# EFFECTS OF BUSHFIRE EXPOSURE ON PRENATAL AND EARLY LIFE DEVELOPMENT IN HUMANS: A LIFE HISTORY PERSPECTIVE

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**at The Australian National University.**

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## *Statement of originality*

This is to certify that, to the best of my knowledge, the content of this thesis is my own work. This thesis has not been submitted for any degree or other purpose excepting the work published in the article listed below, which is cited as necessary.

> O'Donnell, M.H. & Behie, A.M. (2015). Effects of wildfire disaster exposure on male birth weight in an Australian population. *Evolution, Medicine and Public Health.* 1, 344-354.

I certify that the intellectual content of this thesis is the product of my own work and that all the assistance received in preparing this thesis has been acknowledged appropriately.

Megan Hartley O'Donnell

## *Ethics compliance*

This research was undertaken in accordance with the following:

- Australian National University Human Research Ethics Committee Protocol Number 2013/143;
- Victorian Department of Health Human Research Ethics Committee Protocol Number 20/14; and
- Australian Capital Territory (ACT) Health Directorate Human Research Ethics Committee Protocol Number ETHLR.13.330.

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This thesis uses data from the perinatal data collections of The Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM) and the ACT Health Directorate. The findings and views reported in this dissertation are mine and should not be attributed to CCOPMM or the ACT Health Directorate.

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# *Abstract*

Taking a life history theory perspective, this dissertation considers the effects of bushfire-related stress on a suite of human reproductive outcomes. Life history theory suggests that human reproduction is responsive to environmental pressure and that this responsiveness can be detected via changes to reproductive strategy, such as degree of maternal investment.

Using multivariate analysis, this dissertation examines the effects of maternal fire exposure in two populations on gestational age, birth weight, secondary sex ratio, plurality rates, fertility rates, and behavioural indicators. It hypothesises that decreases will be observed in those measures that indicate degree of maternal investment, such as birth weight, gestational age, and plurality, as well as predicting decreases in sex ratio associated with differential loss in early pregnancy. It suggests that behavioural indicators will worsen as sequela of *in utero* stress. Using a mixed-methods approach, the dissertation also documents the experience of mothers who have lived through bushfires while pregnant, hypothesising that they will have experienced considerable subjective and objective stress.

The dissertation finds that reproductive responses vary considerably between the populations studied and potentially in relation to the intensity of fire exposure. Contrary to the hypothesis, it finds that birth weights increased in the population exposed to a smaller fire (the 2003 Canberra fire), while remaining unchanged in the population exposed to a larger fire (Black Saturday fire, Victoria, 2009). However, the reverse was true of secondary sex ratio, which decreased in the population exposed to the Black Saturday fire, while remaining unchanged in the Canberra population. Gestational age was unaffected in both cases. Behavioural indicators and fertility rates were largely unaffected by fire exposure, although they both appeared sensitive to other

environmental factors. Mothers in both fires reported feeling stressed, although mothers exposed to the Black Saturday fire reported higher perceived and objective stress. Mothers in both fires reported that public support and information were, at times, insufficient.

Taken together, the findings indicate the functioning of finely tuned evolutionary mechanisms that adjust to environmental conditions only where a threshold of severity is met, thereby protecting reproductive strategy from the influence of more transient stressors.

*For David*

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## *List of Abbreviations*

| | |
|---|---|
| AALSAC | Australian Alps Longitudinal Study of Australian Children |
| ACT | Australian Capital Territory |
| ANOVA | Analysis of Variance |
| ANU | The Australian National University |
| ASR | Adult Sex Ratio |
| CCOPMM | Consultative Council for Obstetric and Paediatric Mortality and Morbidity |
| ELP | Early Life Programming |
| GA | Gestational age |
| GLM | General Linear Model |
| HREC | Human Research Ethics Committee |
| LBW | Low Birth Weight |
| LHT | Life History Theory |
| LSAC | Longitudinal Study of Australian Children |
| OSR | Operating Sex Ratio |
| PEDS | Parents' Evaluation of Developmental Status |
| PSS | Perceived Stress Scale |
| SSR | Secondary Sex Ratio |
| TFR | Total Fertility Rate |

# 1. INTRODUCTION

Bushfires, both wild and anthropogenic, have long been a significant part of Australian society and ecology. In Australia, anthropogenic fire has been used to manage native ecosystems and this long-term practice of burning has likely influenced the modern landscape (Pyne 1991). Bushfires appear regularly in the myths and traditions of Aboriginal Australians and intentional fires were (and still are) used as a tool to boost food supply. Fires are also well documented in the records of the first white settlers (Pyne 1991). With colonisation and urbanisation, deaths, injuries and property losses from wildfire are now a regular occurrence and are well documented throughout Australia's colonial and post-colonial historical record (Griffiths 2001).

Despite this long-standing history with fire, Australia's changing climate is transforming the nature of Australian bushfires, and fire conditions of unprecedented magnitude and intensity are now being regularly recorded (Climate Council 2014). One example of the increasing severity of wildfire is the 2009 Black Saturday bushfires, which killed 173 people. This more than doubled the previous highest Australian death toll (Australian Emergency Management Institute 2012) making Black Saturday the ninth most deadly wildfire world-wide (Cameron et al. 2009). An example of the effects of fire in urbanised populations is the 2003 Australian Alps fires, which culminated in a fire tornado moving through southern and western Canberra, Australia's capital city.

The majority of research into bushfires has focused on prevention (e.g. natural resource management), mitigation (e.g. fire suppression) and managing public preparedness. However, the likely severity and frequency of future fires means that it will be impossible to prevent, contain or prepare adequately for more fires (Griffiths 2012). As a consequence, more people will be exposed to bushfires and the stressors that follow them. Therefore, it is becoming increasingly important to understand the effects of bushfire exposure on long-term population health.

While some work has considered the psychological impact of fire exposure (Camilleri et al. 2012; Kolmus et al. 2009; Macdonald et al. 2010; McFarlane et al. 1997), none consider the human biological consequences of exposure from an evolutionary standpoint. Although humans have the capacity to protect themselves partially from environment pressures (e.g. by building shelter and importing resources), they remain fundamentally dependent on and subject to their environment (Attenborough 2002). Exposure to different types of environmental disasters, such as storms and earthquakes, and other stressful stimuli, such as wars and familial deaths, in early life are known to correlate with a range of developmental changes, many of which are detrimental to health and wellbeing across the lifespan (Auger et al. 2011; Glynn et al. 2001).

Research in relation to other environmental disasters indicates an increased likelihood of preterm birth, low birth weight and the development of psychiatric disorders in exposed children (Auger et al. 2011; Dancause et al. 2011; Xiong et al. 2008). Similarly, exposure to other stressors in early life have demonstrated negative effects on neurological development, the emergency of behavioural disorders and the incidence of chronic disease (Chisholm et al. 2005; Eriksson 2010; Ulijaszek 1998).

As the intensity and frequency of Australian bushfires increases, and more Australians live in fire-prone regions, a growing cohort of Australian children will experience the stress of bushfire exposure either *in utero* or in early life. Research conducted following other environmental disasters suggests that this exposure may increase their risk of experiencing a broad range of negative life outcomes, but little known research exists on any such effects of wildfire. This dissertation builds on research undertaken as part of my Master's degree dissertation, which also considered the effects of the Black Saturday bushfire on birth outcomes. This dissertation substantially extends that earlier work.

### 1.1. *Significance*

Studying the effects of disasters on foetuses and young children is important for two reasons. Firstly, as the climate changes and populations grow, more people will be impacted by disasters. Human populations are being increasingly exposed to environmental disasters with estimates of up to 375 million people exposed per annum (Oxfam International 2009). Climate forecasts indicate specific risks to Australia (Climate Commission 2012), making it increasingly important to understand the scope of this impact, particularly beyond those effects easily observed and measured, such as infrastructure damage and acute health risks.

While there is a small but growing body of literature that considers the effects of environmental disasters on human reproductive outcomes, little anthropological work exists. This dissertation examines the impact of foetal and childhood disaster exposure in an evolutionary context, specifically in the context of life history theory. It is unique in that it examines bush- or wild-fires in this light and considers the reproductive and developmental outcomes of disasters in an Australian context. As exposure to bushfires increases, a thorough understanding of the long-term implications of fire exposure on public health becomes increasingly important and urgent.

Secondly, many, if not most, women will experience some degree of stress during pregnancy. The study of disasters provides a "natural experiment" that allows a discrete stressor to be studied at a whole-of-population level. This is useful in improving our understanding of the broader mechanics and outcomes of stress in pregnancy. Greater knowledge of prenatal stress should assist in determining the longer-term costs of environmental disasters, as well as deepening our understanding of the effects of other, more commonplace, stressful events (Devakumar et al. 2014).

## 1.2. *Research questions*

This dissertation therefore seeks to analyse reproductive responses to environmental challenges in a modern human population. Specifically, it examines the changes in birth rate, birth weight, gestational age, plurality rates (the rate of multiple pregnancies), secondary sex ratio and selected childhood development indicators arising from prenatal bushfire exposure through the theoretical lens of life history theory. It also seeks to record the experiences of women who lived through bushfires while pregnant.

The dissertation asks four key research questions:

1. What is the effect of prenatal bushfire exposure on birth weight, gestational age, plurality and secondary sex ratio in populations exposed to the Canberra and Black Saturday bushfires?
2. What is the effect on fertility rate in populations exposed to the Canberra and Black Saturday bushfires?

3. What is the effect of prenatal bushfire exposure on developmental indicators in on populations exposed to the Canberra bushfire?
4. What is the lived experience of bushfire exposure while pregnant? This section focuses on the populations exposed Canberra and Black Saturday bushfires but includes some participants who experienced other fires.

## 1.3. *Theoretical basis*

This dissertation argues that human psychological and physiological responses to stress are guided by evolved mechanisms and, thus, can be predicted by evolutionary theories. Accordingly, the dissertation uses the theoretical platform of life history theory (LHT), which proposes that all animals are engaged in, and their behaviour informed by, a cycle of survival, maturation and reproduction (Chisholm 1999). Within this cycle, environmentally-informed trade-offs will occur that alter the balance of resources invested in maturation and reproduction in order to aid survival. In terms of human gestation, environmental influence on developmental biology should arise from both the maternal and foetal/child assessment of the environment. These assessments may be conscious and/or unconscious for the mother and child and the mother's assessment is communicated hormonally to the foetus (Chisholm 1999; Ellison 2009; Gray 2010).

Within the context of this thesis, stress arising from bushfire exposure is the environmental influence being examined. If fire exposure leads to high stress then this environmental stress may theoretically be predicted to trigger reproductive trade-offs. For example, investment in current reproduction might be decreased or abandoned to reserve reproductive potential for a future time when conditions are more favourable.

Such a trade-off would theoretically result in decreases in foetal weight, gestation length, plurality rates and secondary sex ratio, as well as increases in subsequent birth rate. Because of the potentially maladaptive consequences of changes in maternal investment, childhood behavioural disruptions related to the epigenetic effects of early stress exposure on future stress responsivity could also be expected.

### 1.4. *Hypotheses*

Consistent with this theoretical basis, the hypothesis proposed here is that exposure to bushfires, and attendant stressors, will alter reproductive strategy and developmental outcomes in the short and long-term. As these changes are expected to accord with the predictors of LHT. This generates further auxiliary hypotheses: (1) that gestational length will shorten in exposed mother-child couples and birth weight will decline; (2) that the fertility rate will increase; (3) that secondary sex ratio and plurality rates will decline in response to disaster exposure; and (4) that greater developmental comorbidity will be observed in children exposed to bushfires in utero. Finally, due to the documented effects of disasters, a further hypothesis is (5) that the maternal experience of bushfires while pregnant will be highly stressful.

### 1.5. *Dissertation overview*

This research focuses on two central cohorts: mother-child dyads exposed to the 2003 Canberra fires (and aftermath) during gestation or shortly after birth and mother-child dyads exposed to the 2009 Black Saturday fires (and aftermath) during gestation. A small amount of data from other fires was included in Chapter Eight).

Studying the potential effects of bushfires on human reproduction raises three principal methodological challenges. Firstly, the study design must necessarily be retrospective because exposure cannot be predicted. However, because much of the data used in the study is drawn from government collections, pre- and post-disaster data were available and have been used in an attempt to mitigate this limitation.

Secondly, it is neither feasible nor desirable to expose mother-child couples to stress intentionally or to limit their access to services, such as counselling, preventative prenatal healthcare and neonatal health care, which might reduce the observable effect of bushfire stress. This limits the degree of control over the characteristics of the exposed group or the confounding factors to which they are exposed. Wherever possible, this challenge has been managed through analytic methods.

Thirdly, the information required – such as gestational age, birth weight, behavioural data and maternal characteristics – is specific, sensitive and voluminous, making it difficult for a single researcher to collect on a large-scale or with sufficient accuracy. To manage these difficulties, standard government data collections have been used wherever possible. This approach has allowed a broad-scale quantitative comparison between the population directly affected by fire exposure and a geographically proximate comparison population which was not directly affected by fire exposure.

### 1.5.1. Dissertation outline

The dissertation begins by outlining its theoretical approach – life history theory (LHT). It then provides an overview of current understandings of human reproductive strategy, the influence of stress in early life and the relevant literature. The dissertation then presents its method and results and discusses these results in the theoretical context. It concludes by outlining recommendations for future research and practice. This dissertation is divided into nine chapters, described below.

Chapter One (this chapter) introduces this study and specifies the significance of the research, its research questions and hypothesis. It then provides the context for the five research chapters, by providing a background to the disasters studied in this dissertation. This chapter provides a brief overview of bushfire in Australia, including contemporary community and public policy responses to fire. The chapter then outlines the current evidence for worsening future bushfire conditions due to the effects of climate change. Lastly, the chapter provides a brief description of the fires studied in this dissertation (the Black Saturday fire and the Australian Alps fires – i.e. the fire complex that includes the Canberra fire).

Chapter Two expands on the theoretical underpinning of the dissertation. It includes the historical development of LHT, the benefits and challenges of applying LHT to modern human populations and the role of LHT in explaining the phenomena examined here. The chapter then considers human reproductive strategies in more detail before concluding with a brief overview of human reproductive physiology. In this respect the chapter focuses on those elements which are relevant to LHT (i.e. the costs of reproduction) and discusses evolutionary paradoxes, such as human altriciality and dangerous parturition.

Because stress is both a central aspect of this dissertation and a complex topic, Chapter Three considers the human stress response in more detail. The chapter concludes by detailing more recent theoretical developments around the role of early life environmental stress in shaping and programming health in later life. This includes issues of developmental plasticity, foetal origins of adult disease and epigenetic change.

Chapters Four to Eight constitute the dissertations analytic chapters with one chapter apiece addressing the five hypotheses. To aid clarity and ease of navigation, each chapter presents an aspect of the research in a research article format, commencing with an overview of relevant literature, providing specific hypothesis and detailed research methods, followed by results and a discussion of those results.

Chapter Four presents a quantitative cohort study of birth records examining fire effects from the Canberra and Black Saturday fires on birth weight and gestational age among children exposed to fire while *in utero*.

Chapter Five presents a quantitative cohort study examining two indicators of early pregnancy loss: changes to plurality rates and secondary sex ratio.

Chapter Six presents a quantitative cohort study of total fertility rates following the Canberra and Black Saturday fires.

Chapter Seven presents a quantitative cohort study of the indicators of developmental morbidity in children exposed to the Australian Alps fire in early life. Unfortunately, suitable data were not available to conduct this analysis in relation to the Black Saturday fire.

Finally, Chapter Eight presents a mixed-methods examination of the maternal experience of bushfire stress, trauma and recovery.

Conclusions drawn from the body of research as a whole are presented in Chapter Nine, which acts to unify the research presented in the five previous chapters. This chapter also makes recommendations for future research and public policymaking.

## 1.6. *Background to the disasters studied*

Australia experiences a range of environmental disasters. While cyclones and monsoonal flooding are common in the north of the country, bushfires and droughts are common in the south. The arid centre of the country also experiences seasonal floods and droughts. Although the north and south are in different climatic zones, they both experience riverine and flash flooding, droughts, storms, heatwaves, pest plagues, dust storms and minor earthquakes (Australian Emergency Management Institute 2012). Population residential patterns and increases in extreme weather frequency are contributing to increases in the scale, impact and frequency of bushfires (Teague et al. 2010d).

### 1.6.1. Southern Australian ecology

Victoria, the most south-eastern state of mainland Australia, has a long history of bushfires dating to before white settlement. The most significant Australian fire disaster is the Black Saturday bushfire, which killed 173 people. In terms of fatalities, the next most significant bushfires are: the 1983 Ash Wednesday fires, which killed 47 people in Victoria and 32 people in the neighbouring state of South Australia; the 1939 Victorian Black Friday fires, which killed 71 people; and the 1967 Black Tuesday fires which killed 62 people in the state of Tasmania (Australian Emergency Management Institute 2012; Bushfire Cooperative Research Centre 2009; Teague et al. 2010d). Of all

Australian bushfires, Victoria has been the site of the greatest number of significant fires (in terms of fatalities). Although Victoria occupies only 3% of the Australian land mass, it accounts for about half of bushfire-related economic losses (Pyne 1991). Not only do bushfires occur more frequently in Victoria, they are also more intense in nature and more deadly. Between 1900 and 2008, Victoria accounted for 53.6% of bushfire deaths, despite accommodating only about a quarter of Australian residents (Haynes et al. 2010).

Temperate Australian rural landscapes predominantly comprise high- and low-land eucalyptus-dominated bushland interspersed with open grasslands and agricultural land. Eucalyptus is a largely indigenous genus which generally refers to over 700 Australian tree species. Eucalyptus species have been a key ecological force in Australia, with their pollen evident in the palynological record as far back as 34 million years. As such, fire has long been a prominent force in Australian ecology (Pyne 1991). Victoria and, to a lesser extent, Canberra are within a conflagration-prone zone that Pyne (1991) terms the fire flume, due to dominant eucalypt forests. Eucalyptus is an active pyrophyte, combining oil-rich foliage, an evergreen growth habit, thick and easily-shed protective bark, and lignotuber-based reproduction (Pyne 1991). In Victoria, eucalyptus forests commonly have scleromorphic understoreys; these understoreys of persistent dry matter combine with heavy and oil-rich leaf-litter from the eucalyptus canopy to create a potent fuel source. The forests that flourish in much of eastern and central Victoria, in particular, are typical of this ecology. As Hansen and Griffiths (2012) state, “the essential biological imperative of this place is to burn”.

### 1.6.2. Climate change and future predictions

Driven by climatic changes, bushfire risk appears to be increasing in south-eastern Australia (the states of South Australia, Victoria, Tasmania and southern New South Wales, as well as the Australian Capital Territory). Climatic trends, along with population and residency patterns, suggest that the intensity and frequency of fires will increase with greater impact on human settlements. The International Panel on Climate Change (2012) puts the likelihood that changes to the global climate will result in increased severity and frequency of heatwaves in southern Australia at 90%. Further modelling by the Australian Bureau of Meteorology (BoM) and the Commonwealth Scientific and Industrial Research Organisation (CSIRO) predicts an increase in temperatures and reduction in rainfall across southern Australia, resulting from climate change (Bureau of Meteorology 2012).

Climatically, south-eastern Australia is mostly temperate, experiencing wet winters (permitting fuel growth) and warm-to-hot as well as dry summers (which support ignition), see Figure 1-1.

[redacted]

Figure 1-1: Australian climate zones – Canberra and Victorian capital Melbourne are labelled (Bureau of Meteorology 2012)

Climate trend data shows that Victorian maximum summer temperatures have been increasing over the past 30 years, as have mean temperatures (Bureau of Meteorology 2012), while daytime cloud cover has decreased. The Climate Commission's 2012 report *Off the Charts* notes that summer temperatures experienced across Australia during the summer in 2012 were unprecedented (Climate Commission 2012). Temperatures reached above 45 degrees Celsius in central South Australia and inland Western Australia, as well as southern and central parts of the Northern Territory and resulting in a new colour being added to BoM's colour-coded temperature maps. Average temperatures show a strong rising trend, with eight of the ten hottest years on record (since 1910) occurring since 2002 (Bureau of Meteorology 2016). In 2012–13, the hottest year since records began was recorded nation-wide (Climate Council 2014), with the summer of that year termed the Angry Summer.

These changing conditions worsen the threat and magnitude of bushfires, while also increasing deaths from heat exhaustion. Between 1973 and 2010 the Forest Fire Danger Index (which rates the threat of bushfire) increased at 16 out of 38 measurement locations with no locations recording significant declines. These 16 measurement locations are mostly concentrated in south-eastern Australia (Climate Commission 2013). Not only does increasing fire threat raise the direct risk of fire, it also limits the degree to which mitigation techniques, such as back burning and fuel reduction burning, can be employed (Climate Commission 2012).

Changes in climate and existing flammable native forest are now combined with population increases in many peri-urban areas – where dense bush and housing are intermeshed. This creates increased risks to human life, health and infrastructure. For example, strong migration into the peri-urban arc surrounding Greater Melbourne (a phenomenon known colloquially as "tree-changing") has meant that this area, which extends through central and north-eastern Victoria, now has the highest density of people living in a fire-prone area in Australia (Kissane 2010).

### 1.6.3. Bushfire response in Australia

Bushfires are an inherently traumatic type of environmental disaster: fast-moving, unpredictable, loud, hot and powerfully destructive. However, Australian emergency community preparedness policy has been relatively non-interventionist. This dissertation focuses on the Canberra 2003 and Black Saturday 2009 fires; however, these are only two examples from a long and continuing history of fire in Australia.

Australian bushfire response policy is unique because it has never recommended broad-scale evacuation, unlike the policies of other countries with similar threat profiles (Haynes et al. 2010). The Australasian Fire and Emergency Service Authorities Council's policy (AFAC) advocates that people in fire-threatened areas make an individual choice either to defend their property or to leave the area of their own accord (with inconsistent government support to do so). This policy is known formally as the "prepare, stay and defend or leave early" policy, more commonly known as "stay-or-go" (Haynes et al. 2010 p.185). It has been in place in some form for over 100 years and is based on the observation that deaths most commonly occur when people are caught in the open while trying to flee a fire at the last moment. This observed pattern of death-location has resulted in a policy that advocates either leaving early (well ahead of the fire front) or remaining in a structure, thereby affording some protection from radiant heat.

Griffiths (2012), however, argues that the research which supports the stay-or-go policy misinterprets the patterns of deaths which occur following fires. For instance, bodies found near dwellings are determined to be late evacuators, rather than active defenders, although there is no strong empirical evidence to support this distinction (Hansen and Griffiths 2012). Additionally, people found dead inside dwellings are determined to be people sheltering, rather than active defenders sheltering at the last moment when defence becomes impossible. Griffiths contends that the misinterpretation of the location of death leads to a significant overstatement of the capacity of people to defend properties and survive. Indeed the argument for active defence may owe more to Australian cultural mythology of self-reliance than clear interpretation of available evidence (Griffiths 2012).

In terms of stress, the stay-or-go policy exposes residents to a range of potential stressors even as it protects them from others. Forcible evacuations are known to create significant stress, as well as lasting damage to social support networks, and thus the stay-or-go policy avoids those stressors (Oliver-Smith, 1999). However, the self-reliance and determination that is at the heart of stay-or-go creates other stresses as residents (particularly Victorian residents) are left largely unguided in making final decisions and have to bear the stress of both those decisions and their consequences. In the Australian Capital Territory, advice to evacuate is provided more readily by government authorities (Bushfire Recovery Taskforce 2003) and this may act to reduce stress on individuals.

In Victoria, the Country Fire Authority has been strongly criticised for its publicly available information, specifically that its information misrepresents the dangers of defence and that its policies require people to make an assessment of their own physical and psychological wellbeing without sufficient guidance (Kissane 2010). The stay-or-go policy asks residents to make decisions as to their preparedness for defence with little insight into what they will be required to do, the scale of the fire or the time of its likely arrival. The emergency statute in Victoria is ambiguous regarding compulsory evacuation and, as a result, compulsory evacuations are not part of the practice of emergency service agencies (Teague et al. 2009). This leaves crucial decisions entirely in the hands of residents.

Although official advice is to prepare and follow a bushfire plan, in an assessment of community preparedness at the Murrindindi fire (part of the Black Saturday fires), McLennan and Elliot (2011 p.4) state that "commitment to a bushfire plan may be a path to disaster – especially a plan to stay and defend". The same study notes that many community members lacked sufficient information about the fire threat to make sound decisions regarding their own safety.

Importantly, the study notes that children are at very high risk and should not be present at the fire front (McLennan and Elliot 2011). However, CFA publications from that period of time included illustrations of families with children remaining to defend properties – implying that the fire front is potentially a safe and reasonable place for children (Kissane 2010). Although some fires are mild and survivable for adults and children, it is reasonable to expect that government advice will follow a conservative approach. Following the Black Saturday fires, the Victorian Bushfires Royal Commission (2009) recommended that clearer advice and greater support be provided to support relocation in the future, while this recommendation has been acted on by Government, there has not yet been another major fire able to test the effectiveness of the improvements.

Despite the longevity of the stay-or-go policy, there has been relatively little assessment of its effectiveness. One study of the Ash Wednesday fires of 1983 (albeit conducted almost 10 years later) found that most people who perished in the fire either did not recognise the danger or chose an ineffective survival strategy (Krusel and Petris 1992). The smallest proportion of people killed were those who were physically unable to implement their survival plan, in contradiction to other disaster mortality rates that are often higher in the very old, very young and women (Krusel and Petris 1992; Neumayer and Plumper 2007). In a study of all bushfire deaths between 1900 and 2008, distinct gender patterns emerged. Firstly, male deaths were consistently higher. But deaths also reflect prevalent gender-based patterns in behaviour. While men are more likely to die defending properties, women more commonly die in homes, often while sheltering children (Haynes et al. 2010). This remains the case in recent fires, although the difference has declined (Haynes et al. 2010; McLennan and Elliot 2011).

The Royal Commission made a number of policy recommendation in the wake of the Black Saturday fires. These included changes to stay-or-go, land buy-back schemes for homes in high-risk areas, new building codes and standards, and the addition of a new uppermost level in the Fire Danger Rating warning system (the new level is Code Red/Catastrophic) (Department of Justice 2012). Victorians are exposed to considerable advertising campaigns aimed at increasing bushfire awareness and promoting safety messages; however, there is relatively little information on their levels of awareness.

Research conducted by the Country Fire Authority following the Black Saturday bushfires suggests that awareness of recommended actions is moderately high in residents of fire-prone areas. Over 90% of people in fire-prone areas understood that they were at risk of bushfire and that they would need to be self-sufficient when threatened (Strahan Research Pty Ltd 2009). However, only 18% of respondents had a written bushfire plan, while a further 63% had an unwritten plan. While 66% of respondents planned to leave their property if it was threatened by a bushfire, 26% of people planned to wait until they were advised to leave by authorities. This is problematic because all these people were resident in a state that does not provide specific advice on when to leave. Only 62% of residents could identify the highest risk warning category (Strahan Research Pty Ltd 2009). Because these data were collected in the aftermath of the Black Saturday fires, it is possible that respondents had a heightened awareness of fire at that time.

Since the Black Saturday fires, Bushfire Neighbourhood Safer Places have been introduced into most Victorian country towns. These are signposted outdoor locations (e.g. a park, football oval or principal streets) at which people can congregate in the event of a fire. *Figure 1-2* shows the signage outside Victory Park in the Central Victorian town of Castlemaine. However, signposting at these Safer Places clearly states: that residents cannot expect to be safe in these locations; that they should not expect any provisions, amenities or emergency services to be available; that the location has limited capacity; that no provision for people with special needs or medical needs will be available; and that the safer place may offer less protection that the residents' own homes. As such, their utility as a public safety mechanism is limited.


Figure 1-2: Bushfire Neighbourhood Safer Place signage in Castlemaine, Victoria

In the ACT, unlike Victoria, the *Emergency Management Act 1999* (ACT) gives emergency services the authority to order people to leave their homes for their own safety. Although the ACT also uses a stay-or-go policy, the clearer provisions around evacuation meant that ACT residents are provided with clearer evacuation directions. Accordingly, in 2003, areas under direct threat were evacuated (via doorknocking) by the Australian Federal Police (Murray 2003). Unlike Victorians, ACT residents were clearly directed in their response which may have reduced both the degree of stress and the number of people who defended properties in the direct fire line. Evacuation centres were available at the time the fire first impacted the city and ACT Housing was available to assist with relocate (Murray 2003).

In both fires, residents who decided to stay and defend their properties, were likely to be directly exposed to extreme physiological and psychological stressors. Even in situations where the fire front does not reach the property, defence may still entail several vigilant days of patrolling against ember attack, both before and after the direct fire threat passes. This means that even those who escape direct exposure to the fire are exposed to days of reduced sleep and increased arousal. For many people in fire-prone areas, exposure to a large fire will have been preceded by weeks (if not months) of smaller fires and days classified as high-risk, meaning that bushfire plans should be enacted. As such, residents will have already experienced substantial periods of high-stress patrolling or will have already relocated. Relocation also entails its own series of stressors, related to dislocation, anxiety and financial costs (Oliver-Smith, 1999). Therefore, living in a fire region means exposure to stressors that may create long-term Hypothalamus-Pituitary-Adrenal (HPA) arousal, even during mild seasons with few fires.

In both the Canberra and Black Saturday fires, deaths were concentrated in relatively small communities. In the Black Saturday fire, multiple members of family groups died and proportionate population loss in small communities was very great. For these reasons, it is likely that evidence of disaster stress will be seen more acutely in people who were living in these small communities at the time of the fires, compared to people living in areas which experienced the fires but which experienced few or no deaths (Adams et al., 2009; Green, 2009; Scaramella et al., 2009).

