuperscript{3}) for the dry season of 2005 against measured data for Darwin (adapted from Bowman et al)(3)
measured values was $-2\mu g$ with a standard deviation of 3.6. The PM$_{10}$ exposure estimates are shown in Figure 6.2.

Potential confounding variables were included in all analyses using previously established protocols for air pollution health studies (226). These were: average daily temperature; average daily relative humidity; influenza epidemics (above the 90th percentile); and holiday periods. Meteorological data were obtained from the Bureau of Meteorology (227). Influenza rates were provided by the Northern Territory Department of Health and Community Services from the surveillance data derived from a network of sentinel General Practitioners.

**Statistical modelling**

In time-series analysis, statistical modelling is used to account for the effects of both long-term trends and medium-term fluctuations (for example due to season) in hospital admissions or other outcomes of interest. The effect of the covariates in these models is highly non-linear. This non-linear response can either be modelled using parametric
methods, such as generalised linear models (GLMs) with cubic spline (or similar) smoothing functions for covariates, or by using non-parametric smoothing functions of covariates in generalised additive models (GAMs). Some specific problems have occurred with the implementation of GAMs. These have included inappropriate setting for convergence criteria in the S Plus statistical software that was used for the analysis of many studies in the USA leading to elevated effect size estimates (228). Another problem with this method has been under-estimation of the standard errors. In datasets with concurvity, that is datasets in which a function of one of the independent variables can be approximated by a linear combination of functions of the remaining variables, this too can lead to over estimation of the point estimate (112, 229). As over or under-estimation of the effect of air pollution of health outcomes has considerable ramifications for public health policy, particularly the setting of air quality standards, many major studies have been reanalysed as such problems have been identified and addressed. Methods continue to be refined. For example the default convergence criteria setting in S Plus have been rectified, methods have been developed for calculating ‘exact’ standard errors in GAMs and methods for selecting the optimal amount of smoothing continue to be developed (110, 111, 138, 230).

The analysis presented here follows currently accepted parametric approaches using GLMs. It has two stages; (a) development of the statistical model and (b) a sensitivity analysis to select the amount of smoothing of time. The sensitivity analysis provides a systematic and conservative approach for minimising confounding due to the relationship between PM$_{10}$ and time. All analyses were conducted using the statistical software package R version 2.3.1 (231).

**Stage one**

For the first stage we used an over-dispersed Poisson GLM with parametric smoothed functions for time and meteorological data using natural cubic splines following the methods used by the American Medicare Air Pollution Study (MCAPS) (138) and the National Morbidity, Mortality and Air Pollution Study (NMMAPS) (111). In 2006 the authors of MCAPS made their analysis code available on the internet and invited other teams to use this to increase the comparability of results (232). Their code was adapted for our data as
follows: we did not stratify by age; we included an indicator variable for Indigenous status; and we included indicator variables for influenza epidemics, holidays and the change between ICD editions. We used separate regression models to analyse the association of same day PM$_{10}$ and lags up to three days with admission counts for each diagnostic group. All models included the explanatory variables as shown in Equation 6.1 and described in Table 6.2. Terms for same day and lagged meteorological data are included to account for the known immediate and delayed effects of heat and cold on hospital admissions (233). We included an interaction term between Indigenous status and PM to test the relationships between PM and health outcomes in Indigenous people.

**Equation 6.1 The core model**

\[
\log[E(Y_t)] = \beta_0 + \beta_1 \text{PM}_{10} + \text{ns(Time df=40)} + \text{ns(AvDailyTemp df=6)} + \text{ns(AvDailyTempLag1-3 df=6)} + \text{ns(RHumAv df=3)} + \text{ns(RHumAvLag1-3 df=3)} + \text{Indigenous} + \text{DOW} + \text{Flu Epidemic} + \text{ICD10 change} + \text{Holidays} + \log(\text{Population})
\]

Where $E(Y_t)$ = expected admission count on day $t$, ns = natural cubic spline (smoothing function), $\beta$ = beta coefficients represented as $\beta_0$ for the intercept, $\beta_1$ for PM$_{10}$ and not listed thereafter, df = degrees of freedom.
Table 6.2 –Covariates included in the GLM models

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM10</td>
<td>Estimated ambient PM$_{10}$ for lags 0-3 (µg/m$^3$)</td>
</tr>
<tr>
<td>Time</td>
<td>Time in days, represented by a natural cubic spline with 40 degrees of freedom (df). This equates to 4 df per season - an effective smoothing span of 2 months.</td>
</tr>
<tr>
<td>AvDailyTemp</td>
<td>Average daily temperature (calculated by averaging the max and min temperatures), in degrees Celsius (°C) represented by a natural cubic spline with 6 df.</td>
</tr>
<tr>
<td>AvDailyTempLag1-3</td>
<td>Moving averages of average daily temperatures at lags 1, 2 and 3 days. Natural cubic spline with 6 df.</td>
</tr>
<tr>
<td>RHumAv</td>
<td>Average daily relative humidity in percent (%). Natural cubic spline with 3 df.</td>
</tr>
<tr>
<td>RHumAvLag1-3</td>
<td>Moving averages of relative humidity at lags 1, 2 and 3 days. Natural cubic spline with 3 df.</td>
</tr>
<tr>
<td>Indigenous</td>
<td>An index of counts for Indigenous status where Indigenous = 1 and non-Indigenous = 0.</td>
</tr>
<tr>
<td>DOW</td>
<td>Day of the week. Factor with 7 levels.</td>
</tr>
<tr>
<td>Flu Epidemic</td>
<td>Influenza epidemics. Indicator variable for days above the 90th centile.</td>
</tr>
<tr>
<td>ICD10 change</td>
<td>Indicator variable for the change between ICD editions.</td>
</tr>
<tr>
<td>Holidays</td>
<td>Indicator variable for public holidays.</td>
</tr>
<tr>
<td>Population</td>
<td>The estimated yearly population for Indigenous or non-Indigenous as appropriate was included to control for changes in the population denominators over time.</td>
</tr>
<tr>
<td>School holidays</td>
<td>This was included as an indicator variable when we modelled admissions for the outcomes of (1) all respiratory conditions, (2) asthma and (3) respiratory infections because these groups contained a high proportion of children (see Table 6.1).</td>
</tr>
</tbody>
</table>
Stage two

In the second stage of analysis we used a sensitivity analysis (the bandwidth selection method) described by Dominici et al 2004 (110) to select the optimal degrees of freedom (df) for the smoothed function of time. Alternative statistical approaches to selecting the amount of smoothing used in time-series models have included use of the Akaike Information Criterion, minimising the absolute autocorrelation in residuals or other test statistics for departure of the residuals from white noise (234). The amount of smoothing used is an important decision as it can potentially have a major influence on the results. The setting of a greater number of degrees of freedom for the smoothing function will result in many more fluctuations being modelled as part of time and reduce the potential bias in the effect sizes attributed to the effects of pollution. However it also increases the standard error of these estimates. Conversely, fewer degrees of freedom in the smoothed function of time will tend to increase bias and decrease the standard error of the point estimates of the impact of air pollution (110, 234).

We adjusted the amount of smoothing on the time variable by applying different values of a multiplier (\(\alpha\)) that ranged from 0.2 to 3 times the df which had been chosen a priori. An \(\alpha\) of 1 (4 df per year) thus equated to a smoothing period of 2 months, an \(\alpha\) of 0.2 (0.8 df per year) corresponded to a 10 month smoothing interval while an \(\alpha\) of 3 (12 df per year) corresponded to a smoothing interval of just 2.6 weeks. The impact of different levels of \(\alpha\) on the smoothed function of time for the outcome of all respiratory admissions is shown in Figure 6.3.

Figure 6.4 demonstrates how the amount of smoothing of time affects both the absolute value and precision of the point estimate for the association between all respiratory admissions and \(\text{PM}_{10}\).
Figure 6.3 The count of daily admissions for respiratory conditions with three smoothed functions of time. Darwin, April–November 1996-2005.

Figure 6.4. Percentage increase and 95% confidence intervals in daily admissions for all respiratory conditions with 10 µg/m$^3$ rises in same day PM$_{10}$ for alpha values ranging from 0.2 to 3, Darwin, April-November 1996-2005. This demonstrates how the amount of smoothing of time affects both the absolute value and precision of the point estimate.
We applied the sensitivity analysis as follows: Firstly, we selected the model for the PM$_{10}$ lag with the greatest absolute t-value, positive or negative. This is demonstrated in Figure 6.5 for the outcome of all respiratory admissions. In this example the same day estimate (lag 0) was of the greatest magnitude so this was used for the subsequent sensitivity analysis for the effect of changing the smoothing interval ($\alpha$).

![Figure 6.5 Percentage increase and 95% confidence intervals in daily admissions for all respiratory conditions with 10 $\mu$g/m$^3$ rises in PM$_{10}$ for lags of 0 – 3 days at alpha=1, Darwin, April-November 1996-2005.](image)

The influence that the amount of smoothing had on the effect estimate was assessed by plotting the change in the mean squared error (MSE) of the $\beta$ estimates. This is relative change in the square of the difference of the $\beta$ estimate produced when $\alpha=3$, which is accepted to be the least biased, with the $\beta$ estimates produced with sequentially lower values of $\alpha$. An alpha of three is accepted to be the least biased because time varying outcomes are modeled in such great detail that the effects of pollution are less likely to be wrongly attributed to other time varying confounders in comparison to lower levels of alpha in which fewer time varying fluctuations are included in the core model. These were standardised against a $\beta$ estimate corresponding to a 15 percent change in admissions with rises of 10$\mu$g/m$^3$ in PM$_{10}$, the upper limit of the 95 percent confidence interval for all respiratory admissions, our primary outcome of interest. The level of $\alpha$ selected was the
lowest that produced a similar $\beta$ estimate to that produced when $\alpha=3$, as assessed by the MSE. Figure 6.6 demonstrates this for the outcome of all respiratory conditions. There is minimal change in the MSE from $\alpha=3$ to $\alpha=1$ but MSE increases at values of $\alpha$ below one. The a priori choice of $\alpha=1$ was therefore retained.

![Figure 6.6](image)

**Figure 6.6.** Relative change in the mean squared error (MSE) of the $\beta$ estimates obtained when alpha $= 3$ for alpha levels ranging from 0.4 to 3.

**Results**

There were 8,279 admissions during the 10 dry seasons (2,410 days) of the study period. The total numbers of hospital admissions stratified by clinical grouping, Indigenous status and proportion less than 15 years old are shown in Table 6.1. Despite Indigenous people representing 11% of the population of Darwin, they comprised 23% of these admissions.

Figure 6.7 shows the point estimates and 95% confidence intervals for the association between hospital admissions with PM$_{10}$ as the percentage change in admissions per 10µg/m$^3$ rise. There was a borderline positive association for all respiratory admissions with same day PM$_{10}$ (4.81%, 95%CI -1.04, 11.01). The subgroups of respiratory infections, asthma and COPD all had positive associations with same day PM$_{10}$. However, due to small
numbers in these groups the confidence intervals were wide and included the null. The small associations for all cardiovascular diseases and IHD were all negative and not statistically significant.

Figure 6.8 shows the point estimates and 95% confidence intervals for the association between hospital admissions with PM$_{10}$ for Indigenous and non-Indigenous groups. The point estimate for all respiratory admissions in Indigenous residents was much higher than the estimate for non-Indigenous residents, 9.40% (95%CI 1.04, 18.46) compared with 3.14% (95CI% -2.99, 9.66) respectively. For asthma admissions and same-day PM$_{10}$ there was a non-significant estimated increase of 12.6% (95%CI -6.83%, 36.07%) for Indigenous compared with 7.76% (95%CI -6.41%, 24.07%) for non-Indigenous people.

A strong positive association was observed for respiratory infections in Indigenous people of 15.02% (95%CI 3.73, 27.54) at a lag of 3 days while no association was evident for this condition in non-Indigenous people at this lag (0.67%, 95%CI -7.55, -9.61). There was no association for same day PM$_{10}$ with COPD admissions in either group. This is in contrast to negative associations with COPD admissions and lagged PM$_{10}$.

There were no clear associations with PM$_{10}$ and all cardiovascular admissions or IHD. In non-Indigenous people there were negative non-significant estimates for these conditions at all lags, while Indigenous people had some positive non-significant estimates at lags 2 and 3 (see Figure 6.8).

**Discussion**

These results of generally positive associations between PM$_{10}$ and respiratory but not cardiovascular admissions and disproportionately higher associations for Indigenous people are broadly similar to those of the study reported in Chapter Five. Effect estimates in this study for respiratory and cardiovascular admissions tended to be smaller and have greater precision with the 95% confidence intervals generally about half the width of those in the
Figure 6.7 – Point estimates and 95% confidence intervals for the percentage change in hospital admissions per 10 µg/m³ rise in PM₁₀. Darwin, April-November 1996-2005. Alpha (α) represents a factor multiplying the a priori degree of smoothing for the function of time (2-month span).
Figure 6.8 – Point estimates and 95% confidence intervals for the percentage change in hospital admissions per 10 µg/m³ rise in PM$_{10}$ for Indigenous and non-Indigenous people in Darwin, April-November 1996-2005. Alpha (α) represents a factor multiplying the a priori degree of smoothing for the function of time (2-month span).
case-crossover study. For example, the association with respiratory admissions was of borderline statistical significance in both studies with 4.81% (95%CI -1.04, 11.01) increase in admissions per 10 µg/m³ rise in PM₁₀, in this study compared with a 7.77 % increase (OR 1.077  95%CI 0.984, 1.178) per 10 µg/m³ rise in PM₁₀ in the case-crossover study. The smaller effect estimates in this study could partly have been due to lower accuracy of the exposure estimates for daily PM₁₀ as these were modelled rather than directly measured. It could also have been due to the larger sample size, better power and more stable estimates of the time-series study.

The positive associations with respiratory admissions are consistent with the few available studies of ambient particulates derived from biomass combustion (132, 155, 235) that tend to have reported larger associations than generally reported for studies of PM₁₀ primarily derived from other sources. (45, 118, 138, 139). The lack of aeroallergen data for this and the previous hospital admissions study (Chapter Five) is a limitation of this work and possibly also the previous published studies cited above. In our setting, both particulate matter and pollen loads are seasonal and predominantly occur during the tropical dry season and there is potential for peaks in pollen and smoke to coincide. Several studies, including two from Australia in Darwin and Melbourne, have shown relationships between ambient pollen or fungal counts and hospital admissions for respiratory conditions (236-239) and it is not possible to determine how inclusion of these data might have affected the positive associations with respiratory admissions described here or in Chapter Five.

The pattern of absent associations for cardiovascular outcomes was generally consistent with results of studies of biomass smoke from Malaysia (155), Sydney (142), Christchurch (124, 132) and the case-crossover study reported in Chapter Five, in which the associations tended to be negative. This contrasts with the wider air pollution literature in which positive association between non-biomass derived particulates and cardiovascular admissions have been well established (1). However, this could reflect the fact that most biomass smoke studies are small, single city studies and their effect estimates are relatively unstable in contrast to smaller but more precise estimates from large multi city studies such as APHEA.
The effect of biomass smoke on non-respiratory outcomes including cardiovascular diseases has been highlighted as an area requiring further research (4).