### 1.6.4. Black Saturday – 2009

During the lead up to the Black Saturday bushfires, in late January 2009, Victoria had experienced a prolonged heatwave with peak temperatures repeatedly rising above 43 degrees Celsius with very low humidity. By early February, the State's bushland was dry and combustible (Teague et al., 2010). Several large fires burnt during late January and the first week of February, when 43 degrees Celsius maximum temperatures occurred on three consecutive days. On 7 February, a further 316 fires ignited. Temperatures peaking at 46.4 degrees Celsius combined with high winds of up to 120 kilometres per hour propelled 15 of those 316 fires into large and uncontrollable wildfires. It is these fires that are collectively known as the Black Saturday fires (Teague et al. 2010d).

The 15 principal fires were the Delburn, Bunyip, Kilmore East, Horsham, Coleraine, Pomborneit–Weerite, Churchill, Murrindindi, Redesdale, Narre Warren/Harkaway, Narre Warren/Lynbrook (Coral Drive), Narre Warren/Lynbrook (Golf Club Road), Upper Ferntree Gully, Bendigo and Beechworth-Mudgegonga fires. Combined, these 15 fires resulted in 173 deaths and losses of approximately $4 billion in property damage (Teague et al. 2010d). Five of the fires led to a loss of life: the Kilmore East fire (119 deaths), the Murrindindi fire (40 deaths), the Churchill fire (11 deaths), the Beechworth-Mudgegonga fire (2 deaths) and the Bendigo fire (1 death) (Teague et al. 2010d). The fires burnt 450,000 hectares and destroyed over 3,500 structures. It took 31 days to extinguish the last of the fires. Figure 1-3 shows the fire footprint as dark shaded areas.

[redacted]

Figure 1-3: Black Saturday fire footprint (source: Teague et al. 2010b)

As noted earlier, bushfires are a regular seasonal occurrence in Victoria; however, fires of this scale and intensity are rare. Due to erratic and strong winds along with extreme temperatures, the Black Saturday fires were unpredictable and highly dangerous, thereby restricting the efficacy of fire-fighting efforts (Teague et al. 2010d). Fire-fighting was limited by winds that drove spot fires (small fires resulting from wind-born embers) well ahead of the main fire fronts and created crowning fires and flash-overs (fire storms burning in the tree canopy and igniting trees ahead of them). Ground-crews were unable to tackle the fires effectively, while high winds increased the dangers of aerial fire-fighting (Teague et al. 2010d). Because the fire direction was difficult to predict and public communications were impeded by bureaucratic errors and fire damage, many people did not receive warnings and did not leave the fire areas early enough to ensure their safety (Teague et al. 2010b).

The Black Saturday fires are an example of the likely nature of major fires in the future. These fires killed more than twice the number of people killed in the next most deadly fire (the 1983 Ash Wednesday fires). Notably, the proportion of injuries to deaths was almost inverted from those reported following the Ash Wednesday fires, suggesting that the Black Saturday fires were of a much greater intensity. Of the 173 people killed, 163 were very badly burnt and required forensic identification (Kissane 2010). Deaths resulting from the fires included that of Ms Nicole Jefferson who was over eight months pregnant at the time of the fire. The Victorian Coroner found that her death was likely related to her pregnancy-related physical incapacity (Teague et al. 2010a). A further 16 of the deaths were children under 12 years of age (Teague et al. 2010c). The high death toll likely represents the intersection of growing peri-urban populations, poor fire management and extreme weather conditions.

Following the 2009 bushfires, publicly funded mental health services were available for affected people. Over 190,000 people received some form of treatment and, among these, there was a notable increase in demand for perinatal depression services (Bassilios et al., 2011). Several authors have documented extreme stress, trauma, grief and loss following the Black Saturday fires, as well as an increased occurrence of Post-Traumatic Stress Disorder in adults and children (Griffiths 2012; Hansen and Griffiths 2012; Hyland 2011; Kissane 2010).

Notably, some individuals who survived the Black Saturday fires, but were exposed to circumstances that could have resulted in death, reported an absence of fear for the duration of the fire (McLennan et al. 2011). McLennan et al. (2011) argue that for these near-miss survivors their psychological ability to manage extreme stress was life-saving and allowed them to make key survival decisions.

Due to the widespread perception of mishandling of the fire by emergency services, the Victorian Government ordered a Royal Commission into the fires, the highest level of independent inquiry available in Australia. The Victorian Bushfires Royal Commission found a number of weaknesses in the State's fire management systems, particularly with regard to fuel load management and public communications, and made recommendations accordingly (Teague et al. 2010b). The Black Saturday fires and the Royal Commission's recommendations have resulted in the revision of Victoria's bushfire safety policies, public communications, fuel reduction strategy and the construction standards for buildings in bushfire-prone areas (Country Fire Authority 2011a; Country Fire Authority 2011b; Teague et al. 2010b).

### 1.6.5. Canberra Fires – 2003

In January 2003, drought and heatwave conditions affected the whole south-eastern corner of Australia. A line of fires, including the 2003 Canberra fires, extended across the Australian Capital Territory (ACT) and the neighbouring states of New South Wales and Victoria. These fires are collectively known as the 2003 Australian Alps fires. During January, the fires joined together to create a total fire front over 800 kilometres (497 miles) long (Camilleri et al. 2012; Worboys 2003). Steep alpine terrain and erratic fire behaviour complicated fire-fighting activities in all areas. In the ACT, extreme heat and low humidity had created ideal fire conditions that enabled a series of fires throughout early January. On 18 January, these fires merged to form a fire front which then crossed the New South Wales/ACT border and burnt through the southern and western suburbs of Canberra (Camilleri et al. 2012).

Colloquially known as the “Bush Capital”, Canberra is heavily treed, even in urban areas. The ACT Government manages over 700,000 urban trees (Canberra had a population of around 322,000 in 2003), with many more trees on private land (Australian Bureau of Statistics 2003; Territory and Municipal Services 2013). At the time of the fire, Canberra was bounded to the south by over 106,000 hectares of heavily-forested alpine national parks and 7,500 hectares of pine plantations. Overall, only 19% of the Territory’s landmass is classified as built environment (Australian Natural Resources Atlas 2013). Close proximity of trees to residences is also usual in Canberra suburbs, with strict regulations governing the removal and lopping of larger urban trees (Territory and Municipal Services 2013). Despite the common perception of bushfires as a rural concern, these conditions place Canberra city and its suburbs at considerable risk of suburban bushfire.

In total 1,600 houses were damaged, including 488 that were destroyed. Approximately, 160,000 hectares were burnt, accounting for around two-thirds of the ACT’s total landmass. This included 16,000 hectares of plantations and 31,000 hectares of farmland (Camilleri et al. 2012). Four people were killed in the fire and 489 injured, including 49 injuries which were severe enough to require hospitalisation. Over 5,000 people evacuated to official support centres and many more are understood to have evacuated to the homes of friends and family (Macdonald et al. 2010). More than 50,000 residents lost essential services during and following the fire. The estimated financial cost of the fires was $350 million (in terms of public and private property loss and damage) (Macdonald et al. 2010). *Figure 1-4* shows the fire footprint, indicated by flame motifs.

[redacted]

Figure 1-4: Fire areas in the Canberra 2003 fire. Fire affected areas are marked with flame motifs. Source: *The Sydney Morning Herald*, 19 January 2003

The 2003 fire crisis was the most significant ever experienced in Canberra, in part due to the speed of the fire – all the damage occurred in just a few hours – and in part due to unusual firestorm behaviour. The Canberra fires included one of the first film-recorded instances of a fire tornado in Australia, in which convection air currents drive a circulating column of fire.

In populations exposed to the fire directly (i.e. those who experienced loss, including property damage), levels of psychological distress were higher than the general population three years later (19.5% of exposed residents had high to very high levels of psychological distress on the Kessler-10 scale compared to 13.4% in general Canberra population) (Camilleri et al. 2012). Levels of overall health were also lower in the exposed population (25% rated their health as fair or poor compared to 11.3% in the general Canberra population) and 39% of respondents reported that they perceived lasting negative consequences in their lives resulting from the fire in their everyday lives (Camilleri et al. 2012). Many survivors of the fire experienced powerful sensory memories which persisted over several years, consistent with much trauma research. Survivors also noted that large bushfires elsewhere in the country (e.g. the 2009 Victorian fires) triggered a return of memories of the fire (Macdonald et al. 2010).

## 1.7. *Chapter summary*

This dissertation uses two natural experiments – the Canberra and Black Saturday bushfires – to examine the effects of maternal disaster exposure on human reproductive outcomes. Both fires were substantial events that affected many individuals, as well as highlighting strengths and weakness in the official disaster response.

Taking a life history theory perspective, the dissertation predicts that the stress associated with this disaster exposure will trigger adaptive responses to maternal stress. Evidence of such responses is sought by examining the average birth weights, gestational age, plurality rates, fertility rates and the secondary sex ratio in exposed cohorts. Similarly, developmental and behavioural indicators are examined. In order to better understand what maternal stress might be experienced following fire exposure, a mixed-method study documents the experience of some exposed mothers.

Chapter Two expands on the theoretical approach used in this dissertation. It discusses the application of LHT to humans, as well as providing greater detail on human reproduction. It concludes by applying the trade-offs theorised by LHT to human reproduction.

# 2. THEORETICAL APPROACH

This dissertation seeks to explain the potential links between stress and birth or developmental outcomes from a bioanthropological perspective using the theoretical basis of life history theory (LHT) – a common theoretical approach in the disciplines of biological anthropology, human ecology and evolutionary biology. Although it is a well-established theory within evolutionary ecology, LHT is less commonly applied to humans. Application to humans is more challenging because of the complexities of human life history (particularly the high degree of heterogeneity across the species), as well as a more philosophical reluctance to apply biological theories of reproductive ecology to human behaviour.

In this dissertation, LHT provides a theoretical framework for assessing how environmental stressors might relate to changes in reproductive outcomes (including birth outcomes and fertility) and child development. It also allows for an examination into how the quality of the environment is communicated to the body and thus influences reproduction and maturation at a biological level. This dissertation argues that the changes in life history that can be observed in humans exposed to stress, such as changes in birth weight, are candidates for evidence of a biological adaptation to environmental variation. Such mechanisms are likely to have developed during periods of human evolution when there were fewer protective factors against environmental change than are currently available – that is, that these adaptations likely arose in response to the environment of evolutionary adaptedness (EEA). Thus, the dissertation proposes that humans who experience environmental disasters may respond biologically in a manner which is consistent with LHT, because they are subject to conditions which mimic, to some extent, conditions experienced in the EEA (Fink 2010). The ability to make alterations to reproductive strategy in response to environmental change suggests an environmental sensitivity and a developmental plasticity which is potentially a product or by-product of natural selection (Voland 1998).

This approach assumes that humans have mechanisms for monitoring their environment which lead to biological responses. The principal method by which the body responds to the external environment is via the stress response. Due to the complex nature of the human stress response, Chapter Three focuses on the stress response and its effect on maturational triggers. This chapter provides an overview of LHT and how it is applied to humans. Because reproductive strategy is a central element of the application of LHT to this research, the chapter briefly summarises those aspects of human reproductive physiology that are particularly relevant and outlines the predicted effects of environmental variation on reproductive strategy, in particular the reproductive trade-offs that are theorised within LHT.

## 2.1. *Overview of life history theory*

Although LHT traces its development to the work of pioneering 19th century theorists Charles Darwin and Alfred Wallace, LHT was not applied to hominins until much later (Kennedy 2003) and has developed further since that time (West et al. 2011). Because of the importance of reproduction to evolution, reproductive strategy has been studied and theorised widely, with LHT becoming a dominant approach (Rogers 1989; Schuiling 2003).

LHT is the study of life cycles and life events in an ecological and evolutionary context (Chisholm 1999). It concerns individuals' allocation of resources to the primary activities of surviving, growing, maintaining (collectively termed *somatic effort*) and reproducing (*reproductive effort*) over the lifespan (Chisholm 1999). Because natural selection acts via reproductive success, all existing organisms have been selected on the basis of reproductive ability, and the same selective pressure will apply to any offspring that they produce (Chisholm 1999). LHT proposes that organisms will undertake these activities requiring somatic or reproductive effort in the manner that is most likely to increase their direct or inclusive fitness or, in fact, their overall reproductive success (Lickliter and Honeycutt 2003).

Because most important resources are limited, and collecting resources incurs costs, LHT proposes that all organisms must make trade-offs between competing resource demands in order to reproduce most successfully. The connection between LHT and reproductive strategy is thus very close. Chisholm (1999) argues that life cycles are themselves reproductive strategies, because the only outcome of survival, growth and maintenance that is subject to selective pressure is reproduction. The need to divide resources between competing interests, such as growth or reproduction, forms the core of LHT: the principle of allocation (Chisholm 1999). These trade-off decisions will vary across the lifespan due to the shifting balance between realising present gains and the estimated value for potential future gains. In this sense, LHT can be understood as an economic theory of resource allocation, or cost-benefit analysis, in an environment constrained by resource availability and individual mortality risk. LHT is also consistent with game theory, in which organisms may "hedge their bets" against future uncertainty by selecting precautionary reproductive strategies. In terms of reproduction, the principal trade-offs are between mating and parenting efforts, between high quality offspring and high quantity offspring, and between future and present reproduction (Lickliter and Honeycutt 2003). The most all-encompassing of these trade-offs is that between future and present reproduction, also known as the General Life History Problem (Schaffer 1983).

## 2.2. *Applying life history theory to humans*

LHT has developed substantially in response to discoveries emerging from the fields of behavioural ecology, evolutionary psychology, epigenetics and neuroscience. These advances have increased the sophistication of the theoretical model and have therefore allowed the model to be more meaningfully applied to complex organisms, such as humans. Two key developments have been a broader conceptualisation of the environment to include social resources and pressures and the development of stronger theoretical links with evolutionary psychology (Lickliter and Honeycutt 2003). Modern interpretations of LHT propose that reproductive decisions are informed by the social, as well as physical, environment. The impact of these external environments then informs the organism's internal physiological and psychological state (Lickliter and Honeycutt 2003). Therefore, reproductive decisions change not just over the life course but also as these four environments – the physical, social, physiological and psychological - change and interact. Chapter Three applies these principles specifically to the subject of this dissertation by focusing on the utility of the stress response in communicating environmental quality to the growing foetus

In humans, the cognitive process of developing assessments of environmental quality (across the four environment domains) can be analysed by linking LHT and evolutionary psychology. While LHT assists in determining what consequences might flow from environmental assessments, evolutionary psychology is useful in understanding how and why those assessments are made (Lickliter and Honeycutt 2003). This intersection of LHT and evolutionary psychology creates a more complete, and certainly more useful, prism through which human responses and adaptations to environmental change may be analysed.

Modern LHT can also be applied in light of epigenetic research (which is covered in more detail in Chapter Three) to better account for the sources of complexity and variation in human behaviour. One of the theoretical challenges to assimilating these approaches is the increasingly outmoded conceptualisation of nature and nurture. Because of the constant interplay between genes, environment and behaviour, it is not meaningful to consider development as a dichotomous process. Lickliter and Honeycutt (2003 p.821) suggest that there remains within evolutionary psychology "a deep-seated ontological separation between …nature (genes) and … nurture (all else)". Such a separation is increasingly not supported by biology or genetics and severely limits the holistic understanding of human reproductive behaviour in an environmental context. The next section considers some of the specific limitations and challenges in applying LHT to humans.

An understanding of the role of environmental quality in influencing reproductive strategy must necessarily be grounded in the reproductive physiology of human mothers and foetuses. The metabolic impact of conception and gestation is a particular focus here, as metabolic load provides a likely pathway through which gestation and stress might interact in a way that explains the changes to gestational age, birth weight, secondary sex ratio and fertility that are observed following environmental disasters. In this dissertation, fertility is defined, as in demography, as the number of children a woman actually bears over her lifetime, while fecundity describes a woman's reproductive potential.

### 2.2.1. Ovulation, conception and implantation

Compared with those of other placental mammals, the biological functions of the reproductive systems of Homininae differ substantially. While many other mammals' reproductive systems support conception without evidence that conception has occurred, the female reproductive organs of Homininae require the fertilised ovum to signal its presence in order to avoid menstruation (Ellison 2001). The presence of menstruation is also relatively unusual among mammals (although not restricted to Homininae) and an unresolved area of debate. It is unclear whether the costs of maintaining the endometrium following an unfertilised ovulation exceed those of shedding and regrowing the endometrium. Similarly, it is unclear whether the relatively long menstrual cycle present in Homininae is due to the time required to regrow the endometrium or whether this time is required for the processes that support ovulation and that this long delay therefore makes it costly to maintain the endometrium (Ellison 2001). With the endometrium only able to receive a conception for part of the cycle, the timing of ovulation becomes important.

In humans, ovulation is highly sensitive to both life history and environmental conditions. Ovulation commences with menarche, although anovulatory periods are very common in the years immediately following menarche, and the actual frequency of ovulation steadily increases over the ten years following menarche. This delay is, perhaps, related to skeletal immaturity in girls and may act to defer reproduction until pelvic growth is complete in the late teens (Ellison 2001). Additionally, it may allow girls and women to develop substantial adipose fat stores prior to embarking on reproduction. Certainly, early reproduction incurs risks, with low birth weight and pre-term birth more common in teenage mothers (Ellison 2001).

Girls who commence ovulation early (under 12 years of age) show more consistent and frequent ovulation across the lifespan (Ellison 2001), although they may run a higher risk of reproductive cancers (Milne et al. 2011). Girls whose menarche comes later (after 13 years) show less frequent and consistent ovulation, potentially consistent with a hypothetical strategy aimed at maintaining long-term energy balance in a slow growth environment in order to avoid maternal decline (Ellison 2001). In an Australian twin study, the median age of menopause was found to be earlier in girls with late-onset menarche thereby reducing overall fecundity (Kim-Anh et al. 1998). However, a comparative study of 26 countries shows that lifetime fertility was the strongest determinant of menopause, with women of higher fertility reaching menopause earlier (Thomas et al. 2001). This relationship which is possibly related to age at first birth rather than fertility *per se* (Thomas et al. 2001). Although, these findings also support a pattern in which earlier menarche leads to earlier first birth and greater fecundity and fertility.

Both ovulation and menarche appear highly sensitive to energy balance and adipose fat stores. Several studies in women who undertake intensive exercise or physical labour show that patterns of hormones responsible for ovulation (e.g. follicle stimulating hormone) respond to the energetic demands being made of the women's bodies, with greater physical demands decreasing the frequency and regularity of ovulation (Ellison 2001). Common examples of this effect include anovulatory cycles or amenorrhoea in female athletes, professional dancers or in women enduring starvation, although there is substantial individual variation in this response. Similarly, a comparative study of age of menarche in 67 countries showed that girls in countries with better nutrition had an earlier age of menarche, indicating a relationship between energy balance and fecundity (Thomas et al. 2001). From an evolutionary perspective, this suggests that maternal nutritional status (as determined by energy balance) is a reliable biological indicator of the ability of the mother to sustain gestation and lactation in her current environment.

However, other (non-nutritional) forms of stress also play a role in governing ovulation, with increased cortisol levels related to reduced ovarian function (Ellison 2001). This appears to function similarly to nutrition as an indicator of the suitability of the external environment for reproduction.  Further stress as early as *in utero* can affect the onset on menstruation, with stress acting to accelerate maturation and create a front loading of developmental effort (Behie and O'Donnell 2015).

Men also demonstrate some environmental sensitivity, such as deleterious effects of psychosocial stress on sperm motility, an important factor for conception (Fukuda et al. 1998). However, male physiology appears to be less responsive, likely due to the absence of the demands of gestation and lactation following conception. The female reproductive system is under greater selective pressure because women bear most of the physical risks and costs of reproduction. The complexities of conception are a further illustration of this selectivity.

An important aspect of human conception is the rate at which conceptions are lost through failed implantation or where the endometrium is not sustained. Estimates of early foetal loss vary widely, largely due to the difficulty of studying very it empirically (especially given that some loss occurs before fertilisation can be detected medically).

In an American study,  among women who tested positive three consecutive times for Human Chorionic Gonadotropin (HCG), around a third of those pregnancies did not result in a live birth. Of those pregnancies that failed, around 60% failed before the pregnancy was clinically recognisable (Ellison 2001). In an Australian study 78% of women who tested positive for Early Pregnancy Factor did not go on to have a clinically recognisable pregnancy. Studies from less developed countries are rare, but one conducted in rural Bangladesh found that 34% of women who tested positive for HCG experienced early foetal loss (Ellison 2001). Ellison (2001) also gives a very broad likely range of between 30–90% of conceptions being lost before they are clinical recognisable, while Forbes (2005) suggests rates of between 50–96%. Despite the wide variation in rates of loss, even the lowest estimates are considerable, particularly when combined with the more widely agreed estimates that between 15–25% of recognised pregnancies will end in miscarriage (Ellison 2001).

Studies of abortuses lost very early in pregnancy show high rates of genetic abnormality (Ellison 2001). Similarly, studies of later spontaneous abortions (at a median of 10 weeks) show detectable genetic abnormalities in 39% of cases (Del Fabro et al. 2011).

Attribution of over a third of losses to genetic causes indicates that genetic abnormality plays a substantial but incomplete role in early foetal loss. Congenital abnormality also explains the higher rates of loss in plural conceptions as abnormalities are more common in multiple pregnancies than among singletons (Pharoah et al. 2009). Such genetic abnormalities are also implicated where some of the multiple conceptuses are lost but one or more siblings are carried to term and born live (Pharoah et al. 2009). The loss of genetically abnormal conceptions appears to be selective, in that it acts to remove conceptions that have a low chance of reaching maturity. However, because genetic abnormalities do not account for all losses, the actions of chance and misadventure are also important.

Human offspring require substantial and long-term parental (and principally maternal) care and place heavy demands on the mother during gestation. This is due to the demands of encephalisation in human infants which appear to require that inter-birth intervals are sufficient to allow some maternal recovery. Potentially, one of the functions of the difficulty of human conception and environmental sensitivity of ovulation is to protect against frequent pregnancy which might push mothers into an energetic crisis.

### 2.2.2. **Gestation**

During gestation human mothers bear a very substantial metabolic load (Dunsworth et al. 2012). The unusual structure of the human placenta gives the foetus significant access to the maternal bloodstream. Due to the structure of the placenta, the foetal tissue is separated from maternal blood only by a single layer of cells. Consequently, the foetus not only has extensive access to maternal nutrition but is also able to release into the maternal blood stream hormonal triggers that increase the amount of circulating nutrients further  (Ellison 2001). In practice, this means that the mother has less control over the amount of nutrition and oxygen that the foetus uses. For example, a human mother's maximal glucose transfer is ten times higher than that of gestating sheep (Ellison 2001). Accordingly, some researchers describe the metabolic state of pregnant women as one of accelerated starvation (Ellison 2001) as the body responds to meet the increased nutrient and calorie demands of gestation (Picciano 2003).

In early pregnancy, the maternal basal metabolic rate drops, allowing mothers to accumulate greater fat stores even where calorie intake remains stable. While this buffers the foetus against future nutritional stress, it has short-term costs for the mother as energy is diverted away from other functions, such as immune response (Ellison 2001). In later pregnancy, the mother's basal metabolic rate increases: blood volume (and hence pressure) lifts to support placental blood flow, respiratory rate increases to meet foetal oxygen demands and circulating levels of maternal nutrients increase to meet foetal nutritional demands. These changes push the female body into a difficult balance between satisfying foetal demands while not overwhelming maternal metabolism or endocrine system (Dunsworth et al. 2012; Ellison 2001). Gestational diabetes mellitus and preeclampsia are relatively common examples of the failure of this balance.

Unlike many other mammals, humans cannot reabsorb pregnancy except in very early gestation. Once a foetus has survived the initial process of selection (i.e. the first weeks of gestation) selective pressure steadily declines as maternal investment increases

(Ellison 2001). With each passing day of gestation, the likelihood of spontaneous miscarriage falls and female investment increases. Thus, it appears that the greater an investment the mother has made into gestation, the more likely that child's survival becomes.

However, although pregnancies cannot be reabsorbed, potentially the degree of maternal investment can be lowered by spontaneously aborting the foetus, by limiting foetal nutrition (resulting in lowered birth weight) or by shortening gestation (Dunsworth et al. 2012). The more progressed the pregnancy at the point where investment is to be lowered, the more likely that the latter two strategies will be adopted, due to the "sunk costs" of early gestation. If a woman is physiologically stressed during her pregnancy, particularly after the first trimester, carrying the pregnancy to a viable age represents a least cost return on the metabolic energy already invested. As a genetically unique entity with differing needs from its mother (Coall and Chisholm 2003), the foetus also has an increasing ability to act hormonally to decrease the likelihood of abortion (Ellison 2001). Because of the unusually close access to the maternal bloodstream that human foetuses experience, foetal hormones can be delivered efficiently into the maternal bloodstream and can act to decrease the likelihood of abortion (Ellison 2001).

When the mother experiences stress, there exists a complex interaction between the demands of the stress response and the demands of gestation, as both create substantial metabolic strain.

### 2.2.3. **Parturition**

Human parturition has been a particular area of focus for theorists because it is more difficult and dangerous for humans than for our near living relatives. Unlike humans, orangutans (*Pongo*), gorillas (*Gorilla*) and chimpanzees (*Pan*) have birth canals which are much larger than the skulls of their neonates (Rosenberg and Trevathan 2002). Due to the human maternal pelvic inlet and the neonate's skull being close to the same size, humans experience significant difficulty in birthing, which presents a substantial danger to both foetal and maternal survival. Around 287,000 women per year die in childbirth, with a further 10 million estimated to be injured or infected (United Nations Maternal Mortality Estimation Inter-agency Group 2013). The risks of parturition relate chiefly to a risk of incompatibility between maternal pelvic breadth and neonate head circumference (cephalopelvic disproportion) (Flinn 2010).

Traditionally, the difficulties of human parturition have been conceived as following from an evolutionary trade-off between habitual bipedalism and human brain size. Known as the obstetric dilemma (OD), this theory proposes that birth timing results from a trade-off between neural and skeletal maturity of the foetus (skull size) and pelvic breadth of the mother. If the gestation proceeds beyond the point at which the foetus can move safely through the pelvic opening, then the risks to both mother and infant become extreme. This trade-off is resolved by birthing a smaller, and hence relatively underdeveloped, neonate. The OD has been the principal accepted explanation for human altriciality (Dunsworth et al. 2012). Although earlier birth may ease the risks to the mother, it results in significant declines in neonate survival (Dodds et al. 2001).

However, Dunsworth et al. (2012) and others including Kurki and Decrausaz (2016) have re-examined the OD and particularly the assumption that human pelvic width is constrained by bipedal locomotion. Dunsworth et al. (2012) find that the engineering problem of efficient wide-hipped bipedal movement is overcome, possibly through the action of the hip abductor muscles (the M. gluteus medius and minimus). Further, Kurki and Decrausaz (2016) indicate that the variation found in the shape of the human pelvis is suggestive of a complex interplay between selective forces and other factors, such as genetic drift, rather than the simple relationship between obstetric sufficiency and bipedalism. Studies which compare the running economy of male and female runners, women and men are equally metabolically efficient, thus potentially decoupling the relationship between pelvic width and bipedal efficiency (Daniels and Daniels 1992).

If pelvic width is not constrained by bipedalism, then another factor must influence timing of birth. Dunsworth et al. (2012) propose the energetics of gestation and growth (EGG) hypothesis which suggests that birth occurs when the mother's metabolic load reaches an energetic crisis. Thus, birth occurs as a result of the mother's metabolic constraints rather than her skeletal ones. Although lactating mothers bear an increased nutritional load after birth, the respiratory and glucose demands of the foetus are relieved. This accords with observations that infants carried post-term can begin to lose weight as their nutritional demands exceed the mother's capacity (Ellison 2001).

Relationships between maternal and foetal nutrition are commonly observed. Women with large amounts of available energy, for instance those that are obese or have gestational diabetes (resulting in greater circulating glucose), carry their foetuses for longer and have larger infants (Hillier et al. 2008). It appears that these women's enhanced capacity to provide for their foetus allows them to carry for longer. This excessive gestation, however, has its own risks, particularly during parturition. Conversely, famines result in smaller infants and anorexic mothers also tend to give birth to smaller and premature infants (Lummaa and Clutton-Brock 2002; Yarde et al. 2013). Because obesity and gestational diabetes would have been largely unknown in the EEA, potentially those conditions now act to compromise the usual method of triggering parturition, resulting in increased risk and greater need for medical intervention in modern environments.

The EGG hypothesis also supports the observed relationship between birth timing and multiple types of maternal stress. The stress response is energetically costly: the action of the HPA and the Sympathetic Adrenal Medullary result in both oxygen and glucose consumption increasing (covered in detail in Chapter Three). Potentially, this cost, when combined with the energetic demands of a foetus, may cause the EGG trigger point to be reached earlier than would otherwise be the case, particularly in the third trimester when foetal weight gain is greatest. Furthermore, because stress increases the release of glucose into the maternal blood stream, this increases the availability of nutrition to the foetus, potentially propelling it faster toward the maternal metabolic threshold. The availability of oxygen may be a particular constraint. Although food may be restricted in the aftermath of some disasters, oxygen availability (and therefore also blood pressure and volume) is a routinely fixed constraint. The role of the stress response in consuming oxygen and elevating blood pressure may therefore be an explanatory factor in the relationship between stress hormones and birth timing.

Because both pregnancy and stress raise maternal blood pressure, this may potentially create an extreme strain on the maternal circulatory system.

The EGG hypothesis also accommodates the thrifty phenotype hypothesis. This states that foetuses can make metabolic adjustments *in utero* in response to external environmental cues that allow it to be more metabolically thrifty (Hales and Barker 2001). In reference to Trivers's (1974) work on child-parent conflict, the needs of the foetus are, in this case, contrary to that of the mother. Under the EGG hypothesis, the mother needs to birth the child before the metabolic demands of gestation exceed her capacity; however, the foetus will benefit from longer gestation until the point where the safety of birth is further compromised or it begins to starve. In other words, it is not in the interests of the foetus to be born substantially early although this would reduce the costs to the mother. Therefore, the thrifty phenotype may assist the foetus in remaining below the maternal metabolic threshold when adverse conditions make it possible that this threshold will be reached abnormally early. The thrifty phenotype may therefore function to increase survival not only post-partum but also *in utero* by reducing metabolic demand and thus avoiding the EGG trigger for longer. The impacts of disaster stress may predict the emergence of the thrifty phenotype in foetuses, as their placental communication with the mother tells them that they are likely to enter a tough environment. The emergence of this phenotype may also act to protect foetuses *in utero,* as well as postnatally.

Dunsworth et al. (2012) base their conclusions on the parity of metabolic efficiency between males and females (and thus between narrower and wider pelves). However, there is dispute about the degree to which pelvic breadth affects movement efficiency.

Daniels and Daniels (1992) find that, although male and female runners are equally efficient below maximum speed, male runners are more efficient at maximum speed. If metabolic efficiency is equal at normal speeds, movement efficiency may have played a lesser role in constraining hip breadth. The EGG does not, on its own, appear to explain why human parturition is so dangerous. If the mother's metabolic capacity is the restrictive force on gestation, why are female hips not even broader to allow safer delivery? It is possible that there are constraints on hip width that are related to bipedalism but which only emerge at the extremes of phenotypic variation and high speed movement. While the EGG might control birth timing, other selective forces must constrain the ability of the pelvis to broaden. The timing of birth is therefore likely related to both the OD and the EEG hypotheses, as they do not necessarily contradict each other.

Following birth, human children continue to require substantial nutritional investment. Under normal circumstances, the calorie requirements of lactation exceeds those of gestation (Picciano 2003). However, there appear to be two ways in which the energetic requirements of lactation can be better managed by a physiologically stressed mother than those of gestation. Firstly, lactation removes the burden on mothers' oxygen consumption and blood pressure. Secondly, lactation allows the infant to consume fats directly. This is rather than requiring fat to be metabolised into glucose in order to cross the placental barrier, and later to supplement intake with other sources of nutrition (Ellison 2001). Mothers lactating with insufficient calorie intake are also able to lower their basal metabolic rates in order to divert calories from their bodily functioning and into lactation (Ellison 2001). This lowering of metabolic rate could not occur in pregnancy beyond the first trimester without compromising placental blood supply.

### 4.1.1. Onset of labour

In terms of mechanics, labour is triggered by hormones produced by both the foetus and the mother. Unlike other mammals, humans do not experience a rapid spike in hormones preceding birth, but rather a gradual increase over the final trimester. For humans, there appears to be no exactly correct time to be born, but rather a long window of opportunity which is sensitive to internal and external circumstances. It is also unclear whether there is an absolute maximum gestational length, with gestations of up to 12 months having been recorded (in a case where foetal brain development was severely retarded, and hence energetic demands were low) (Ellison 2001).

Stress hormones, principally cortisol, are included in the suite of hormones that rise over the third trimester, are correlated with the onset of labour, and are important in preparing the foetus for birth (for instance by maturing the lungs). While there is no known way for the foetus to trigger labour alone, it can act against the mother's hormonal response by releasing foetal hormones that act to prolong gestation (Ellison 2001). Both maternal and foetal cortisol are implicated in the timing of labour (Inder et al. 2001; Sandman et al. 2003; Wadhwa et al. 1998).

Ellison (2001) suggests that the increases in foetal cortisol observed prior to the onset of labour may be due to the foetuses' increased stress as it approaches the maternal metabolic threshold and becomes increasingly unable to meet its own need for glucose and oxygen.

However, maternal hormones seem most responsive to external pressures. In addition to altering foetal neuro- and organo-genesis (discussed in more detail below), rising maternal cortisol is an important trigger for birth (Catalano and Bruckner 2006; Inder et al. 2001; Wadhwa et al. 1998). Inder et al. (2001) argue that cortisol functions as a "placental clock" in which rising cortisol levels in the third trimester trigger the commencement of parturition. Accordingly, they find that elevated corticotrophin-releasing hormone (CRH) is a moderate predictor of preterm birth, accounting for about 50 % of preterm deliveries. Similarly, Wadhwa et al. (1998) find that elevated CRH levels during weeks 28-30 of gestation predicted reduced gestational length, which has been replicated in later studies (Graignic-Philippe et al. 2014). Studies of births following environmental disasters also show this effect (Glynn et al. 2001). High CRH levels during months four and five of gestation are also significantly higher in women who go on to experience preterm labour (Graignic-Philippe et al. 2014). Importantly, spikes in maternal cortisol trigger increased placental CRH production in the third trimester (Torche and Kleinhaus 2012). However, while studies suggest that higher maternal stress at the end of pregnancy may relate to greater difficulties during birth (including the duration of labour and use of anaesthesia), it is stressful events earlier in gestation which appear to play a greater role in determining the timing of parturition, potentially laying out a timetable for the pregnancy from very early on (Graignic-Philippe et al. 2014).

Adrenaline release can delay the onset of labour. Additionally, adrenaline levels during labour correlate with a slower delivery (possibly by reducing the supply and efficacy of oxytocin) (Wadhwa et al. 1998). O'Donnell and Behie (2013) find that there was an increase in post-term (born at greater than 41 weeks) infants born to mothers who lived in parts of Victoria that were burnt during the 2009 Black Saturday fires. Patterns of post-term delivery are further found after earthquakes and terrorist attacks (Margerison-Zilko et al. 2015; Oyarzo et al. 2012). It is possible that the experience of bushfire and presumed increase in maternal adrenaline production may have delayed the onset of labour.

### 2.3. *Reproductive strategy*

Reproductive strategy governs when, with whom, and how often, an organism should reproduce in order to maximise its inclusive fitness. Human reproductive strategy reflects both the allocation of resources over the lifetime and the metabolic intensity of human reproduction. Sexual selection and mating strategies, although a large and important area of research and debate, fall outside the scope of this dissertation. Accordingly, this chapter focuses on elements of reproductive strategy relevant to sexual maturation, gestation and parturition.

Under the theory of natural selection, individuals are expected to seek those reproductive strategies that confer the greatest chance of passing on their genes at the least cost. Such strategies will be informed by that individual's health, social status and environment. At a species level, general consistency in reproductive strategy allows a species to be characterised as exhibiting a particular type of strategy (Gangestad and Simpson 2000). Reproductive strategies can be broadly defined as lying on a spectrum between *r*-selection, which makes maximum investment in mating and minimum investment in parenting, and *K*-selection, which maximises parenting effort. However, defining reproductive strategy as simply either *r*- or *K*-selected is now less common, as it underplays the complexity of the bet-hedging strategies employed by species in response to future uncertainty (Chisholm 1999).

Nevertheless, organisms can only respond (in an unconscious biological sense) to environmental pressure within the biological constraints of their underlying *r*- or *K*-selection. The energetic demands of encephalisation tie humans to a *K*-strategy. Despite a limited degree of inter-population variation (e.g. average family size varies between societies), as a species humans are very clearly placed at the *K* end of the spectrum (Bogaert and Rushton 1989).

Nevertheless, humans do show variability in the specific reproductive strategies that they employ. Differences in reproductive strategy reflect differences in the individual's internal, external, psychological and physiological environments. Figueredo et al.(2005) relates the variation that exists in humans to differences in personality; however, environmental stability, resource availability and extrinsic mortality rate are stronger contenders to explain variation in reproductive strategy and may, in themselves, also be influential on personality. Human reproductive strategies are thus both sophisticated and plastic (Chisholm 1999).

### 2.4. *Application of LHT trade-offs*

The principal strategic reproductive trade-offs proposed by LHT are those between offspring quality and quantity (which involves the trade-off between mating and parenting efforts) and between future and present reproduction (Lickliter and Honeycutt 2003). It is a fundamental assumption of LHT that organisms' resources are constrained. Because of these constraints, there must be an ongoing balancing of the organism's needs against resource constraints and all trade-offs will result in both losses and gains (Kuzawa 2005). For instance, delaying reproduction may allow the growth of a larger, stronger maternal body (and the fitness benefits that confers) but will delay reproduction resulting in fewer offspring (a fitness cost) (Kuzawa 2005).

### 2.4.1. Quality: quantity and mating: parenting trade-offs

In humans, every child requires substantial parental investment of resources to reach maturity, but the nature and extent of this investment can vary widely above and beyond the minimum needed for survival. Such variation demonstrates behavioural plasticity (Bribiescas and Muehlenbein 2010). In terms of the number of children born, human parents, like all animals, face the need to seek a balance between the quantity of children they have and the quality of those children. However, unlike other animals, humans have the capacity to make conscious decisions about reproduction, even if this does not always occur (Coall et al. 2016).

LHT proposes that organisms will seek to strike the best possible balance between quality and quantity that the environment will allow (Rogers 1989; Vining 2011). Higher quality children require greater investment; where a greater number is sought, a reduction in parental investment in each must result. Even though humans reproduce within a complex social and technological context, the fundamental constraints on resources, and therefore the need for trade-offs, remain.

For any human parent, the share of resources available for each child must necessarily diminish as the total number of children increases (at least in early childhood, before children themselves can make a contribution to the attainment of resources), although the resource share per child may remain adequate for good child wellbeing. However, in environments where people do not have sufficient access to resources, division of resources between children can create inequities in survival rates (Irudaya Rajan et al. 1992). Such circumstances are not usual in the population examined in this dissertation. In more prosperous environments, sharing of resources between siblings is unlikely to affect survival. Nevertheless, parents in prosperous environments still need to allocate resources between their children, particularly parental time, which is necessarily under fixed constraints.

There are important environmental reasons why human reproductive strategy, despite favouring high parental investment to produce high-quality children, might need to increase quantity (and thus lower quality) in some circumstances (Coall et al. 2016). In environments where there are high mortality risks, LHT predicts that parents will have a greater number of children to compensate for potential losses (Figueredo et al. 2005). LHT theorises that higher quality offspring can be expected to have a greater chance of successfully reproducing, but are a risky proposition in an environment which poses a high risk of non-selective mortality to those offspring. In risky environments, higher fecundity is a hedging strategy aimed at increasing the chances that some offspring will survive and reproduce (Pasztor et al. 2000). Although this mechanism might appear counter-intuitive, in truly dangerous environments it is unlikely that parents will have the ability to protect even very high quality offspring, therefore heavy investment in few offspring does not offer good chances of offspring survival (Coall and Chisholm 2003; Coall et al. 2016).

The quality-quantity trade-off also reflects the conflicts proposed by Trivers (1974) between the needs of the parent, particularly mothers, and the needs of the child. While it is beneficial for the child to receive maximal investment, the parents must balance the needs of the child with the demands of their own health (particularly maternal health), the demands of future children and the need for future reproduction. As Coall and Chisholm (2003 p.1776) succinctly state, the foetus has a "100% genetic interest in itself" but no more than a 50% interest in its parents or future siblings.

Levels of maternal investment can also vary during gestation, with shorter gestations indicative of lowered maternal investment. For example, mothers who are stressed show shorter gestations, which has been connected to the role of stress hormones in triggering birth (Inder et al. 2001). This connection between stress and parturition may arise from a reduction in maternal investment in the foetus due to unfavourable environmental conditions, communicated via the stress response. Because unassisted onset of labour is not reflective of conscious decisions, it is an indicator of reproductive strategy not obscured by culture or technology (Dunsworth et al. 2012).

As shortened gestation indicates lowered investment, spontaneous abortion demonstrates a withdrawal of maternal investment. While spontaneous abortion is often associated with genetic abnormalities (as discussed earlier in this chapter) such failures of fitness do not account for all pregnancy loss. Further, spontaneous abortion becomes more common where women are stressed (Lazinski et al. 2008; Quinlan and Quinlan 2007). The high levels of genetic abnormalities in lost foetuses and the relationship between maternal stress and pregnancy loss suggests that such pregnancy losses may have adaptive features, rather than being exclusively the result of non-selective failures or pathologies. Rates of foetal loss range from 30-90% in humans. This indicates that selective pressure is very strong in early life and that this pressure works effectively to help protect the mother from unwise maternal investment (Ellison 2001).

Foetuses may be able to take protective measures against reductions in maternal investment. For example, by developing a more efficient metabolism, foetuses can reduce their maternal cost and thus increase their chances of survival (Pike and Milligan 2010). This thrifty development also potentially delays triggering parturition in accordance with the EGG hypothesis (Dunsworth et al. 2012), as well as speeding foetal physical maturity and thereby increasing post-partum survival. Reduced birth weight is an outcome of this strategy. Alternatively, foetuses may divert their diminishing resources to greater placental development in order to bolster their access to resources over the pregnancy, resulting in a higher ratio of placental to foetal weight (Coall and Chisholm 2003; Haig 1993).

### 2.4.2. **Future and present trade-offs**

Because reproduction is resource-intensive, reproducing now may limit the capacity of an organism to reproduce later. This is particularly, and more crucially, applied to females due to their high metabolic investment. But there are also, albeit much lesser, energetic costs for males. Although the demands of gestation are significant, so too are the demands of lactation; therefore, maternal investment continues to increase following birth, meaning that the needs of current young children continue to compromise the possibility of future children (Pike and Milligan 2010).

Any present reproduction may create costs by reducing the resources available for future reproduction. In a risky environment, this means that present reproduction reduces future reproduction, even if conditions become more favourable  (Coall et al. 2016). However, delaying reproduction is also risky as the organism may die before it is able to reproduce. Thus mortality risk emerges as a key influence on reproductive decisions. Where mortality risk is (unconsciously) perceived to be high (through either current or early life stress), the future is heavily discounted and reproduction is likely to occur earlier (Coall et al. 2016). Where mortality risk is perceived to be low, later reproduction is favoured which should allow for greater investment in each offspring under the quality-quantity trade-off.

An uncertain environment in which the mother assesses that her own survival is threatened is likely to favour earlier reproduction and higher fecundity. For example, women raised in psychologically stressful environments demonstrate a front-loading of maturation, including early menarche, which increases their fertility. These effects also appear when female foetuses are stressed *in utero* by exposure to maternal cigarette smoking (Behie and O'Donnell 2015).

Behaviour indicative of sexual strategy also alters. Girls and women exposed to stress in childhood also demonstrating younger age at first birth, a shorter inter-birth interval and a greater number of sexual partners even where social conditions are accounted for (Chisholm et al. 2005). While younger age at first intercourse and first birth, a shorter inter-birth interval and a greater number of sexual partners are reflective of social factors, such as vulnerability and disordered attachment, menarche may act as a reliable indicator of a biological response to a stressful environment that acts to increase lifetime fertility. However, where foetuses experience nutritional stress *in utero,* the same effects have not been found (Yarde et al. 2013), suggesting that the absence of sufficient calories during gestation may predict different outcomes.

Rapid maturation may also incur costs that further favour early reproduction. Some evidence suggests that early menarche exposes women to increased likelihood of reproductive cancers (Chisholm et al. 2005; Milne and Judge 2009; Milne et al. 2011). Such costs associated with early maturation strengthen the importance of earlier birth and shift future-present trade-off decisions in favour of earlier reproduction.

Even in modern human populations, lifespan expectations (i.e. the length of time that the individual expects to live) influences conscious reproductive intent. In an individual-level experiment, participants underwent mortality priming (using an internet-based program to increase their perception of their own mortality) and then answered questions about the number of children that they wished to have. Males who had received mortality priming nominated higher numbers of offspring than controls. Yet the response was not present in females, likely due to their differential resource-investment concerns (Sear and Mathews 2008).

In environments where resources are scarce, however, delayed reproduction may confer greater benefits. Conception is sensitive to energetic and hormonal balances, meaning that environments which restrict calorie intake should also impede fecundity. Not only does energetic flux lower ovarian function, increases in circulating cortisol can also down regulate progesterone and ovulation (Ellison 2001). This means that stressed women are less likely to conceive and, as noted earlier, may also be less likely to bring a baby to term. That outcome, although deeply distressing, acts as a biological means of delaying reproductive investment until a more favourable time when return on reproductive investment can be maximised.

### 2.4.3. **Sex-selective trade-offs**

Sex ratio (the ratio of males to females in a population) can be measured at several points in the lifespan: the primary sex ratio (PSR) is measured at conception; the secondary sex ratio (SSR) is measured at birth; and the adult sex ratio (ASR) is measured in adulthood. The operating sex ratio (OSR) measures the ratio of sexually active adults in a given population (Del Giudice 2011).

The causes of changing PSR and SSR remain under investigation, however, there are strong theoretical evolutionary factors favouring one sex over another under certain environmental conditions (Catalano and Bruckner 2006). Although the ratio persistently remains close to 1:1, even small diversions might indicate the mechanism of selective pressures during conception and gestation. In most human populations, males slightly exceed females in the SSR. This section examines briefly some of the principal ideas related to biological and evolutionary constraints on the sex ratio: Fisherian selection, theories of male frailty and the Trivers-Willard theory. A more comprehensive discussion of the effects of stress and particularly early pregnancy loss on sex ratio is presented in Chapter Six. This discussion considers biological causes of sex ratio disruption, rather than behavioural causes, such as sex selective abortion or differential child rearing practices.

R.A. Fisher, writing in 1930, proposed that women have a pre-set genetically encoded ratio for their offspring and that women unconsciously maintain this ratio by either allowing biased access of the sperm to the ovum (e.g. through timing of intercourse and cryptic female choice) or through miscarrying foetuses of the non-preferred sex (Argasinski 2012; Mittwoch 2000; Wedekind and Evanno 2010). The preference for one sex over another is informed by the existing sex ratio of the mother's social group, with the least represented sex being favoured. This came to be known as the Fisherian principle. The least represented sex has the greatest chance of successful reproduction because the ratio imbalance acts to increase the less represented sex's access to breeding opportunities (Argasinski 2012).

Some studies lend partial support to the suggestion that women exhibit the capacity to balance the sex ratio of their offspring. Dyson (2012), for example, notes that the chance of conceiving a male is increased by earlier female children, and vice versa. These changes might equally reflect the greater metabolic load of male offspring and a reflexive preference for subsequent female children. Carl Dusing, in the 19th century, argued that the number of grandchildren achieved could be used as a measure of a female's genetically encoded sex ratio (Argasinski 2012).

However, whether the ratio alters at conception or later in gestation remains uncertain (at least in part due to methodological challenges). As such, understanding the roles of cryptic female choice, sperm motility and foetal loss remains incomplete. Further, such selection is likely to respond to a range of environmental pressures that affects maternal condition, rather than just the sex of previous children. Consequently, there exists a likely role for effects of stressors on sex selection.

One common evolutionary explanation for this is that males face greater survival risks during gestation and early life and consequently have lowered survival rates at sexual maturity. This effect then requires a compensatory excess of males at birth to deliver a balanced ASR (Catalano and Bruckner 2005; Catalano and Bruckner 2006; Catalano et al. 2005).

However, the causes of higher early life mortality in males remain an area of debate. Some propose that the Y-chromosome borne by male conceptions creates inherent genetic weakness. However, there is little evidence that the presence of the Y-chromosome itself is relevant to higher rates of loss among male foetuses. Neither Bellver et al. (2010) nor Venkatesh et al. (2011) find relationships between rates of recurrent spontaneous abortion and Y-chromosome microdeletions. However, the absence of a second X-chromosome does appear relevant, since damage to the X-chromosome cannot be compensated for by its pair (Howerton et al. 2013; Martinez et al. 2008).

Nevertheless, the Y-chromosome and its role in testosterone production may play a role in increased childhood mortality. Male children can exhibit greater risk-taking behaviours, which increase the risk of childhood mortality. Although also enculturated, there is potentially an underlying role for testosterone and other androgens in driving a propensity for physical risk-taking and higher levels of aggression (Gray 2010). The role of chromosomal differences is discussed further in Chapter Six. Because of the increased risks of disease and accidental death, carrying male children may not act to maximise the reproductive success of mothers, particularly where dangerous or unpredictable environments heighten the risk of illness or accidental death.

Secondly, female children may also be selected for under unpredictable environmental conditions. As with the quality-quantity and future-present trade-offs, sex selection supports Trivers' (1974) theory of parent-offspring conflict. Daughters place lesser metabolic demands on the mother due to their smaller average body size and reduced postnatal growth rates. In times of low resource availability, this lesser demand may lead to a greater likelihood of female foetuses being sustained throughout gestation (Gray 2010; Helle et al. 2009; Martin and Festa-Bianchet 2011). Accordingly, the more metabolically costly production of sons is selected against (Hardy 2002; James 2000; Rickard 2008). Subsequent offspring are slightly smaller than their predecessor (on average) with this effect stronger in offspring which follow a male child, reflecting the strain on the mother (Rickard 2008; Venkatesh et al. 2011). For example, Rickard (2008) finds a decrease in birth weight of 9% where siblings follow sons. There is an inverse relationship between offspring numbers and sex ratio suggesting that mothers do not quickly recover from this increased load (Jacobsen et al. 1999). Older mothers (over 35 years of age) and very young mothers are also more likely to bear girls, suggesting a trade-off between maternal condition and offspring sex (Dickson and Parker 1997; Windham 2001).

Trivers and Willard (1973) also argue that weak sons produce fewer offspring than weak daughters. Notably, work in humans shows that women of a high social status have a greater number of sons (Cameron and Dalerum 2009; Grant and Yang 2003), although other studies find no such effect (Keller et al. 2001). More recent work has extended this theory to focus on the conflicts that occur between mothers and foetuses during conception and gestation (Bribiescas and Muehlenbein 2010). Consistent with this work, female foetuses who require fewer resources have a higher chance of survival, while greater reproductive potential should offer the mother the best available investment where future reproduction is compromised by stochasticity.

## 2.5. *Limitations and challenges*

As noted earlier, humans display wide variations in their reproductive behaviour, a characteristic which makes applying LHT to human behaviour especially complex. One of the principal assumptions of LHT is that, in general, individuals seek to maximise their fitness and, hence, the degree to which their genes progress into the next generation. Theories of inclusive fitness suggest that individuals without offspring may instead advance their genes by contributing to the care of near relatives' offspring (Hamilton 1964a; Hamilton 1964b). However, human populations often show far more variability than a simple application of these theoretical models would allow.

Any assessment of reproductive strategy in humans is complicated by the cultural and technical confounds that exist in human societies. In comparison with other primates, human reproduction is less opportunistic and more guided by restrictive social rules. Social rules vary between cultures but can include: the maintenance of sex as a private act; specific rules guiding when marriage can occur; rules guiding the practice of polyamory, polyandry and polygyny; dowry competition; and demi-parental care for siblings (Quinlan and Quinlan 2007; Schuiling 2003).

Although rules can vary widely between cultures, most appear intended to control who reproduces and when. Sanctions for violating such rules can be severe. Although social rules are understood to guide reproduction in other primates, this level of scope and variety in social behavioural constraint appears to be common only in humans

(Schuiling 2003). Consequently, cultural and social influences strongly influence observable reproductive strategy in humans, meaning that any analysis of human reproductive strategies is subject to a significant range of social confounds that act to obscure underlying biological drivers. This dissertation aims to mitigate this challenge by including measures that are largely beyond social control, such as birth weights, gestational age and (in Australia, where selective abortion is illegal) SSR.

Technological ability further complicates human reproductive strategies. Unlike other species, humans have more recently been able to control their reproduction without limiting intercourse (through the use of deliberate timing, as well as contraceptives, pharmaceutical abortifacients and invasive terminations where these technologies are accessible). Humans can also, albeit to a more limited extent, assist reproduction though assistive reproductive technologies in situations where an individual's biology renders them infertile or limits their fecundity, although access to these technologies is limited and success variable. Similarly, medical technology has very substantially improved infant and mother survival rates during gestation and immediately following parturition. However, because both need for and access to reproductive technologies remains limited (Li et al. 2013), it is unlikely that their influence is able to overcome naturally arising changes in reproductive outcomes when large populations are studied, such as in the research presented in this dissertation.

## 2.6. *Chapter summary*

This chapter argues that LHT is a useful theoretical base for understanding changes to fertility, birth outcomes and developmental changes following environmental disasters.

Because of the interactions between the substantial metabolic load of reproduction and maturation, exposure to additional stresses causes problematic rises in allostatic load (which are expanded upon in Chapter Three). The organism must respond to this pressure by making alterations in its life history.

A small but growing body of empirical evidence supports this theoretical assertion, which are presented in the following chapters. Chapter Three examines how humans understand their environment in order to inform their reproductive strategy. It builds on the theoretical base of LHT outlined in this chapter and then incorporates the very recent research on the effects of early life stress and allostatic load in programming growth and development. This programming is increasingly linked in the literature to a greater propensity to later life disease.

# 3. EARLY LIFE PROGRAMMING

The application of LHT to human reproduction requires the existence of biological mechanisms to convey the quality of the external environment to the body and, in terms of *in utero* effects, through the body of the mother to the foetus. As noted in the previous chapter, stress appears to be a likely mechanism by which the external environment can be communicated biologically to the mother and foetus. Stress is an elusive and often poorly defined concept. Within this dissertation, stress refers to the stress response, that is, a psychological and physiological process that arises as a result of exposure to a stressor. Although the stressor (or stressors) that trigger the stress response may be either psychological (e.g. emotional trauma) or physiological (e.g. starvation), the resulting stress response is almost inevitably both psychological and physiological.

One function of the stress response appears to be communication of environmental quality. In early life, be it *in utero* or in early childhood, these environmental assessments help tailor maturation to the receiving environment. Issues related to this process, often termed early life programming, constitute a rapidly growing field of research, particularly in terms of the mechanics of change (principally epigenetics) and the results of change (such as the foetal origins of adult disease).

This chapter discusses how humans understand their environments. It then focuses on the human stress response and its role in communicating environmental quality. The chapter closes by providing an overview of emerging research into epigenetic change and the lifelong effects of environmentally-informed alterations in maturation. Issues related to the measurement of stress and substantial variation in stress response between individuals are examined in Chapter Eight.

## 3.1. *Understanding the environment*

In order to make reproductive trade-offs or to tailor maturation, an organism must first understand its environment. The fundamental problem faced by all organisms in this context is the "uncertain futures problem" – that is, that no organism can be exactly certain of its future environment (Chisholm 1999). Consequently, organisms must allocate their resources according to their best prediction of the future environment and must make these assessments according to the knowledge that they are able to gain about their environment. However, because total resolution of the uncertain future problems is not possible, the best available solutions are adaptive strategies that are based on the organisms' estimation of the future environment. In order to make this estimation, all organisms require an internal model that allows their phenotype and behaviour to shift in response to information about the past, current and potential future environment (Chisholm 1999). Since survival and growth are key points of allocation, organisms need to make predictions about their future environment early in life in order to formulate an ontogenic response.

While humans evidently make conscious and deliberate decisions regarding resource allocation, their biology is also informed by their environment and can accordingly respond to the allocation of biological resources. This biological response can include up or down regulation of metabolic, immunological and reproductive functions, among others physiological functions.

One way in which environmental pressure can be communicated biologically is through the experience of psychological and physiological stress. The experience of stress appears to have an influential role in shaping reproductive strategy, particularly where significant stress is experienced in early life (Behie and O'Donnell 2015; Chisholm et al. 2005; Milne and Judge 2009; Milne et al. 2011). It appears that, through neural and endocrine pathways, the experience of stress is capable of altering development and the timing of life history traits, and thus reproductive strategy (Bribiescas and Muehlenbein 2010).

Environmental disasters provide useful natural experiments regarding the impact of stress at the population level. One of the advantages of studying the responses of populations to environmental disasters is that such natural experiments temporarily reduce the capacity of individuals to control and direct their environment, thereby exposing large groups to consistent and stressful conditions (Fink 2010). Although any population experiencing a disaster will vary at an individual level in terms of resilience and resource availability (Musazzi and Marrocco 2016), there should nevertheless be a high degree of shared experience of stress and deprivation which may result in similar biological responses. Despite the capacity of disasters to deepen pre-existing inequities in the longer-term (Oliver-Smith 1996), in the immediate aftermath of disasters, individuals of different social status can share somewhat similar experiences of disruption and chaos. In recent work, objective and subjective stress measures were found to be largely uncorrelated with each other. Objective stress measures show the strongest association with reproductive outcomes (King and Laplante 2015).

Interactions between perceived and objective stress are discussed further in Chapter Eight.

However, one of the challenges in considering the influence of both environment and environmental disaster on reproductive strategy is the degree to which humans construct and control their environments. In order to understand the development of human life history and reproductive strategy, it is necessary to understand the contexts in which these strategies develop.

The deliberate modification of an organism's environment by that organism to boost survival and reproduction is described as ecological niche construction. Odum (in Hardesty 1972 p. 458) describes a niche as an organism's "profession or way of life … while noting that the habitat is equivalent to its address". While niche construction is by no means a uniquely human behaviour, humans construct extraordinarily complex niches (Hardesty 1972; Laland 1999). Indeed, human niche construction has had such a profound effect on global ecosystems that some authors refer to our current epoch as the Anthropocene (Crutzen 2006). This ability to construct environments is not recent and may have already had profound impacts on the course of human development. For example, Flannery (2006) argues that, while climate stability supported the Neolithic transition, human action through farming and clearing also acted in tandem to prolong the "long summer" and thus increase the benefits of agriculture.

The development of niche-building behaviours, including cultural practice, are also subject to selective pressure thereby creating an ongoing and interdependent relationship between the natural and constructed environments (Gangestad and Simpson 2000). Humanity's capacity for niche construction appears to have dramatically benefited its survival, resulting in large recent population increases (Crutzen 2006). In practice, this means that humanity's recent evolution may owe as much to its constructed environment as to the natural one (Laland and Brown 2006). However, adaptation to niches also confers risks. Because niches remove some selective forces and promote others, they function to alter the frequency of alleles. This can result in "evolutionary inertia and momentum" which can act to fix alleles that might otherwise have been lost, to maintain stable polymorphisms (i.e. alternative phenotypes) contrary to natural selective pressures and to remove polymorphisms that would have been retained in an unconstructed environment (Laland 1999 p.10242; Laland and Brown 2006).