While the outcomes for the smaller diagnostic subgroups of asthma, COPD, respiratory infections, and ischaemic heart disease had greater variation between the studies, confidence intervals around the point estimates from the two studies reported here were widely overlapping. The outcomes for asthma were the most consistent with both studies reporting non-significant positive associations of similar magnitude. There was no association between same day PM$_{10}$ and COPD in the time-series study. In contrast, COPD was the outcome with the strongest association observed in the case-crossover study and has been well documented to be associated both biomass and non-biomass derived particulates (99, 240, 241). While the time-series study found borderline associations with respiratory infections, this outcome had no association with PM$_{10}$ in the case-crossover study. There is less information about this outcome in relation to ambient air pollution in the wider literature, however indoor biomass smoke has been clearly associated with the prevalence and mortality due to respiratory infections (83). Associations with ischaemic heart disease tended to be negative in both studies with the inverse association attaining statistical significance for non-Indigenous people in the case-crossover study. These were unexpected findings and difficult to explain as particulate air pollution from primarily non-biomass sources has been clearly associated with admissions for ischaemic heart disease and many other cardiovascular outcomes (1). However the case-crossover study did find positive association in Indigenous people that reached statistical significance at a lag of 3 days. The positive associations in Indigenous people might reflect the fact that this population subgroup is at particularly high risk for ischaemic heart disease (242). However results from the analyses of the smaller diagnostic subgroups are based on analysis of much smaller patient numbers. The effect estimates are therefore much less stable in these groups and the findings require replication.
In both studies the risk of admission for respiratory conditions in Indigenous people was two to three times higher than the non-Indigenous estimates and reached statistical significance in spite of the smaller population subgroup being studied. Although the actual differences between these outcomes are unlikely to be of statistical significance due to the less precise outcomes in the smaller Indigenous group, the findings are consistent with the wider literature. Socio-economic disadvantage, chronic cardio-respiratory diseases and diabetes all modify the effects of ambient PM$_{10}$ on cardio-respiratory admissions (206, 208). Further, Indigenous Australians collectively experience many barriers to accessing and utilising appropriate health care services. These include cultural and educational differences that affect the decision to seek health care, language differences that limit communication and clinical decision making and practical issues such as limited access to transport (243). Collectively these reduce the likelihood of early diagnosis and early effective treatment further placing them at risk. Residential segregation is less likely to explain the difference in this setting as exposure is relatively uniform across the city (212). This consistent observation has implications for local public health policy and practice, that need to take into account the impact of air pollution on individuals and communities at higher risk (210). For example, if a high proportion of people at risk from air pollution are Indigenous, health information and warnings will need to be provided in an appropriate and accessible way for people in that group, particularly in settings such as Darwin where they form a sizeable proportion of the population. Health service providers also need to be well informed about higher risk groups to provide appropriate clinical and preventive care.

The improved precision of the effect estimates in this study can be attributed to two factors, the most obvious being the much larger sample size with 10 rather than 3 years of admissions data examined. The improved precision attributable to using a different method for the statistical analysis was not directly quantified, but is likely to have been relatively small in comparison. A case-crossover analysis with conditional logistic regression modelling has been shown to represent a specific case of time-series modelling when there is a common exposure, as is usually the case in air pollution studies (116). However, despite the equivalence of estimates from these two methods, they can give different
standard errors because time-series analysis allows for over-dispersion of the Poisson variance, while the case-crossover design does not (116). Künzli and Schindler have also pointed out that case-crossover methods may have decreased power to detect associations because the variability in the difference between air pollution concentrations on event and control days can be much smaller than the full distributions of air pollution values that are used in time-series methodology (244). In the case-crossover study reported in Chapter Five, I matched the referent days on three variables: day of week, month and calendar year. Some case-crossover studies also use this approach to control for the influence of other environmental variables such as co-pollutants or meteorological factors. This further reduces the number of control days available for the analysis results in the loss of some statistical power (116, 222). All of the factors described above could contribute to the slightly wider confidence intervals reported for case-crossover studies when this approach has been directly compared with Poisson time-series generalized linear models (220-222).

Even though the results of this time-series study had greater precision than the results of the smaller case-crossover study, most results still included the null apart from the findings for Indigenous people. In spite of this there was consistency in the major findings of associations between bushfire derived PM$_{10}$ and respiratory but not cardiovascular hospital admissions. There was also consistency in the much larger effect estimates observed for Indigenous people. The consistency in the main results between the two approaches that used different exposure estimates, study duration and methods of analysis, further strengthens the evidence of harmful population health impacts from biomass smoke even at the relatively low concentrations experienced in the city of Darwin.
Ambient biomass smoke and presentations to the Emergency Department of Royal Darwin Hospital during 2004 and 2005.

Output from smoke dispersal model June 16th 2004 showing the location and predicted smoke plumes from regional fires. (Photo Alan Wain. Bureau of Meteorology)
Chapter Seven

Thesis chapter

Ambient biomass smoke and presentations to the Emergency Department of Royal Darwin Hospital during 2004 and 2005

This chapter reports the relationship between particulate air pollution and emergency department presentations for cardio-respiratory conditions, over a two year period using a case-crossover analysis.

Author contributions
For this work I acknowledge the support of research assistant Ivan Hanigan who assisted with data cleaning and database management.
Abstract

Introduction

The studies presented in chapters Four and Five of this thesis reported associations between particulate air pollution asthma symptoms, asthma medication use and respiratory and cardiovascular hospital admissions. Here I present the final study for this research program, an analysis of air pollution and attendances to the Emergency Department (ED) of Royal Darwin Hospital.

Methods

I examined the relationship between atmospheric loadings of particles <10 microns (PM$_{10}$) and <2.5 microns (PM$_{2.5}$) in diameter, and emergency department attendances for cardio-respiratory conditions (ICD10 diagnosis codes J00-J99 and I00-I99) over the two fire seasons of 2004 and 2005. I used a case-crossover design and conditional logistic regression modelling to calculate odds ratios for an attendance with 10 µg/m$^3$ rises in PM$_{10}$ and 5 µg/m$^3$ rises in PM$_{2.5}$. These estimates were adjusted for pollen and fungal spore counts, influenza rates, meteorology and holiday periods. Indigenous presentations were examined separately and compared with the overall findings.

Results

In total 3664 respiratory and 1195 cardiovascular presentations were examined. Atmospheric particulate loadings were low throughout the study period and Australia’s air quality standards for PM$_{10}$ and PM$_{2.5}$ were rarely exceeded. There were no significant associations between PM$_{10}$ or PM$_{2.5}$ and respiratory attendances overall or with any diagnostic subgroup. Same day negative associations bordering on statistical significance were observed for all cardiovascular conditions (PM$_{10}$ OR=$0.92$ 95%CI $0.81$, $1.05$ and PM$_{2.5}$ OR=$0.93$ 95%CI $0.85$, $1.01$) and for ischaemic heart disease (PM$_{10}$ OR=$0.87$ 95%CI $0.69$-$1.09$ and PM$_{2.5}$ OR=$0.88$ 95%CI $0.76$-$1.02$). The results for Indigenous people were similar to the overall findings.
Conclusions

The restricted availability of ED attendance data limited the study to just two years during which airborne particulate matter demonstrated less variation and peaked at lower levels than in previous years. This limited the statistical power of this study. No positive associations were found with any outcomes. However tendencies towards inverse associations with cardiovascular outcomes were again observed, similar to findings of hospital admissions reported in previous chapters.
Introduction

This thesis has examined a range of health outcomes including symptoms and medication use in a cohort of people with asthma (Chapter Four) and cardio-respiratory hospital admissions (Chapters Five and Six) in relation to ambient particulate pollution from vegetation fires in the adjacent savanna landscape. This chapter presents the final component of my project, an analysis of presentations to the Emergency Department (ED) of Royal Darwin Hospital for the two dry seasons of 2004 and 2005, the period for which both ED attendance and particulate data were available. ED presentations were examined because they have previously been shown to be associated with air pollution in a number of different settings including Darwin (8, 158, 237, 245, 246) and they reflect a level of morbidity that is neither captured by measuring individual symptoms nor hospital admissions data. Additionally, emergency departments place an important and increasing demand on health services (247). An understanding of the additional burden related to external environmental factors such as air pollution is useful for planning services and allocation of resources.

Methods

This analysis followed the case-crossover design described in detail in Chapter Five. Comparison of environmental data was made between the days patients attended hospital, and several referent days on which they did not attend. The referent days were selected from the same month and year and matched by day of week of the admission.

Exposure measures

PM$_{10}$ and PM$_{2.5}$ were measured using a Rupprecht and Patashnick Partisol plus model 2025 sequential air sampler located close to the main residential areas. This provided 24 hour gravimetric measures of PM ($\mu$g/m$^3$). These data were validated by inter-laboratory comparison gravimetric analyses conducted with the Marine and Atmospheric Research Division of Australia's Commonwealth Scientific and Industrial Research Organisation (CSIRO).
Daily meteorological data were provided by the Bureau of Meteorology and weekly consultation rates for influenza-like illness were provided by the Northern Territory (NT) Department of Health and Community Services from data routinely gathered from sentinel general practitioners.

**Outcome measures - Patient emergency department attendances**

Hospital admission data were collected by the Royal Darwin Hospital (RDH), the single major public hospital and referral centre servicing the northern half of the Northern Territory. It provides the only emergency department service available in Darwin. At discharge from the emergency department, whether this be for admission to hospital or discharge home, the patient’s medical diagnoses are directly entered into an electronic register by the treating doctor who must chose the most appropriate diagnosis from a menu coded according to the International Classification of Diseases version 10 (ICD10) (213). De-identified emergency data for 2004 and 2005 with a principal ICD10 diagnosis code for respiratory and circulatory conditions (Table 7.1) were extracted from this database for analysis. Each presentation included details of date of birth, gender, ethnicity, ICD principal diagnosis code, occupation, place of residence, dates of attendance, admission and discharge and a unique identifier. These data were cleaned by identifying gaps and errors in the data extraction process and by finding and eliminating duplicate records. Those whose primary residential address was not in Darwin were excluded. The excluded group comprised residents of rural communities adjacent to Darwin, remote towns and Indigenous communities within the NT and interstate travellers. We included the first presentation only for each episode of illness by excluding re-presentations within 4 weeks.

**Data analysis**

Respiratory and cardiovascular admissions were described according to the same diagnostic groups examined for hospital admissions in Chapter Five. We used conditional logistic regression models to calculate odds ratios (OR) and 95% confidence intervals (CI) for hospital admission in relation to variation in PM adjusted for weekly influenza rate, days with rainfall > 5mm, same day mean temperature and humidity, the mean temperature and humidity of the previous three days and public holidays. These potential confounders were
Table 7.1 Presentations to the Emergency Department of Royal Darwin Hospital for respiratory and cardiovascular conditions. Darwin, April – November 2004 and 2005.

<table>
<thead>
<tr>
<th>Condition</th>
<th>ICD10 codes</th>
<th>Total</th>
<th>Daily mean</th>
<th>Std Dev</th>
<th>Daily min</th>
<th>Daily max</th>
<th>Percent &lt;= 15 yrs old</th>
<th>Percent Indigenous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory diagnoses - all</td>
<td>J00-J99</td>
<td>3333</td>
<td>6.8</td>
<td>2.8</td>
<td>0</td>
<td>17</td>
<td>43.7</td>
<td>27</td>
</tr>
<tr>
<td>Asthma</td>
<td>J45-J46</td>
<td>540</td>
<td>1.1</td>
<td>1.1</td>
<td>0</td>
<td>6</td>
<td>41.3</td>
<td>23</td>
</tr>
<tr>
<td>COPD</td>
<td>J40 –J44</td>
<td>201</td>
<td>0.4</td>
<td>0.6</td>
<td>0</td>
<td>3</td>
<td>2.0</td>
<td>24</td>
</tr>
<tr>
<td>Respiratory infections</td>
<td>J00-J22</td>
<td>2167</td>
<td>4.4</td>
<td>2.1</td>
<td>0</td>
<td>11</td>
<td>52.4</td>
<td>30</td>
</tr>
<tr>
<td>Cardiovascular diagnoses - all</td>
<td>I00 – I99</td>
<td>1116</td>
<td>2.3</td>
<td>1.6</td>
<td>0</td>
<td>9</td>
<td>2.1</td>
<td>17</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>I20 – I25</td>
<td>344</td>
<td>0.7</td>
<td>0.8</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>16</td>
</tr>
</tbody>
</table>

chosen a priori and included in all models. School holidays were additionally included as a dummy variable for all respiratory conditions, asthma, and respiratory infections as these groups included a substantial proportion of children. The Indigenous and non-Indigenous sub-populations were also examined separately.

**Results**

*Exposure data*

Atmospheric particulate loadings were low throughout the study period and Australia’s air quality standards for PM$_{10}$ and PM$_{2.5}$ were rarely exceeded. Daily PM loadings, meteorology and weekly GP consultations for influenza are summarised in Table 7.2.

*Patient emergency department attendance data*

In total 3664 respiratory and 1195 cardiovascular presentations were examined. While 11% of the population is Indigenous (192), this group accounted for 22% of all presentations. The relationships between attendances and atmospheric PM for all patients are shown in Figure 7.1 and for Indigenous patients only are shown in Figure 7.2. The results for non-Indigenous people are not shown because they were not appreciably different from the overall results. There were no significant associations between PM and respiratory attendances overall or

<table>
<thead>
<tr>
<th>Variable</th>
<th>Percentiles</th>
<th>Mean</th>
<th>Std Dev</th>
<th>Min</th>
<th>10th</th>
<th>25th</th>
<th>50th</th>
<th>75th</th>
<th>90th</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily PM$_{10}$ (µg/m$^3$)</td>
<td></td>
<td>18.3</td>
<td>7.0</td>
<td>1.1</td>
<td>10.3</td>
<td>13.6</td>
<td>17.4</td>
<td>22.5</td>
<td>27.7</td>
<td>68.7</td>
</tr>
<tr>
<td>Daily PM$_{2.5}$ (µg/m$^3$)</td>
<td></td>
<td>10.1</td>
<td>5.6</td>
<td>0.3</td>
<td>4.2</td>
<td>6.5</td>
<td>9.4</td>
<td>13.1</td>
<td>16.5</td>
<td>57.7</td>
</tr>
<tr>
<td>Daily temperature °C</td>
<td></td>
<td>27.4</td>
<td>2.3</td>
<td>20.3</td>
<td>24.3</td>
<td>25.7</td>
<td>27.5</td>
<td>29</td>
<td>29.9</td>
<td>31.7</td>
</tr>
<tr>
<td>Daily relative humidity (%)</td>
<td></td>
<td>64.9</td>
<td>10.8</td>
<td>22.0</td>
<td>50.5</td>
<td>60.9</td>
<td>66.7</td>
<td>71.5</td>
<td>75.3</td>
<td>90.1</td>
</tr>
<tr>
<td>Daily precipitation (mm)</td>
<td></td>
<td>1.3</td>
<td>5.4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1.0</td>
<td>46.8</td>
</tr>
<tr>
<td>Weekly GP consultations for influenza-like illness per 1000 consultations</td>
<td></td>
<td>16.1</td>
<td>11.2</td>
<td>2.4</td>
<td>5.2</td>
<td>9.2</td>
<td>14.8</td>
<td>20.1</td>
<td>30.6</td>
<td>61.9</td>
</tr>
</tbody>
</table>

with any diagnostic subgroup. Same day negative associations bordering on statistical significance were observed for all cardiovascular conditions. We observed an 8% decrease in presentations with 10 µg/m$^3$ rises in PM$_{10}$ (OR=0.92 95%CI 0.81,1.05) and a 7% decrease for 5 µg rises in PM$_{2.5}$ (OR=0.93 95%CI 0.85, 1.01). Results were similar for ischaemic heart disease: for PM$_{10}$ OR=0.87 (95%CI 0.69, 1.09) and for PM$_{2.5}$ OR=0.88 (95%CI 0.76,1.02).

Outcomes in Indigenous people were less precise due to the small number of cases, but were not substantially different from the overall results.

Discussion

No significant associations between PM and hospital emergency department attendances for respiratory conditions were observed in this analysis. There was however, a borderline inverse relationship with cardiovascular admissions particularly for same day PM$_{2.5}$. Results for Indigenous people were similar to the overall findings.

This study had the advantage of a greater daily presentation rate compared with admissions to hospital and therefore a higher number of cases was included in this two year analysis than the in the three year study of hospital admissions (Chapter Five) apart from the two smaller subgroups of COPD and Ischaemic heart disease. In addition aerobiology and PM$_{2.5}$ data were available for inclusion in the models. The main limitation of this study was the
Figure 7.1 Adjusted odds ratios and 95% confidence intervals for all respiratory and cardiovascular presentations to the Emergency Department of Royal Darwin Hospital per 10µg/m³ rise in PM₁₀ and 5µg/m³ rises in PM₂.₅ for the same day and lags up to 3 days. Darwin, April–November, 2004 and 2005
Figure 7.2 Adjusted odds ratios and 95% confidence intervals for respiratory and cardiovascular presentations to the Emergency Department of Royal Darwin Hospital. per 10µg/m³ rise in PM₁₀ and 5µg/m³ rises in PM₁₅ for the same day and lags up to 3 days, Indigenous presentations only. Darwin, April–November, 2004 and 2005.
lower overall levels of pollution during the study period compared with both the three year (Chapter Five) and ten year (Chapter Six) hospital admissions studies.