Niche construction provides a useful prism through which to view reproductive outcomes in human populations following environmental disasters and the stochasticity they create. Disasters cause the constructed niche to become physically and socially unpredictable. Although it is rare to have disasters that are sufficient to disrupt niches beyond the point of recovery and repopulation – even the devastating Toba Eruption in South-East Asia during the Quaternary period had a limited long-term effect (Louys 2007) – environmental disasters can disable niches and thus expose traits that are maladaptive outside the niche environment.

Following environmental disasters, where whole populations experience significant and similar stress, it may be possible to observe population-wide changes in life history traits. Because disasters act to temporarily disable protective niches, they allow measurement of biological stress effects which might otherwise not be evident. Thus, by inhibiting human dependency on tailored niches, disasters create a useful method for studying the stress-related reproductive and maturational changes that are predicted by LHT.

### 3.2. *Human stress response*

A principal method through which humans biologically understand their environment is via the experience of stress. Contemporary understanding of stress has its origins in Walter Bradford Cannon's theory of homeostasis and Hans Selye's subsequent General Adaptation Syndrome (GAS) (Ganzel et al. 2010). In these theories, stress is understood as a perceived or actual threat to an organism's homeostasis, caused by an internal or external stressor. Cannon (1915) proposed that, in response to a stressor, animals' bodies prepare to either fight or flee – the "fight or flight" response. Selye later substantially developed the understanding of the fight or flight response and first demonstrated a link between exposure to stressors and an autonomic response in the Hypothalamic-Pituitary-Adrenal (HPA) axis (McEwen 1994). Selye theorises that organisms respond to stressors through the HPA axis and the release of a range of hormones and neurotransmitters. The endocrine system is responsible for many of the physiological changes that occur in a stressed organism, creating a cascade of effects that are triggered by hormone secretion in the brain. This response is metabolically costly to the organism and, when prolonged, ultimately results in its exhaustion and death.

More recently, further work has demonstrated that GAS oversimplifies the stress response and that the implicated neuroendocrine pathways act (at least partially) independently of each other in order to regulate the stress response and avoid critical levels of exhaustion (McEwen 1994; Sapolsky 1998). Additionally, while the stress response is broadly consistent, there is still considerable variation in both the responses of different humans to the same stressor and the responses of individual humans to stressors over time. These variations are sufficient to result in inconsistent long-term responses to stress (McEwen 1994). The load which an ongoing stress response places on the organisms is termed allostatic load and it is the magnitude of this load which is now considered most important in predicting deleterious side effects of the stress response.

Exposure to stressors do indeed stimulate the HPA axis as preparation for an adaptive response that seeks to protect the organism from the stressor by fleeing the stressor (flight), defending against the stressor (fight), placating the stressor (appease) or hiding from the stressor (freeze). This response results in the secretion of a suite of stress hormones, including: corticotrophin-releasing hormone (CRH) (and/or vasopressin), adrenocorticotrophic hormone (ACTH), corticosteroids and adrenaline (Dallman et al. 1994). Only anthropoid primates release CRH. Corticosteroids can act to inhibit the action of the adrenocortical system (through a negative feedback loop with the pituitary gland), thereby regulating the stress response. Although the adrenocortical system can release corticosteroids, this is down-regulated if corticosteroids are already present in the bloodstream. CRH results in an increase in blood cortisol (a steroid hormone) that promotes physical changes including increased blood glucose, accelerated heart rate and peripheral vasoconstriction. These changes prepare the body for exertion and injury (Alder et al. 2007b). Indeed, it is this preparation which allows an effective protective response (Ganzel et al. 2010). Because stress is a neuroendocrine response, once triggered it is decentralized from the brain and is sustained through the endocrine system (Sapolsky 1992). Neural hormones are required to reduce the response and thus return the organism to homeostasis. Corticosteroids act as a feedback mechanism and instruct the endocrine system to cease its response (Koob et al. 1994). Figure 3-1 shows the HPA axis.

[redacted]

Figure 3-1: HPA axis with feedback loop indicated by arrows (Alschuler 2016)

In terms of the influence of stress on life history traits, the relatively stable and consistent nature of the stress response among primates suggests that the stress response has been a durable characteristic (Gray 2010). Anthropoid primates have a common stress response which initiates the neuroendocrine cascade described above as a reaction to a stressor. These stressors can be physiological or psychological as well as internal or external (Ganzel et al. 2010; Segerstrom and Miller 2004). The endocrine system, which controls much of the influence of stress on foetal development, is from an evolutionary perspective a very highly conserved system with basic endocrine function shared between all vertebrates (Bribiescas and Muehlenbein 2010). This indicates little change over time (Bribiescas and Muehlenbein 2010). Because the action of hormones appears stable, it is likely that the endocrinological response of modern humans to stress differs little from that of early hominids (Bribiescas and Muehlenbein 2010). The consistency and durability of the endocrine system's response to stress suggests that the stress response has been subject to long-term balancing selection.

Part of the role of the endocrine system may lie in controlling and maintaining phenotypic variation. There appears to be relatively little variability in the neural and endocrine stress response and degrees of individual difference appear only moderately hereditable (Bribiescas and Muehlenbein 2010). The consistency of the stress response suggests that its role in pregnancy and early life may be similarly long-standing (Gray 2010). Therefore, the effects of stress on pregnancy may also have been both subject to and a product of natural selection. Although humans construct ecological niches that protect them from many stressors (Laland et al. 2008), their stress response is likely still adapted to an earlier and more precarious existence (i.e. the environment of evolutionary adaptedness or EEA). Thus, while glucose secretion, accelerated heart rate and other aspects of the stress response are costly, the ability to flee or fight would have been crucial to the avoidance of predation and death in the EEA.

### 3.3. *Maturation and stochasticity*

The ability to make adjustments in reproductive strategies suggests that humans express phenotypic plasticity in their biology, as well as very substantial plasticity in their behaviour. It is common for organisms to be able to modulate their reproductive strategy in response to environmental pressure (Bribiescas and Muehlenbein 2010) as a method of addressing the uncertain futures problem (Chisholm 1999). The degree of variation in human behaviour surrounding reproduction, both historically and inter-culturally, provides evidence that there exists very considerable plasticity in human reproductive behaviour. However, there is also evidence suggesting that reproductive biological plasticity occurs, particularly in response to early life experience. Changes in life history traits, such as the timing of sexual maturity, provide examples of such biological plasticity in humans (Chisholm et al. 2005). Phenotypic plasticity allows organisms to hedge their bets against the uncertain futures problem.

Human can also express behavioural flexibility that is the product of conscious thought. For example, while age at menarche is a biological variable age at first sex is a behavioural variable. Other variables, such as age at first birth, represent an intersection of both the biological and the conscious, as neither menarche nor intercourse are sufficient without the other. However, behavioural variables which appear to be socially determined are also subject to biological imperatives. Factors which might influence age at first sex, such as stress responsiveness and resilience to peer group pressure can be affected by the functioning of a more reactive HPA. Thus, in practice, it is quite difficult to determine whether any variable can be adequately described as wholly one or the other.

In organisms that demonstrate early life plasticity, including humans, early life stress allows adaptations that enable immediate survival or reduce the costs of later stressors to the organism over its life course. For example, studies on rats have shown that prenatal stress is related to developmental neurological changes that increase fear of novelty, a useful adaptation in a dangerous environment (Weinstock 2007). Recent work on butterflies also suggests that the offspring of stressed mothers are able to better tolerate stress as adults and do not experience the shortened lifespans that their mothers and unexposed peers do when stressed (Saastamoinen et al. 2013).

In terms of the effects of stress during pregnancy on humans, there is some work suggesting that these are considerable and in some cases damaging. The foetal programming hypothesis is the most widely accepted. Also known as the Foetal Origins of Adult Disease, Developmental Origins of Health and Disease or Early Life Programming (ELP, used here after), the hypothesis postulates that stimulation and insults in early life modulate the normal course of development and that these changes can result in significant and permanent alterations to adult outcomes, such as the likelihood of developing chronic diseases (Bilbo and Schwarz 2009; Boersma et al. 2014; Eriksson 2010). The implications is that many adult disorders have a developmental origin (Bale et al. 2010).

First described by Barker et al. in 1989, ELP hypothesises that the intrauterine environment may permanently change organ structure and function, including brain development, particularly during critical periods of organogenesis (Drever et al. 2010). While social and environmental influences on gene expression have also been demonstrated in adults (Cole 2009), such influences during foetal development may profoundly affect life outcomes. Cole (2009) argues that the environment “not only gets inside the body, but stays there”. This appears to be especially true for a developing foetus.

From a bioanthropological viewpoint, animals (such as humans) that occupy a broad range of ecological niches must be able to respond to a wide variety of environmental demands (Meaney et al. 1994). Given this, much attention has been paid to the potential evolutionary source of human plasticity (Kuzawa 2005). ELP, and the responsiveness to the environment that it allows, may help give humans the plasticity they require in order to survive in a huge range of environments. Three broad theories offer explanations for how adverse consequences occur as a result of stress-related plasticity. Firstly, the 3-hit concept suggests that changes occur due to the combined effects of genetic predisposition, adverse early life events, and adverse later life events. Secondly, the cumulative stress theory argues that the effects of stress accumulate over the lifespan until a tipping point is encountered. And thirdly, the mismatch hypothesis posits that early life programming can be maladaptive to actual later life events (Musazzi and Marrocco 2016). Importantly, these theories relate rather than compete. The 3-hit theory is a specific conceptualisation of the cumulative stress theory which only specifies that stress experiences interact with underlying genetics, thus requiring that genetic predisposition be viewed as a form of cumulative stress. The mismatch hypothesis does not exclude the requirement for life stress exposure but extends this theory to incorporate the risks of ontogenic plasticity in changing environments. Indeed, it is well supported by bioanthropological theory. Essentially, organisms encounter the uncertain futures problem when the stressors that have been adapted to *in utero* do not manifest in later life. Mismatches between the natal and adult environments are identified as the cause of many of the negative downstream effects of programming.

In terms of stress, poor nutrition is an area often examined in the literature surrounding ELP. Accordingly, foetuses are viewed as being able to gauge their likely future nutritional environmental from the one they experience *in utero*. Uterine nutrition is somewhat buffered from that of the mother and the foetus is able to hormonally override some of the mother's efforts to conserve nutrition. This results in a gap between foetal and maternal nutritional status. According to one study, even in situations where maternal nutrition varied by up to 118,000 kcal per day, the weight of the resulting infants varied by less than 500g (Kuzawa 2005). Other research suggests that foetuses are able to adapt to poor natal nutrition by expressing a thriftier metabolism which allows growth to continue despite low nutrition, this is termed the "thrifty phenotype" (Boersma et al. 2014; Hales and Barker 2001). However, if food shortages affect gestation but do not continue throughout the lifetime, the *in utero* adaptation toward lower metabolic rate may become maladaptive in adulthood and increase obesity and associated disease risks (Boersma et al. 2014; Hales and Barker 2001). For example, association between poor natal growth and later life metabolic syndrome has been found and attributed to a mismatch between the food availability experienced *in utero* and during childhood (Hales and Barker 2001).

Higher birth weight within the normal range is a positive predictor of a number of functions including immune response, lean body mass, and distribution of adipose tissues. This suggests that foetal growth rates are implicated across a suite of developmental domains. Because the impacts of growth are widely spread, Kuzawa

(2005) argues that preparation for the post-natal environment must begin prenatally to allow all relevant domains to be altered. This proposition assumes that the prenatal environment is a useful predictor of the post-natal one and that the foetus is capable of making and responding to this prediction (Kuzawa 2005). Given that future predictions are troublesome (the uncertain futures problem), mismatches between the prenatal prediction and the actual post-natal environment are a risk for any long-lived species.

For long-lived species, foetal predictions of the post-natal environment seem to be a high-risk strategy. To make this strategy a useful one, the foetus requires a capacity to judge average environmental standards as distinct from short-term fluctuations that might occur during gestation. Some work on prenatal nutrition does indeed suggest that the capacity of foetuses to judge nutrition is not based on the experience of that pregnancy alone. Kuzawa (2005) hypothesises that, because seasonal fluctuations in food availability is a poor signal of long-term food availability, foetuses must receive a more integrated suite of cues regarding nutrition, including the long-term nutrition balance and birth weight of the mother. Paternal birth weight also correlates with infant birth weight, albeit less strongly than maternal birth weight (Coutinho et al. 1997). One of the results of such an indicator is that the way foetuses experience of uterine nutrition could be understood within the context of the mother's long-term nutritional status, making changes less susceptible to short-term fluctuations and more adaptive to longer-term patterns (Kuzawa 2005).

While Kuzawa (2005) focuses on the matriline, other studies find effects through the patriline in which the quality of food access in childhood appears to create intergenerational associations with the development of metabolic diseases (Bygren et al.

2001; Kaati et al. 2007). Pembrey (2008a; 2008b; 2002) and Bygren et al. (2001) argue that grandparental nutrition can make predictable differences in grandchildren's longevity and morbidity. Grandparental experience of both nutritional restriction and nutritional excess appears related to responsive changes in adult grandchildren, particularly with regard to metabolic disorders, even where there has been no nutritional stress in the intervening generation. Related work indicates that transgenerational effects can be sex-specific and alter depending on which grandparent experienced nutritional stress or excess (Kaati et al. 2007; Pembrey et al. 2006). Girls appear to be more responsive to stress experienced by parents and grandparents (Pembrey et al. 2006), which possibly reflects sex-specific differences in placental structure (as discussed in the following section).

Developmental responses also occur in response to non-nutritional prenatal stressors, including life events, war and environmental disasters. In several studies, this stress exposure is linked to problematic outcomes, including increased disease and increased disease risk (Boersma et al. 2014; Dancause et al. 2015; King et al. 2009; Laplante et al. 2008; Turcotte-Tremblay et al. 2014). Such effects indicate that *in utero* conditioning is not functioning to enhance the adaptation of the foetus to modern environments. For instance, greater HPA reactivity observed in children exposed to prenatal stress may confer a more reactive and thus, in a stochastic environment, safer phenotype. However, in modern society, this high stress responsiveness may also lead to a range of behavioural patterns which inhibit social and scholastic achievement (Boersma et al. 2014). Thus, the adaptation becomes problematic because of a misalignment between the requirements of the natal and adult environments. Notably, some studies find that exposure to mild neonatal adversity results in reduced HPA reactivity or early maturation of the amygdala-prefrontal cortex connectivity, leading to greater resilience to stress. Only severe neonatal adversity results in deleterious changes. This suggests that an adaptive process of *in utero* adjustment becomes maladaptive only where stressors are severe and the capacity of the cortex to adapt to corticosteroid exposure is overwhelmed (Musazzi and Marrocco 2016).

There are energetic costs associated with plasticity and risks associated with misreading the environment. Because organisms can only predict the likely future environment based on past and current environments, these predictions can be faulty (Chisholm 1999; Coall and Chisholm 2003). Despite the risk that environmentally responsive phenotypes will have later life costs (e.g. chronic disease related to changes in the dispersal of adipose tissue), these may be necessary costs and may not limit reproduction if they occur in post-reproductive life. As such, these costs are not subject to selective pressure. Selection is strong in early life and a response which increases immediate survival at the cost of later life illness or maladaptation can be expected to be positively selected (Muehlenbein 2010).

Likely due to the costs of plasticity and the risk of misperception, plasticity appears to occur only within more rigid parameters (Bribiescas and Muehlenbein 2010). In humans, alteration in the length of gestation, birth weight, age of sexual maturity, and age at first birth, demonstrate a bounded flexibility as they occur only within discrete ranges. Plasticity that exceeds those ranges (e.g. birth weights below 1500 grams) presents a very substantial mortality risk. For any species which inhabits a range of ecological niches, and therefore encounters a range of differing pressures, phenotypic plasticity is likely to aid survival and reproduction. For humans, who inhabit an extremely wide range of environments (humans are represented in almost every terrestrial environment), selective pressures are likely to favour the ability to be both behaviourally and biologically flexible in reproductive strategy. If the timing of life history traits are understood to be a product of evolution, plasticity in these traits should be a similarly evolved capacity (Pike and Milligan 2010). This does not mean that all changes to life history are the product of direct selection, however, as many may be by-products of other adaptations (Gould and Lewontin 1979; Gray 2010; Pike and Milligan 2010).

In very young children (e.g. in the first thousand days from conception), infantile emotions likely serve as signals about the quality of their environment (Chisholm 1999). In very early life, there is no neurological connection between the amygdala and the prefrontal cortex; therefore, all early memories are emotional products of the limbic system untempered by the contextualisation later provided by the prefrontal cortex. Because of the involvement of the amygdala, feelings of fear, and the events that have aroused such fear are most prominent. The activation of this region may signal to children the quality of their environment biologically. This early period may play an important role in the development of further neurological function, particularly that of the HPA (Hane et al. 2010). Just as prenatal stress may create adaptive cascades, so too can stress in early post-natal life. These changes, particularly when they affect stress responsivity, can be expected to manifest in behavioural and psychosocial disruptions throughout childhood and adulthood (Shonkoff and Garner 2012).

### 3.3.1. Epigenetic change

The field of epigenetics (the study of environment–gene interactions and extra-DNA mitotical or meiotical heredity) is a rapidly growing one (Bird 2002; Cao-Lei et al. 2016; Haig 2006). Epigenetics is concerned with the gap between genotype and phenotype, which is theoretically complex. Classical selection acts on phenotype only, but reproductive advantage has been understood to be transmitted through genotype alone. In terms of stress and reproduction, some evidence now suggests that stress experienced *in utero* can be influential on phenotype and that this influence can be transmitted, without alteration to genotype, to subsequent generations (Pembrey 2002). This indicates that some form of intergenerational transmission of phenotypic variation is occurring.

For Neo-Darwinist theories, including LHT, this presents a challenge as it suggests that Weismann's Barrier, a key tenet of Neo-Darwinism, is being breached. However, Weismann's Barrier, proposed by evolutionary biologist August Weismann in 1886, only states that germ cells cannot be influenced by somatic cells (Oxford University Press 2008; Princeton University 2013). This does not exclude epigenetic changes, which do not alter the germline, but rather its expression. Therefore, evidence for the intergenerational transmission of acquired characteristics does not imply a fault in Weismann's Barrier, because epigenetic change requires alterations to methylation, rather than DNA sequence (Kelly and Katz 2009).

Environmental changes *in utero* can alter patterns of methylation, causing genes to be silenced or formerly silenced genes to be expressed (Radtke et al. 2011). Some studies show that methylation alters in response to *in utero* stress (Howerton et al. 2013;

Janssen et al. 2016; Radtke et al. 2011) while others indicate that such changes are transmissible to subsequent generations (Morgan et al. 1999; Pembrey 2008a; Pembrey 2008b).

Incomplete zygotic demethylation may explain why the effects of developmental stress appear to persist intergenerationally. For example, Morgan et al. (1999 p.314) find that, in mice, an epigenetic modification affecting coat colour and metabolism is inherited due to an "incomplete erasure of an epigenetic modification". In rats, female foetuses who experience nutritional deprivation have offspring who demonstrate foetal growth restriction. These offspring have reduced glucose utilisation capacity, as do their own subsequent offspring, even when the intermediate generation has received appropriate nutrition (Lummaa and Clutton-Brock 2002). Similarly, Anway et al. (2005) find that female rats exposed to endocrine disrupters have a greater number of infertile male offspring and that this effect is heritable though the paternal line via their fertile male offspring. Anway et al. (2005) suggest that the observed patterns correlate with methylation disruptions in the germ line. However, these results have not be replicated (Renner 2009).

Fear may also be heritable. Dias and Ressler (2014) subject mice to odour fear conditioning (in which they were exposed to acetophenone paired with mild electrical shocks) prior to conception. These mice were then bred and the next two resulting generations also showed an aversion to the odour of acetophenone, despite experiencing no noxious exposure themselves. Such transfer may allow fear of dangerous stimuli to be inherited – the authors suggest that human phobias may be inherited similarly (Dias and Ressler 2014). Other animal studies appear to suggest that some epigenetic changes are beneficial. Saastamoinen, Hirai and van Nouhuys (2013) find that butterflies stressed by nutritional deficiency or inability to rest have shortened lifespans. However, their offspring, after experiencing the same stressors, did not suffer the same fate. The maternal experience of stress appears to have improved the ability of the subsequent generation to experience stress with lesser costs.

Animal studies provide useful but imperfect models of human responses. In studies of stress, the differences between basal cortisol and cortiocosterone levels between humans and rodents, as well as metabolic differences, are important modifiers (Graignic-Philippe et al. 2014). However, Radtke et al. (2011) find epigenetic effects on *in utero* exposure to domestic violence in humans, and that these alterations of expression continued as the exposed children grew. Similarly, Janssen et al. (2016) find effects of maternal mood on methylation within the placenta and propose that these changes may affect placental function in a manner that adversely affects the child's later development. Similarly, the Project Ice Storm team find evidence of epigenetic change in infants exposed to the 1998 Quebec ice storm while *in utero*. In particular, maternal hardship during the storm was associated with methylation levels in 1675 CpG sites affiliated with 957 genes, largely related to immune function (Cao-Lei et al. 2014). Associations between maternal stress and epigenetic changes which potentially decrease the risk of central adiposity in the exposed children indicate protective potential against obesity-linked diseases (Cao-Lei et al. 2016). Conversely, Rijlaarsdam et al. (2016), in their meta-analysis of studies examining genome-wide epigenetic effects of prenatal stress, find no large or consistent effects of prenatal stress of neonatal DNA methylation.

In another potentially epigenetic pattern observed in humans, a mother's own preterm birth is a strong indicator of the risk of prematurity in her own offspring (Ananth and Vintzileos 2006; Foster et al. 2000; Goldenberg et al. 2008; Moster et al. 2008). Pembrey (2002) notes that heritability in the maternal line is complicated because one generation resides inside the other during early development. Arguably, two generations actually reside inside the other because ova are fully formed *in utero*. This means that the natal environment could directly affect the development of two subsequent generations as the ova carry methylation patterns into these subsequent generations. Incomplete demethylation during early gestation could therefore result in " a cascade of … effects down the generations" (Pembrey 2002 p.670).

Importantly, there are also non-epigenetic links between low birth weight and prematurity. For example, the inhibitory effect of oxytocin on the maternal stress response is reduced, or some cases reversed, in mothers who themselves have encountered early life stress. This means that their foetuses are more likely to be exposed to higher steroid hormone levels with the attendant increased (but non-epigenetic) risk of low weight or prematurity (Musazzi and Marrocco 2016).

Assuming that stress-related *in utero* change confers some sort of adaptive benefit, epigenetic heritability may act selectively because it reflects the fitness of the parental generation. Some studies indicate that the existence of apparently epigenetic inheritance patterns is sex-specific (Kaati et al. 2007; Morgan et al. 1999; Pembrey 2008a; Pembrey 2008b; Pembrey 2002; Pembrey et al. 2006; Wossidlo et al. 2011), which indicates the possible presence of sex differentiated selective pressures. Although such intergenerational patterns are far less clear in humans and human evidence is more frequently contradictory (Lummaa and Clutton-Brock 2002), a growing body of research suggests that the effects of perinatal reprogramming can cross multiple generations (Musazzi and Marrocco 2016).

### 3.4. *Chapter summary*

The application of LHT to humans requires that humans are systemically responsive to their environments, in both their maturation and reproduction. This chapter has provided evidence both that this responsivity occurs and that the experience of stress is a key mechanism through which the environment is communicated to the body. The effects of stress on gene expression result in changes to development which can be detected through clinical measures, such as anthropometrics. Further, it is possible, though certainly not conclusively demonstrated, that the effects of stress on foetal development will create intergenerational echoes. This means that effects of stressful events may have effects beyond the immediately affected generation.

The next chapter, Chapter Four, is the first of the analytic chapters presented in this dissertation. It examines the role of prenatal stress on two indicators of the prenatal environment: gestational age and birth weight. After presenting a review of the relevant literature, the chapter compares gestational age and birth weights between infants affected by the Canberra 2003 fires and the Black Saturday fires against those of their unexposed peers.

# 4. EFFECTS OF FIRE EXPOSURE ON BIRTH WEIGHT AND GESTATIONAL AGE IN TWO POPULATIONS

## 4.1. *Introduction*

This chapter is a retrospective cohort study of birth records in Victoria and the Australian Capital Territory (ACT). It aims to explore changes in average birth weights or gestational age in relation to maternal bushfire exposure. The approach taken in this chapter is informed by the pilot study to this research, which was reported in detail in O'Donnell and Behie (2013). Unlike the pilot study, the research design used here allows for the inclusion of a substantial number of confounding maternal characteristics; it also includes birth weight and gestational age as continuous variables rather than categorical variables. Given the predictions of LHT (outlined in Chapter Two) that highly stressed mothers should experience a decreased capacity to invest in offspring and that this could manifest biologically in reduced birth weight and shortened gestation. This chapter outlines the underlying biology of these hypothesised effects through a review of the literature and an investigation into birth outcomes following fires in Victoria and Canberra.

## 4.2. *Research question*

The central question of this chapter asks what is the effect of prenatal bushfire exposure on birth weight, and gestational age in populations exposed to the Canberra and Black

Saturday bushfires? The chapter uses the natural experiment of these two fires to make a quantitative analysis of these cohorts.

## 4.3. *Effects of stress in pregnancy*

In pregnant women, the stress response can have a profound impact on the foetus and is understood to affect neural and physical development. This is believed to affect life outcomes, including disease propensity, reactivity to stress, and lifespan (Shonkoff and Garner 2012). Despite a significant body of research in other mammals (Boersma et al. 2014), evidence of stress effects in humans remains less well understood and human research shows greater variability.

Although the exact mechanisms of the effects of stress in pregnancy remain poorly understood, increased activation of the maternal HPA axis seems the most likely pathway for impact on the foetus. In pregnancy, women naturally have heightened levels of cortisol, which is essential for normal foetal development since it assists in preparing the foetus for the extra-uterine environment (Charil et al. 2010). However, when the mother is exposed to stressors, levels of cortisol can deviate from levels considered to be beneficial to development. The transfer of maternal cortisol to the foetus is regulated through the placenta which produces a glucocorticoid barrier enzyme (11-HSD2) that neutralises some maternal cortisol. However, increased maternal cortisol levels reduce the efficacy of this enzyme resulting in greater transfer of cortisol across the placental barrier (Charil et al. 2010). Several studies have demonstrated that maternal cortisol levels account for as much as 40% of variation in foetal levels (Lazinski et al. 2008; Sandman and Davis 2010; Sandman et al. 2003). This increased foetal exposure can alter foetal development in ways which persist into adulthood (Radtke et al. 2011).

Placental (and endometrial) CRH production is understood to be important in foetal development, particularly in the early development of the placenta itself (Torche and Kleinhaus 2012). The role of the placenta is important in mediating the transfer of maternal cortisol (and other substances) to the foetus. Torche and Kleinhaus (2012) suggest that the role of the placenta may help explain the greater sensitivity to maternal stress observed in female foetuses. Because the placenta arises from foetal tissue, it differs according to the sex of the foetus. In girls, X-chromosome inactivation is low in placental tissue, allowing a greater role for the strength of dual X-chromosomes (Torche and Kleinhaus 2012). Females appear to have a greater capacity to respond to maternal stress signals than their male counterparts, potentially due to the adaptability of the double X genome (Torche and Kleinhaus 2012). Such a mechanism would be consistent with the greater magnitude of epigenetic response, which has been observed in girls in recent studies into environmental predictors of the emergency of the thrifty phenotype (Pembrey et al. 2006).

Other work proposes a different mechanism of impact: vasoconstriction resulting from increased release of maternal catecholamine. Such vasoconstriction would likely lead to low foetal oxygen levels and may provide a mechanism for changes in neurodevelopment and cognitive performance observed in children of prenatally stressed mothers (detailed below) (Graignic-Philippe et al. 2014). As increases in cortisol and catecholamines are not contradictory, both processes may occur.

The effects of stress on the development of sexual reproductive cells (Fukuda et al. 1998), foetuses, infants, and children (Charil et al. 2010; Laplante et al. 2008) are increasingly documented in the literature and appear both negative and profound (Perry 2004; Radtke et al. 2011). In studies which use animal models, prenatal exposure to stress is linked to changes in brain development (Merlot et al. 2008; Rodrigues et al. 2011; Velisek 2005), immune function (Charil et al. 2010), stress response (Matthews 2002), and the development of asthma (Drever et al. 2010). These studies suggest that it may be possible to extrapolate such findings to humans. In fact further work in humans suggests that childhood obesity (Li et al. 2010), the development of psychiatric disorders (Armstrong 2009; Graignic-Philippe et al. 2014; King et al. 2005; Kinney et al. 2008a; Kinney et al. 2008b), decreased linguistic functioning (Graignic-Philippe et al. 2014; Laplante et al. 2008), and susceptibility to other diseases (Maulik 2007) may be linked to prenatal exposure to stress. Other research in animal models suggests that some of these changes can persist intergenerationally (Matthews and Phillips 2012).

However, some studies have not found such links. In a large study in two populations (Swedish and English), Rai et al. (2012) find no relationship between prenatal and early life stress and development of Autism Spectrum Disorder (ASD). Other studies which have found links between prenatal stress and later cognitive performance find that, although significant, prenatal stress accounts for a relatively low (10-17%) amount of variance in cognitive performance (Graignic-Philippe et al. 2014). This underscores the complexity of cognitive development, performance, and measurement.