The only previous study of emergency department attendances in Darwin (8) examined asthma diagnoses and found a strong association, but only when PM$_{10}$ exceeded 40 µg/m$^3$, a threshold rarely breached during this study. The findings of this study are also concordant with the asthma panel study presented in Chapter Four (216). That study found associations between particulate levels and symptoms and medication use but not health care attendances during the same time period with similarly low particulate pollution levels. I did not test for non-linear associations in this study because particulate levels remained lower than the threshold identified in the previous emergency department study and because testing for thresholds would have further reduced the power of this small study.

Studies of larger populations and more severe bushfire pollution episodes have found associations with outpatient attendances for asthma and other respiratory conditions (144, 145, 158). However, Darwin’s population is 110,000 (192), the study duration was limited by the availability of health data and pollution levels remained lower than expected during this period. All of these factors limited our ability to detect small associations that might have been present. Indeed, in a post hoc analysis of these data, I calculated that we only had sufficient power (80%) to detect associations between respiratory presentations and 10 µg/m$^3$ rises in PM$_{10}$ with odds ratios of 1.5 or more. Our power to detect an association with an odds ratio of 1.1 was just 11%. Similarly, a study in Vimercate, Italy, with a population of just 25,000 was unable to detect associations even with higher levels of pollution in a one year study of emergency department attendances (248), despite the well established association between particulate air pollution and hospital attendances in urban industrial settings (130).

While the association between urban derived PM and cardiovascular conditions has been reasonably well established, the association between cardiovascular outcomes and biomass smoke has been less well studied (249). This study was not sufficiently powered to detect
associations with cardiovascular ED presentations as these were much fewer in number than respiratory presentations. The borderline inverse associations between PM and cardiovascular presentations were unexpected and is most likely to represent a chance finding, although for non-Indigenous people it is consistent with the tendency towards negative or absent associations described in our two hospital admissions studies. While two large studies of pollution episodes from vegetation fires in Malaysia and Sydney (142, 155), both failed to detect associations between PM and cardiovascular admissions, the current evidence is insufficient to draw conclusions about the role of ambient biomass smoke in cardiovascular diseases.

The results from this study also differed from the findings of the two hospital admissions studies (Chapters Five and Six) in that there was no clear difference in outcomes in the Indigenous sub-population (235). The lower levels of pollution observed during this study in which PM$_{10}$ generally remained below 40 µg/m$^3$, compared with the admissions studies in which there were prolonged episodes during which PM$_{10}$ fluctuated between 40 and 70 µg/m$^3$, would have further limited the ability to detect an association in this smaller population, despite their higher risks of chronic diseases and hospitalisation (242). It could also reflect differences in health services utilisation by urban Indigenous people, who might choose to have their condition managed by specific Indigenous health services in preference to the emergency department, or might present relatively late in the course of their illness making it more likely that a relationship with admissions rather than ED attendances is identified. However data about the utilisation of primary health care services in Darwin are not available for either the Indigenous or non-Indigenous populations although barriers to the use of mainstream health services and relatively late presentations for other conditions have been reported in the wider literature on Indigenous health (250).

**Conclusions**

These findings are generally consistent with previous work in Darwin. No clear public health impact in terms of emergency department presentations could be demonstrated during this
time period when PM$_{10}$ usually remained well below 40 µg/m$^3$. However, this study lacked sufficient power to detect any such association unless its magnitude was far greater than would be expected from the available literature. Even though the null result is consistent with the contention that low levels of air pollution from small deliberate burns have minimal public health impact, the findings should be interpreted with caution.
Summary, synthesis and discussion.

Melbourne covered by a smoke haze, 20 January 2007 (Photo, The Age)

Satellite image of Victoria showing the location of several bushfires and widespread smoke haze. 20 December 2006
Chapter Eight

Thesis chapter

Summary, synthesis and discussion

Here I summarise and discuss the main contributions of the work presented in this thesis to the body of research evidence on the health effects of particulate air pollution derived from bushfires. The chapter is divided into the following sections:

- Summary of the research
- Synthesis of the main findings
  - Asthma
  - Hospital admissions and emergency department attendances for respiratory and cardiovascular conditions
- Discussion
  - Contribution to the epidemiology of exposure to ambient biomass smoke
  - Contribution to land and fire management in Australia
    - Understanding the risks of air pollution from severe wildfires
    - Understanding and managing the risks of air pollution from prescribed burns
    - The role of Indigenous fire management in Australia
  - Global implications of fire management
- Future directions

Author contributions

I am the sole author of this work.
Summary of the research

Patterns of air pollution in Darwin provide a rare opportunity for prospective studies of clinical and public health impacts of particulates arising from landscape fires. This thesis aimed to contribute to the evidence base for land management and public health interventions in Australia and other fire prone regions of the world.

I examined the relationship between the mean daily PM$_{10}$ and PM$_{2.5}$ and a range of health outcomes including:

- the daily frequency of symptoms, medication use and health care attendances in a cohort of people with asthma followed for eight months;
- daily hospital admissions for respiratory and cardiovascular conditions over three years during which particulates were directly measured in Darwin;
- daily hospital admissions for respiratory and cardiovascular conditions over a ten year period during which particulates were estimated from daily visibility records; and
- daily emergency department presentations for two dry seasons

Synthesis of the main findings

**Asthma**

Health outcomes relating to asthma was examined in all of the above studies. The *Darwin Asthma Study* (Chapter Four) found that increases in PM$_{10}$ and PM$_{2.5}$ were both correlated with the onset of symptoms in people with asthma and the commencement of inhaled reliever medication and oral steroids. However, neither PM$_{10}$ nor PM$_{2.5}$ were correlated with increased asthma attacks, exercise induced asthma or health care attendances. This study was adequately powered to detect changes in these outcomes; indeed strong positive associations were found for less frequent outcomes such as commencing oral steroids. The lack of association with health care attendances in this study where PM$_{10}$ rarely exceeded 40 µg/m$^3$ and PM$_{2.5}$ rarely exceeded 25 µg/m$^3$ is concordant with results from my earlier work in
Darwin which found a strong association with emergency department attendances above but not below a PM$_{10}$ threshold of 40 µg/m$^3$ (8). The two year study of emergency department attendances presented in this thesis (Chapter Seven) also failed to find an association with asthma presentations at similarly low particulate concentrations in spite of the larger sample size. It is plausible that health attendances for asthma could have a non-linear association with particulate pollution as most people will initially try to manage exacerbations of their own or their child’s asthma before seeking professional help. A limitation of the studies in this thesis is that the concentration response associations were not explicitly tested for any outcome. In the wider literature, mortality has been shown to have a linear relationship with particulate air pollution with no lower threshold (1, 195, 251), however the concentration response relationships for other outcomes, such as asthma exacerbations have not been well characterised (252).

In view of the well documented evidence of the association of both biomass and non-biomass derived particulates with hospital admissions for asthma (45, 125, 253-255), it was surprising that the time-series and case-crossover studies (Chapters Five and Six) did not find clear associations between PM$_{10}$ and this outcome. The two studies examined 3 and 10 dry seasons respectively and both captured many episodes during which air quality guidelines were exceeded for several days. There were positive associations of similar magnitude in the two studies, (12% vs 14% increase per 10µg rise in PM$_{10}$), although the confidence intervals were wide and included the null in both cases. The most likely explanation for these results is that the low daily admission rates for asthma (approximately one patient every two days - half that of the emergency department attendance rate) limited the statistical power to detect an association.

**Hospital admissions and emergency department attendances for respiratory and cardiovascular conditions**

Admissions to hospital for respiratory and cardiovascular conditions were examined in Chapters Five and Six. There was consistency in the main findings of these studies for the
primary outcomes studied. Admissions for all respiratory conditions were positively associated with \( \text{PM}_{10} \) with much greater effect sizes for Indigenous people, while admissions for cardiovascular conditions tended to have absent or negative associations. The Emergency Department study conducted over two years of relatively lower particulate levels, found no association with presentations for respiratory conditions. However in keeping with the studies of admissions, cardiovascular attendances tended towards negative associations with \( \text{PM}_{10} \) and \( \text{PM}_{2.5} \).

The tendency towards inverse associations of \( \text{PM}_{10} \) and \( \text{PM}_{2.5} \) with cardiovascular diseases was the most surprising outcome from all these studies because previous research has demonstrated an association between particulate matter and a range of adverse cardiovascular outcomes, underpinned by the identification of plausible patho-physiological mechanisms (1, 138, 256, 257). My findings may be anomalous due to the small size of the studies presented here. However, three other much larger studies have examined the relationship between ambient biomass smoke and both cardiovascular and respiratory hospital admissions. The first was conducted in Christchurch, New Zealand where outdoor particulates are primarily derived from wood heaters (132), while the other two examined bushfire smoke events in Malaysia and Sydney (142, 155). All three studies found relatively large positive associations with respiratory admissions but generally absent associations with cardiovascular admissions. The hypothesis that particulate matter derived from biomass combustion has greater respiratory and lesser cardiovascular toxicity than particulates derived from other sources invites further examination.

**Discussion**

*Contribution to the epidemiology of exposure to ambient biomass smoke*

Health effects of air pollution tend to be of small magnitude and relatively large populations are required to be able to detect these statistically (58). However, the larger the population
centre, the more likely it is that ambient particulates will arise from a diversity of sources especially emissions from motor vehicles and industrial plants. Apportioning different sources, such as the proportion of particulates derived from wood heaters in major population centres is technically difficult and there are few published epidemiological studies in which this has been attempted (134, 135). Identifying episodes of severe smoke pollution from vegetation fires is easier, and large population centres can be affected. However, such episodes can be difficult to study as they are generally unexpected, infrequent and of short duration, limiting the data available for analysis. All these factors contribute to the relative paucity of studies concerning ambient biomass smoke.

Although bushfire smoke is the only important source of air pollution in Darwin, its small population is an important limitation for epidemiological studies. I attempted to overcome this constraint by using two different strategies. The first was to conduct a statistically well powered panel study of adults and children with asthma that found clear associations between ambient biomass smoke and asthma symptoms and medication use. It had the further strength of the inclusion of aeroallergen data in the analysis. Secondly, as my primary study of hospital admissions was based upon just three years of directly measured exposure data, I conducted additional analyses of hospital admissions based on a longer (ten year) time period using estimated ambient PM$_{10}$. The main findings from the longer study were generally consistent with the short-term study of hospital data. Both studies demonstrated associations between daily bushfire derived PM$_{10}$ and respiratory admissions that were of much greater magnitude in Indigenous people.

The structure of the population of Darwin needs to be considered when interpreting the findings of the hospital studies. The relatively young population (258) would mean that the proportion of people in known risk groups for air pollution, such as the elderly and those with chronic diseases, is likely to be smaller than in other capital cities. However the much higher proportion of Indigenous people would have countered this. Although we controlled for Indigenous status in all analyses, the hospital studies relied on information routinely collected for administrative purposes. While an audit of the accuracy of recording of
Indigenous status in hospital records found that of 94% were correctly identified, when misclassification did occur it was nearly always in the direction of wrongly identifying an Indigenous person as non-Indigenous (259).

An overall strength of this research program has been the general consistency of findings between the different studies. Additionally my findings of associations between bushfire smoke and exacerbations of asthma and respiratory hospital admissions are also supported by the limited available international literature, much of which has been recently published. They are also supported by two recent Australian studies that had the advantage of being able to directly compare bushfire derived particulate matter with other sources of particulate matter in cities with large populations (141, 142). There is now little doubt from the available evidence that high concentrations of airborne particulates derived from vegetation fires are associated with exacerbations of respiratory diseases, hospital admissions and deaths (see Chapter 3, Table 3.2). My further contributions to this field have been in two areas:

- I have provided new evidence that adverse effects from bushfire smoke, particularly in relation to asthma, are measurable at low concentrations of pollution (ie PM$_{10}$ < 40µg/m$^3$ and PM$_{2.5}$ < 25µg/m$^3$); and
- I have provided the first Australian evidence that Indigenous people are particularly at risk from particulate air pollution.

This work has provided important local evidence for policy and planning of health services. It also fulfills my overall aim of increasing the evidence base for land and fire management practices. Below I discuss how my work could contribute to land and fire management in Australia and elsewhere.

**Contribution to land and fire management in Australia**

**Understanding the risks of air pollution from severe wildfires**

Severe bushfires are occurring more often than ever before in Australia (5). With many major population centres situated close to large areas of native vegetation, it is conceivable that a
substantial part of the health and economic costs associated with bushfires could be attributable to smoke pollution, which can affect millions of people, rather than the fires themselves (219). In spite of the well established wider evidence concerning particulate air pollution, and the more recent evidence specifically concerning smoke from vegetation fires, Australians to date have given relatively little attention to this issue. In Australia for example, the summer of 2006-7, was a particularly bad fire season associated with widespread and prolonged droughts in many regions (260). While there is little scientific commentary and evaluation available so soon after these fires, public perception of them can be gleaned from an online summary of media reports chronicling the six months of severe fires around Australia from September 2006 (261). These described the extent and location of fires, the many homes that were lost and the death of a man who fell from a trailer while fighting fires. Although there were many descriptions of severe smoke pollution, no reports from this particular summary referred to smoke as a hazard in itself. As demonstrated by such reporting, smoke pollution was primarily newsworthy because it reduced visibility, in some cases necessitating road closures, or disrupted electricity supplies. However, in the context of severe wildfires, journalists appear to have failed to grasp, or be less motivated to report the linkage between human health and particulate pollution.

Media reporting of fire might also reflect the perceptions of experts. While public health authorities have a detailed understanding of the health risks of air pollution and have established protocols for responding to severe pollution episodes (262, 263), knowledge of these health risks does not appear to be widespread amongst experts and policy makers in the field of bushfire management. For example, recent technical reports by both the Australian Government (5) and Australia’s Cooperative Research Centre (CRC) for Bushfires (264) have failed to include substantive consideration of smoke haze in their assessments of the overall community impacts and estimated economic losses from bushfires in Australia. Smoke pollution has been acknowledged by the Bushfires CRC as an ‘emerging issue’ but they have not tackled this problem directly. Their small health research program has focused on issues such as documenting the toxic components of bushfire smoke (265) and evaluating masks for the protection of the health of fire-fighters (266), rather than examining
broader population health impacts. A recent detailed examination of the social, political and scientific influences on fire management in both Australia and the USA, also lacked any reference to the public health or economic impacts of air pollution from wildfires (267).

The risk of omitting any consideration of smoke pollution from policy discussions about bushfires in Australia is illustrated by two economic analyses of forest fires. The first was conducted by the International Development Research Centre and the Institute of Southeast Asian Studies in 1999, ‘Indonesia’s fire and haze: The cost of the catastrophe’ (268). In a wide ranging analysis, the cost of the Southeast Asian fires in 1997-98 was estimated to be between US$4.4-9.7 billion, of which about US$1 billion was attributed to short-term health costs caused by harm from the air pollution. When the book was updated in 2006, the estimated health costs were acknowledged to be a major underestimate in the light of evidence that had subsequently became available (268, 269). A recent analysis has been conducted in a setting more analogous to Australia’s situation than the extreme forest fires of South East Asia. The economic impact of a single forest fire in Canada that burned for one week and produced a smoke plume affecting two population centres for just one day was studied in detail. This included a valuation of timber losses, environmental harms, fire fighting costs and the health costs of exposing 1.1 million people to a mean PM$_{2.5}$ concentration of 55 µg/m$^3$ for 24 hours (219). The authors found that the health costs, although less than the timber losses, exceeded losses due to infrastructure damage and fire fighting costs (Table 8.1).

The estimates in Table 8.1 are subject to many assumptions and limitations. Although the authors followed established protocols for evaluating the health costs of air pollution, for the purposes of this study they assumed that the health impacts were entirely due to PM$_{2.5}$ and that the epidemiology of particulates derived from forest fires was the same as for particulates from other sources (219). Nonetheless, this economic analysis, coupled
Table 8.1: Estimated values of health impacts related to increased PM$_{2.5}$ levels on 24 May 2001 and other losses from the Chisholm Fire in Canada. Adapted from Rittmaster et al (219).