Studies conducted after birth and in very early life suggest that heightened maternal responsivity to stress alters infants' stress responses. Davis et al. (2011) find that infants' cortisol response to a heel pin-prick is greater in the infants of mothers who were stressed during pregnancy (based on self-report) where pin-prick tests are conducted at 24 hours and 15, 19, 25, 31 and 36 weeks after birth. These differences cannot be accounted for by mode of delivery, prenatal medical history, socioeconomic status or child race, sex or birth order. This suggests that maternal and child stress is related, but does not provide evidence for whether this relationship is biological (e.g. the result of epigenetic changes *in utero*) or social (e.g. stress communicated through the interactions of mother and child post-birth). Indeed, it is not clear if this differentiation can be made while allowing for the potential genetic underpinnings of maternal anxiety.

Several other studies show similar positive relationships between maternal stress experience and the infant's degree of irritability or fussiness in early life (Graignic-Philippe et al. 2014). Radtke et al. (2011) find that maternal exposure to domestic violence during pregnancy alters the expression of the glucocorticoid receptors in infants. In this study, the methylation status of the glucocorticoid receptor in the children exposed to domestic violence *in utero* remained altered into adolescence.

Similarly, changes to the glucocorticoid receptor co-chaperone protein are associated with experience of abuse in childhood; these changes appear to increase vulnerability to depressive disorders (Bale et al. 2010). Such studies provide evidence for the links between exposure and specific developmental alterations to basal HPA activation. Additionally, *in utero* and childhood exposure to domestic violence, neglect and other abuse are linked to significant retardation in neurogenesis at three years of age (Perry 2004; Perry 2009). However, despite suggestions that there may be a direct relationship between prenatal stress and neurogenesis independent of birth weight, studies of head circumference have not found significant relationships (Graignic-Philippe et al. 2014).

Other studies find that early life stress, such as both psychological and physical maternal stress, to increase the likelihood of infection and later development of chronic disease (Behie and O'Donnell 2015; McEwen 1994). Further work also suggests that prenatal stress creates changes in placental phenotype, which also results in changes to foetal development (Charil et al. 2010). Alterations in foetal development arising from excessive exposure to maternal cortisol may be termed “epigenetic” because they are understood to arise from the interaction of the foetus’ genetic make-up and its environment to promote or demote the expression of certain genes. Emerging evidence suggests that maternal experience of stress might predict such epigenetic changes in offspring via DNA methylation, histone acetylation and noncoding RNAs (Bale et al. 2010).

Changes to brain development appear associated with stress-related hormonal changes to the uterine environment. This includes the production of glucocorticoid-sensitive proteins and increased production of hormones such as progesterone, oestrogens, and the glucose-transporter GLUT-1. In animal studies, changes in development (principally, reduced tissue volume) in the hippocampus, amygdala, corpus callosum, cerebral cortex, cerebellum, and hypothalamus have been detected. In rodents, such studies link the timing of maternal stress exposure to the neurogenesis of brain regions that are associated with the cognitive changes shown by offspring in later life (Bale et al. 2010). In humans, studies show reductions in grey matter density have been found in 6–9 year-old children exposed to prenatal stress at 19 weeks gestation (Charil et al.

2010). To date, however, no human studies have linked the timing of stress exposure to specific brain regions. Evidence regarding timing of exposure in humans is varied but suggests that the first trimester is the most vulnerable period (Glynn et al. 2001; Lazinski et al. 2008; O'Donnell et al. 2009). This is also the period with the most rapid rate of organogenesis.

There are exceptions to this general trend, however. Two related studies find an increase in the incidence of ASD in children exposed to hurricane-induced maternal stress-related during the second and third trimesters (Kinney et al. 2008a; Kinney et al. 2008b). Conversely, later studies do not demonstrate a link between ASD and maternal stress (Rai et al. 2012). Additionally, nutrition stress appears to have different timing effects. According to one study (Lummaa and Clutton-Brock 2002), exposure to famine has a greater impact in the third trimester, most likely due to the high foetal calorie demands encountered in this trimester. While the methodological challenges of human studies limit the available evidence, if similar changes to those observed in animal models exist in humans, the implications are wide-ranging.

Maternal experience of life stress during gestation also correlates with decreases in sex ratio in humans (Catalano 2003; Catalano and Bruckner 2005; Catalano et al. 2005; Hansen et al. 1999; Helle et al. 2009) as discussed in Chapter Six.

### 4.4. *Preterm stress from traumatic events*

There is a growing body of research which documents changes in birth outcomes following environmental disasters and other stressful events. Premature birth (here defined as less than 37 weeks gestation) as well as associated low birth weight (defined as less than 2500 grams) and very low birth weight (defined as less than 1500 grams) substantially decrease the likelihood of infant survival (Callaghan et al. 2006). As discussed in Chapter Two, early birth and low birth weight may represent changes in reproductive strategy which seek to make the best trade-offs between quality and quantity of offspring, as well as between current and future reproduction. The literature regarding more general causes of prenatal stress, and the impacts on foetal development and birth timing, is very substantial (Alder et al. 2007a; Alder et al. 2007b; Anderson 2005; Brummelte et al. 2011; Lazinski et al. 2008). Increases in infants' stress response (Davis et al. 2011; Davis et al. 2005; Emack and Matthews 2011; O'Donnell et al. 2009; Radtke et al. 2011; Sandman et al. 2003; Sandman et al. 1999; Velisek 2005), decreased infantile immune response (Merlot et al. 2008) and poorer longer-term developmental outcomes (Bussa et al. 2010; Charil et al. 2010; Clarke et al. 1996) (Gutteling et al. 2005) (Rice et al. 2010; Sandman and Davis 2010) (Tegethoff et al. 2011) are all been associated with increases in maternal prenatal stress levels.

The links between stress and poor birth outcomes, particularly preterm births and low birth weight, are well-established in the literature (Alder et al. 2007b; Anderson 2005; Benitzhak and Verny 2004; Dancause et al. 2011; Glynn et al. 2001; Harville et al. 2009; Inder et al. 2001; Lazinski et al. 2008; Maric et al. 2009; Mulder et al. 2002; Obel et al. 2003; Peacock et al. 1995; Tegethoff et al. 2011; Torche and Kleinhaus 2012; Wadhwa et al. 1998; Xiong et al. 2008). This section reviews the literature which examines preterm birth and low birth weight in relation to discrete traumatic events, such as wars, terrorism and environmental disasters.

Two studies focus on the 9/11 terrorist attacks in the United States on September 11, 2001. Lederman at al. (2004) shows that infants born to women who lived within four kilometres of the World Trade Centre in New York during the attacks had babies who were, on average, 149 grams lighter than those born to women outside a four kilometre radius. Similarly, in an unpublished dissertation, Eccleston (2011) finds that, for those children who were exposed during the first or second trimester, the children born to mothers who were living in New York during the attacks were born between 12-14 grams lighter and 1-1.5 days earlier than those children born to mothers living in nearby counties. The smaller magnitude of change found in the second study likely relates to the inclusion of women further from ground zero who were not included in the earlier study.

Evidence from the 1999 bombing of Belgrade, Yugoslavia, is also consistent with this trend. Children born following the bombings were lighter at birth with a mean difference of 86 grams (95% CI = 67–104 grams), than children born in the years immediately before the bombing (Maric et al. 2009). The authors attribute this effect to the stress of the 3-month bombing campaign. However, no specific trimester of exposure appeared relevant and the length of gestation was not altered.

Following environmental disasters, the trend of lighter and (often) earlier birth persists. A series of studies examine the impact of the January 1998 ice storm in Quebec, Canada (Auger et al. 2011). The storm caused extensive damage and disruption in a geographically-defined region, including loss of power and heating for up to four weeks. Results of these studies show that preterm birth in the affected cohort increased in the two months following the storm (Auger et al. 2011) compared to births in unaffected regions or in the affected region in previous years. The covariate adjusted odds of preterm birth for January and February 2008 were 27% higher in the affected region than in unaffected regions, but this effect was not sustained beyond February. Adjusted odds of preterm births in January and February 2008 were also 28% higher relative to the same time period in 1999–2003 and again these odds were not elevated over the longer-term (Auger et al. 2011). Although the study shows only a weak association, further investigation of this event finds evidence of an effect. Dancause et al. (2011), for example, show that women exposed to the storm during pregnancy experienced significant stress and that gestational age differed with ice storm exposure. Gestations were shorter among women exposed in their first (mean gestation = 38.9 weeks) or second trimester (38.7 weeks) compared with those exposed in their third trimester (39.7 weeks) or during the preconception period (39.4 weeks) ($p = 0.05$). Birth weights were similarly lower with subjective stress accounting for 2.3% of variation in birth weight ($p = 0.008$) compared to women who were exposed immediately prior to conception or in the third trimester of their pregnancy (Dancause et al. 2011).

Mothers who experienced Hurricane Katrina, in the US Gulf Coast states, showed a similar pattern of preterm births, with the frequency of low weight birth being 14% in women who experienced a greater degree of hurricane exposure, compared with 4.7% in women without high hurricane exposure (95% CI = 1.13–9.89, $p = 0.01$) (Xiong et al. 2008). Similarly, the incidence of preterm birth was 14% in the high exposure group, compared with 6.3 % in women with lower exposure (95% CI = 0.82–6.38, $p = 0.05$) (Xiong et al. 2008). The study found no worsening of outcomes in mothers with Post-Traumatic Stress Disorder (PTSD) or major depression diagnoses, although other studies do find a relationship between maternal PSTD and infant temperament (Tees et al. 2009).

Two further studies examine the impact of earthquakes on birth weight and timing. Torche and Kleinhaus (2012) examine both prenatal birth and sex ratio changes by comparing births following a Chilean earthquake with those that occurred a year earlier. They also examine the role of exposure at different time points in gestation. Earthquake exposure in the first trimester of gestation resulted in the probability of preterm birth increasing by 0.038 (95% CI = 0.005–0.072) and 0.039 (95% CI = 0.002–0.075) respectively for female children exposed in months two and three of gestation, but changes in male children were not significant. A decline in the SSR was also recorded following exposure toward the end of the first trimester; however, because no sex ratio for foetal deaths was available, the study is unable to state whether the falling SSR and higher female rates of preterm birth were due to excess male mortality. Glynn et al. (2001) also briefly report a correlation between decreased gestation length and exposure to the Northridge, California (US) earthquake during the first trimester.

O'Donnell and Behie (2013) look at birth weight, gestational age and the SSR following the 2009 Black Saturday bushfire in Victoria, Australia. This study finds a significant increase in preterm births (z-value = 2.0, $p = 0.04$) and low birth weight (z-value = 2.8, $p = 0.001$) in the cohort exposed toward the end of gestation, when compared to births elsewhere in the state during the same period. Consistent with this, Auger et al. (2014) find that extreme heat episodes (4–7 days where the ambient air temperatures exceeded 32 degrees Celsius) increased the hazard of births preterm birth. As very hot weather always precedes bushfires, temperature may contribute to the overall effect.

O'Donnell and Behie (2013) also find a secondary trend of increased over-term births. This may due to either previous research designs that did not highlight over-term births, or actual differences in the women's access to, or use of, medical intervention.

### 4.4.1. Research hypothesis

Because of the documented effects of stress on birth weight and gestational age, the chapter hypothesises that gestational length will shorten in exposed mother-child couples and birth weight will decline.

## 4.5. *Method*

### 4.5.1. Ethics approval

The ethical aspects of this dissertation were approved by The Australian National University Human Research Ethics Committee (ANU-HREC) on 23 September 2013 (project reference: 2013/143). Following approval from the Chief Health Officer, further necessary approvals were granted by the ACT Government Human Research Ethics Committee's Low Risk Sub-Committee on 5 December 2013 (project reference: ETHLR.13.330). Approval was granted by the Victorian Department of Health Human Research Ethics Committee on 4 December 2014 (project reference: 20/14), which allowed approval of data access by the Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM).

### 4.5.2. **Collection methods**

Birth record data is collected by hospital staff, midwives or medical staff in accordance with the directives of the Perinatal National Minimum Data Set. This means that clinical birth records in Australia are collected and administered for virtually all births by hospitals and midwives (rather than requiring the parent to submit a form as is required for birth registration), ensuring the sample contained the entire target population (or as close as possible). Anthropometric measurements are also taken by clinical staff using appropriately maintained equipment. Gestational age is a clinical estimate based on maternal recall of last menstrual period, ultrasound imaging, or a combination of both. Measures of maternal factors, including cigarette smoking, alcohol consumption, marital status, and Aboriginal or Torres Strait Islander status, are collected via self-reporting and, as such, may contain inaccuracies (Peacock et al. 1995).

ACT birth records between 2000 and 2010 were requested from the Epidemiology Section (Population Health Informatics) of the Health Directorate in the ACT Government. In total, 48,408 births were represented in the ACT birth record data extract.

Table 4-1: Total ACT sample by baby's year of birth and mother's residence in a fire area

| Year of birth | Heavily affected | Moderately affected | Least affected | Combined |
|---|---|---|---|---|
| 2000 | 216 | 2805 | 1139 | 4160 |
| 2001 | 256 | 2495 | 1131 | 3882 |
| 2002 | 275 | 2592 | 1171 | 4038 |
| 2003 | 255 | 2583 | 1269 | 4107 |
| 2004 | 271 | 2579 | 1255 | 4105 |
| 2005 | 288 | 2664 | 1326 | 4278 |
| 2006 | 295 | 2821 | 1455 | 4571 |
| 2007 | 304 | 2846 | 1468 | 4618 |
| 2008 | 313 | 2874 | 1600 | 4787 |
| 2009 | 318 | 2946 | 1626 | 4890 |
| 2010 | 300 | 3081 | 1591 | 4972 |
| Totals | 3091 | 30286 | 15031 | 48408 |

Sample size calculations (conducted through the National Statistical Service website) indicate that an insufficient number of births occurred in the heavily affected area to meet the sample required for a confidence interval of 5% where the proportion is set to 50% (National Statistical Service 2017). This is discussed further in the limitations section (see section 4.6).

Victorian birth records between 2006 and 2012 were requested from CCOPMM via the Victorian Department of Health and Human Services. Due to the policies of the Department, data were not released, but rather data analysis were conducted by a departmental epidemiologist under my direction. In total, 514,995 births were represented in the Victorian birth record data extract.

Sample size calculations (conducted as above) indicate that a sufficient number of births occurred in the heavily affected area to meet the sample required for a confidence interval of 5% (National Statistical Service 2017); however, this is not the case when divided by trimester. This is discussed further in the limitations section (see section 4.6).

Table 4-2: Total Victorian sample by baby's year of birth and mother's residence in a fire area

| Year of birth | Heavily affected | Threatened | Surrounding | Unaffected | Total |
|---|---|---|---|---|---|
| 2006 | 539 | 1383 | 5416 | 62492 | 69830 |
| 2007 | 517 | 1416 | 5616 | 64879 | 72428 |
| 2008 | 488 | 1481 | 5539 | 64994 | 72502 |
| 2009 | 511 | 1374 | 5783 | 65300 | 72968 |
| 2010 | 471 | 1470 | 5706 | 66674 | 74321 |
| 2011 | 433 | 1574 | 5734 | 66264 | 74005 |
| 2012 | 446 | 1660 | 5983 | 70203 | 78292 |
| Totals | 3405 | 10358 | 39777 | 460806 | 514346 |

The following dependent variables are used in the analysis: (1) estimated gestational age; (2) birth weight. The following independent variables are used in the analysis (1) baby's sex; (2) mother reporting a residential address in a fire area; (3) maternal age; (4) maternal marital status; (5) maternal Indigenous status; (6) maternal smoking status; (7) previous pregnancy status and (8) baby plurality.

### 4.1.1. Data cleaning procedures

Any numeric code for missing values was replaced with a single code and excluded from any further analysis. Impossible responses, such as negative numbers, were removed and some variables were combined to create sufficient group sizes for analysis. Indigenous status was recoded from Aboriginal, Torres Strait Islander, or both, to create a combined binary variable in which the mother or baby was coded as either Indigenous or not. Similarly, marital status was combined to create a binary (coupled or not) due to small cell sizes in other categories. Termination cases were also removed from the data and were not included in further analysis.

### 4.5.3. New variables

In the ACT sample, two new derived variables have been created. The first is the timing of birth related to the fire (*birthtiming*). This variable describes the timing of births in relation to the fire event. Births in years other than 2003 and 2004 are grouped by year only. Births in 2003 and 2004 were divided into quarters for clarity. Distributions of *birthtiming* are shown in Figure 4-1. The second variable is the geographic fire area (*firearea*). This refers to the statistical subdivision of the mother's residential address grouped by degree of fire exposure. The statistical subdivisions of Belconnen (0510), Tuggeranong (0525) and Woden Valley (0515) are collectively termed moderately affected as fire damage occurred in at least one suburb in these regions and all were directly under threat of fire (Murray 2003). Weston Creek-Stromlo (0520) is termed heavily affected because of the concentrated loss of property in this region. The remaining ACT populated subdivisions of Gungahlin-Hall (0540), North Canberra (0505) and South Canberra (0535) are classified as least affected as no fire damage was reported in these regions and they were not under direct threat, although they were affected by smoke. Figure 4-2 shows the subdivision areas.

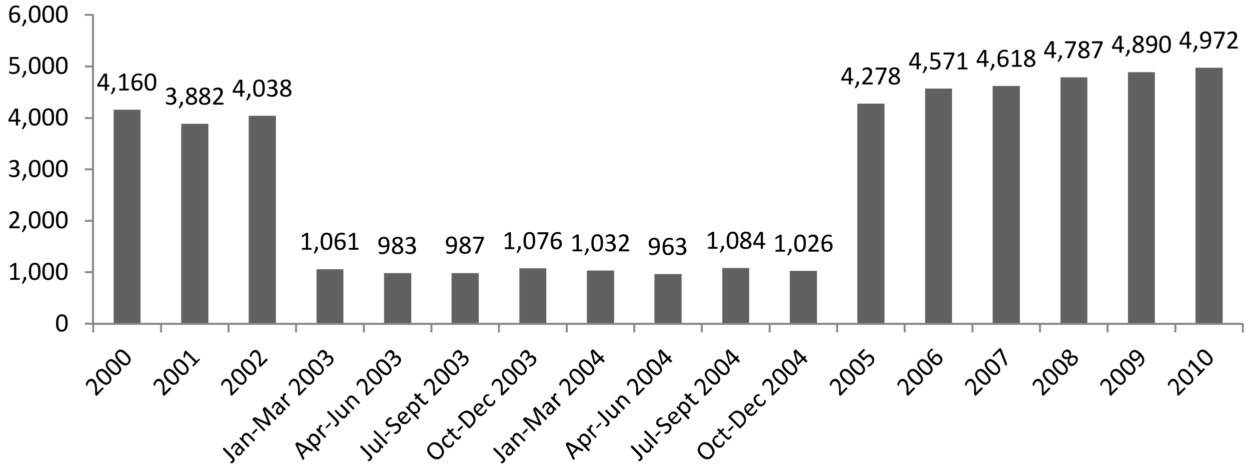


Figure 4-1: Distribution of births between *birthtiming* categories for all ACT births 2000 - 2010

[redacted]

Figure 4-2: Statistical subdivision areas in the ACT as produced by the Australian Bureau of Statistics

In the Victorian sample, the timing of fire exposure is calculated by creating an approximate trimester of exposure variable using an estimated conception date calculated by deducting the gestational age from the birth date. This gives an estimated conception date, the estimated days elapsed of gestation at the time of the fire are calculated using 7 February 2009 as the fire onset date. *Table 4-3* shows the number of births in each trimester category for exposed births in 2009. Exact trimester of exposure is not calculated in the ACT sample due to data access requirements related to privacy concerns.

Table 4-3: Population for each trimester divided by fire exposure level in 2009 for the Victorian population.

| | Exposed in the first trimester | Exposed in the second trimester | Exposed in the third trimester | Total |
|---|---|---|---|---|
| Heavily affected | 137 | 104 | 169 | 410 |
| Affected | 356 | 283 | 488 | 1127 |
| Surrounding | 1456 | 1185 | 2137 | 4778 |
| Rest of State | 16976 | 13443 | 23347 | 53766 |
| Total | 18925 | 15015 | 26141 | 60081 |

This method gives an approximate trimester of exposure only because it assumes that fire exposure occurred on 7 February for all births; however, different populations were impacted (and re-impacted) over almost a month, depending on location, and a small number of fires began prior to 7 February, although they did not become large until after that time. Therefore, trimester of exposure will be inaccurate in some cases, meaning that some first trimester exposed dyads will appear in the analysis as second trimester exposed.

[redacted]

Figure 4-3: Fire areas affected by the 2009 Black Saturday fires, the darkest areas of shading indicate the fire footprint (Encyclopedia Britannica 2009).

A fire area variable is created to define degrees of fire exposure. Based on the description of the 15 principal fires given in the Victorian Bushfires Royal Commission Final Report (Teague et al. 2010c), relevant local government areas were mapped to postcodes which provide geographical categories that are finer-grained than local government areas. Heavily affected areas are defined as those where multiple deaths and significant property damage occurred. Affected areas are those ones where destruction occurred but was more limited as well as those areas that were directly adjacent to heavily affected areas and were threatened by fire. Surrounding areas are those neighbouring affected areas, they are rural but did not experience direct fire threat from Black Saturday. Finally, rest of state is the remainder of Victorian postcodes. A list of heavily affected, affected and surrounding postcode areas can be found in Appendix A.

#### 4.5.4. **Analysis strategies**

In the ACT sample, unbalanced Analyses of Variance (ANOVA) are used to determine the influence of a mother reporting a residential address in a fire area (firearea) and other factors considered relevant to birth weight and gestational age (Goldenberg et al. 2008). Unbalanced ANOVAs are used because the ACT sample does not meet the assumptions of a balanced ANOVA due to the presence of smaller group sizes, particularly related to the small Aboriginal and Torres Strait Islander population. Following advice from the statistical adviser to this dissertation (located at the ANU Statistical Consulting Unit), unbalanced ANOVAs are conducted with reporting of predicted means to show magnitude of effects as well as statistical significance. While significance (reported as *p*-values) are used extensively in this research to indicate the presence of effects, it is important to note that statistically significant effects do not necessarily have practical or clinical relevance. This consideration is explored further in the discussion of the results presented in this dissertation.

Following advice from the statistical adviser, these were used instead of a general linear model because they show independent effects rather than effects against a reference category. ANOVAs are conducted separately for each year to identify the effect of the listed variables on birth weight and gestational age. Thereafter, an ANOVA of data from all years is conducted using an interaction term between fire area and the timing of birth.

Chi-square tests of independence are used to examine rates of gestational diabetes and maternal smoking.

In Victoria, the analysis was conducted by Department of Health staff under direction. Multiple linear regressions are used as ANOVAs with predicted means cannot be successfully conducted by the Department of Health staff on available software. Regression models are constructed with the above variables to account for their effects on birth weight and gestation age. The analysis strategies in this dissertation are guided and reviewed by The Australian National University Statistical Consulting Unit.

### 4.6. *Limitations*

This dissertation uses a geographical definition of disaster impact which might inadvertently exclude some affected people because disaster impact is not necessarily geographically limited. For instance, a person not resident in the disaster-affected area may still be affected by the disaster through relatives or friends, or through the loss of property or stock. Additionally, in both cases only small samples were available. Importantly, this is a whole-of-population study designed to include as close to all potential participants as possible. In both cases, it is neither possible nor ethical to increase the number of mother-child dyads affected by the fires. Thus, this is an inherent limitation of 'natural experiment' designs which has been considered in the discussion of the results of this dissertation.

The requirements of privacy legislation mean that place of residence is only identifiable at a high level, for example at council boundary level or at the Australian Bureau of Statistics' statistical geography levels (is detailed in section 6.3). These can be insufficiently specific making the construction of a disaster footprint more difficult. Such issues are common to population-based disaster research of this type.

Due to the restrictions of the available data, the study assumes that residential location at the time of birth is the same as residential location during pregnancy. However, because this research studies a disaster area, levels of inward migration are likely to be low. Therefore, women who were disaster-exposed are likely to be under-represented in the disaster-exposed cohort, resulting in the risk of a type two error (i.e. accepting a false null hypothesis).

Further, neither study is able to determine trimester of exposure with complete accuracy because the fire event occurred over a number of weeks. Because of reasonably small groups, particularly in the Victorian sample, timing effects should be interpreted cautiously.

Highly restrictive data access conditions imposed by the data owner posed considerable challenges to the conduct of the Victorian analysis. Because no direct access to the data was allowed, there opportunity to extend or refine the analysis was limited. Postcode 3658 was inadvertently coded as surrounding rather than heavily affected; however, as one of 41 postcodes in the surrounding category it is unlikely to have a substantial effect. Unfortunately, it was not possible to correct this owing to time limits on data access. Importantly, because the area was miscoded in such a way that it was excluded from the heavily affected category, the miscoding will have the effect of understating rather than overstating effects. Thus, any analysis that finds an effect can be understood to be reflecting a substantial effect.

## 4.7. *Preliminary Statistics – Canberra fire*

This section presents preliminary statistics to provide a brief overview of the ACT Health birth record data (see Table 4-4 and Table 4-5).

Table 4-4: Overall descriptive statistics by year – Canberra fires

| 2000 | | | 2001 | 2002 | 2003 | 2004 | 2005 | 2006 | 2007 | 2008 | 2009 | 2010 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Birth weight | mean | 3414 | 3391 | 3419 | 3408 | 3397 | 3368 | 3373 | 3379 | 3387 | 3400 | 3372 |
| | s.d | 617.3 | 610.2 | 620.3 | 620.3 | 636.8 | 630 | 605.2 | 611.8 | 604.1 | 595.1 | 600 |
| Mother’s age | mean | 29.68 | 29.88 | 30.04 | 30.23 | 35.52 | 30.54 | 30.66 | 30.57 | 30.94 | 30.77 | 30.91 |
| | s.d | 5.26 | 5.26 | 5.38 | 5.28 | 5.34 | 5.33 | 5.26 | 5.36 | 5.28 | 5.35 | 5.26 |
| Gestation | mean | 39.03 | 38.99 | 39.06 | 39.03 | 38.97 | 38.97 | 38.96 | 38.99 | 39.02 | 39.08 | 39 |
| | s.d | 2.23 | 2.25 | 2.22 | 2.33 | 2.28 | 2.26 | 2.30 | 2.23 | 2.27 | 2.15 | 2.24 |
| Percentage ATSI | | 0.91 | 1.11 | 1.25 | 1.42 | 1.39 | 1.87 | 1.69 | 1.47 | 1.57 | 1.38 | 1.21 |
| Percentage primipartum | | 32.09 | 37.29 | 37.87 | 35.11 | 32.42 | 32.22 | 33.43 | 33.72 | 31.82 | 33.44 | 31.84 |
| SSR | | 1.05 | 1.05 | 1.05 | 1.08 | 1.08 | 1.02 | 1.08 | 1.04 | 1.08 | 1.15 | 1.04 |
| Percentage multiple births | | 2.81 | 3.92 | 3.19 | 2.73 | 4.43 | 2.78 | 4.20 | 3.29 | 3.38 | 2.90 | 3.14 |
| Percentage mothers smoking | | 15.38 | 14.94 | 14.53 | 12.02 | 15.36 | 13.77 | 13.67 | 12.47 | 12.88 | 10.7 | 10.41 |
| Percentage mothers coupled | | 90.83 | 89.80 | 90.54 | 90.75 | 91.23 | 92.47 | 93.48 | 94.06 | 94.83 | 94.1 | 94.99 |

Table 4-5: Descriptive statistics for children born in 2003 divided by fire exposure status – Canberra fires

| | | Heavily affected | Moderately affected | Least affected |
|---|---|---|---|---|
| Birth weight | mean | 3549 | 3394 | 3409 |
| | s.d | 616.2 | 634.2 | 588.5 |
| Gestation | mean | 39.28 | 38.97 | 39.1 |
| | s.d | 1.76 | 2.50 | 2.05 |
| Mother's age | mean | 30.96 | 29.82 | 30.92 |
| | s.d | 4.85 | 5.30 | 5.24 |
| Percentage ATSI | | 0.39 | 1.48 | 1.51 |
| Percentage primigravida | | 30.20 | 34.49 | 37.35 |
| Percentage male | | 54.51 | 52.15 | 50.19 |
| Percentage multiple births | | 0.78 | 3.14 | 2.29 |
| Percentage mothers smoking | | 9.02 | 13.79 | 9 |
| Percentage mothers coupled | | 92.16 | 90.44 | 91.10 |

## 4.8. *Preliminary Statistics – Black Saturday*

This section presents preliminary statistics to provide a brief overview of the Victorian perinatal collection 2006–2012. All figures are shown in *Table 4-6* and *Table 4-7*.

Table 4-6: Whole-of-population descriptive statistics for the Victorian perinatal data extract

| | 2006 | 2007 | 2008 | 2009 | 2010 | 2011 | 2012 |
|---|---|---|---|---|---|---|---|
| Birth weight | 3350.7 (637.5) | 3348.2 (639.4) | 3355.4 (637.5) | 3346.5 (645) | 3343.7 (642.2) | 3348.4 (641.1) | 3350.7 (623.6) |
| Mother's age | 30.6 (5.4) | 30.7 (5.4) | 30.8 (5.5) | 30.7 (5.5) | 30.7 (5.5) | 30.7 (5.5) | 30.7 (5.4) |
| Gestation | 38.8 (2.4) | 38.8 (2.4) | 38.7 (2.4) | 38.7 (2.5) | 38.7 (2.4) | 38.7 (2.4) | 38.7 (2.4) |
| Percentage ATSI children | 0.82 | 0.97 | 1.01 | 1.69 | 1.67 | 1.84 | 1.79 |
| Percentage primipartum mothers | 32.9 | 33 | 33.5 | 33.3 | 33.5 | 33.9 | 33.9 |
| Percentage male | 51.6 | 51.3 | 51.5 | 51.2 | 51.3 | 51.6 | 51.4 |
| Percentage multiple births | 3.7 | 3.5 | 3.4 | 3.4 | 3.2 | 3.2 | 3.1 |
| Percentage mothers smoking at any time during gestation | No data | No data | No data | 11.03 | 4.28 | 5.43 | 11.73 |
| Percentage mothers coupled | 86.6 | 86.3 | 86.7 | 85.6 | 85.3 | 85.6 | 86.5 |

Table 4-7: Descriptive statistics of Victorian births in 2009 divided by fire exposure

| | | Heavily affected | Affected | Surrounding | Rest of state |
|---|---|---|---|---|---|
| Birth weight | Mean | 3378.65 | 3396.48 | 3369.78 | 3343.11 |
| | s.d | 610.16 | 699.65 | 649.10 | 643.58 |
| Gestation | Mean | 38.78 | 38.64 | 38.77 | 38.72 |
| | s.d | 2.76 | 2.76 | 2.45 | 2.50 |
| Mother's age | Mean | 31.19 | 30.39 | 29.75 | 30.82 |
| | s.d | 5.54 | 5.53 | 5.57 | 5.49 |
| Percentage ATSI | | 2.15 | 2.03 | 1.95 | 1.66 |
| Percentage primigravida | | 27.08 | 30.67 | 31.07 | 33.63 |
| Percentage male | | 46.58 | 52.40 | 52.41 | 51.12 |
| Percentage multiple births | | 2.34 | 3.48 | 3.50 | 3.36 |
| Percentage of mothers smoking | | 15.04 | 13.83 | 16.31 | 10.48 |
| Percentage of mothers coupled | | 85.35 | 84.86 | 82.18 | 85.94 |

## 4.9. *Results – Canberra fire*

### 4.9.1. **Gestational age**

Unbalanced Analyses of Variance (ANOVA) was used to analyse the impact of being resident in a fire area along with other variables shown in the literature to influence birth weight and gestational age. This analysis is conducted seperately for each year in the cleaned sample (2000–2010) and also conducted by offpsring sex. All full analyses are shown in Appendix A.

The analysis shows no significant influence on average gestational age of the mother being resident in a fire affected area during 2003. The ANOVAs finds no significant differences in average gestational age between male or female infants whose mothers were resident in differently affected geographical areas in the year of the fire (2003).

Although not significant, the male heavily affected cohort shows a higher predicted gestational age (39.57 weeks) compared to 39.05 weeks in the moderately affected areas and 39.14 weeks in the least affected areas.

Among male neonates, there is an increase in gestational age which correlated with the rise in birth weight (discussed below); however, this is not statistically significant for those babies exposed mostly in the third or late second trimester (i.e. those born between January and March 2003) and those exposed very early in the first trimester or conceived in the fire aftermath (i.e. those born between October and December 2003) (see Figure 4-4).

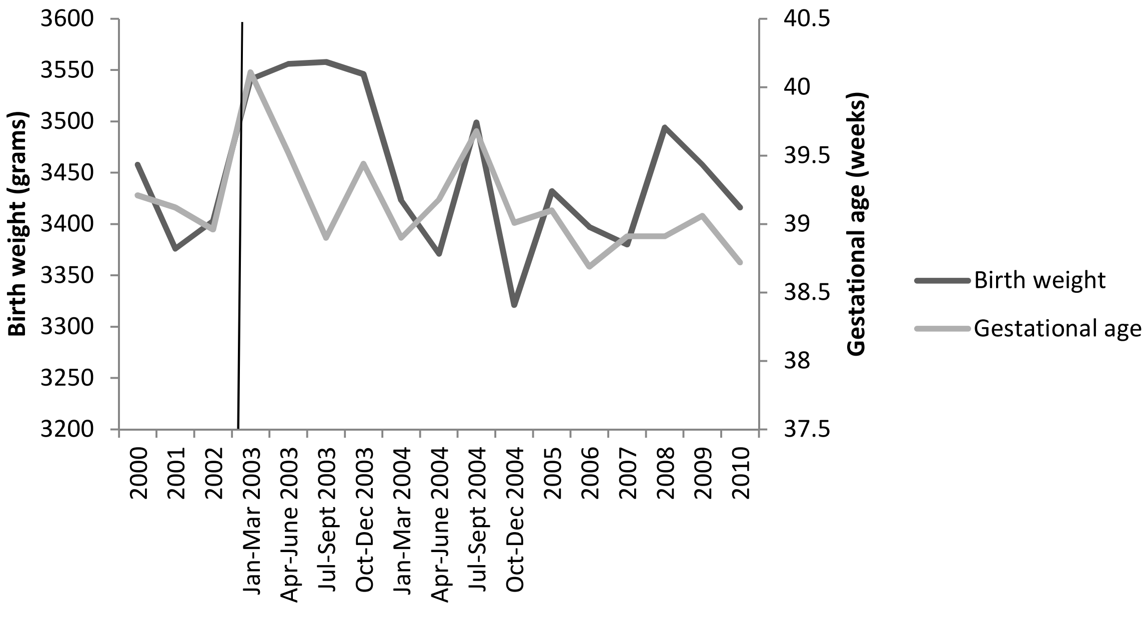


Figure 4-4: Predicted means (arising from ANOVA) of birth weight and gestational age among male infants in affected areas. Birth weight and gestational age disunite during 2003 and early 2004. The vertical line indicates the time of the fire.

A concordant relationship between birth weight and gestational age is not observed for neonates exposed later in the first trimester or in the second trimester (i.e. those born between April and September 2003). Due to this, it appears that the usual relationship between birth weight and gestational length may have been disrupted. While infants who were exposed later in gestation (in the third or late second trimester at the time of the fire) show a consistent relationship between gestational length and birth weight, that relationship lessens over the remainder of 2003 (i.e. children exposed earlier in gestation or conceived in the fire aftermath). The relationship between gestation and birth weight becomes more typical and consistent again from early 2004 onwards.

For both sexes, other significant influences on gestational age are consistent with the literature (Goldenberg et al. 2008). This includes: with older mothers (over 36 and older), those that identified as Aboriginal and/or Torres Strait Islander, mothers who smoked during gestation, and unmarried, widowed, divorced or seperated mothers reporting shorter gestations. Also consistent with the literature, twins, triplets and greater are shown to have shorter average gestations than their singleton peers.

### 4.9.2. **Birth weight analysis**

Unbalanced Analyses of Variance (ANOVA) is used to analyse the impact of being resident in a fire area and other variables which have been shown in the literature to influence birth weight and gestational age. This analysis is conducted seperately for each year in the cleaned sample (2000–2010) and also conducted by offspring sex.

Unbalanced ANOVAs for each year show that, when compared to those reported in the moderately affected area, average male birth weights were 197 grams heavier in the severely affected area in the year of the fire (2003) ($F = 5.73$, $p < 0.003$, $df = 2$), 18 grams heavier in the severely affected area in 2005 ($F = 3.06$, $p = 0.05$, $df = 2$), 47 grams heavier in the severely affected area in 2008 (F = 3.19, $p$ = 0.04, $df$ = 2) and 56 grams lighter in the severely affected area in 2010 (F = 3.52, $p$ = 0.03, $df$ = 2).

When conducted for female neonates, the analysis finds that, when compared to those reported in the moderately affected area, average female birth weights were 163 grams greater in the severely affected area in 2008 (F = 5.71, $p < 0.003$, $df$ = 2), 117 grams greater in the severely affected area in 2009 (F = 3.47, $p$= 0.03, $df$ = 2) and 160 grams greater in the severely affected area in 2010 (F = 5.66, $p < 0.004$, $df$ = 2).

The analysis finds that effects on neonates born to mothers who resided in the severely affected area in the year of the fire were only significant only for male offspring (F = 5.73, $p$ = 0.003, $df$ = 2). A further unbalanced ANOVA which included data from all years (2000–2010) indicates that exposed male neonates were heavier throughout 2003 than those males born to mothers residing in the same areas in other years (F-value = 1.58, $p$ = 0.01, $df$ = 2), while the exposed cohort was also born heavier on average than males born elsewhere in the ACT during 2003. In 2003, male neonates born to mothers who resided in severely affected areas had a predicted mean birth weight of 3,657 grams, compared with 3,460 grams for those born to mothers in affected areas and 3,454 grams for those born to mothers in unaffected areas (see Figure 4-5).

Other significant factors detected in the 2003 ANOVA on birth weight are consistent with the literature: those mothers who smoked during gestation (F = 49.75, $p < 0.001$, $df$ = 1) or reported as single mothers (F = 7.47, $p$ = 0.006, $df$ = 1) had babies with lighter mean birth weights (Goldenberg et al. 2008; Population Health Research Centre 2004). Also consistent with the literature, multiples were significantly lighter than their singleton peers (F = 153.68, $p < 0.001$, $df$ = 1) and primigravida mothers had lighter offspring than multigravida mothers (F = 14.97, $p < 0.001$, $df = 1$) (Goldenberg et al. 2008). Older maternal age increased average predicted mean birth weight (F-value = 6.3, $p = 0.01$).

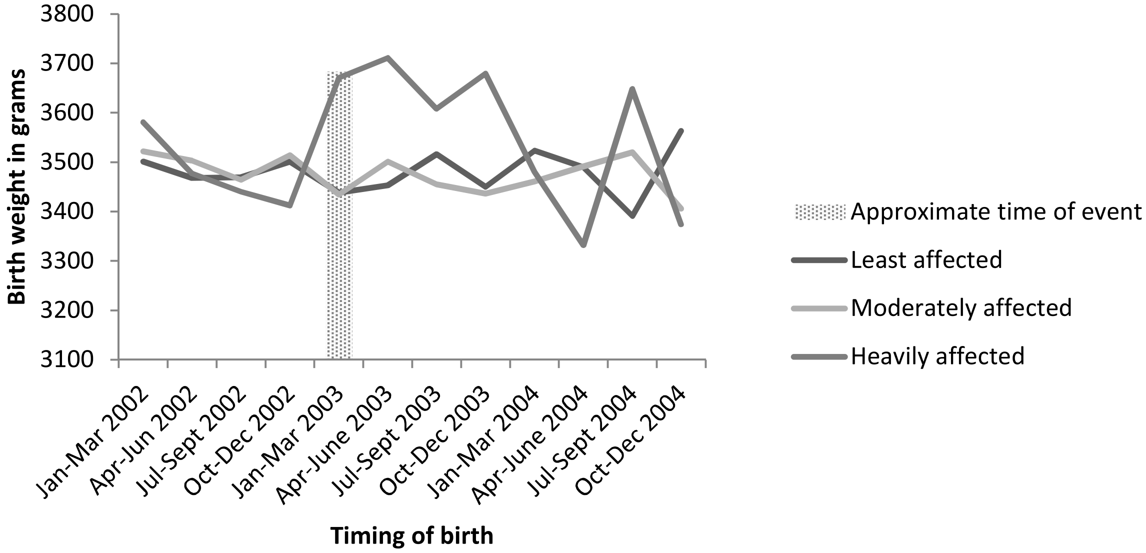


Figure 4-5: Means for birth weight arising from ANOVA analysis of males (only) at three levels of estimated fire exposure between 2002 and 2004, with the fire and fire aftermath (2003) shown by the shaded area.

A chi-square test of those effects finds that the increase in average birth weight was due to a significant increase in the number of male neonates born at greater than 4,000 grams (permutation corrected $p = 0.02$, uncorrected $p = 0.01$) with the largest difference occurring in individuals born between 4,501 and 5,000 grams (shown in Figure 2). Although, there is also an increase in severely underweight exposed male infants (born at between 500 and 1,500 grams), this change is insufficient to offset the overall increase in average weights (see *Figure 4-6*).

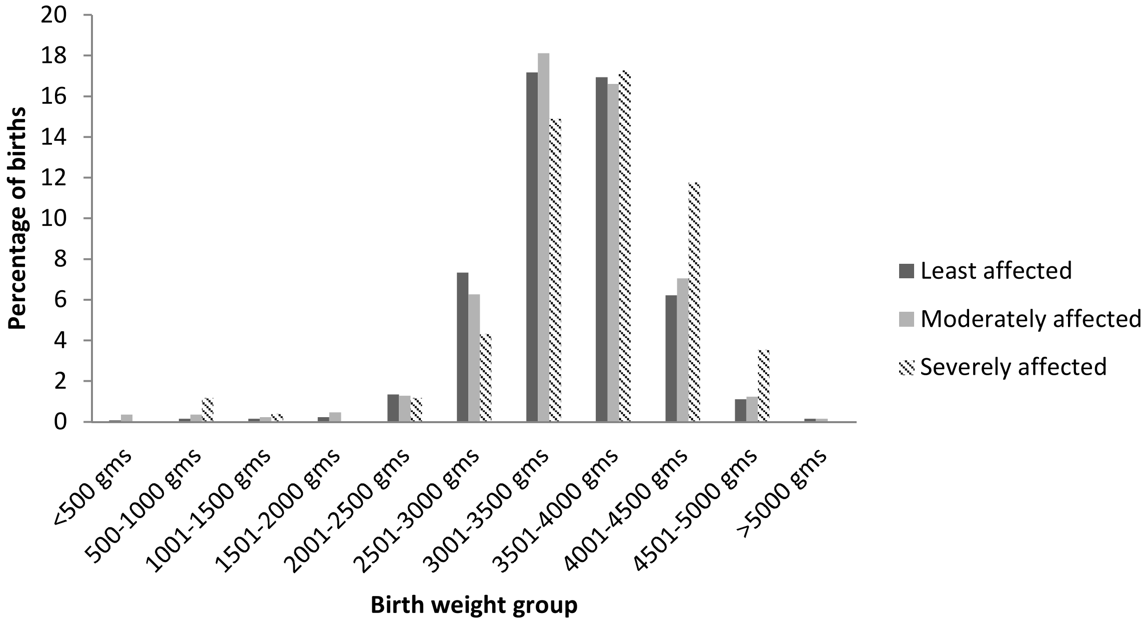


Figure 4-6: Plot of the percentage of births in each weight category by fire exposure in 2003, a chi-square test indicates that these differences are significant at p = 0.02. In the severely affected area, there were increases in birth weights between 4000 and 5000 grams
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#### 4.9.2.1. Maternal health

Because of the unusual pattern of higher birth weight, the analysis explores the presence of other factors with a known relationship to birth weight changes, such as gestational diabetes and maternal smoking. Chi-square tests find no significant differences in the rate of gestational diabetes mellitus (GDM) in mothers of males between the three defined areas of fire exposure in ($p = 0.81$) and indicate that maternal smoking declined in the ACT during the first six months of 2003 ($p < 0.001$) although this effect is mainly outside the severely affected area.

## 4.10. *Results – Black Saturday*

### 4.10.1. **Birth weight**

Multiple linear regression of birth weight shows that identifying as either Aboriginal or Torres Strait Islander (Coef. = -91.27; $p < 0.00$), offspring being of female sex (Coef. = 108.68; $p < 0.00$), maternal smoking (Coef. = 221.57; $p < 0.00$) and plurality (Coef. = -1009.98; $p < 0.00$) significantly lowered birth weight. Partnered marital status (Coef. = 86.11; $p < 0.00$) elevated birth weight to a small degree.

The analysis shows no significant effects on birth weight of being born to a mother residing in the affected areas in 2009 when timing of birth is included in the model.

Multiple linear regression is again used to explore the effect on each sex of fire exposure in 2009 on birth weight by sex. Regression analysis shows that there were no significant effects of fire exposure on gestational length in males or females.

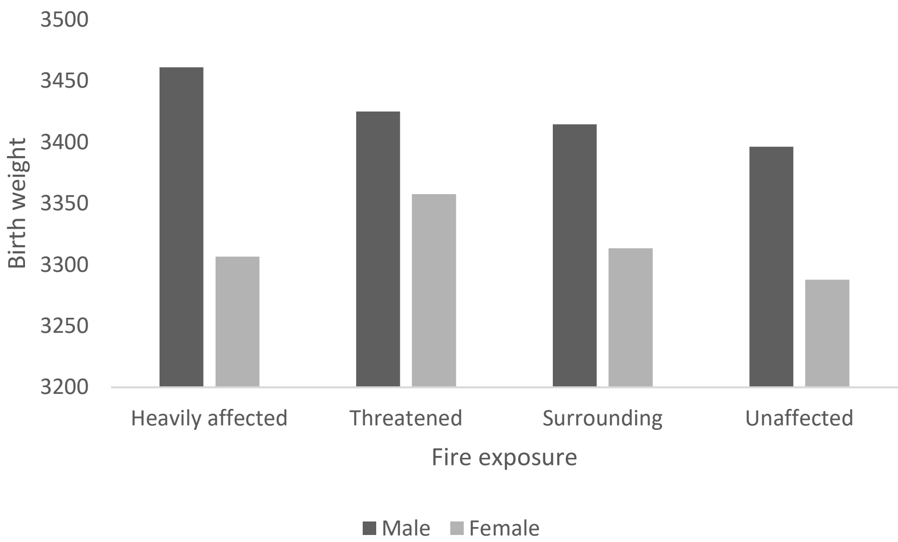


Figure 4-7: Average birth weight for males and females in 2009 divided by level of fire exposure (heavily exposed, threatened, surrounding or unaffected). Average birth weights is highest in the heavily affected area for males and is similar to those of the unaffected area for females.

Effects of other maternal characteristics were consistent between the sexes with a history of previous pregnancies (Coef. = 112.01 for two pregnancies, Coef. = 135.74 for more than two pregnancies, $p < 0.00$ for males and Coef. = 86.73 for two pregnancies, Coef. = 102.91 for more than two pregnancies, $p < 0.00$ for females) and coupled status raising birth weights (Coef. = 85.48, $p < 0.00$ for males and Coef. = 88.16, $p < 0.00$ for females). Identifying as Aboriginal or Torres Strait Islander (Coef. = -83.47, $p < 0.00$ for males and Coef. = -97.57, $p < 0.00$ for females), smoking in pregnancy (Coef. = -219.39, $p < 0.00$ for males and Coef. = -221.8, $p < 0.00$ for females) and being a multiple birth (Coef. = -1015.84, $p < 0.00$ for males and Coef. = 1003.03, $p < 0.00$ for females) are shown to have decreased birth weight.

### 4.10.2. **Gestational age**

Multiple linear regression is used to explore changes in gestational age. The analysis finds there are no significant effects of the mother residing in a fire effected area at any point during gestation on gestational age. Influences on gestational length include shortening of gestation associated with being: identified as either Aboriginal or Torres Strait Islander (Coef. = -0.27; $p < 0.00$), being the mother's third or subsequent child (Coef. = -0.16; $p < 0.00$), being a multiple birth (Coef. = -3.65; $p < 0.00$), rising maternal age (Coef. = -0.01; $p < 0.00$), offspring being male (Coef. = -0.10; $p < 0.00$) and being born to a mother who smoked cigarettes during gestation (Coef. = 0.25; $p < 0.00$). Conversely, partnered marital status lengthened gestation (Coef. = 0.35; $p < 0.00$).

Multiple linear regression is used to explore the effect on each sex of fire exposure in 2009 on gestation. Regression analysis shows that there were no significant effects of fire exposure on gestational length in males or females at any point in gestation, consistent with previous analysis.

Effects of other maternal characteristics were consistent between the sexes, with decreases in gestational length associated with identifying as Aboriginal or Torres Strait Islander (Coef. = -0.21, $p = 0.04$ for males and Coef. = -0.33, $p = 0.00$ for females), smoking in pregnancy (Coef. = -0.24, $p < 0.00$ for males and Coef. = -0.24, $p < 0.00$ for females), more than two previous pregnancies (Coef. = -0.12, $p < 0.00$ for males and Coef. = -0.21, $p < 0.00$ for females), rising maternal age (Coef. = -0.01, $p < 0.00$ for males and Coef. = -0.02, $p < 0.00$ for females) and being a multiple birth (Coef. = -3.68,

$p < 0.00$ for males and Coef. = -3.63, $p < 0.00$ for females). Coupled status lengthened gestation (Coef. = 0.35, $p < 0.00$ for males and Coef. = 0.34, $p < 0.00$ for females).

## 4.11. *Discussion*

### 4.11.1. **Canberra fires**

This analysis indicates that the mother-foetal dyad being present in the heavily impacted areas during gestation (as indicated by reporting a residential address in fire affected area at birth) had a significant effect on the birth weights of male foetuses. This effect resulted in males showing a pattern of higher birth weights, accompanied by a partially corresponding increase in gestational length. For those babies born between April and September 2003 (those exposed in the first trimester) there was no corresponding increase in gestational length, suggesting accelerated *in utero* growth. Although significantly higher and lower birth weights are detected in the heavily affected area between 2008 and 2010, the inconsistency and temporal dislocation of this pattern suggests it is not fire related and may not relate to a common cause (O'Donnell and Behie 2015).

Macrosomia poses important health risks to both mother and neonate. Although the influence of fire on birth weight was smaller than the influence of other known factors, such as maternal smoking, the gain of almost 200 grams detected is likely to be of clinical relevance in many cases. In the immediate-term, macrosomia heightens the risk of unplanned caesareans, assisted delivery, shoulder dystocia, newborn asphyxia and perineal tears – presenting risks to both mother and child (Henriksen 2008). As adults, macrosomic infants report higher rates of obesity and metabolic disorders, indicating the possible action of foetal programming of bodyweight (Calkins and Devaskar 2011; Henriksen 2008). As such, the potential causes of macrosomia in this cohort deserve close attention.

The findings of this study are contrary to the general trend of prematurity and lowered birth weights that have been documented by several previous studies and, as such, may be an effect created or exacerbated by the small available sample arising from this disaster, particularly because significant trends only emerged in one sex.

Other maternal behaviours appear not to explain the trend. Maternal smoking reduced across the ACT in the fire year, although to a lesser degree in the heavily affected area. Maternal smoking has a strong depressive effect on baby weight. Although reported smoking was included in the model, unreported smoking could not be accounted for. However, assuming that unreported smoking followed similar patterns to reported smoking, changes in maternal smoking do not adequately explain the increase in birth weights immediately after the fire (between January and March) (O'Donnell and Behie 2015).

The following three previous studies report patterns of higher birth weights and lengthened gestation: Oyarzo et al. (2012), Margerison-Zilko et al. (2015) and O’Donnell and Behie (2013). O’Donnell and Behie (2013) hypothesised that patterns of longer gestation might reflect altered maternal decision making regarding obstetric intervention following disasters (O'Donnell and Behie 2013). Psychologically, disasters are understood to negatively affect an individual's sense of control, meaning that women may potentially seek to reassert this control through health decisions. Further some studies report that women feel their babies are safer *in utero* following disasters (Badakhsh et al. 2010). Such psychosocial effects might lead to women delaying intervention and thus prolonging gestation. However, in this case, lengthened gestation only partially correlates with birth weight changes and it thus seems unlikely that alterations in maternal decision making would explain the sex differentiation in gestation and birth weight  (O'Donnell and Behie 2015).

In their recent study, Margerison-Zilko et al. (2015) find extended gestation and higher birthweights following the September 11 terrorist attacks. The authors suggest that prolonged gestation is a protective adaptation to a highly stressful environment. Certainly, this finding is consistent with the effects of maternal adrenaline in delaying the onset of parturition, a likely defence mechanism against threats at the end of gestation.

Oyarzo et al. (2012) suggest that the secondary pattern of foetal macrosomia found in their study reflects the prevalence of GDM in the study population. Their analysis was able to show that GDM prevalence was not significantly different in relation to fire exposure and that, in 2003, 4.4% of women had a diagnosis of GDM, which is consistent with national levels (Li et al. 2013). Importantly, this dissertation finds no increased prevalence in GDM among the mothers of male neonates between the three levels of fire exposure. Thus, diagnosed GDM does not appear to explain the observed macrosomia in this instance  (O'Donnell and Behie 2015).

However, as GDM is usually only screened for once during gestation for otherwise low-risk pregnancies and has few obvious symptoms, it is plausible that mothers whose blood glucose has increased following screening or who presented with subclinical elevations could be missed by standard screening. Hence, elevation of maternal blood glucose via the effects of stress on both a somatic mechanism and a behavioural mechanism could explain the observed covariance (O'Donnell and Behie 2015).

In terms of somatic mechanisms, increased cortisol levels in humans drive higher blood glucose via gluconeogenesis (Khani and Tayek 2001). It is known that a complex relationship exists between maternal psychosocial stress in pregnancy and blood cortisol levels (Wadhwa et al. 1996). Any cortisol mediated relationship between stress and blood glucose in pregnancy is necessarily complicated by alterations in hormonal function during pregnancy and changes in maternal responsiveness to exogenous stress. This is communicated physiologically through the action of both the Hypothalamus-Pituitary-Adrenal (HPA) axis and the Hypothalamus-Pituitary-Ovarian (HPO) axis (Wadhwa et al. 1996).

Maternal endocrinology undergoes systematic changes during pregnancy. The placenta acts as temporary endocrinal agent and secretes the necessary placental steroid hormones to support foetal maintenance, growth, maturation, and parturition. During human pregnancy, corticotropin-releasing hormone (CRH) is released by the placenta in increasing quantities as the pregnancy progresses. Although the placenta metabolises much maternal cortisol, and foetal cortisol concentrations are 5–10 times lower than maternal levels, around 10% of maternal cortisol crosses the placental barrier (Ellison 2009). Placental CRH stimulates the production of maternal cortisol, establishing a positive feedback loop which supports the increase of CRH, ACTH, and cortisol together. Although a placental product, placental ACTH remains bioactive in the maternal bloodstream (Wadhwa et al. 1996) and thus functions to raise maternal blood glucose.

Mothers experience a compensatory dampening in cortisol responsivity as pregnancy progresses (Glover 2015), although the exact timing of clinically significant reductions in responsivity remain unclear (O'Donnell et al. 2009). This alternation of responsivity means that data from non-pregnant participants cannot be assumed to be representative of endocrine responsivity in pregnant women (O'Donnell and Behie 2015).

There is evidence of a modest relationship between maternal self-reporting of anxiety or stress and maternal stress hormones in pregnant women (O'Donnell et al. 2009; Obel et al. 2005; Wadhwa et al. 1996). For example, while the interaction between stress and blood chemistry is variable across gestation, Wadhwa et al. (1996) find that variation in production of cortisol and placental ACTH is related to maternal experience of stress even into the third trimester. Likewise, maternal salivary cortisol levels are also positively associated with reports of psychosocial stress (Nepomnaschy and Flinn 2009; Obel et al. 2005).

Because evidence suggests that some degree of maternal hormonal responsivity to psychosocial stress persists throughout pregnancy (although moderated by falling responsiveness), maternal experience of psychosocial stress may also be associated with heightened blood glucose levels due to the glucogenic effects of cortisol. Since glucose is able to cross the placental barrier, a hyperglycemic intrauterine environment may result from higher maternal blood glucose (O'Donnell and Behie 2015).

Secondly, with regard to behavioural mechanisms, both increased food intake and increased consumption of low-nutrition but high-calorie foods have been documented as a behavioural response to stress during pregnancy (Hurley et al. 2005; Lobel et al. 2008). Increased maternal food consumption in response to stress has only been found significant in energy-dense food categories, such as bread, sweets, oils, and fats (Hurley et al. 2005). The HPO axis can act to upregulate steroid hormone production when energy is abundant (Ellison 2009). As the Canberra fires disaster was not sufficient to interrupt food supply, mothers in the analysis would have had abundant food access. Under conditions of high energy availability, excess macro-nutrition combined with the actions of stress hormones may result in higher maternal blood glucose, potentially creating a higher-than-usual supply of energy to the foetus (O'Donnell and Behie 2015). Further, official advice to stay indoors, combined with smoky and hot weather conditions, may act to reduce usual maternal exercise levels (O'Donnell and Behie 2015).

Lastly, greater responsiveness of male foetuses to maternal stress, often measured in terms of sex differentials in early pregnancy loss under conditions of stress, is well documented in the literature (Catalano and Bruckner 2006; Peterka et al. 2004). Male foetuses also display more rapid natal growth and higher average birth weight than females. Retnakaran et al. (2015) show that mothers carrying male foetuses have increased odds of developing GDM (odds ratio: 1.39, 95% CI: 1.01–1.90) when compared to mothers carrying female foetuses. Having male offspring also increases the effects of other GDM risk factors, such as maternal age over 35 years and non-white ethnicity (Retnakaran et al. 2015). The increased birth weight observed in the male children of exposed mothers may represent an intersection of male foetal stress response and heightened risk factors for GDM among exposed women due to stress, potential nutritional changes, and carrying a male child (O'Donnell and Behie 2015).

Torche and Kleinhaus (2012) find that females showed greater declines in birth weight when exposed to an earthquake *in utero*, although this is an uncommon finding in the literature. Importantly, however, they note that sex-linked differences in birth weight suggest differential functioning of the placenta under stressful conditions. As the placenta originates from foetal cells, it is “inherently sex-specific” and, accordingly, aspects of the placenta’s functioning, such as epigenetic expression may differ by sex (Torche and Kleinhaus 2012 p. 563). Changes in placental functioning and growth are also identified by Coall and Chisholm (2003) as a key component of the maternal-foetal response to environmental pressure. Thus, placental function under stress and potential sex-specific aspects of functioning warrant future work.

### 4.11.2. Black Saturday

This analysis finds little or no effect of bushfire exposure on the gestational age or birth weight of neonates exposed to the Black Saturday fires. This is in contrast to O’Donnell and Behie (2013) which finds effects on both gestational age and birth weight. According to this earlier study, being born to a mother resident in the Black Saturday fire affected areas during gestation is associated with a small but significant increase in preterm births ($p$ = 0.04) and a decrease in birth weight ($p$ = 0.01). These effects appeared among infants exposed in the later stages of pregnancy. However, the O’Donnell and Behie (2013) study is not able to account for maternal factors, such as age or smoking status, and therefore limited in its ability to separate true fire effects from other types of regional variation.