<table>
<thead>
<tr>
<th>Losses</th>
<th>Estimated value (Canadian dollars)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Value of health impacts of exposure of 1.1 million people to exceed PM$_{2.5}$ from the fire for one day.</td>
<td>$10,111 911 – $12,137 043</td>
</tr>
<tr>
<td>Loss of 75 buildings including 21 homes.</td>
<td>not estimated.</td>
</tr>
<tr>
<td>Lost infrastructure (bridges)</td>
<td>$2,000 000</td>
</tr>
<tr>
<td>Loss of electrical power infrastructure</td>
<td>$2,000 000</td>
</tr>
<tr>
<td>Fire-fighting costs (7 days)</td>
<td>$10,000 000</td>
</tr>
<tr>
<td>Timber supply lost</td>
<td>$20,000 000</td>
</tr>
</tbody>
</table>

With the emerging epidemiological research, serves to highlight the potentially large magnitude of the public health and economic impacts of smoke pollution from wildfires.

By extension, the evidence from the Canadian study described above (219) would suggest that the public health and economic consequences of the relatively prolonged exposure of Melbourne’s 3.7 million residents to the smoke haze of December and January 2006-7 might have been considerably greater than the public health and economic costs of the actual flames and fire-fighting response. Victoria’s Environment Protection Association (EPA) reported that during December 2006 the PM$_{10}$ maximum target of 50 µg/m$^3$ was exceeded for up to 11 days (depending on the region of Melbourne) ranging from 60-254 µg/m$^3$ during this period. Data for PM$_{2.5}$ were less complete but the number of days exceeding the reporting target of 25 µg/m$^3$ was similar with the maximum 24 hour mean PM$_{2.5}$ ranging from 93 µg/m$^3$ to 116 µg/m$^3$ (270), about double the level reported for the Canadian case study. Figure 8.1 shows daily visibility presented as an index of maximum airborne particles in Melbourne throughout 2006. This clearly illustrates that bushfire smoke was the major reason that Melbourne did not achieve its air quality objectives for particulate air pollution in 2006 (270).
Understanding and managing the risks of air pollution from prescribed burns

Clearly, an understanding of the harms and economic costs associated with severe pollution events is essential for understanding the potential contribution, relative costs and acceptability of preventive activities. Indeed, the main intervention, setting fires under controlled conditions to reduce fuel loads, is neither free of risk nor universally accepted (271). Smoke pollution is a reason frequently cited by land managers for public opposition to deliberate burns (40, 267, 271-273). As the Bushfires CRC has noted: “While there is a general acceptance within the community of smoke generated by uncontrolled bushfires, smoke from prescribed fires can generate considerable criticism of the land managers, agencies and governments concerned” (265). This apparently contradictory situation can be understood in terms of the well described factors that shape public perception of, and responses to risk (274). Wildfires, on one hand, are feared because they are catastrophic and unpredictable, have the capacity to destroy homes and livelihoods and in contrast to

Figure 8.1 Visibility, as daily maximum airborne particle index at selected air quality monitoring stations in Melbourne 2006. Adapted from EPA Victoria. (270)
smoke pollution, the associated casualties are clearly identifiable. As discussed above, these factors tend to dominate media reporting and community concerns during severe bushfires. Prescribed burns, on the other hand, do not (usually) threaten life or property, yet the associated air pollution can affect entire communities, is deliberately imposed, and the ‘benefits’ of this exposure, in terms of preventing later fires, may be poorly understood.

The fear that public opposition might remove their one effective tool for countering severe fires, could be one reason why the Bushfires CRC and other land management agencies in Australia have been reluctant to quantify or publicise the potential health risks of exposure to bushfire smoke, preferring to describe it in terms of being a ‘nuisance’ or ‘inconvenience’ (265, 267). The Bushfires CRC has supported the development of sophisticated models that predict the likely path of smoke plumes (275). While such modelling is an invaluable tool for fire managers it cannot predict the absolute level of particulates expected at a given location. Nor does it overcome the lack of epidemiological evidence about the likely harms of exposure, or the effectiveness of interventions to reduce this.

The research presented here has made new contributions to understanding the health impacts of biomass smoke at lower levels, analogous to the pollution from low intensity prescribed burns (i.e. particulate levels at and below Australia’s current air quality standard of 50µg/m$^3$ for PM$_{10}$). For example the exacerbation of asthma and admissions to hospital for respiratory conditions, were measurable even at the lower range of exposure levels associated with vegetation fires. There is uneven distribution of the impacts of air pollution among the population and the elderly, infants and those with chronic diseases have been all been previously recognised as being at higher risk (175, 183, 205, 276, 277), and in this work, Indigenous people in Darwin were also found to be a higher risk group. As there is no evidence for a lower level of particulate matter from biomass smoke that could be considered safe for all people, the health risks of air pollution need to be understood and incorporated into plans for deliberate vegetation burns.
The guidelines suggested below are based upon the pragmatic acceptance that it is impossible to exclude fire and therefore smoke from Australian environments, and that there is a role for using planned fires to reduce the threats from unplanned fires. However, it is important that land managers can justify the argument that prescribed burns are effective in preventing major fire disasters, particularly in the context of changing climatic conditions. Fundamentally there is a trade-off between the adverse health impacts of low levels of smoke pollution from frequent planned fires against that of higher particulate levels and other public health dangers associated with wildfires. In relation to deliberate vegetation fires, the evidence presented in this thesis supports the following guidelines:

• Managers of deliberate vegetation burns should minimise the amount of air pollution affecting urban centres as much as possible because, regardless of the level of pollution, there will be an increased risk of adverse health impacts for some people.

• The impact of deliberate vegetation burning on air quality in urban areas should be measured to provide information for evaluating potential health impacts and planning future burning programs.

• Adequate advance public notification and health information should be integral to any deliberate burning program, to allow people at higher risk from exposure to bushfire smoke to take appropriate action.

Australia’s current ambient air quality standard for PM_{10} is 50µg/m^3 and the reporting advisory standard for PM_{2.5} is 25µg/m^3 (278). Studies need to be replicated in other settings to provide a more substantial evidence base before a separate air quality standard for biomass derived particulates can be justified. Nevertheless it is notable that the limited available evidence suggests that a more stringent target could be appropriate. For example the magnitude of reported associations with respiratory outcomes was greater for ambient particulate matter derived from biomass combustion compared with particulate matter derived from other sources in the only three studies that have sought to make this
comparison (45, 141, 142). The results from two Darwin studies suggest that 40 µg/m$^3$ could be an appropriate standard below which the adverse impacts for asthma are less serious. An earlier study observed a threshold of 40µg/m$^3$ of PM$_{10}$ for detecting emergency department attendances for asthma (8) while the asthma panel study (Chapter Four) found no association with asthma health attendances to general practitioners, specialists or hospital emergency departments in a period during which PM$_{10}$ generally remained below 40 µg/m$^3$.

Guidelines will need to be refined as the evidence base improves, and information about a wider range of health outcomes (such as long-term health outcomes, perinatal morbidity and mortality) and exposure measures (such as PM$_{2.5}$) becomes available.

**The role of Indigenous fire management in Australia**

The role of Indigenous Australians in fire management, and thus smoke pollution, deserves special mention for a number of reasons. As discussed in Chapter One, it is generally accepted that the skilful use of fire over millennia maintained Australia’s biodiversity and minimised catastrophic wildfires. However, severe wildfires have become a regular feature of Australian landscapes since Indigenous management ceased following European colonisation (14, 279). Areas of the Northern Territory remaining under Indigenous management have better preserved biodiversity and lower fuel loads than other regions of our tropical savannas (25, 280). Moreover, air pollution in Darwin has worsened in association with the relatively recent transition from Indigenous to predominantly non-Indigenous land management (212). However, in addition to the disruption of environmental management provided by Indigenous people, the human consequences of the transition away from customary life styles following European colonisation have been catastrophic (281). As discussed in Chapters Five and Six, the social disadvantage and chronic ill health now experienced by this population are the most probable reasons that Indigenous people in Darwin now suffer a disproportionate burden of adverse health outcomes associated with particulate air pollution. Social disadvantage is not only an important risk factor for the development of chronic diseases, it is associated with reduced access to health services and the attendant opportunities for early diagnosis and management, further increasing the
health risks associated with these conditions and environmental hazards that may exacerbate them (277, 282, 283).

**Global implications of fire management**

There are many other regions of the world where fire and smoke are becoming increasingly serious public health problems. A dramatic example of this is the annual smoke haze from fires in South East Asia. This phenomenon could be viewed as a more extreme consequence of the transformation of land use associated with cultural transitions than that which I described for Australia in Chapter One. As fires are lit to clear land for new commercial or farming enterprises, Indigenous groups are marginalised and governments are reluctant to enforce the laws constraining the wide scale deliberate use of fire (284-286). The South East Asian haze has the added complexity of affecting many countries, some of which (e.g. Singapore) do not substantially contribute to the problem. Far more attention has been given to the haze from South East Asian fires than elsewhere presumably because of its sheer magnitude, recurring nature and the large populations affected. As discussed earlier in this chapter, economic assessments have explicitly included the health affects of smoke (269). Similar to Australia, the main population impacts are in the areas of health, welfare and economic losses and these are magnified in settings where a higher proportion of the population is impoverished and access to health care is more limited (268). However, the main interventions to avoid adverse heath and social impacts are in the realm of land use and environmental management and both these areas require political direction and international cooperation. An example of this is the Association of South East Asian Nations Agreement on Trans-boundary Haze Pollution which has yet to be ratified by all countries (285, 287).

The roles that fire emissions play in the global carbon and water cycles are a subject of increasing research and beyond the scope of this thesis. Alteration in global environmental systems will have far reaching human health implications and it is important to acknowledge that the impacts of smoke pollution go beyond the direct adverse health effects that I have
Landscape fires currently emit an amount of carbon, mostly in the form of CO₂, approximately equivalent to two thirds that of carbon emissions from fossil fuels (288, 289). Increasing wildfires are thus likely to contribute to, as well as be a consequence of, global environmental change. They are expected to become more frequent and intense as global warming produces more frequent droughts and high fire danger weather conditions (56, 290). This is further exacerbated by the fact that increased atmospheric CO₂ concentrations promotes the growth of woody vegetation leading to more rapid accumulation of fuel loads (289, 291).

The particulate matter emitted from vegetation fires can also affect global climate systems by contributing to global dimming, the term used to describe the reduced penetration of sunlight through the earth’s atmosphere. This occurs via many complex and interrelated mechanisms that depend on the composition and chemistry of the particles. Carbon based particles, the largest single source of which is vegetation fires (292), contribute to both surface cooling and atmospheric warming by absorbing sunlight in the earth’s atmosphere (293). Sulphates, about 80% of which are derived from the combustion of coal (294), primarily cause cooling by scattering sunlight and reducing the amount that reaches the earth’s surface (295). The net effect of all such processes has so far been one of cooling and this has offset the global warming associated with greenhouse gas emissions by about 50% (296). Particulates also affect global water cycles though regional cooling associated with haze and by providing the nuclei for cloud formation. The effects described above have been demonstrated in South East Asia where particulates from forest fires, the use of domestic biomass fuels and fossil fuel combustion all contribute to haze formation that has been termed the Asian Brown Cloud (295). This haze has been shown to change the balance of water and cloud patterns and has caused reduced regional rainfall in South East Asia and possibly increased rainfall in northern Australia (297).

All of the above factors add considerable complexity to local land management issues and highlight the need to be proactive in the management of fire at local and regional levels. This is not only to minimise the direct health impacts of biomass smoke but to decrease the
contribution to large scale fires to global environmental change from the emission of both particulate matter and greenhouse gases, especially CO₂.

**Future directions**

There are many complex interactions between fire, environments and people and the human health impact of bushfire smoke is just one of many related issues. However, in Australia, it has been a neglected part of the picture. Further research in this field needs to better integrate studies of the practicalities of landscape and fire management in relation to both the risks of fires and the risks of pollution and these risks need to be placed in the wider contexts of global environmental changes and public health policy.

Specific areas for which there are substantial gaps in the available evidence include:

- The effectiveness of fuel reductions burns and other methods of preventing severe landscape fires and analysis of their feasibility, particularly in the context of the predicted increase in extreme fire weather conditions (298). Additionally the ecological, economic and human health impacts of these strategies need to be evaluated;

- The health effects of biomass smoke at different concentrations including the examination of perinatal and cardiovascular outcomes and risks associated with prolonged or recurrent exposure. This should include evaluation of the relative public health importance of biomass smoke in comparison with other sources of air pollution and assessment of the need for specific air quality standards and guidelines for deliberate vegetation burns;

- The biological mechanisms through which biomass smoke causes ill health;

- The economic evaluation of severe bushfires in Australia including the health impacts of bushfire smoke; and
• The effectiveness and feasibility of interventions such as access to air conditioned facilities or high efficiency particle air filters, the use of preventive medication, the avoidance of outdoor physical activity and evacuation from affected regions to protect individuals from the impact of episodes of severe air pollution.

In collaboration with other epidemiologists and landscape and fire ecologists I am planning to contribute to some of these areas by conducting a multi-site study in several Australian regional towns and capital cities affected by biomass smoke pollution from a variety of sources. We will examine the environmental and epidemiological associations with particulate air pollution. This will include the relationship between particulate air pollution and the frequency of landscape fire activity in a range of different vegetation types relative to other sources of biomass smoke. We will also determine meteorological conditions for which it is safe to undertake planned fuel reduction control programs but not likely to cause unacceptable levels of air pollution. The study will have multiple locations to provide improved statistical power to detect health effects from biomass smoke above noisy background data. The geographic range of locations will also provide insights into the role of both weather and climate in influencing health outcomes and fire risk. This will provide empirical (as opposed to modelled) data for better understanding of the potential impacts of climate change on achieving socially acceptable landscape fire management in Australia. The research will be done in collaboration with lead government agencies with a stake in sustainable fire management, public health and the regulation of air quality. The overall aim is to provide a basis for informing and improving existing environmental, public health and fire management policies and practice in Australia.
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Canadian forest fire 2005. (Photo AJ Chapman AP)
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APPENDICES

A. The Darwin Asthma Study: Documentation, protocols and reports

Recruitment flyer
Project Information sheet
Consent forms - adult and child
Questionnaires - adult and child
Asthma risk screening questionnaire
Appointment procedure including protocols for spirometry, cotinine collection and decontamination
Daily dairy
Project feedback for participants


B. The Darwin Smoke Project: Newsletters and presentations

Newsletter 1. June 2004
Newsletter 2. October 2004
Newsletter 3. March 2005
Newsletter 4. July 2005
Newsletter 5. December 2005

Appendix A.

The Darwin Asthma Study: Documentation, protocols and reports
Asthma in the Tropics

Do you have asthma?

Have you had symptoms in the last 12 months?

Would you like to be part of a research project about environmental factors that trigger asthma?

Researchers from the Darwin Asthma Study are looking for volunteers to be a part of a project investigating asthma in our tropical environment.

Adults and school-aged children with asthma who live in Darwin or Palmerston are eligible to participate.

You will be asked to keep a track of your asthma symptoms for seven months during the dry season of 2004. Over the same period the researchers will be recording many different environmental factors to see how these influence your asthma symptoms.

The aim of the research is to help find ways to reduce asthma for people living in Darwin and similar environments.

Interested in participating?

Contact the Darwin Asthma Study for an information pack

To contact us you can:
- Email asthmastudy@tedgp.org.au with your contact details
- Post this form to the Darwin Asthma Study, Darwin Centre for Disease Control PO Box 40596 Casuarina NT 0811
- Fax this form to 89228310
- Phone 89228401
- SMS 'asthma' to 0407612247 or call to leave a message

☐ Please send me an information pack about the Darwin Asthma Study

Name .......................................................... Address ..........................................................

Telephone b/h ........................................... a/h ................................

The Darwin Asthma Study is supported by the Australian Research Council in partnership with Charles Darwin University, the Menzies School of Health Research, Flinders University of South Australia, Australian National University, Northern Territory Government, Bureau of Meteorology, Commonwealth Scientific and Industrial Research Organisation, the Top End Division of General Practice and the Asthma Foundation of the Northern Territory.
Information for people interested in participating in the Darwin Asthma Study

What is the asthma study about?
Asthma is a chronic lung condition where inflammation of the airways causes symptoms such as cough, wheeziness and shortness of breath. Symptoms can be exacerbated by many causes including pollens, animal dander and smoke or chemicals. This project is to help us learn more about local environmental factors that might be important triggers for asthma.
The study involves following you or your child’s daily symptoms of asthma during the dry season of 2004, that is from the 1st of April until the 31st October 2004. This information will be compared with daily pollen counts, smoke levels, air temperature and humidity, and the amount of coughs and flu present in Darwin.