The results of this more detailed analysis suggest that the effects on gestation and birth weight previously detected might be better attributed to regional differences in maternal demographics, such as maternal age, couple status, or ethnicity, or maternal behaviours, such as the prevalence of smoking during pregnancy. Where these factors are controlled, there are very limited effects of fire exposure. This dissertation finds no effects when the sexes are analysed separately. Noting that only a small sample was available to this analysis, it appears that any effect on neonates was too small or infrequent to be detected by the methods used here.

Moreover, this analysis finds that there is consistent annual regional variation in birth weights between the four areas defined in this study (those areas that were heavily affected, threatened, surrounding, or unaffected by the Black Saturday fire). Regional variation may be due to a range of socioeconomic and demographic factors which cannot be captured and analysed by this study. Further, maternal smoking could only be included to a limited degree as this was not collected prior to 2009 and the initial years of collection suggest some underreporting may have occurred as reported rates are lower than national averages.

The important influence of smoking is demonstrated by the role of identifying as Indigenous within this analysis. Although being Indigenous was significant prior to 2010, it is not significant from 2010 onwards. This suggests a link between Indigenous status and smoking behaviour. As maternal smoking is better accounted for in the analysis, the importance of Indigeneity wanes indicating that it may have been acting as a proxy variable for smoking.

Rates of smoking among Indigenous Australians are nearly double those of non-Indigenous Australians (Purcell 2015). Studies of smoking among pregnant Aboriginal women find very high rates, ranging from 41% (in Far North Queensland) to 67% (in Perth) of mothers reporting smoking during pregnancy. In Victoria, around 40% of Aboriginal women admitted to hospital a month prior to delivery are smokers compared to 8% of non-Aboriginal women (van der Sterren and Goreen Narrkwarren Ngrn-toura Project Team 2010). The high incidence of smoking among Indigenous women also likely represents an intersection between class, youth and ethnicity. Both mothers under 25 years and mothers from the lowest socioeconomic quintile are more likely to smoke (Dennis 2016), and these groups are over-represented among Indigenous mothers (Australian Institute of Health and Welfare 2009).

Previous research shows that although Aboriginal women are aware of the general health risks of smoking they are less aware of the specific risks to the foetus (van der Sterren and Goreen Narrkwarren Ngrn-toura Project Team 2010). While pregnancy is an important motivating factor for smoking cessation in Aboriginal women, smoking cessation interventions that are effective in mainstream communities do not appear to be as effective for Aboriginal mothers, particularly where they are also struggling with issues of poverty and violence (van der Sterren and Goreen Narrkwarren Ngrn-toura Project Team 2010). Smoking cessation may also have greater costs for Indigenous mothers than for mainstream mothers. Within some Indigenous communities, smoking reflects both traditional practice and a cultural response to dispossession. Further, in communities where smoking is the norm, cessation may lead to social isolation (Dennis 2016). More generally, smokers confronted with health messages do not always make connections between the impact of smoking on other people's bodies and the likelihood of a similar impact on their own, believing instead that they will not come to harm (Dennis 2016).

Smoking during pregnancy presents direct survival risks to the foetus, raising the likelihood of both miscarriage and stillbirth (Rogers 2009; Shiverick and Salafia 1999). In terms of this dissertation, maternal smoking is strongly and consistently associated with preterm birth and lowered birth weight (Peacock et al. 1995; Rogers 2009; Shiverick and Salafia 1999). There are also lifelong effects on the health and wellbeing of the foetus (Behie and O'Donnell 2015; Hackshaw et al. 2011). These are discussed in more detail in Chapter Seven. Significantly, some effects of smoking are lifelong and may only emerge in later life. This contradicts the observations made by some smoking mothers that the babies of smoking peers (or themselves, if born to a smoking mother) have been born healthy and therefore the risks of smoking are overstated (Dennis 2016).

The Canberra fires highlight the potential for environmental disasters to also affect smoking, with smoking declining in the fire year without other apparent causes (e.g. there were no new preventative health campaigns, policies or legislative changes enacted that year to reduce smoking). Unfortunately, further examination of smoking was not possible in the Victorian sample due to insufficient data.

While this dissertation does not find the effects of stress arising from fire to be an important factor for healthy birth weight in the Victorian population, it affirms the influential role of smoking in foetal wellbeing and also restates the importance of more effective cessation supports for Indigenous women and girls. The absence of a strong fire effect differs from the effects of other disasters reported in the literature and discussed extensively in this chapter (Auger et al. 2011; Dancause et al. 2011; Glynn et al. 2001; Harville et al. 2010; Xiong et al. 2008). It also differs from the results shown earlier in relation to the Canberra fires (O'Donnell and Behie 2015).

The difference between the effects of the two fires studied here is perhaps related to the nature of fire disasters themselves and the very different natures of the two fires studied. Wildfires, unlikely floods, tsunamis, or earthquakes, are often highly erratic in their impact. For example, fire can raze one house while leaving its neighbours undamaged. As such, it is difficult to estimate how stress is distributed at a population level. This is particularly the case of a fire like Black Saturday which threatened homes over many weeks. In contrast to the Canberra fire, which occurred on a single day and impacted one medium-sized community (Canberra had a population of around 322,000 in 2003) (Australian Bureau of Statistics 2012a), the Black Saturday fires occurred over almost a month and affected dozens of smaller communities and townships, as well as a regional city, differentially (Teague et al. 2010d). While stress effects on gestation may have occurred as a result of the Black Saturday fires, these effects may be too disparate to detect using whole-of-population methods. Chapter Eight in this dissertation looks at women's experience of stress more closely and finds this varies greatly both between women within the same fire and between survivors of different fires. Whole-of-population methods necessarily encounter difficulties in accurately reflecting highly heterogeneous experiences accurately.

Further research is needed to determine the role of disaster type with regards to the effects of stress during pregnancy. Such research will assist in casting light on the specific effects of stress *in utero*, including developing current understandings of when stress is either adaptive or toxic and how these thresholds might differ from person to person.

## 4.12. *Chapter summary*

The two fires studied in the section show different biological responses to external environmental threats. While mothers exposed to the Canberra fire delivered higher weight infants, mothers exposed to the Black Saturday fires did not show a detectable response. The reasons for these differences may lie in actual effects – particularly regarding the nature of fires and the stress they create – or in methodological weaknesses. This is also related to the difficulties of determining just who is affected when fires behave in a manner that leaves a patchwork of burnt areas through numerous communities, such as the pattern created by the Black Saturday fire. These issues are addressed in Chapter Nine; which presents conclusions from both this analysis and the dissertation as a whole. The next chapter examines the impact of the two fires on indicators of early pregnancy loss: plurality rates and secondary sex ratio.

# 5. EFFECTS OF FIRE EXPOSURE ON EARLY FOETAL LOSS IN TWO POPULATIONS

## *5.1. Introduction*

As a retrospective cohort study of birth records in Victoria and the Australian Capital Territory (ACT), this chapter investigates changes in secondary sex ratio (SSR) and plurality rate among live born neonates in relation to maternal bushfire exposure. Both the SSR and plurality rate can act as post-partum indicators of underlying rates of spontaneous abortion and stillbirth in populations. The approach taken in this chapter has been informed by a pilot study to this research (see O'Donnell and Behie 2013).

LHT predicts that highly stressed mothers should experience a decreased capacity to invest in offspring (see Chapter 2). Biologically, this manifests as a heightened propensity toward spontaneous abortion of male foetuses and increased loss among multiple conceptions. While proximate evidence of changes in the rates of loss is difficult to measure empirically – as many losses occur before the pregnancy is clinically recognised (Ellison 2001) – the results of the increase in rates of loss can be observed through decreases in sex ratio or in multiple birth rates.

### 5.1.1. Research question

The aim of this chapter is to examine the effects of bushfire-related maternal stress on foetal loss. It uses population measures of the SSR and plurality rate as evidence of prenatal losses. The central question concerns the effect of prenatal bushfire exposure on birth weight, gestational age, plurality, and secondary sex ratio on populations exposed to the Canberra and Black Saturday bushfires. This chapter especially focuses on plurality and SSR because these both relate to underlying loss of conceptions.

Because of the documented effects of stress on early loss and SSR, the hypothesis is that both SSR and plurality rates will decline in response to disaster exposure. Where SSR declines are expected, plurality rates should also fall due to the effects of lost conceptions.

## *5.2. Background*

### 5.2.1. Early foetal loss

In humans, conception is fragile and foetal losses in early pregnancy are both substantial and a fundamental aspect of human evolution. In part, this high rate of loss is due to characteristics of the human menstrual cycle. Because the endometrium is shed and regrown monthly, if an ovum is fertilised before or after the endometrium is ready for implantation, the conception will necessarily be lost as the blastocyst will be either unable to implant in the immature endometrium or will be shed along with the endometrium.

Early pregnancy loss, and the selective pressures that drive it, are important in understanding how loss and miscarriage might interact with stress. Notably, from the studies presented earlier, there is little difference between rates of loss in women living in developed and developing nations (if anything, in these studies, losses in industrialised nations were higher, potentially due methodological factors, such as better access to accurate testing facilities) (see Chapter Two). This suggests that differences in resource availability or health care access are not necessarily primary factors in early foetal loss.

In multiple pregnancies, early loss does not always affect all of the conceptuses (Landy and Keith 1998). Rather, it is understood to be relatively common for one or more of multiples to be lost and the remaining conceptions to continue to term. Known as the "vanishing twin", these conceptions can be either reabsorbed into the endometrium or can present as a vaginal bleed in early pregnancy. The rate of loss among multiples is high. Where two embryonic sacs are observed in an unassisted pregnancy, the loss of one twin is expected in around 40% of cases. The loss rate falls to around 7% once two embryos are observed and loss rates are higher where a greater number of conceptus are observed (Landy and Keith 1998). Where reabsorption occurs, it can be impossible to detect the existence of a vanished twin unless early ultrasounds are conducted or products of conception (such as additional gestational sacs) remain. Even with modern substantial improvements in sonography, it can be difficult to distinguish evidence of a lost twin from other normal embryonic structures (Landy and Keith 1998). From an evolutionary viewpoint, the pressures of selection act on multiple conceptions just as they do on single conceptions (or even more powerfully because of the additional maternal strain) and that selective pressure that creates a lower chance of survival in the more fragile conceptions (Catalano et al. 2013).

Because human offspring require substantial investment to survive, it is important that parents, and particularly mothers, are highly selective about which offspring they invest in. One of these biological "decisions" concerns whether the mother carries to term, with selection pressures particularly pronounced during gestation (Brown et al. 2013). The high rates of early loss observed in humans suggests a strategy that selects only the strongest conceptions, allowing parents to maximise potential benefits. In particular, mothers must aim to limit any investment in foetuses carrying genetic abnormalities which severely limit their lifespan (e.g. genetic conditions such as trisomy 16, which commonly causes prenatal death). Because such conditions prevent the child from reaching reproductive age, they present a genetic "dead-end" by preventing the passing on of the parents' genes. Even among survivors of multiple conceptions, rates of congenital abnormalities are higher than with singletons (Pharoah et al. 2009), suggesting that selection is removing only those conceptions unable to survive gestation and birth. Any investment made in children unable to survive will not have a biological benefit but does impose substantial biological costs. This biological accounting, of course, does not allow for the social and emotional benefits that these children confer to their parents.

### 5.2.2. Sex-selective loss

Both the higher metabolic cost of males and their genetic frailty indicate greater selective pressure on males during gestation. While the genetic determination of sex is relatively clear, how sex selection occurs, and thus how changes in primary and secondary sex ratio might occur, remains an area of considerable debate. In animal studies, the process of *in utero* sex selection has been shown to be responsive to environmental pressure and able to adapt relatively quickly under certain conditions (Marin and Baker 1998). For example, the common housefly, *Musca domestica,* shows a wide variation in sex determination. In some variants, males and females are chromosomally indistinguishable, while in others sex determination is dependent on a masculising Y-linked gene (Marin and Baker 1998). Because these closely related variants show diffing methods of sex determination it implies recent evolutionary change. Potentially, genes related to sex determination are able to respond rapidly to positive pressures. This is because changes in these genes result in changes in the population sex ratio which are advantageous for the adapting organism. Accordingly, such genes are able to rapidly establish a genetic foothold (Marin and Baker 1998). However, what is true of short lived insect species may not necessarily be true of longer-lived mammal species. Further, the role of the Y-chromosome as the sex determinant in human species appears relatively conserved (Marin and Baker 1998).

In humans, some authors suggest that stress can change paternal factors, such as differential sperm motility, which leads to fewer male conceptions and hence a reduced PSR (Fukuda et al. 1998). Fukuda et al. (1996) find that among men with previously normal sperm motility, sperm motility was significantly decreased in the month after their houses were destroyed in an earthquake. Normal motility in the men was gradually restored over two to nine months. Fukuda et al. (1996) propose that one outcome of reduced motility is lowered SSR. Others propose increased male foetal loss as the most substantial contributor to the observed change and thus a decline in the SSR (Catalano and Bruckner 2006). Still others suggest that the ovum is able to make a choice between X- or Y-chromosome bearing sperm (known as cryptic female choice), potentially in response to a balancing or ratio-dependent (i.e. Fisherian) selective pressure (Ellison 2001).

A principal challenge in determining where sex ratio changes occur is the difficulty of studying conception and the PSR in humans. In this respect, the existing evidence appears contradictory. Some studies find an excess of male conceptions, while others suggest that unity at conception is most probable. For example, Garenne (2011) finds that a PSR of 1.6 results in an SSR of 1.05, attributing this change to a higher rate of loss among male foetuses. However, studies which examine the distribution of Y- and X-chromosomes in sperm samples (as a method of determining the likely PSR) find that Y- and X-chromosomes are equally distributed and that, because of this, there is no reason not to assume unity (or near unity) at conception (Goldman et al. 1993; Lobel et al. 1993). In accordance with this, Kano (2010) argues that, if the primary sex ratio can be assumed to be 1.0 (unity), then the usual SSR observed of around 1.05 must mean that more loss occurs among female foetuses.

The direct evidence for arguments supporting increased male (rather than female) foetal loss is insufficient. Although one early study shows an increased rates of male foetal loss in otherwise outwardly observably healthy abortuses, the ratio of loss in abortuses with observable abnormalities favours female foetuses (Byrne and Warburton 1987). More recent studies examining detectable genetic abnormalities in abortuses do not find strong evidence of selection against males. These studies either do not detect any male excess (Del Fabro et al. 2011) or find a female excess (Kano et al. 2010). However, both Kano et al. (2010) and Del Fabro et al. (2011) limited their studies to couples with histories of recurrent spontaneous abortion and genetically abnormal abortuses, suggesting that these results may not be generalisable to other populations.

Despite the difficulty in demonstrating male frailty *in utero* in a clinical setting, declines in the SSR at a population level are quite commonly observed following extreme population stressors (Catalano and Bruckner 2006). These are documented in more detail below. Although little work exists, sex selective effects should present equally in single and multiple conceptions.

### 5.2.3. Effects of environment on conception

Although SSR in human populations has, in recent history, remained stable at around 1.05, resulting in a slight excess of males at birth, some of the literature records a decline in SSR in the past five decades in North America and parts of Europe. This appears most pronounced in industrialised nations (Del Giudice 2011; Dickson and Parker 1997; Dodds and Armson 1997; Dyson 2012; Grech et al. 2003; Hamilton and Rasmussen 2010; Irudaya Rajan et al. 1992; James 1996; James 1998; MacKenzie et al. 2005; Magnuson et al. 2007). However, others argue that this decline has ceased. James (1996) states that, although the SSR in the United Kingdom fell between 1973–1990, it then increased between 1990 and 1994. While agreeing that there was an increase in SSR between 1990 and 1994, Dickson and Parker (1997) contend that this increase was insufficient to arrest the broader trend.

A wide range of factors are either causal to or associated with changes in SSR. These include: technological changes, such as the use of fertility treatments (Gutiérrez-Adán et al. 2001); biological changes, such as reductions in sperm motility arising from exposure to war and disaster (Bisioli 2004; Fukuda et al. 1998; Fukuda et al. 1996; Grant and Yang 2003; James 2003; O'Donnell and Behie 2014; Suzuki et al. 2015);

differing hormone levels in parents at the point of conception (Bisioli 2004); environmental changes, particularly persistent environmental pollutants (James 1998; MacKenzie et al. 2005; Terrell et al. 2011) and climate changes (Fukuda et al. 2014); and flaws in research method, particularly the use of inaccurate census data (Irudaya Rajan et al. 1992) or flawed methods of analysis (Dickson and Parker 1997). Some older studies link declines in SSR to the father's profession, with stressful professions (such as being a fighter pilot or deep-sea diver) resulting in declines (Lyster 1982; Snyder 1961). Some suggest causes, such as environmental temperature (Grech et al. 2003), environmental lead exposure (Jongbloet and Roeleveld 2007) and maternal diet (Cramer and Lumey 2010), however, these have not been supported. In some countries, such as India and China, substantial changes in SSR appear to have resulted from sex-selective termination of female foetuses, rather than biological or environmental factors (Irudaya Rajan et al. 1992).

Grech et al. (2003) best summarise the state of the evidence when they suggest that there are no reasonably certain causes of changes to SSR and that the causes are, in any case, most likely to be multifactorial. However, even in the developed world, environmental disasters still appear to act as a sufficient pressure to alter the SSR. Limited but growing research has documents declines in the SSR following environmental disasters. Similarly, wars, acts of terrorism, economic decline and workplace stress are shown to influence the SSR. An obvious commonality between these situations is that they are stressful for parents.

Economic decline can be expected to be a cause of widespread population stress, as average personal incomes diminish and people suffer financial hardship. With regard to the collapse of the East German economy in 1991, Catalano (2003) found that the SSR fell in East Germany compared to that of the more affluent West Germany, even when pre-existing differences in affluence are controlled. Similarly, Catalano and Bruckner (2005) find that the Swedish SSR correlated to the health of the economy between 1862 and 1991. They find that for every 1% decrease in the consumption of consumer goods, there were 25 fewer males born. Similarly, Greenland's SSR shifted from a traditional excess of females (a low SSR) to an excess of males (a high SSR) as industrialisation and urbanisation increased the material wealth and healthcare access of the population (Hamilton and Rasmussen 2010). This suggests that as the selective pressure in a subsistence economy with constrained healthcare decreased, a greater number of male foetuses were brought to term. More recently, a decrease in the SSR has been detected in the US following the Global Financial Crisis (Grech 2015).

War and terrorism similarly depress the SSR in most instances (Catalano et al. 2005; Zorn et al. 2002); although war is inconsistent in its effect (Helle et al. 2009). Because terrorism has been less studied, it is not possible compare the consistency of effects. However, in terms of terrorism, Catalano et al. (2005) find that the SSR in California declined by December 2001 to its second lowest rate in 14 years, is linked to the effect of the September 11 terrorist attacks. In terms of war, James (2003) finds that the SSR increased following both world wars, but fell following the Iraq and Slovenian wars. Zorn et al. (2002) also report a significant SSR decline following the 1991 Slovenian ten-day war. SSR declines were also noted following the Nagasaki and Hiroshima bombings (Peterka et al. 2004). Helle et al. (2009) review the effects of Second World War, periods of economic decline, and famine in several nations. They find, consistent with James (2003), that SSR had declined in all instances apart from the Second World War, during which male births increased in the UK. James (2003) suggests that the two world wars may show different patterns in UK populations because the UK was geographically removed from the conflict (not withstanding it was bombed during both wars). James (2003) also suggests that high coital rates associated with soldiers on leave might influence the PSR. Conversely, both the Iraqi and Slovenian declines are measured in the resident populations and might be therefore more related to the maternal experience of stress. Helle et al. (2009 p.1230) states that prenatal stress is the "most compelling" argument for changes to SSR.

Hansen et al. (1999) examine the role of life stressors and find that mothers who had experienced traumatic life stress (such as a familial death) had offspring with a lower SSR. Additionally, exposure near conception appeared to have the greatest impact (trend significant at $p = 0.013$). Similarly, life patterns and health behaviours can influence the SSR. Windham (2001) finds that SSR increased with maternal education status and, unusually, with maternal smoking. In the same review, SSR decreased with younger parental age, greater parental age difference, parental minority status, winter conception, and prior multiple births. Seasonality of SSR is also reported in another study, with lower SSR in spring births compared to autumn births (Magnuson et al. 2007). This contradicts Windham's (2001) findings.

Early studies consider the role of paternal occupational stress. Snyder (1961) examines fighter pilots who were found to father a significantly greater proportion of female children ($p = 0.01$) compared to men in other occupations. Snyder attributed this to the impact of their stressful occupation. Lyster (1982) finds a similar trend in the children of fighter pilots and professional deep-ocean divers. Unfortunately these limited studies do not consider other possible influence on sex ratio, such as the environmental extremes experienced by the fathers. As discussed in Chapter Two, women who hold high status social positions appear to carry a greater number of males, potentially related to lower overall fertility and the fitness benefits of stronger sons (Grant and Yang 2003). In later research, Magnuson et al. (2007) find that paternal occupation level (defined as owner, managerial or other) is more relevant to the SSR than industry sector, with owners and managers more likely to have high offspring SSR. Accordingly, other studies show that lower social class (although harder to define consistently) leads to lower SSR (Navara 2010).

Environmental factors, such as pollution and persistent environmental endocrines, are considered by several studies, but none find convincing evidence of an effect (Dodds and Armson 1997; Jongbloet and Roeleveld 2007; Terrell et al. 2011). Similarly, paternal and maternal testosterone levels may be a contributing factor to SSR (Bisioli 2004) but appears to be not well studied in humans, although maternal levels have been found to be relevant to both stress and atypical sex ratios in other mammals (Grant 2007). At the same time the evidence is, in many cases, also insufficient to refute the possibility of an effect (James 1998). A more recent study examining the steady rise in the sex ratio of foetal deaths in Japan finds an association with climate change and exposure to extreme weather conditions. This rise in the foetal death ratio leads to decreases in the SSR (Fukuda et al. 2014).

The limited number of studies that consider environmental disasters continue to support this general trend. Earthquakes in Kobe, Japan in 1994 (Fukuda et al. 1998), Kerman, Iran in 2003 (Saadat 2008) and the East Japan Earthquake (Tōhoku) in 2011 (Hamamatsu et al. 2014; Suzuki et al. 2015) were linked to reduced SSRs that were statistically significant. A similar, though not significant trend is also found following a 2005 Chilean earthquake (Torche and Kleinhaus 2012). Likewise, Grech and Scherb (2015) find a dose-response relationship between disaster exposure and SSR declines after 2005's Hurricane Katrina. Following the Chernobyl nuclear disaster (1986), there was a decline in the SSR, amounting to the loss of around 400 boys (Peterka et al. 2004). However, an earlier study of the 2009 Black Saturday bushfire in Victoria found no change to the SSR although it is likely that the study period was insufficient (O'Donnell 2012; O'Donnell and Behie 2013).

Internationally, SSRs in industrialised nations are declining and there is no clear reason for this decline. Davis (2007) find that the SSR is declining in Japan and in white population in the US, even as there is no decline in the African-Americans population. This difference may relate to the SSR already being substantially lower in African-Americans. Similarly, Canada shows a small decline in population SSR, which is sometimes attributed to increasing migration from minority groups with lower SSRs. Dickson and Parker (1997) document a steady decline in the SSR in the UK between 1973 and 1990, although James (1996) argues that this decline has since ended and the SSR is now rising. Grech (2003) details SSR declines across Europe, North America, and Mexico. Although European SSRs declined on average, declines were marked in North Europe even as SSR increased in the Mediterranean countries (between latitudes 35–40 degrees). Marginalised ethnicities and populations consistently report lower SSRs, including African-Americans (Davis et al. 2007; Grant and Yang 2003), Canada's First Nations communities (MacKenzie et al. 2005), and immigrant groups (Dodds and Armson 1997).

## 5.3. Method

Because of the methodological difficulties in assessing primary sex ratio, this chapter focuses on the SSR. Because the SSR is measured at birth based on physical characteristics, it is relatively free of cultural determinations of gender. Although some babies do not have a determinable sex at birth, this rate is extremely low in Australian populations (less than 1%) (Australian Institute of Health and Welfare 2009; O'Donnell 2012). Also, since it is a measure that is relatively free from cultural and technological confounds, the SSR, its variation, and norms, may provide an insight into the functioning of selection and adaptation in human populations as they respond to environmental and social changes.

In Australia, recording of sex at birth is routine and accurate, providing reliable and consistent data. In addition, sex-selective abortion is both illegal and socially taboo. Because of this, the SSR provides a measurable variable which is likely to indicate any biological response to bushfire exposure which has altered the selective pressures of males and females *in utero*.

### 5.3.1. Ethical approval

Approval of the ethical aspects of this dissertation was granted by The Australian National University Human Research Ethics Committee (ANU-HREC) on 23

September 2013 (project reference: 2013/143). For the Canberra sample, further necessary approvals were granted by the ACT Government Human Research Ethics Committee's Low Risk Sub-Committee on 5 December 2013 (project reference: ETHLR.13.330). For the Victorian sample, further necessary approval was granted by the Victorian Department of Health Human Research Ethics Committee on 4 December 2014 (project reference: 20/14).

### 5.3.2. Sampling strategies and collection methods

The sampling strategy and collection method applied to this study are the same as those applied in Chapter Four. The following variables were used in the analysis: baby's sex; plurality; mother reporting a residential address in a fire area, created from statistical subdivision codes provided in the raw data; and timing of exposure.

Total ACT sample numbers are shown below, there was a total of 48,408 births represented.

Table 5-1: Total ACT sample by baby's year of birth and fire exposure. The mother's residential location is used to estimate fire exposure at three levels as either heavily, moderately or leat fire affected

| Year of birth | Heavily affected | Moderately affected | Least affected | Total |
|---|---|---|---|---|
| 2000 | 216 | 2805 | 1139 | 4160 |
| 2001 | 256 | 2495 | 1131 | 3882 |
| 2002 | 275 | 2592 | 1171 | 4038 |
| 2003 | 255 | 2583 | 1269 | 4107 |
| 2004 | 271 | 2579 | 1255 | 4105 |
| 2005 | 288 | 2664 | 1326 | 4278 |
| 2006 | 295 | 2821 | 1455 | 4571 |
| 2007 | 304 | 2846 | 1468 | 4618 |
| 2008 | 313 | 2874 | 1600 | 4787 |
| 2009 | 318 | 2946 | 1626 | 4890 |
| 2010 | 300 | 3081 | 1591 | 4972 |
| Totals | 3091 | 30286 | 15031 | 48408 |

In total, 514,346 births were represented in the Victorian analysis, shown in Table 5-2.

Table 5-2: Total Victorian sample by baby's year of birth and mother's residential location. Locations were defined as heavily fire affected, threatened with fire damage, surrounding fire affected or threatened areas (but not directly affected or threatened), and the remainder of the state.

| Year of birth | Heavily affected | Threatened | Surrounding | Rest of VIC | Combined |
|---|---|---|---|---|---|
| 2006 | 539 | 1383 | 5416 | 62492 | 69830 |
| 2007 | 517 | 1416 | 5616 | 64879 | 72428 |
| 2008 | 488 | 1481 | 5539 | 64994 | 72502 |
| 2009 | 511 | 1374 | 5783 | 65300 | 72968 |
| 2010 | 471 | 1470 | 5706 | 66674 | 74321 |
| 2011 | 433 | 1574 | 5734 | 66264 | 74005 |
| 2012 | 446 | 1660 | 5983 | 70203 | 78292 |
| Totals | 3405 | 10358 | 39777 | 460806 | 514346 |

### 5.3.3. Data cleaning procedures

Baby's whose sex was unable to be determined are excluded from the analysis, representing less than 0.02% of the sample.

### 5.3.4. Analysis strategies

Chi-square tests are used to examine changes to both the baby's sex and plurality variables in relation to the following factors: mother reporting a residential address in a fire area and the time of birth in relation to the fire event. The construction of the fire area and birth timing variables are described in detail in Chapter Four.

## *5.4. Preliminary statistics*

Descriptive statistics for both study populations (ACT and Victoria) are presented in Chapter Four.

## *5.5. Results – ACT birth records*

### **5.5.1. Secondary sex ratio**

Analysis shows there was no decrease in SSR associated with fire exposure, although a non-significant decrease in male births occurred in the cohort exposed in the third trimester in the most and least affected fire areas. This decline occurred too late in gestation to represent early foetal loss and, as such, is more likely to represent a change in the sex ratio of still-born babies (see Figure 5-1). There was a significant increase ($p = 0.04$) in the percentage of male births among the cohort born to mothers in the heavily affected areas between April and June 2004, which represents babies that were conceived in the immediate aftermath of the fire.

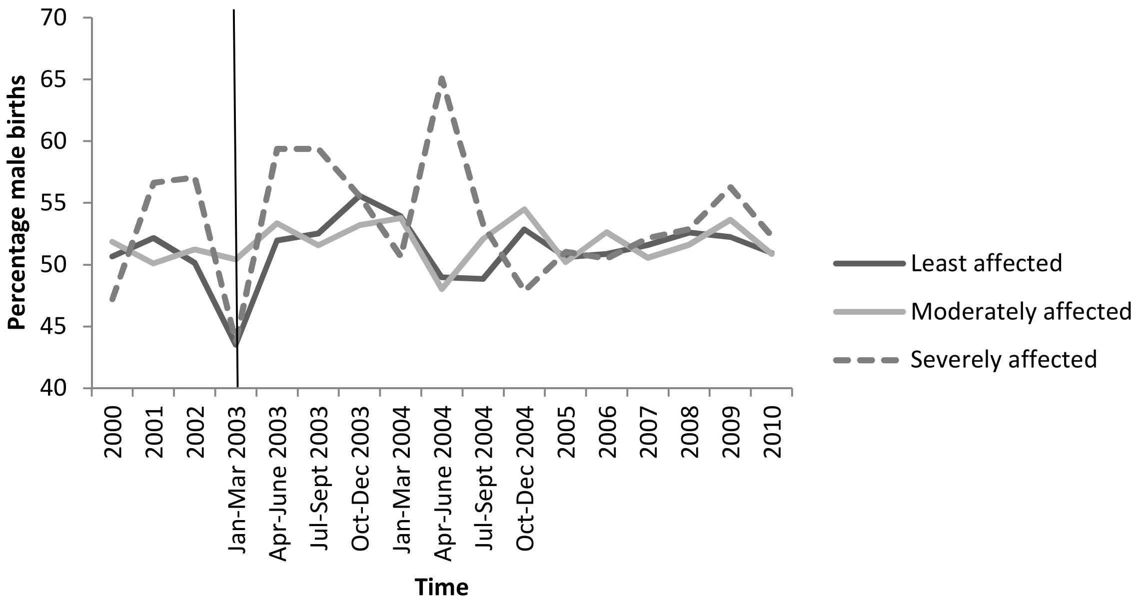

Figure 5-1: The percentage of male live births between 2000 and 2010 with the fire year (2003) and aftermath (2004) split into quarters for clarity. The vertical line indicates the time of the fire.