What do I need to do to be in the asthma study?
You need to be 8 years old or over and have had asthma diagnosed by a doctor to be eligible to participate in the study. Participation will entail the following:
(1) At the start of the study we will ask you to fill in a questionnaire about your or your child’s asthma, and to blow into a meter that will record you peak flow rate when you breath out, and your forced expiratory volume which is the greatest volume of air you can blow out in one second. We will do these measures before and after you have an inhaled dose of reliever medication such as Ventolin.
(2) We will use a vacuum cleaner to sample and measure the levels of known household allergens such as house dust mite, cockroaches and cat dander and ask for a sample of your (or your child’s) saliva to measure the amount of cotinine, a substance which indicates your level of exposure to cigarette smoke.
(3) During the study we will ask you to keep a daily diary of your (or your child’s) asthma symptoms, medication use, exercise, outdoor time and visits to the doctor or hospital. We will provide this for you to use as an aid to help you remember you symptoms over each two week period.
(4) We will give you a peak flow monitor and ask you to write down your peak flow rate each day.
(5) You will be telephoned by a person from a market research company every second week and asked a set of standard questions about your asthma such as your symptoms, medications use, and visits to the doctor on each day for the previous two weeks. See attached sheet for the weekly record form to be used for this.

Will I receive any benefits by being in the study?
There is no direct benefit from participating in the study although you may get a better idea about how much of your asthma is triggered by pollen, smoke or other allergens. Being in the study will not interfere in any way with the care you receive from your normal general practitioner for your asthma. You will not be expected to take any additional treatment, or receive any additional advice about your asthma from the researchers. The purpose of the study is to improve our knowledge of the relative importance of several environmental triggers of asthma. For example, the type of pollens common in tropical regions of Australia and their role in allergic conditions has not been studied before.

What will be done to make sure the information is confidential
All participants in this study will be given a code number and information collected will be kept under the code number on a secure database at Northern Territory University with access only to the study investigators.
What will be done with the study results?
The results of the study will be written in a report for local and government use and published in medical journals. It is important to inform policy makers and others of any new information about bushfire smoke and asthma, so that air pollution can be appropriately monitored and controlled. You or your child will not be identified in any of the study results.

What are my rights as a participant?
(1) You can decide whether or not you would like yourself or your child to take part in this research project.

(2) You can decide whether or not you would like to withdraw yourself or your child from the study at any stage without having to give an explanation for your decision.

(3) All identifiable information about you will remain completely confidential. No identifiable medical information will be made available to anyone who is not on the project team.

(4) This project follows the guidelines of the National Statement on Ethical Conduct in Research involving Humans 1999 and has been approved by the ethics committees of the Northern Territory University and the joint committee of the Menzies School of Health Research and the Northern Territory Department of Health and Community Services. You can receive a copy of these documents if desired by contacting the project team. See next page for contact details.

(5) You may wish to discuss the research project with your family or your doctor. You can also ask for more information before deciding to take place in the project.

How can I find out about the results when the study is finished?
If you would like a written copy of the summary of the results of the study sent to your home address then complete the relevant section on the consent form and a result summary will be sent to you in late 2004.

Who has funded this study?
The Darwin Asthma Study is funded by the Australian Research Council in partnership with the Northern Territory University, Flinders University of South Australia (FUSA), the Bureau of Meteorology, the Commonwealth Scientific and Industrial Research Organisation (CSIRO), the Menzies School of Health Research and the Northern Territory Government Departments of Health, Environment and Business.

Who do I contact?
For more information or for any urgent matters that may relate to the asthma study contact:
Dr Rosalind Webby, Darwin Centre for Disease control on 892 28401
Dr Fay Johnston, Northern Territory University on 89467015.

If you have any concerns about the project at any stage you are invited to contact the executive officers of the Ethics Committees of either the Charles Darwin University on 89467064, or the Menzies School of Health Research on 89228624.
CONSENT TO PARTICIPATE IN A THE ASTHMA STUDY
Darwin 2004

I, .............................................................. , agree to participate in the asthma study. In giving my consent I acknowledge that:

1. The procedures required for the project have been explained to me, and any questions I have about the project have been answered to my satisfaction.

2. I have read the Information Statement and have been given the opportunity to discuss the information and my involvement in the project with family and /or friends.

3. I am aware of the risks and inconveniences associated with the project.

4. I understand that I can withdraw from the study at any time, without affecting my treatment or my relationships with the researcher(s) now or in the future.

5. I understand that my involvement is strictly confidential and no information about me will be used in any way that reveals my identity.

Signed:.................................................. Date: ....................................

Name:....................................................

Please indicate below if you would like to receive written results of the study

Yes I would like to be sent results of the study.

No I do not wish to be sent results of the study.

Mailing address for results____________________________________________________
____________________________________________________
CONSENT FOR A CHILD TO PARTICIPATE IN A THE ASTHMA STUDY
Darwin 2004

I, .................................................... , agree to my child
...........................................(full name) participating in the asthma study. In giving
my consent I acknowledge that:

1. The procedures required for the project have been explained to me, and any
questions I have about the project have been answered to my satisfaction.

2. I have read the Information Statement and have been given the opportunity to
discuss the information and my involvement in the project with family and /or
friends.

3. I am aware of the risks and inconveniences associated with the project.

4. I understand that I can withdraw my child from the study at any time, without
affecting my treatment or my relationships with the researcher(s) now or in the
future.

5. I understand that my involvement is strictly confidential and no information about
me will be used in any way that reveals my child’s identity.

Signed:.............................................(parent or guardian)     Date: ......................

Name:..................................................

Please indicate below if you would like to receive written results of the study

Yes I would like to be sent results of the study.

No I do not wish to be sent results of the study.

Mailing address for results____________________________________________________
______________________________
Asthma in the Tropics

QUESTIONNAIRE

for Adults and Children 12 years and older

Thank you for being a part of this important research. Please complete this initial questionnaire and mail it to the asthma research team in the pre paid envelope. All information provided will be kept strictly confidential and it will be used for the research project only.

1. Name ________________________________________________

2. Sex  male  female

3. DOB  /  /  /

4. Ethnic background  Indigenous  Non Indigenous

5. Residential address
   Number and street _______________________________
   Suburb and postcode_____________________________

6. What telephone number should we use to contact you each fortnight?  ____________________________

7. What is your suburb of workplace or major daytime activity?  ____________________________

DIAGNOSIS OF ASTHMA

8. Have you been diagnosed with asthma by a doctor?  Yes  No

9. Have you had any asthma symptoms, such as wheeze, coughing, chest tightness or shortness of breath or used any asthma medication at any time during the last 12 months?  Yes  No

ASTHMA CONTROL

10. Are you using regular preventive medication such as pulmicort, beclomforte, qvar or flixotide puffers?  Yes  No

11. Have you been woken from sleep by asthma in the past month?  Yes  No
12. Have you been admitted to hospital because of asthma in the past year?
   Yes  No

13. How many different GPs have you seen for your asthma in the past 12 months?
   ........................................

14. How would you rate the severity of your asthma in the last month?
   Mild
   Moderate
   Severe

15. Have you taken oral steroid medication (eg prednisolone tablets) for asthma in the past month?
   Yes  No

MEDICAL HISTORY

16. Have you ever had hay fever?     Yes  No

17. Have you ever had eczema?      Yes  No

HOUSEHOLD

18. Counting yourself how many people live in your house?
   1  2  3  4  5  6  7  8
   If more than 8 please write how many ________

19. How many bedrooms are there in your house?
   1  2  3  4  5
   If more than 5 please write how many ________

20. What is the highest grade or educational level that any adult member of your household has completed? (tick one of the following)

   Did not complete primary school
   Completed primary school
   Did not complete highest year of secondary school
   Completed highest level of secondary school
   Completed a trade certificate or university degree

SMOKING

21. Do you smoke cigarettes, cigars, pipes or tobacco (rolled)?   Yes  No
IF NO go to Question 24

22. How many cigarettes do you usually smoke a day? .............

23. Do you smoke inside the house or car? Yes No

24. Do other people living in your house smoke cigarettes, pipes, cigars or tobacco (rolled)? Yes No

IF NO go to Question 28

25. How many other people in the household smoke?
None 1 2 3 4 5 6 7 8

26. Do they smoke in the house or car? Yes No

27. How many other people smoke inside the house or car?
None 1 2 3 4 5 6 7 8

ASTHMA TRIGGERS

28. Do you have a cat at your home? Yes No

29. Do you have a dog at your home? Yes No

30. Do you have a gas stove/ oven or cook-top in your home? Yes No

If YES  Does it have a range hood with an extractor fan that you use when cooking? Yes No

31. Please estimate the number of hours per weekday you spend in air-conditioning? ................. hours

32. Please estimate the number of hours per weekend day you spend in air-conditioning? ................. hours
33. What triggers your asthma? (tick as many as relevant)

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<tr>
<th>TRIGGERS</th>
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<tbody>
<tr>
<td>Exercise</td>
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<td>Pollens</td>
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<td>Dust</td>
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<td>Swimming pools</td>
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<td>Colds/ viral infection</td>
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<td>Foods</td>
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<tr>
<td>Change in weather, exposure to cool air</td>
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<tr>
<td>Drugs. eg aspirin or non steroid anti-inflammatory tablets</td>
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<tr>
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<tr>
<td>Animal hair/fur</td>
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<tr>
<td>Other ……………………………………….</td>
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***THANK YOU***

THIS IS THE END OF THE QUESTIONNAIRE

Please return in the prepaid envelope to: Dr Rosalind Webby
Centre for Disease Control, PO Box 41326 Casuarina NT 0811

Office use only

| Cotinine | FEV1pre | FEV1post |
Asthma in the Tropics

QUESTIONNAIRE
for children eleven years old or younger

Thank you for being a part of this important research. Please fill out the following information for your child and mail it to the asthma research team in the prepaid envelope. All information provided will be kept strictly confidential and it will be used for the research project only.

1. Name of parent/guardian filling out this form: ________________________________

2. Name of your child: ________________________________ __________________

3. Sex of child    male    female


5. DOB of child    /    /    /    /

6. Residential address of child
   Number and street    ______________________________
   Suburb and postcode    ______________________________

7. What telephone number should we use to contact you each fortnight?
   ________________________________

8. What is the suburb of your child’s school? ________________________________

DIAGNOSIS OF ASThma

9. Has your child been diagnosed with asthma by a doctor?    Yes    No

10. Has your child had any asthma symptoms, such as wheeze, coughing, chest tightness or shortness of breath or used any asthma medication at any time during the last 12 months?    Yes    No

ASTHMA CONTROL

11. Is your child using regular preventive medication such as pulmicort, beclol forte, qvar or flixotide puffers?    Yes    No

12. Has your child been woken from sleep by asthma in the past month?
13. Has your child been admitted to hospital because of asthma in the past year?
   Yes  No

14. How many **different** GPs has your child seen for asthma in the past 12 months?
   ..................................

15. How would you rate the severity of your child’s asthma in the last month?
   Mild  Moderate  Severe

16. Has your child taken oral steroid medication (eg prednisolone tablets) for asthma in the past month?
   Yes  No

MEDICAL HISTORY

17. Has your child ever had hayfever?
   Yes  No

18. Has your child ever had eczema?
   Yes  No

HOUSEHOLD

19. How many people live in your home including yourself?
   1  2  3  4  5  6  7  8
   If more than 8 please write how many  .................

20. How many bedrooms are there in your house?
   1  2  3  4  5
   If more than 5 please write how many  ......................

21. What is the highest grade or educational level that any adult member of your household has completed? *(tick one of the following)*
   Did not complete primary school
   Completed primary school
   Did not complete highest year of secondary school
   Completed highest level of secondary school
   Completed a trade certificate or university degree
SMOKING

22. Does any one in your household smoke cigarettes, pipes, cigars or rolled tobacco?  
   Yes  No  
   IF NO Go to Question 26

23. How many people in your household smoke cigarettes, pipes or rolled tobacco?  
   0  1  2  3  4  5  6  7  8

24. Do any of these people smoke inside the house or car?  
   Yes  No  
   IF NO Go to Question 26

25. How many people smoke inside the house or car?  
   0  1  2  3  4  5  6  7  8

ASTHMA TRIGGERS

26. Do you have a cat at your home?  
   Yes  No

27. Do you have a dog at your home?  
   Yes  No

28. Do you have a gas stove or cook-top or oven in your home?  
   Yes  No  
   IF YES  
   Does it have a range hood with an extractor fan that you use when cooking?  
   Yes  No

29. Please estimate the number of hours per \textbf{weekday} your child spends in air-conditioning?  
   .................. hours

30. Please estimate the number of hours per \textbf{weekend} day your child spends in air-conditioning?  
   .................. hours
31. What triggers your child’s asthma? *(tick as many as relevant)*

<table>
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<tbody>
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<td>Other</td>
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**THANK YOU**

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Please return in the prepaid envelope to: Dr Rosalind Webby, Centre For Disease Control, Po Box 41326 Casuarina NT 0811

Office use only

| Cotinine | FEV1pre | FEV1post |
### ASTHMA RISK SCREENING QUESTIONNAIRE

<table>
<thead>
<tr>
<th>QUESTION</th>
<th>SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you been woken from sleep by asthma in the past month?</td>
<td>Yes=15 No=0</td>
</tr>
<tr>
<td>Have you been admitted to hospital because of asthma in the past year?</td>
<td>Yes =25 No=0</td>
</tr>
<tr>
<td>How many <strong>different</strong> GPs have you seen for your asthma in the past 12 months?</td>
<td>One = 0</td>
</tr>
<tr>
<td></td>
<td>More than one =25</td>
</tr>
<tr>
<td>How would you rate the severity of your asthma in the last month?</td>
<td>Nil/Mild=0</td>
</tr>
<tr>
<td></td>
<td>Moderate/Severe=1</td>
</tr>
<tr>
<td>Have you taken oral steroid medication (eg prednisolone tablets) for asthma in the past month?</td>
<td>Yes=25</td>
</tr>
<tr>
<td></td>
<td>No=0</td>
</tr>
</tbody>
</table>

A total score of 30 or more indicates a high likelihood of hospital admission for asthma within 12 months.

Appointment Procedure for the Darwin Asthma Study

Check participant has returned questionnaire and consent form. If not, consent form needs to be completed before taking saliva or performing spirometry.

Taking cotinine samples
- Ensure participant has fasted for 30 mins prior to taking Cotinine sample
- Take sample ASAP after arrival
- Get participant to remove swab from salivette and either chew or place under tongue until they feel that they can no longer prevent swallowing saliva produced (approx 30 – 45 secs).
- Return swab to salivette and label with name, DOB, date of collection and study number.
- Place in esky until can be placed in fridge
- Offer drink of water prior to spirometry

Using the spirometer – following NAC protocol 2004
Spirometer used – Welsh Allyn Pneumocheck Spirometer #61000
The spirometer consists of two parts – the handset and the printer/charger.

- Measure participant against height chart
- Enter age, height and sex into printer/charger
- Explain and demonstrate procedure to participant (P)
- Attach disposable mouthpiece
- Switch handset on, wait till reads SELECT and press test button
- Screen will read FVC, then STEADY, then DO FVC1
- Ask P to blow following participant instructions as below
- When handset beeps and reads BUSY then GOOD FVC. Push test button again and screen reads DO FVC2
- Repeat until 3 good FVC’s are recorded.
- Place handset back in printer/charger, wait 3 seconds till it beeps and green data light on printer/charger stops flashing. Press print button on printer/charger.
- Deliver Ventolin via spacer – 1 puff, 4 breaths x 2
- Wait 10 minutes (check through Questionnaire while waiting)
- Repeat spirometry as above
- Write results on questionnaire and stick printouts on backing sheet.
- Dispose of cardboard tube and put spacer in bucket for washing and decontaminating later

Participant Instructions
- P must be standing comfortably
- P instructed to breathe in fully and seal lips around mouthpiece
- P instructed to “blast air out as fast and as far as they can”
- Need to have a rapid start, a vigorous effort all the way through and continue until absolutely no more air can be exhaled (at least 6 second expiration)
- No leaning forward during the test
- Minimum of 3 acceptable blows
- Smooth, rapid take off with no hesitation, cough, leak, tongue obstruction, glottic closure, valsalva or early termination.

**Care of Spacers**
- Wash in bucket with hot water with kitchen detergent
- DO NOT RINSE
- Air dry on towel in Anne’s room
- When dry, wipe with an Isowipe (70% alcohol wipe), paying particular attention to the inside and outside of the mouthpiece.
- If any device is contaminated with blood it must be discarded.  
  (taken from Asthma Victoria’s Victorian Schools Policy 2003)

**Decontamination of Spirometer**
- Manual recommends visually inspecting screen of mouthpiece before each use
- Instruct P’s NOT to inhale via mouthpiece
- Manual recommends cleaning and sterilising pneumotach after every 50 manouvres. Pneumotach can be scrubbed with a small brush, boiled for 30 minutes and left to air dry for 4 days or blown with a hair dryer on high for 10 minutes.
Please mark this diary each day to help you during the telephone interview with us.
- Tick the box if you have a symptom, leave it unmarked if you did not have a symptom.
- Information about your day time symptoms are best recorded in the evening and night time symptoms in the morning (for example: Monday’s daytime symptoms count from the time you get up until you go to bed. Monday’s night time symptoms count from when you go to bed until Tuesday morning when you get up).

### Symptoms during the day?
- Chest wheezing
- Breathlessness
- Chest tightness
- Cough

### Symptoms during the night?
- Chest wheezing
- Breathlessness
- Chest tightness
- Cough

### Activity today?
- Any physical exercise today?
- If yes, mark if you had difficulties with your breathing afterwards

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<tr>
<th>Day of the week</th>
<th>M</th>
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<tr>
<td>Slept overnight away from Darwin / Palmerston region?</td>
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</tbody>
</table>

**Any asthma attacks today?** Definition: Any asthma episode involving breathlessness and/or wheezing and/or chest tightness and/or coughing that interrupts ongoing activities or requires some procedure, such as resting or use of a nebuliser for a return to normal and comfortable breathing.

- During the day?
- During the night?

**Missed school / normal work today? (exclude weekends and holidays)**

**Visit to a health care facility today because of your asthma? (GP, hospital emergency dept, hospital outpatient, paediatrician)**

**Asthma Medication:** Write down the name of your medication in the following free spaces of the first column and note underneath your regular dose of this medicine (eg. Ventolin 3 puffs). In the daily columns note how often you have taken this medication at that dose (eg. 3 x).

1. medication
   ………………………
   Regular dose: …………..

2. medication
   ………………………
   Regular dose: …………..

3. medication
   ………………………
   Regular dose: …………..

4. medication
   ………………………
   Regular dose: …………..
Thank you for being a part of the *Darwin Asthma Study* in 2004.

Please find enclosed:
- a summary of the results of the study
- a print out of your own results
- an example to help explain what your results mean.

If you would like any further information or help understanding these results please call Anne on 8922 8540.

The Asthma Study was one part of a bigger research program known as the Darwin Smoke Project. If you want to stay in touch and receive our newsletters ask Anne to add you to our email distribution list. Alternatively you can obtain the newsletters from the web, see [www.wildlife.ntu.edu.au](http://www.wildlife.ntu.edu.au/)

Thank you once again for your participation. The findings are contributing to the safe management of bushfire prone land in Australia and also to our understanding of the effects of different types of air pollution on asthma.

Yours sincerely,

Dr Fay Johnston     Anne Myerscough RN     Dr Rosalind Webby
Darwin Asthma Study Results

Introduction
The Darwin Asthma Study aimed to determine which local environmental factors might contribute to worsening of asthma in the communities of Darwin and Palmerston. 251 volunteers with known asthma kept track of their symptoms and medication during the dry season of 2004. During this time we monitored weather conditions, influenza rates in the community and atmospheric levels of smoke pollution, pollen and fungal spores.

Environmental conditions during the study

Weather
2004 was an unusual dry season with rainfall persisting well into June and delaying the drying out of bush grasses. This resulted in lower than usual fire hazard conditions persisting until September.

The maximum daily air temperature ranged from 25.7 to 36.1°C (average 31.8). The minimum ranged from 13.3 to 27.5°C (average 21.2) and the relative humidity at 9.00am varied from 9% to 96% (average 64%).

Smoke levels
Smoke pollution remained low throughout the study period. We measured pollution as PM$_{10}$. This is the mass of particles less than 10 microns across per cubic meter of air. Our average daily level was 20 µg/m$^3$ and it ranged from 3 - 46 µg/m$^3$. Most of the time the PM$_{10}$ were well below Australia’s national air quality standard of 50 µg/m$^3$.

Pollen levels
Our pollen counts also remained very low by Australian standards. Pollen levels ranged from 0.5 – 61 grains per cubic meter of air with an average daily count of just 16. Our grass pollen counts peaked first in April and then again in June. The range for grass pollen was 0-22 grains per cubic meter with an average of 3.

In comparison, total pollen counts in Brisbane have been measured at 700-800 grains per cubic meter and in Melbourne the count can exceed 2000. Grass pollen counts in both these cities often peak at around 300.

Fungal spores
Darwin’s fungal spore counts during the dry season of 2004 were in the moderate range. The counts ranged from 108 to 6049 spores per cubic meter with an average of 1843. In the wet season our counts are much higher.

A similar study in Brisbane in the winter of 2000 recorded counts in the high range. Their average count was 7352 and ranged from 546-67301 spores per cubic meter.

Influenza
Consultations to Darwin GPs for influenza-like illnesses varied from 0.5% to 3% of all consultations during the study. In large outbreaks of influenza consultation rates usually exceed 3% and may approach 10%.

Anne Myerscough, Judy Manning and Dr Fay Johnston attending one of the rooftop pollution monitors in Palmerston
Environmental influences on asthma

While a few people in the group were particularly sensitive to bushfire smoke, most participants were not affected by the low levels of pollution present during the study. We found small but statistically significant associations between smoke pollution levels (measured as PM$_{10}$ and PM$_{2.5}$) and:

- the average number of **asthma symptoms** experienced by participants each day.
- the average amount of **releiver medication** (eg doses from a Ventolin puffer) used by participants each day.
- the proportion of participants starting a course of **steroid tablets** such as prednisolone. Steroid tablets are reserved to treat more serious exacerbations of asthma.

The biggest association was with the use of steroid tablets which rose by approximately 50% with each rise of 10 in PM$_{10}$. Only 40 out of the 251 participants needed to use steroid tablets during the study, with 17 participants needing more than one course of tablets.

All findings were particularly apparent in adults and in people with more severe asthma. Children with mild asthma were mostly not affected by bushfire smoke pollution.

We also found small associations between asthma symptoms and:

- the minimum air temperature
- GP consultation rates for influenza.

Individual factors affecting asthma

As expected, individual factors were important predictors of asthma symptoms and medication use. Regardless of the environmental conditions the following groups all were significantly more likely to have symptoms or use medication for their asthma:

- smokers (compared with non-smokers)
- adults (compared with children)
- people who lived in less crowded circumstances (compared with more crowded)
- non-Indigenous participants (compared with Indigenous)
- those with (or whose parents had) lower compared with higher education levels

We did not find any associations between pollution levels and exercise induced asthma, asthma attacks or health care attendances for asthma. Nor did we find any health effects to be associated with pollen levels, fungal spores, rainfall, relative humidity, dew point or maximum air temperature.

What do the results mean?

Most people with asthma can be reassured that low levels of smoke pollution will have only have small effects on their asthma which can be easily managed with reliever medication. However, for more susceptible people, low levels of smoke pollution can cause significant worsening of their symptoms and the need to use steroid tablets to regain control of their asthma.

As the pollution levels rise, our analysis would predict more widespread and serious impacts on people with asthma. Pollution levels exceeding the national air quality targets are likely to constitute an important public health hazard.

The **main messages** from our study for people with asthma are:

1. If you are sensitive to bushfire smoke you can reduce the impact by avoiding strenuous outdoor exercise on hazy days which will cause you to breathe in a much higher amount of pollution. Staying inside air-conditioned buildings, if this is practical for you, is also likely to be helpful.

2. If you smoke it’s a good idea to consider quitting. Smoking was the biggest avoidable contributor to symptoms and medication use for asthma in this study.

3. Have a written action plan worked out with your GP so you know when to increase reliever medication or start steroid tablets to keep your asthma under control.
YOUR RESULTS

HOW TO READ YOUR RESULTS: an example

Triangles mark the days when treatment included taking steroid tablets such as prednisolone.

Squares mark the days of visits to a health professional about your asthma.

Circles mark the days when asthma attacks were recorded.

The height of the blue lines represents your symptom score each day. For each reported symptom (such as ‘wheeze during the day’ or ‘cough at night’) you were given one point. The highest score possible each day is 8. If you had no symptoms the score was zero and there will be no blue line.
New Australian Research on

Dr. Faye Johnston and Prof Ross Bailey, Menzies School of Health Research, Institute of Advanced Studies, Charles Darwin University. Prof Louis Pilotta, Faculty of Medicine, University of New South Wales and Prof David Bowman, School for Environmental Research, Institute of Advanced Studies, Charles Darwin University.

Natural disasters on a world-wide scale are becoming increasingly evident and are reported daily in the media. The effects of tsunamis, earthquakes, volcanic activity and other natural phenomena are faced by members of the global community each day.

In Australia, the driest continent on the planet, severe vegetation fires are becoming more and more frequent. Understanding the health effects of bushfire smoke is critically important to maintaining the general wellbeing of Australians, particularly those with respiratory problems.

Below is a summary of a recent research project from Darwin that aimed to determine the impacts of bushfire smoke on asthma. The full report is due to be published later this year in the International Journal of Environmental Health Research [1].

Introduction

While severe vegetation fires are becoming more and more frequent in Australia and around the world [2], there are only a dozen or so published studies that have examined the health impacts of the associated smoke pollution.

Previous Research

Research from the USA and South East Asia has consistently demonstrated associations with respiratory symptoms and hospital admissions and the extreme pollution generated by the 1997 forest fires in Sarawak and Borneo also had a measurable association with deaths [3].

Australian Research

Of five previously published studies conducted here in Australia, two found associations between bushfire smoke and hospital presentations for asthma, while three studies that examined the 1994 fires near Sydney were unable to demonstrate associations between pollution levels and either measures of lung function or hospital attendances for asthma. [3]

Mixed results are not surprising.

Difficulties faced in this area of research

Studies concerning natural disasters are inherently difficult because they cannot be planned in advance. Finding appropriate health data for the analysis and choosing appropriate time periods to compare with the air pollution episode can present many difficulties. However, understanding the health effects of bushfire smoke is important for us in Australia because we have such highly fire adapted and fire prone vegetation. It is now accepted that avoiding serious bushfire disasters requires active management of fuel loads by deliberate
ERROR: undefined
OFFENDING COMMAND: q
STACK:
Appendix B.

The Darwin Smoke Project: Newsletters and presentations
Welcome to the first newsletter from the Darwin Smoke project, a unique multi-disciplinary research program that aims to identify causes and consequences of high air pollution episodes in the Darwin region. The project has several components:

- **Atmospheric Chemistry** – measuring the concentration of particles arising from smoke pollution in Darwin and Palmerston
- **Aero-biology** – measuring the amount of pollen and fungal spores in our air and identifying the predominant species
- **Meteorology** – measuring the daily temperature, rainfall and humidity, wind speed, direction and temperature inversions to help understand and predict the dispersal of smoke from savanna fires
- **Landscape Ecology** – examining how different land management affects the fuel loads and fire cycles in the savannas and examining the timing and geographic distribution of fires using satellite imagery
- **Epidemiology** – examining the impacts of environmental factors including smoke pollution, weather, fungi and pollen counts on the health of the population of Darwin

After many months of preparation the live phase of the project started in March 2004, with the commencement of health and environmental data collections.

**Atmospheric Chemistry**

The Team: A/Prof David Parry, Francoise Foti and Dr Tony Jong - Charles Darwin University, Dr John Gras – CSIRO Atmospheric Research. Contact – david.parry@cdu.edu.au

Aerosol sampling equipment location and setup:

At the Palmerston campus of Charles Darwin University a tapered element oscillating microbalance (TEOM) for measuring PM10 (ie particles 10 microns or less in diameter) with an Automatic Cartridge Collection Unit (ACCU) for measuring PM2.5 has been operating since 1 April 2004. A plot of the data from the TEOM is shown below: The peaks at the end of April and early May correspond to local fires during that time. The sudden drop in particle level in May was due to intense rain clearing the air and preventing further burning.
At the Casuarina campus of Charles Darwin University a Partisol Dichotomous Sampler is measuring PM10 and PM2.5 and in Darwin City a Microvol has been set up to measure PM10.

The Partisol aerosol sampler for measuring particulate air pollution on the roof of building 18 at CDU

Sporewatch samplers, for pollen sampling, have been co-located with the aerosol samplers at the Palmerston and Casuarina Campuses of Charles Darwin University. The pollen samples are collected on tape that is mounted and stained and sent to Dr Simon Haberle at the Australian National University for counting. The slides have shown a high density of fungal spores together with grass and eucalypt pollen.

The Sporewatch pollen counter on the roof of building 18 at CDU

Landscape Ecology

The team: Prof David Bowman and Don Franklin from the ARC Key Centre for Tropical Wildlife Management.

Contact – david.bowman@cdu.edu.au

The objective for this year’s field season is to discover if land management is influencing the grass biomass. There is some evidence that since the cessation of Aboriginal fire management grass, particularly annual tall grasses like Sorghum, have become more abundant driving a ‘grass-fire cycle’. For instance a common observation is that there is lower grass biomass in Arnhem Land compared to surrounding Darwin.

We are undertaking a systematic assessment of the biomass of grass throughout north west Australia in a common vegetation type (stringbark or *E. tetrodonta* forests). These data allow us to statistically partition out the effect of climate, soil type and land management on grass biomass. Next year we will undertake an intensive sampling around Darwin and some Aboriginal communities further increasing the scope and statistical power of our analysis.

Meteorology

The team: Jim Arthur, Ian Shepherd, and Dr Michael Foley, BOM Darwin and Alan Wain, BOM Melbourne

Contact – a.wain@bom.gov.au

On average, Darwin has been warmer and wetter than average over the period March to May. March was fairly typical, with the monsoon most vigorous around the middle of the month. By April the monsoon flow had relaxed, and rainfall in April was close to normal. May, on the other hand, was unusual. There were isolated heavy falls and a high number of rain days. In Darwin there were ten days of rain, the most in May since 1968 and mostly around the end of the month. This had the effect of dramatically reducing airborne particles in Darwin.

Satellite imagery and smoke dispersal modelling

Alan Wain is analysing board-scale satellite imagery to identify *hot spots* (sites indicative active fire fronts). Using a super-computer the hot spot data is combined with regional daily meteorological information to predict smoke dispersal from fires across the Top End. The actual measurements of air pollution made in this study will be used to refine and validate these models.
The team: A/Prof Ross Bailie (Menzies), Prof Louis Pilotto (Flinders University), Dr Fay Johnston, (Centre for Remote Health), Dr Ros Webby (ANU and Darwin Centre for Disease Control), Anne Myerscough and Janelle Fisher (CDU).

Contact – fay.johnston@cdu.edu.au

The health studies are being conducted in collaboration with the NT Department of Health and Community Services. There are three main components. (1) Hospital studies which will examine daily presentations and admissions for respiratory and cardiovascular diseases, (2) The asthma cohort study which is tracking the daily symptoms, medication use and health care attendances of a group of Darwin people with asthma, and (3) Community based surveillance of presentations to GPs for influenza-like illness and hay fever, and daily pharmacy sales of treatments for hay fever.

Updates from the asthma and hay fever studies are in this newsletter. Data extraction from Royal Darwin Hospital has not yet commenced.

**The Darwin Asthma Study**

Volunteer Lucas Schober with Dr Fay Johnston.

The asthma cohort study started on 1 March 2003. 260 enthusiastic adults and children are enrolled in the study. The participants record a daily diary of asthma symptoms, medication use and health care attendance. This information is compared to the environmental recordings from pollen, particulate air pollution and meteorological variables measured in both Darwin and Palmerston.

All participants completed an initial questionnaire and gave a saliva sample to test for cotinine, a marker of exposure to passive tobacco smoke. All adults also had a spirometry test pre and post salbutamol. The participants are phoned fortnightly during the 6 month period. The study will continue until the end of September 2004.

The study would not be possible without the assistance of many people. Thank you to the Top End Division of General Practice for financial assistance to employ a nurse to assist with spirometry. The Centre for Disease Control has also provided essential resources and financial assistance. Thank you to Asthma NT for the loan of their spirometer and invaluable knowledge.

**The Darwin Pollen and Hay fever Study**

The Darwin Pollen and Hay Fever Study will provide new information about the types of pollens present in the Darwin area and their impact on hayfever. Burkard Sporewatch Counters are measuring the aerobiology over Darwin and Palmerston daily, providing both a quantitative measure of pollens and fungal spores as and identifying predominant species. Hayfever levels in the Darwin population are being monitored through the sales of hayfever products at local pharmacies and consultations with General Practitioners.

A number of pharmacies in the Darwin, Palmerston and rural area are voluntarily recording daily sales of hayfever products. They are Darwin Mall Pharmacy, Barden’s Galleria, Stuart Park Pharmacy, Nightcliff Chemmart, Hibiscus Amcal Pharmacy, Palmerston Soul Pattinson Pharmacy and Coolalinga Amcal Pharmacy. Thank you to you all!

For more information about the Darwin Smoke Project contact Trisha Butler at the Key Centre for Tropical Wildlife Management Charles Darwin University NT 0909 telephone 89466574 or email patricia.butler@cdu.edu.au
Welcome to the second newsletter from the Darwin Smoke project, a unique multi-disciplinary research program that aims to identify causes and consequences of high air pollution episodes in the Darwin region. The project has several components:

- **Atmospheric Chemistry** – measuring the concentration of particles arising from smoke pollution in Darwin and Palmerston.

- **Aero-biology** – measuring the amount of pollen and fungal spores in our air and identifying the predominant species.

- **Meteorology** – measuring the daily temperature, rainfall and humidity, wind speed, direction and temperature inversions to help understand and predict the dispersal of smoke from savanna fires.

- **Landscape Ecology** – examining how different land management affects the fuel loads and fire cycles in the savannas and examining the timing and geographic distribution of fires using satellite imagery.

- **Epidemiology** – examining the impacts of environmental factors including smoke pollution, weather, fungi and pollen counts on the health of the population of Darwin.

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**Atmospheric Chemistry**

**The Team:** A/Prof David Parry, Francoise Foti and Dr Tony Jong - Charles Darwin University, Dr John Gras – CSIRO Atmospheric Research. Contact – david.parry@cdu.edu.au

The measurement of particulate matter (PM10 and PM2.5) is continuing at the Palmerston Campus and Casuarina Campus sites of the Charles Darwin University, together with PM10 measurements in the Darwin City area. PM10 and PM2.5 data from the Partisol Dichotomous Sampler at Casuarina Campus is shown below.

The data up until 28 September 2004 at Casuarina showed no exceedences of the PM10 NEPM standard (daily average 50 µg/m³), however there were 4 exceedences of the PM2.5 NEPM (NEPM daily average 25 µg/m³).
Dr John Gras from CSIRO Atmospheric Research in Melbourne visited on 26 to 28 July 2004 to assist with the calibration of the instruments and to review the aerosol sampling program. The Tapered Element Oscillating Microbalance (TEOM) for PM10 and the Automatic Cartridge Collection Unit (ACCU) for PM2.5 both passed all calibration checks. However, a fault was identified with the Partisol Dichotomous Sampler that could not be rectified on site so it was returned to the supplier in Melbourne. We were fortunate that the supplier had a spare unit which he shipped to us, resulting in minimum down time. The instrument has now been repaired and the supplier has agreed to us retaining the spare Partisol to allow us to do some parallel measurements for quality control purposes at both Casuarina and Palmerston sites.

The MicroVol has also developed a flow problem which has necessitated it be returned to Melbourne for repair.

We are currently conducting quality control on our weighing methodologies by having filters check weighed by the CSIRO Atmospheric laboratory. Results to date show good agreement between our two laboratories.

The model uses a combination of satellite images, GIS, the HYSPLIT program being run by Alan Wain.

The Sporewatch samplers have been running continuously. The pollen samples are collected on tape that is mounted and stained and sent to Dr Simon Haberle at the Australian National University for counting.

**Landscape Ecology**

**The team:** Prof David Bowman and Don Franklin from the ARC Key Centre for Tropical Wildlife Management.

Contact – david.bowman@cdu.edu.au

The aim of the Landscape ecology project is to examine the relative importance of fire management and environmental conditions on grass fuel loads. It has previously been suggested that high frequencies of fire activity have driven a ‘grass-fire’ cycle resulting in bigger, hotter and more polluting bushfires.

Four months of fieldwork during the dry season is now over. Christine Maas and Don Franklin assessed the contribution of grasses and forbs to fuel loads as 211 sites in Stringybark (*Eucalyptus tetrodonta*) dominated savannas. Sites, shown in red on the map, were distributed across north-western Australia, from Kalumburu and the Mitchell Plateau in the north-west to Cobourg and Gove Peninsulas in the north, inland to Larrimah, and south-east beyond Borroloola and almost to Wollogorang.

Owen Price and Don Franklin have now started the data analysis with input from David Bowman.

Some interesting trends have emerged, not all as anticipated. The grass and herb fuel loads were generally rather lower than anticipated, and there is a
weak negative relationship with mean annual rainfall – the highest fuel loads were associated with Spinifex (*Triodia*) in drier regions. Some higher rainfall regions and especially Gove Peninsula had remarkably low fuel loads. But the overwhelming impression is of extraordinary variability between sites often even when separated by just a few kilometres.

In regions where annual spear-grasses (*Sarga intrans* and *S. timorense* - these were formerly in the genus *Sorghum*) occur, the presence and especially dominance of these grasses is associated with higher fuel loads.

However, this doesn’t necessarily mean that spear-grass is invading and increasing fuel loads. It could well be spear-grass occurs where conditions favour more lush growth. This is something we hope to tease out from the data. Nonetheless, the result shows that grass fuel loads are the result of a complex interplay between biophysical conditions, geographic regions, and management regimes. How this variation relates to fire management patterns and associated variation in smoke pollution remains to be determined.

**Epidemiology**

**The team:** A/Prof Ross Bailie (Menzies), Prof Louis Pilotto (Flinders University), Dr Fay Johnston, (Centre for Remote Health), Dr Ros Webby (ANU and Darwin Centre for Disease Control), Anne Myerscough and Janelle Fisher (CDU).

Contact – fay.johnston@cdu.edu.au

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**The Darwin Asthma Study**

The collection of data from our cohort of 236 adult and children with asthma will conclude on November 7. These participants have been completing a daily diary with details of symptoms, medication use and health care attendance since March, providing the study with 36 weeks of asthma data. The first 3 months of data is currently being analysed using STATA.

**The Darwin Pollen and Hay fever Study**

The *Darwin Pollen and Hayfever Study* has commenced analysis of the data between March and August 2004. Though initial findings revealed different sales trends for Telfast, Clarinase, Rhinocort and Beconase, sales tended to reach a peak in May (Telfast) and June (other products).

Between late March and early June there was a conspicuous presence of grass pollens in the pollen count. Other significant plant taxa during this period included *Acacia* (Wattles), palms, *Eucalypts* (Gums) and *Callitris* (Cypress Pine).

Further analysis will be conducted to determine their relationship with pollens, smoke and other environmental conditions.

Plant specimen collection is continuing in both Casuarina and Palmerston areas. Flowering specimens are collected, pressed and dried before being taken to the NT Herbarium for identification. Flower specimens are then sent to Dr Simon Haberle at ANU for comparison with pollen specimens collected by the aerosol samplers and for inclusion in a pollen database. A total of 101 different flowering plants have been sent to ANU to date.

**Hospital Studies**

Planning for the extraction of RDH hospital data has commenced. Presentation and admission data from 2000 to the end of 2005 for respiratory and cardiovascular conditions will be examined in the next stage of the study.
Some results so far.....

Total pollen

Pharmacy sales of products for hay fever

The palynology team at ANU

Meteorology

The team: Jim Arthur, Ian Shepherd, and Dr Michael Foley, BOM Darwin and Alan Wain, BOM Melbourne

Contact -m.foley@bom.gov.au

The 2004 fire season for the northwestern Top End got off to a late start after rainfall encroached into the early Dry Season. (Darwin Airport had a record June rainfall total of 50.6 mm.) The rains reduced fuel curing early in the season and contributed to lower fire dangers in May and June.

The figure below shows the number of days in each month when the peak fire danger index recorded in the Darwin-Daly district exceeded Very High 35 (marginal fire weather warning conditions) or Near Extreme 45. It can be seen that this year, fire dangers for the northwestern Top End peaked during September. A procession of strong high pressure systems moving through the Great Australian Bight during September maintained unusually fresh and dry southeasterly wind flow over the Top End. This combined with high fuel curing and typical hot ‘Build Up’ temperatures to give increased fire dangers during the month.

Days per month with notable fire danger values.

For more information about the Darwin Smoke Project contact Trisha Butler at the Key Centre for Tropical Wildlife Management Charles Darwin University NT 0909 telephone 8946574 or email patricia.butler@cdu.edu.au
Welcome to the third newsletter from the Darwin Smoke project, a multi-disciplinary research program that aims to identify causes and consequences of high air pollution episodes in the Darwin region. The project has several components:

- **Atmospheric Chemistry** – measuring the concentration of particles arising from smoke pollution in Darwin and Palmerston.

- **Aero-biology** – measuring the amount of pollen and fungal spores in our air and identifying the predominant species.

- **Meteorology** – measuring the daily temperature, rainfall and humidity, wind speed, direction and temperature inversions to help understand and predict the dispersal of smoke from savanna fires.

- **Landscape Ecology** – examining how different land management affects the fuel loads and fire cycles in the savannas and examining the timing and geographic distribution of fires using satellite imagery.

- **Epidemiology** – examining the impacts of environmental factors including smoke pollution, weather, fungi and pollen counts on the health of the population of Darwin.

### Atmospheric Chemistry

**The Team:** A/Prof David Parry and Francoise Foti - Charles Darwin University, Dr John Gras – CSIRO Atmospheric Research. Contact – david.parry@cdu.edu.au

Measurements of particulate matter (PM 10 and PM 2.5) are continuing at both Palmerston and Casuarina sites. The data (7/4/04 - 26/1/05) from the Partisol Dichotomous sampler at Casuarina campus is shown below. The graph shows that there have been no exceedances of the PM10 Air NEPM standard of 50 µg m$^{-3}$. In contrast there have been four days on which the PM2.5 Air NEPM standard of 25 µg m$^{-3}$ was exceeded, the highest of 36.5 µg m$^{-3}$ on 6th September 2004. The PM2.5 values from mid-November 2004 to end of January 2005 have remained constantly low.

We have recently purchased a second Dichotomous Partisol sampler, jointly funded by CDU and NTG (DIPE and DHCS). This sampler will initially be used for quality control work with the current samplers,
beginning with the deployment at the Palmerston site in parallel with the TEOM and ACCU systems that are operating there.

The MicroVol sampler that has been deployed in Darwin city area has continued to malfunction which has been traced back to problems with high humidity and water penetration. The system has now been repaired twice and a solution proposed by the manufacturers. As a result very little data has been obtained with this sampler.

Pollen sampling is continuing at Casuarina and Palmerston. The samplers are set up for continuous sampling however we have encountered a few problems with the samplers stopping in mid cycle. We have been investigating the problem but as yet cannot determine what is causing the problem. We have changed the solution applied to the plastics strips used in the samplers. We are now using a Silicon solution that is applied to the strip in one thin coat, as opposed to the old method where two different solutions were applied. The original solution was quite thick when applied and ran in the hot weather. The new method has overcome these problems and also makes mounting of the strips easier and more efficient.

**Aerobiology at ANU, report from Dr Simon Haberle**

The daily pollen and fungal counts for the two sampling stations in Darwin have yielded a diverse assemblage of airborne pollen and fungal spore types. Clear pollen seasons have emerged over the last 9 months of daily count data with Palms, *Callitris* and *Eucalyptus pollen* dominating the Sept-Nov quarter.

Over 40 pollen types are consistently encountered in the daily counts and many of them can be identified to at least genera level. Currently we are processing around 300 herbarium specimens of locally collected flowering plants to develop a Darwin Pollen Reference Collection to assist in refining our identification of pollen taxa encountered during the daily pollen counts.

**Landscape Ecology**

**The team:** Prof David Bowman and Don Franklin from the ARC Key Centre for Tropical Wildlife Management.

Contact – david.bowman@cdu.edu.au

**Spear-grass and fuel loads**

As reported in the last newsletter, our survey revealed that grass fuel loads in Stringybark (*Eucalyptus tetrodonta*) savannas are higher where annual spear-grass (*Sarga* spp.) is prevalent. Since then, we have made considerable progress in considering why this is so. We have identified that spear-grass is more prevalent in areas with higher rainfall, where there are less trees and shrubs, and where soils are heavier. No great surprise there. But of considerable interest is that these effects explained only about 20% of the variation in the prevalence of spear-grass. So what accounts for the other 80%?
We believe that this must be largely an effect of management. But what is the management impact? It’s not as simple as how often the country is burnt, because parts of Arnhemland have little spear-grass (and consequently low grass fuel loads) even though they’re burnt as often as areas near Darwin where spear-grass is abundant. We are currently investigating the idea that the answer lies in the patchiness of burning – where the landscape is burnt with numerous small fires, annual spear-grasses may have trouble tracking the set of circumstances that suits it.

We hope to have a manuscript reporting our results ready for peer review in the next few weeks.

Introducing Louis Elliott

Lou has recently joined the team as an Honours student. His project is to assess grass fuel loads and composition in Woollybutt (*Eucalyptus miniata*) savannas at sites around Darwin, and also in the Daly River region. His work should provide an independent test for the conclusions about spear-grass made during the above study.

Epidemiology

The team: A/Prof Ross Bailie (Menzies), Prof Louis Pilotto (Flinders University), Dr Fay Johnston, (Centre for Remote Health), Dr Ros Webby (ANU and Darwin Centre for Disease Control), Anne Myerscough and Janelle Fisher (CDU).

Contact – fay.johnston@cdu.edu.au

The Darwin Asthma Study: interim results from April – June 2005

The study commenced on March 1st and was completed on November 7th 2005. Environmental data became available from April. Dr Ros Webby has completed an interim analysis of the data from April to June 2004 and the results are summarised below. Analysis of the entire study period has now commenced.

The mean number of people providing data each day was 234 of 251 participants.

The mean daily prevalence of symptoms were as follows; wheeze 11%, breathlessness 11%, chest tightness 10% and cough 21%, asthma attacks 4%, missed school or work due to asthma 1% and health care visits for asthma 0.66%. The mean daily prevalence of medication use was 42% for inhaled steroids, 23% for bronchodilators and 1% for oral steroids.

Multi-variate statistical models were used to examine the effects of:

- particulate matter (PM$_{10}$ and PM$_{2.5}$ recorded at Casuarina)
- dew point temperature at 9am
- daily rainfall
- total pollen count
- total fungal count
- influenza rate in the community
- weekends, public holidays and school holidays
- autocorrelation of symptoms within each participant
These showed no association between air pollutants and asthma symptoms or asthma attacks. Some positive associations were demonstrated between breathing difficulty with exercise and PM$_{2.5}$. No association was shown between particulate matter and missed school or work due to asthma, health care visits for asthma or medication use which was measured as bronchodilator, oral steroid or inhaled steroid use.

These results are reassuring. They suggest that during the initial stages of the study, none of the environmental triggers we monitored were important contributors to asthma symptoms in our study group. During this time levels of smoke pollution remained low with a mean PM$_{10}$ level of 18 ug/m$^3$ well under the national air quality standard threshold of 50 ug/m$^3$.

**Meteorology**

The team: Jim Arthur, Ian Shepherd, and Dr Michael Foley, BOM Darwin and Alan Wain, BOM Melbourne

Contact –m.foley@bom.gov.au

Since the end of October, there have not been any significant fire weather situations over the Top End. A burst of the monsoon around Christmas brought the first substantial rains to Darwin, and by the end of January, fuel curings (as estimated by the Bushfires Council) had dropped from 85% to 60%, effectively removing any chance of raised fire dangers for the time being.

Despite another short monsoon burst at the beginning of February (prior to the formation of TC Harvey in the Gulf of Carpentaria), wet season rainfall has been below somewhat below average. Darwin rainfall currently lies in the 4th decile (see Figure). Monsoon bursts have so far been well correlated with active phases of the Madden-Julian Oscillation (an equatorial disturbance which tends to move around the globe from west to east with a period of 30 to 60 days). Another monsoon burst may be developing in early March.

With two months of the Wet Season remaining, it is too early to predict whether or not wet season rainfall totals will be lower than usual, and hence what the likelihood is of an early start to this year’s fire season.

![Wet Season Rainfall Graph](image-url)
Welcome to the fourth newsletter from the Darwin Smoke project, a multi-disciplinary research program that aims to identify causes and consequences of high air pollution episodes in the Darwin region. The project has several components including Atmospheric Chemistry, Aero-biology, Meteorology, Landscape Ecology and Epidemiology.

**Epidemiology**

**The team:** Dr Fay Johnston (Menzies School of Health Research), Dr Ros Webby (ANU and Darwin Centre for Disease Control), Prof Louis Pilotto (Flinders University), A/Prof Ross Bailie (Menzies), Anne Myerscough.

Contact – [fay.johnston@cdu.edu.au](mailto:fay.johnston@cdu.edu.au)

As part of the Darwin Smoke Project we have been conducting two epidemiological studies. (1) The community based **Darwin Asthma Study** and (2) the **Royal Darwin Hospital Study** which is examining admissions for a wide range of heart and lung diseases in relation to smoke pollution. Analysis of the asthma study is almost complete and our main findings are summarised below. Analysis for the hospital study will commence in 2006.

**Darwin Asthma Study 2004 Results**

**Overview**

We aimed to determine which local environmental factors might contribute to worsening of asthma in the community. 251 residents of Darwin and Palmerston with known asthma kept track of their symptoms and medication use for seven months during the dry season of 2004. During this time we monitored weather conditions, influenza rates in the community and atmospheric levels of smoke pollution, pollen and fungal spores.

**Environmental conditions during the study**

1. **Meterology**

2004 was an unusual dry season with rainfall persisting well into June and delaying the curing of grasses. This resulted in lower than usual fire hazard conditions persisting until September. The maximum daily air temperature ranged from 25.7 to 36.1°C (average 31.8). The minimum ranged from 13.3 to 27.5°C (average 21.2) and the relative humidity at 9.00am varied from 9% to 96% (average 64%).

2. **Smoke levels**

Smoke pollution remained low throughout the study period. The average mass of particulate matter less than 10 microns in diameter per cubic meter of air (PM$_{10}$) was 20 µg/m$^3$ (range 3 - 46 µg/m$^3$). The national air quality target for PM$_{10}$ of 50 µg/m$^3$ was not exceeded during the study. Particulate matter less than 2.5 microns in diameter (PM$_{2.5}$) made up approximately 50% of the measured PM$_{10}$. PM$_{2.5}$ exceeded the national advisory reporting standard of 25 µg/m$^3$ on a total of 5 days.

3. **Pollen levels**

Pollen counts also remained very low by Australian standards. In Darwin our total count ranged from 0.5 – 61 grains per cubic meter of air with an average daily
count of 16. Our grass pollen counts peaked first in April and then again in June. The range for grass pollen was 0-22 grains per cubic meter with an average of 3. In comparison, total pollen counts in Brisbane have been measured at 700-800 grains per cubic meter and in Melbourne the count can exceed 2000. Grass pollen counts in both these cities often peak at around 300.

The American Academy of Allergy, Asthma, and Immunology’s (AAAAI) Aeroallergen Network use the following criteria for grass pollen counts - 0 to 5 low, 6 to 20 moderate, 21 to 200 high and 201 or higher very high.

4. Fungal spores
These remained at low to moderate levels for most of the study, ranging from 108 to 6049 spores per cubic meter with a mean of 1843. A similar study in Brisbane in 2000 recorded higher counts with a mean of 7352 (range 546-67301) spores per cubic meter. The AAAAI criteria for spore counts are: 0 to 900 low, 901 to 2,500 moderate, 2,501 to 25,000 high and 25,001 or higher very high.

5. Influenza
Consultations to Darwin GPs for influenza-like illnesses varied from 0.5% to 3% of all consultations during the study. In large outbreaks of influenza consultation rates usually exceed 3% and may approach 10%.

Environmental influences on asthma
Even at the low levels of particulate pollution present during our study we found small but statistically significant associations between smoke pollution levels (measures at PM$_{10}$ and PM$_{2.5}$) and:

(1) the average number of asthma symptoms experienced by participants each day. This effect was quite small and could be attributed to extra symptoms in just a few participants, rather across the entire group.

(2) the average amount of reliever medication used by participants each day. This was also quite a small effect, that is it was largely due to increasing reliever use in just a few participants. Inhaled reliever medication (eg a Ventolin puffer) is used to manage asthma on a day-to-day basis.

(3) the proportion of participants starting a course of steroid tablets such as prednisolone. This outcome had the biggest association with PM$_{10}$. The proportion of participants starting steroid tablets rose by approximately 50% with each rise of 10 in PM$_{10}$. This is an important outcome because steroid tablets are reserved to treat more serious exacerbations of asthma. Starting a course of this medication indicates there has been clinically significant worsening of asthma control.

All findings were more marked in adults compared with children and in those with more severe asthma.

We also found small associations between asthma symptoms and the minimum air temperature and GP consultation rates for influenza.

We did not find any associations between pollution levels and exercise induced asthma, asthma attacks or health care attendances for asthma. Nor did we find any health effects to be associated with pollution levels at time lags of up to 5 days, pollen levels, fungal spores, rainfall, relative humidity, dew point or maximum air temperature.

Individual factors affecting asthma
As expected, individual factors were important predictors of asthma symptoms and medication use. The following groups all were significantly more likely to have symptoms or use medication for their asthma:

- smokers (compared with non-smokers)
- adults (compared with children)
people who lived in less crowded circumstances (compared with more crowded)
non-Indigenous participants (compared with Indigenous)
those with (or whose parents had) lower compared with higher education levels

What do the results mean?
Most people with asthma can be reassured that low levels of smoke pollution (ie PM$_{10}$ of less than 40 µg/m$^3$) will have little or no effect on their asthma. However, even at these levels, a small proportion of people will experience significant worsening of their symptoms, needing to use more medication, including steroid tablets, to regain control of their asthma. As the pollution levels rise, our analysis would predict more widespread and serious impacts on people with asthma. Pollution levels exceeding the national air quality targets are likely to constitute an important public health hazard.

The main messages for people with asthma are:

(1) If you are sensitive to bushfire smoke you can reduce the impact of hazy days by avoiding strenuous outdoor exercise that will cause you to inhale a much higher amount of pollution. Staying inside air-conditioned buildings, if this is a practical option for you, is also likely to reduce the level of your exposure.

(2) If you smoke you should consider quitting – smoking was the biggest avoidable contributor to symptoms and medication use for asthma in this study

(3) Have a written action plan worked out with your GP so you know when to increase reliever medication or start steroid tablets to keep your asthma under control.

The main message for land managers is that landscape burning should explicitly aim to minimise air pollution. The prevention of large intense fires that are extremely polluting should continue to be of high priority. Prescribed burns should be managed to minimise pollution over urban areas, and to avoid exceeding national air quality standards.

Landscape Ecology

The team: Prof David Bowman and Don Franklin from the ARC Key Centre for Tropical Wildlife Management.

Contact – david.bowman@cdu.edu.au

Grass fuel loads around Darwin

Louis Elliott has recently completed his survey of grass fuel loads and their composition. With the help of volunteers, Lou surveyed 101 sites in Darwin Woollybutt Eucalyptus miniata woodland within about 100 km of Darwin, from within the city limits north to Gunn Point, south to Litchfield National Park and west to Dundee Beach and the Cox Peninsula. The grass samples have now been dried and weighed. Litter fuel loads were also sampled. Over the coming months, Lou will analyse the data and write up the results, with completion due at the end of the year.

Preliminary results suggest that sites dominated by annual spear grass tend to have high fuel loads, but other sites are variable and those dominated by perennial native grasses can also have high fuel loads.
Atmospheric Chemistry

The Team: A/Prof David Parry, Ms Judy Manning and Ms Francoise Foti - Charles Darwin University, Dr John Gras – CSIRO Atmospheric Research. Contact – david.parry@cdu.edu.au

Measurements of particulate matter (PM 10 and PM 2.5) continue at both Palmerston Campus and Casuarina Campus. The data from the Partisol Dichotomous sampler at Casuarina Campus is shown below (7/4/04 – 22/6/05). Once again the graph shows no exceedances of the PM 10 Air NEPM standard of 50 µg m\(^{-3}\). There were however 2 days when the PM 2.5 Air NEPM standard of 25 µg m\(^{-3}\) was exceeded in early February 2005, bringing the total exceedences over the past 12 months to 7.

The second Dichotomous Partisol sampler that was purchased by the project partners has been deployed at the Palmerston Campus site, collocated with the TEOM/ACCU system for QC of that system. There were some early teething problems with this new sampler but these have been overcome. We are starting to get some good data sets that can be compared to the results from the TEOM and ACCU systems already in operation at Palmerston.

Pollen sampling at the Palmerston campus has been concluded after collecting 12 months of data and the pollen sampler has been moved to Casuarina campus. We have overcome the problem of the samplers stopping mid cycle on one sampler but the other remains a source of consternation. We have been in contact with the manufacturers and investigation of the problem is continuing. Despite the problems the pollen and fungal data sets are of very good quality.

For more information about the Darwin Smoke Project contact Trisha Butler at the Key Centre for Tropical Wildlife Management Charles Darwin University NT 0909 telephone 89466574 or email patricia.butler@cdu.edu.au
Project feedback meeting
As the project is drawing to a close with all stages of data collection now complete, we had a meeting on November 11 to share and discuss the findings thus far with our funders and stakeholders. This newsletter presents the content and outcomes from that meeting.

Agenda
Introduction and welcome - David Bowman

Results
- Atmospheric chemistry - David Parry
- Darwin Asthma Study – Fay Johnston
- Hospital studies – Fay Johnston
- Smoke modeling - Michael Foley
- Historical changes in air pollution – David Bowman
- Causes of pollution – How are we managing grass fuels? - Don Franklin and Lou Elliot
- Darwin’s pollen calendar – Simon Haberle
- When is our hayfever season? – Fay Johnston

Discussion – facilitated by David Bowman
- Should air quality and pollen monitoring continue?
- Who pays?
- Does this study affect fire management?
- Implications of health findings for other regions
- A tool for BOM daily dry season warning system?

Key findings
Air Quality
1. Air quality in Darwin largely remained within National air quality guidelines during the last two years (with the notable exception of Territory Day 2005)

2. The air quality in Darwin has deteriorated since the 1950s and particularly since the 1970s. An analysis of meteorological data and airport visibility records has demonstrated a marked increase in air pollution in the first half of the dry season.

Fire ecology
1. Higher fuel loads, especially spear grass, are present in areas managed with greater frequency of burning.
2. Thus our current management may be contributing to a worsening grass / fire cycle where annual widespread burning encourages spear grass and other problem grasses such as mission grass and gamba grass. The risk is that this will gradually cause much more severe fire hazards around Darwin with adverse health and biodiversity outcomes.

Health Outcomes

1. Most people are unaffected by the background levels of bushfire smoke usually present in Darwin.

2. A proportion of people are very sensitive to particulate pollution even at levels well within air quality guidelines. For example during 2004 the level of particulate air pollution was significantly associated with worsening of asthma symptoms and the need for additional medication such as steroid tablets in about 5% of participants in the Darwin Asthma Study.

3. Admissions to RDH for heart and lung diseases were significantly associated with particulate pollution measured during 1996. The air was monitored at Charles Point but there is close correlation between pollution levels measured at Palmerston, Darwin and Charles Point.

4. Our previous work in 2000 demonstrated that asthma presentations for to RDH more than doubled when air quality guidelines were exceeded.

5. Further analyses of hospital data will be done during 2006.

6. On a preliminary analysis, the main contributors to hay fever in Darwin appear to be:

- grass pollens
- acacia pollens
- Alternaria spores
- smoke pollution

Clear seasonal trends (grains/m³) for grass (Poaceae) and wattle (Acacia) pollens Casuarina 2004/2005 with peaks in the early dry season.

Conclusions to date

1. Polluting fires demonstrably have public health consequences. Worsening pollution levels and periods during which air quality guidelines are exceeded have a measurable public health impact in Darwin.

2. In terms of public health there is a trade-off between less polluting controlled burning and more polluting uncontrolled landscape fires.

3. There is also a trade-off of the ecological and economic consequences of controlled and uncontrolled fires that are unclear and require further research.

4. Of particular concern is the possible effect of early season burning on the proliferation of flammable grasses.
Meeting outcomes

1. A media release prepared by CDU with input from NRETA, EPA and NT Health is being finalised and will be released soon.

2. It is hoped that the monitoring of PM10 and PM2.5 at Casuarina will continue beyond 1 January 2006. EPA is exploring funding options to maintain the data flow. The TEOM may be used instead of the Partisol to bring the monitoring in line with national air quality standards.

3. The daily collection of pollen will continue at the CDU site funded by the Bushfire Project grant until 1 July 2006, when hopefully new funding will be available. The pollen samples will be sent to ANU for storage but not analysed until further sources of funding are secured. It is thought important to continue monitoring to extend the sample period thereby allowing more robust aerobiological conclusions to be made.

4. One area of research flagged by the meeting concerned the need to undertake an economic analysis of the health impact of bushfire smoke. This analysis would need to be balanced against the economic benefits and costs of fire management/asset protection. Involvement of both health and environmental economists would be required to tackle this complex problem.

5. Discussion was made of involving the Bureau of Meteorology in providing health warning on days of bad smoke haze. This issue is complicated and requires careful assessment of the cost and accuracy of such warning and the need of the Darwin community for such a warning. An alternative option is to provide specific health education to people at higher risk from air pollution such as those with chronic heart and lung conditions. On days with low air quality as evidenced by poor visibility, people at higher risk should monitor and manage their symptoms particularly closely and avoid vigorous outdoor exercise.

6. The relative ecological costs and benefits of broad-acre early dry season burning demands more research, particularly experimental analyses, given the uncertainties associated with existing correlative analyses.

7. It is proposed to hold a meeting with all stakeholders to deliver the final results of the project in November 2006 in a single presentation. At this meeting ample time will be set aside to discuss the hand over of the project findings to the stakeholders who then can make informed decision about the continuity of the research program and the implications for policy and practice.

For more information about the Darwin Smoke Project contact Carmen Crossing at the School for Environmental Research, Charles Darwin University NT 0909 telephone 08 8946 7104 or email carmen.crossing@cdu.edu.au
Bushfire smoke, health and fire management

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Introduction
We reviewed the international literature about the health effects of wildfire smoke and conducted a series of studies assessing the impact of smoke from regional savanna fires on the health of people living in Darwin. Most of the fires occurring during the study period were of low intensity, analogous to prescribed burns in southern Australia. The pollution they generated rarely caused Australia’s air quality guidelines to be breached.

Summary of results
• As severe fires have become more common around the world, bushfire smoke has been recognised as an increasingly important public health hazard.
• Research has clearly established that airborne particles, the major component of bushfire smoke, can exacerbate heart and lung diseases, cause hospitalisation and death.
• The risks are greater for: (1) people who are exposed to higher levels of pollution, (2) babies and young children, (3) the elderly and (4) people with diabetes, heart or lung conditions.
• Harmful health effects from bushfire smoke, such as worsening asthma, are measurable at pollution levels below current Australian air quality standards.
• Bushfire smoke could be relatively more harmful to the respiratory system than other sources of particulate air pollution. This issue needs further research.

What does this mean for land managers?
• Individuals, corporations and government agencies that manage land to prevent severe bushfires and smoke pollution events over populated areas are performing an important public health service.
• The main intervention for achieving this, namely controlled burning, is not free of risk to the public. While the pollution levels from low intensity prescribed burns is usually far lower than that from severe wildfires, there appears to be no clear lower limit for pollution that is safe for all members of the community. However, these risks need to be balanced against the substantial health risks of severe pollution episodes from intense fires.
• Risks can be minimised by:
  o Managing deliberate burns to (1) minimise the amount of pollution affecting urban areas and (2) avoid exceeding national air quality standards.
  o Good public communication including advance health advisories so that people at higher risk from exposure to smoke can take appropriate action.
• Closer collaboration between public health and fire management researchers will facilitate clarification and communication of the relative risks of severe fires and management interventions to prevent them.