Table 5-3: Results of chi-square tests for the percentage of male births in the ACT between 2000 and 2010. The three areas of impact (least affected, moderately affected, and severely affected) with approximate timing of fire exposure are also shown. Significant p-values are marked with an asterisk.

| Year | Birth timing in relation to fire | Least affected | Moderately affected | Severely affected | p |
|---|---|---|---|---|---|
| 2000 | Pre-fire | 50.66 | 51.84 | 47.22 | 0.38 |
| 2001 | Pre-fire | 52.17 | 50.1 | 56.64 | 0.1 |
| 2002 | Pre-fire | 50.13 | 51.23 | 57.09 | 0.11 |
| Jan-Mar 2003 | 3rd trimester | 43.52 | 50.42 | 43.75 | 0.11 |
| Apr-June 2003 | 2nd trimester | 51.95 | 53.36 | 59.38 | 0.56 |
| Jul-Sept 2003 | 1st trimester | 52.53 | 51.57 | 59.38 | 0.49 |
| Oct-Dec 2003 | Conceived in aftermath | 55.59 | 53.2 | 55.56 | 0.75 |
| Jan-Mar 2004 | Conceived in aftermath | 53.92 | 53.76 | 50.67 | 0.87 |
| Apr-June 2004 | Conceived in aftermath | 48.97 | 48.03 | 65.08 | 0.04 * |
| Jul-Sept 2004 | Conceived in aftermath | 48.85 | 52.08 | 53.23 | 0.58 |
| Oct-Dec 2004 | Conceived in aftermath | 52.86 | 54.49 | 47.89 | 0.55 |
| 2005 | Post-fire | 50.6 | 50.19 | 51.04 | 0.94 |
| 2006 | Post-fire | 50.86 | 52.64 | 50.51 | 0.48 |
| 2007 | Post-fire | 51.6 | 50.56 | 52.15 | 0.75 |
| 2008 | Post-fire | 52.6 | 51.62 | 52.88 | 0.78 |
| 2009 | Post-fire | 52.25 | 53.63 | 56.29 | 0.44 |
| 2010 | Post-fire | 50.94 | 50.88 | 52.33 | 0.89 |

### 5.5.2. Plurality

A chi-square test shows that the proportion of multiples born in the fire area in 2003 dropped significantly ($p$ = 0.05). This includes a decrease in multiples among those infants exposed to the fire in the first trimester.

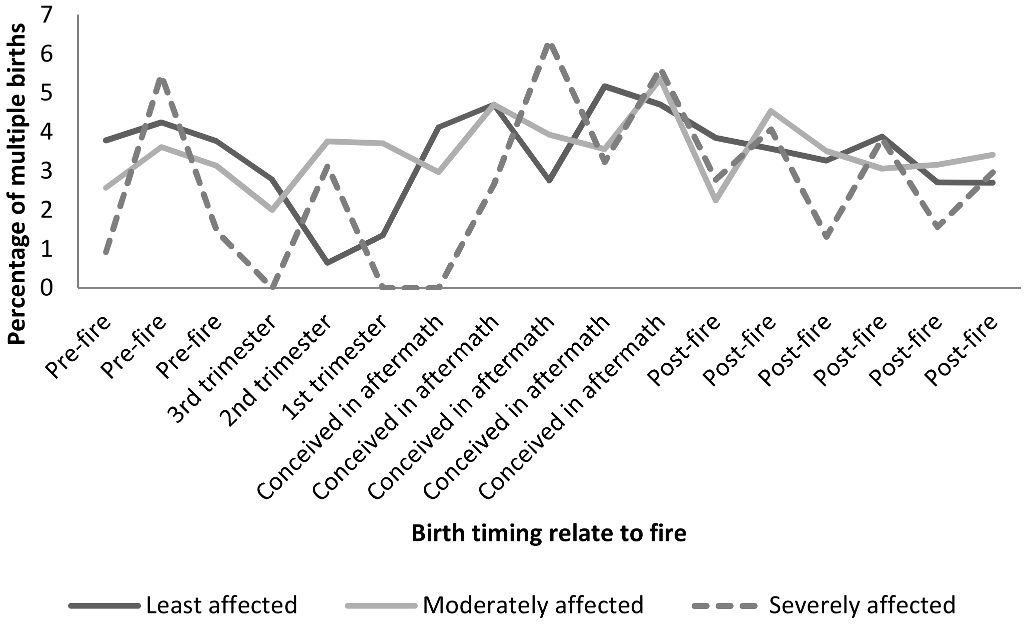


Figure 5-2: Percentages of all births recorded as multiple births (where all types of multiple birth are combined) against approximate timing of fire exposure between the three areas of impact (least affected, moderately affected, and severely affected) based on mothers' residential location.

When divided by trimester, the decline in multiple births in the most exposed cohort occurred among those exposed in the first trimester, which is consistent with predictions of increased early foetal loss.

Table 5-4: Results of chi-square tests for rates of multiple births in the ACT between 2000 and 2010 with approximate timing of fire exposure and the three areas of impact (least affected, moderately affected and severely affected) based on mothers' residential location also shown. Significant p-values are marked with an asterisk.

| Year | Birth timing in relation to fire | Least affected | Moderately affected | Severely affected | p |
|---|---|---|---|---|---|
| 2000 | Pre-fire | 3.78 | 2.57 | 0.93 | 0.03* |
| 2001 | Pre-fire | 4.24 | 3.61 | 5.47 | 0.27 |
| 2002 | Pre-fire | 3.76 | 3.13 | 1.45 | 0.14 |
| Jan-Mar 2003 | 3rd trimester | 2.78 | 2.00 | Nil | 0.35 |
| Apr-June 2003 | 2nd trimester | 0.65 | 3.76 | 3.13 | 0.03* |
| Jul-Sept 2003 | 1st trimester | 1.35 | 3.71 | Nil | 0.04* |
| Oct-Dec 2003 | Conceived in aftermath | 4.12 | 2.97 | Nil | 0.21 |
| Jan-Mar 2004 | Conceived in aftermath | 4.7 | 4.7 | 2.67 | 0.72 |
| Apr-June 2004 | Conceived in aftermath | 2.76 | 3.93 | 6.35 | 0.36 |
| Jul-Sept 2004 | Conceived in aftermath | 5.17 | 3.56 | 3.23 | 0.44 |
| Oct-Dec 2004 | Conceived in aftermath | 4.70 | 5.36 | 5.63 | 0.83 |
| 2005 | Post-fire | 3.85 | 2.25 | 2.78 | 0.02* |
| 2006 | Post-fire | 3.57 | 4.54 | 4.07 | 0.33 |
| 2007 | Post-fire | 3.27 | 3.51 | 1.32 | 0.12 |
| 2008 | Post-fire | 3.88 | 3.06 | 3.83 | 0.32 |
| 2009 | Post-fire | 2.71 | 3.16 | 1.57 | 0.24 |
| 2010 | Post-fire | 2.70 | 3.41 | 2.97 | 0.38 |

## *5.6. Results – Victoria birth records*

### 5.6.1. Secondary sex ratio

Analysis shows there was a significant decrease in SSR associated with fire exposure in 2009 ($p$ = 0.03) with the percentage of male births equalling 46.6% in the severely affected region, compared to 52.4% in less affected regions and 51.1% in the remainder of Victoria when analysed by year (see Table 5-5).

Table 5-5: Results of chi-square tests for the percentage of male live births in Victoria between 2006 and 2012 by year and by the four areas of impact (heavily fire affected, threatened with fire damage, surrounding areas, and rest of state - based on mothers' residential location). Significant p-values are marked with an asterisk.

| Year | Heavily affected | Threatened | Surrounding | Rest of State | p |
|---|---|---|---|---|---|
| 2006 | 49.17 | 51.19 | 51.7 | 51.61 | 0.71 |
| 2007 | 52.61 | 52.68 | 51.26 | 51.29 | 0.70 |
| 2008 | 54.1 | 53.14 | 51.72 | 51.42 | 0.36 |
| 2009 | 46.58 | 52.4 | 52.41 | 51.12 | 0.03* |
| 2010 | 50.96 | 50.75 | 51.12 | 51.31 | 0.97 |
| 2011 | 52.66 | 53.18 | 50.82 | 51.62 | 0.37 |
| 2012 | 50 | 51.08 | 50.33 | 51.48 | 0.35 |

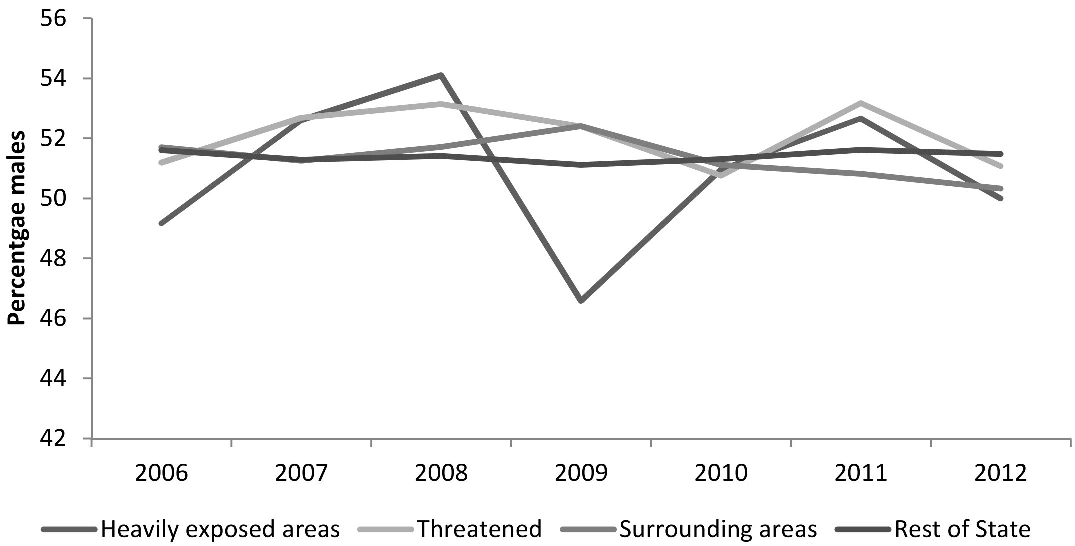


Figure 5-3: Percentage of births which were male by exposure status (heavily exposed, threatened, surrounding, or rest of state) between 2006 and 2009. The decrease in the heavily exposed area was significant in chi-square analysis at p = 0.03.

When divided by trimester of exposure, this decrease is no longer significant (first trimester exposure: $p$ = 0.17; second trimester exposure: $p$ = 0.17; and third trimester exposure: $p$ = 0.59). This is most likely due to smaller group size but still shows a clear trend of decreases in SSR through all three trimesters of exposure for births to mothers in the severely affected area, with the greatest decrease among infants exposed in the second trimester (see Table 5-6).

Table 5-6: Results of chi-square tests  for the percentage of male live births in Victoria between 2006 and 2012 by timing of exposure and by the four areas of impact (heavily fire affected, threatened with fire damage, surrounding areas and rest of state) based on mothers' residential location. Significant p-values are marked with an asterisk.

| Birth timing in relation to fire | Severely affected | Threatened | Surrounding | Rest of state | $p$ |
|---|---|---|---|---|---|
| Pre-fire | 51.86 | 52.29 | 51.62 | 51.41 | 0.65 |
| 3rd trimester | 46.75 | 50.82 | 52.41 | 50.88 | 0.38 |
| 2nd trimester | 44.23 | 56.54 | 51.90 | 51.59 | 0.17 |
| 1st trimester | 45.99 | 51.97 | 50.62 | 51.38 | 0.59 |
| Conceived 300 days after fire | 52.43 | 50.61 | 51.78 | 51.25 | 0.82 |
| Conceived 300-599 after fire | 49.43 | 52.80 | 51.01 | 51.47 | 0.61 |
| Conceived 600-899 after fire | 51.24 | 50.96 | 50.61 | 51.52 | 0.66 |
| Conceived 900-1119  after fire | 49.63 | 52.27 | 49.92 | 51.34 | 0.33 |
| Post-fire | 57.50 | 54.96 | 52.28 | 52.50 | 0.87 |

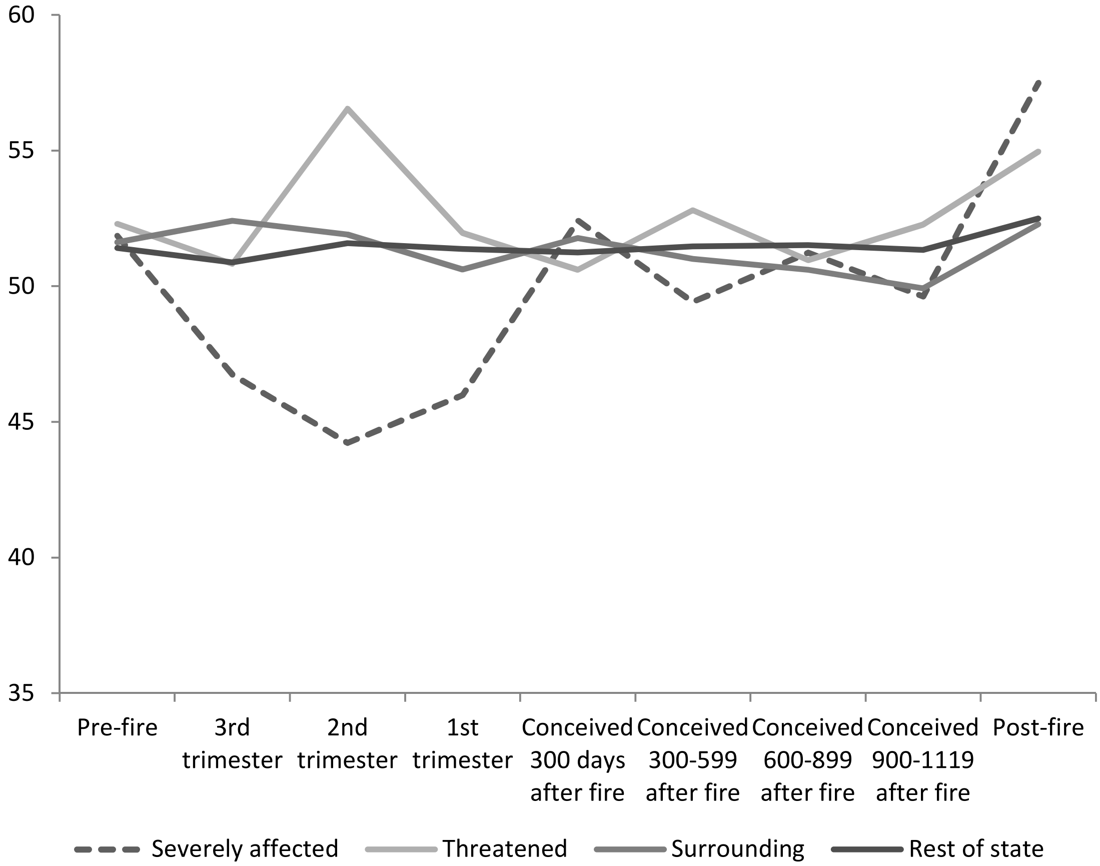


Figure 5-4: Percentage of male births in Victoria between 2006 and 2012 by timing of exposure and fire exposure timing and status (heavily fire affected, threatened with fire damage, surrounding areas and rest of state) based on mothers' residential location. This shows the decrease in male live births among the cohort exposed to fire in the severely affected area. This decrease was not significant.

### 5.6.2. Plurality

The analysis shows there was a significant decreased in the rate of plurality among the cohort exposed to the fire in the second trimester ($p$ = 0.03), which correlates with the largest decline in SSR.

Table 5-7: Results of chi-square tests for the percentage of multiple births in Victoria between 2006 and 2012 by timing of exposure and by the four areas of impact (heavily fire affected, threatened with fire damage, surrounding areas and rest of state) based on mothers' residential location. Significant p-values are marked with an asterisk.

| Birth timing in relation to fire | Severely affected | Threaten ed | Surround ing | Rest of state | p |
|---|---|---|---|---|---|
| Pre-fire | 4.04 | 3.35 | 3.66 | 3.42 | 0.2 |
| 3rd trimester | 1.18 | 2.04 | 3.55 | 3.33 | 0.15 |
| 2nd trimester | 1.92 | 6.32 | 4.39 | 3.57 | 0.03* |
| 1st trimester | 4.38 | 2.23 | 3.49 | 3 | 0.44 |
| Conceived 300 days after fire | 2.91 | 3.1 | 3.03 | 3.38 | 0.55 |
| Conceived 300-599 after fire | 2.27 | 2.03 | 3.47 | 3.21 | 0.06 |
| Conceived 600-899 after fire | 3.31 | 4.37 | 3.26 | 3.13 | 0.08 |
| Conceived 900-1119 after fire | 5.22 | 3.47 | 2.68 | 2.88 | 0.07 |
| Post-fire | 15 | 1.52 | 5.44 | 6.28 | 0.01* |

In the severely affected cohort, comparatively low rates of multiple births are observed among second and third trimester exposed cohorts (see Figure 5-5). There was a considerable increase in multiple births in the severely affected area in the post-fire period, likely arising from the effects of rare events (such as birth greater than twins) on the small sample size available to this analysis.

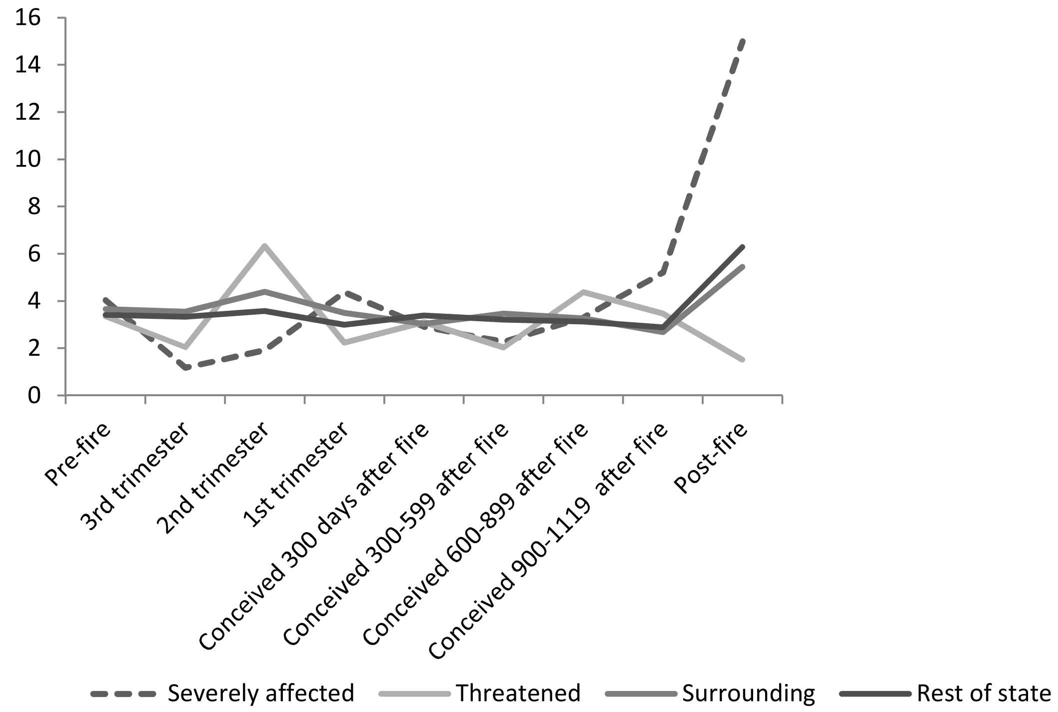


Figure 5-5: Percentage of multiple births in Victoria between 2006 and 2012 by timing of exposure and fire exposure status. Percentage of multiple births in Victoria between 2006 and 2012 by timing of exposure and fire exposure timing and status (heavily fire affected, threatened with fire damage, surrounding areas and the remainder of the state - based on mothers' residential location), showing the decrease in multiple births among the cohort residing in the severely affected area during the second and third trimesters. A chi-square test found significant changes in the second trimester are significant (p = 0.03) reflecting both the decrease in the severely affected area and an increase in the moderately affected area.

## *5.7. Limitations*

It is important to note the limitations of this analysis. Firstly, this analysis uses only an approximate trimester of exposure. The timing variable used assumes that fire exposure occurred on 7 February for all births; however, different populations were affected over the course of almost a month, depending on location. Further, a small number of fires began prior to 7 February – from 28 January onwards – although they were not destructive until after 7 February. Therefore, the trimester of exposure will be inaccurate in some case meaning. Thus means, for instance, that some first trimester exposed dyads will appear in the analysis as second trimester exposed.

Secondly, the analysis identifies exposure by residence at the time of birth. As discussed earlier, there are some inaccuracies in this method but it remains the best available approach.

Lastly, the interpretation of the analysis infers an altered PSR or increased early loss from the SSR. Future research may instead examine the reported rates of stillbirth and reported miscarriage in order to seek direct evidence for increases in stillbirths and miscarriage among exposed women.

More generally, theoretical views of sex ratio, as discussed in Chapter Two, suggest that constant fluctuation should occur in the sex ratio and this is affirmed by empirical work (Catalano 2005). It is therefore a methodological challenge to account for natural variation when examining sex ratios, particularly effects such as other correlations and the echo effects of previous disruptions. Although it was outside the scope of this work to conduct such analysis, this is an important consideration for future research.

## *5.8. Discussion*

This chapter finds declines in the SSR of live births in response to exposure to the Black Saturday bushfires, supporting previous studies of other disasters (Fukuda et al. 1998; Fukuda et al. 2013; Fukuda et al. 2014; Hamamatsu et al. 2014; Saadat 2008; Suzuki et al. 2015; Torche and Kleinhaus 2012), as well as evolutionary and genetic theories of male frailty (Stinson 1985; Trivers and Willard 1973). This chapter uses a more accurate method of determining fire exposure than was possible in previous work

(O'Donnell and Behie 2013). These results are consistent with theories of sex ratio determination; which state that males are more vulnerable to environmental change and, as such, should be more vulnerable to early loss than their female peers.

In the Victorian case study, the severely affected region shows declines in SSR across all three trimesters of exposure, with the greatest decline in the second trimester. The decline is significant when analysed by year ($p = 0.03$), noting that the sample size was sufficient when all cases in the heavily affected area in 2009 are considered together.

Changes to SSR indicate that there are males "missing" from the cohort. There are three usual reasons why these males are absent from the cohort. Firstly, the number of male conceptions may have declined. Secondly, the survival rate of male foetuses may be less than that of female foetuses. And thirdly, it may be a data artefact caused by small groups, sampling error or movement out of the disaster area. In this case, a whole-of-population sample (or very close to) is used and there is no evidence that women carrying male foetuses would have relocated at a greater rate than women carrying female foetuses. Consequently, the pattern is unlikely to be a data artefact. However, because the research was not able to account for natural variations in SSR, it is important that this finding is interpreted as an indicator of the need for further research.

As discussed, there is limited evidence that stress may alter sperm motility in a way that reduces the likelihood of male conceptions by altering the access of Y-bearing sperm to the ovum (Fukuda et al. 1996). However, while changes in the sex ratio of the cohort exposed at conception might be explained through this mechanisms, the pattern of SSR reductions among foetuses exposed in the second and third trimesters cannot have been caused by changes in the primary sex ratio (PSR).

Rather, declines in the cohort exposed in the second and third trimesters are more likely indicative of a rise in the number of miscarried or still-born male babies. However, because the timing variable used in this study is subject to inaccuracies, it is possible that some of the births coded as being exposed in the second trimester may have been exposed in the first trimester. Therefore, timing effects should be interpreted cautiously with more research needed to fully understand timing and the type of loss being observed. Declines in the group exposed in the first trimester exposed group may include some missing males who are the result of altered PSR (Catalano and Bruckner 2006); it may also include males who are missing due to sex differentiated decreased foetal survival rates in early pregnancy.

The statistical significance of this result indicates that it is not due to chance. In this case, arguably all missing males have a clinical or real-world significance; nevertheless, it is important to note that the degree of clinical significance will vary widely from case to case. That is, from examples of loss that are unknown to the parent up to parents who have experience a stillbirth.

The pattern of SSR declines found in both Victoria and, to a lesser extent, the ACT are broadly consistent with disaster-associated declines in SSR; however, changes to multiple births are not consistent with the predicted effects of stress.

Although the relationship between pregnancy stress and stillbirth (i.e. pregnancy loss after 20 weeks gestation) is incompletely understood, some studies also find a relationship between greater maternal stress and increased stillbirth risk (Stephansson et al. 2001; Wisborg et al. 2008). Previous work finds that elevations in stillbirth rates are associated with persistent life stressors such as socioeconomic disadvantage

(Stephansson et al. 2001) and psychosocial stressors (Wisborg et al. 2008). It is, therefore, possible that disaster-associated stress predicated a rise in both miscarriage and stillbirth rates.

As discussed earlier, because males make greater demands on the mother and are more genetically fragile, stress-induced losses should favour the retention of female foetuses as they are more robust and less costly. Such selective pressure should manifest in an altered SSR even where the underlying PSR is unchanged. Although beyond the scope of this dissertation, analysis of still-born babies is an important areas for future research. Despite declines over previous decades, stillbirth rates in Australia have plateaued in the past decade (Hilder et al. 2014). Since stillbirths still occur idiopathically in genetically normal foetuses, research into residual causes remains important.

Maternal stress is also a documented risk factor for miscarriage (i.e. pregnancy loss before 20 weeks gestation) (Arck et al. 2008; Morland et al. 2008). Because not all miscarriages are recorded and some may be unrecognised by the mother, it is challenging to analyse the effects of stress on miscarriage rates. Nevertheless, Arck et al. (2008) examine 1,098 women between four and twelve weeks gestation and find an association between higher CRH levels and eventual miscarriage, as well as associations between more general indicators of poorer physical condition (such as maternal age above 33 years and maternal body mass index below 20) and miscarriage risk. These risk factors are more pronounced in women included who were between weeks 4 and 7 of gestation. Similarly, Morland et al. (2008) find an association between miscarriage risk and domestic violence, potentially due to the interaction between psychosocial stress and direct physical forming a threat to the foetus. Also consistent with Arck et al. (2008), Morland et al. (2008) find that older maternal age increase miscarriage risk.

Changes in the rates of multiple births can also provide evidence of underlying pregnancy loss. Early loss of one or more foetuses may result in a multiple conception leading to only a single live birth, and thus being recorded in the dataset as a singleton. The observations of reduced SSR in Victoria are accompanied by a reduction in the rate of multiple births. Declines are apparent in the cohort exposed to fire-related stress in the third trimester and second trimester, while changes are significant in the cohort exposed in the second trimester ($p = 0.03$). However, although changes to multiple rates are significant in the second trimester exposed cohort, this is also due to an increase in multiple births in the threatened area. The pattern of decreases in multiple births in the heavily affected cohort who were exposed in the second and third trimesters is consistent with the pattern which would be observed if the SSR were being altered by pregnancy losses rather than altered PSR, as it indicates the absence of missing siblings.

However, the increase in multiple births in the threatened area is notable, as this group could also be expected to have experienced higher-than-usual maternal stress during the fire period and thus predicted to experience a lower-than-usual rate of multiple births. Because the effect appears in those children exposed in the second trimester, any fire-related effect in this cohort is due to more multiple pregnancies being maintained to term, rather than a higher number of multiple conceptions. Such a trend has not been reported previously and is not generally consistent with the documented effects of stress on gestation. This increase in multiple births in also associated with an increase in SSR, again suggestive of clement pregnancy conditions allowing more pregnancies to be maintained.

The analysis does not find a strong pattern of early loss arising from the Canberra fires, potentially related to weakness in the method, such as the small available sample. Although the analysis shows a reduction in the number of multiple births in the severely affected area among the cohort exposed in the first trimester (the most vulnerable cohort), this pattern is much less clearly marked than the Victorian pattern and is not significant.

In terms of SSR, the only statistically significant fluctuation among ACT births is an increase in the number of males born to mothers in the severely affected area who were conceived shortly after the fire. Fire effects on SSR are likely to be evident in this cohort, as both sex determination and the bulk of spontaneous pregnancy loss occurs at or shortly after conception. Importantly, such a rising SSR is contrary to both theory and empirical observations of previous studies. A decline in male live births at and immediately after the fire, possibly indicate an increase in still-born males. However, this decline occurred in both the most and least fire affected groups, suggesting that the cause is not related to fire exposure.

Similarly, significant declines in multiple birth rates occurred in both the most- and least- affected fire areas in the ACT. In the least affected area, a significantly lower rate of multiple birth appears in the second trimester exposed cohort, while in the heavily affected area a significant decrease in multiple births (in this case, no multiple births) occurs in the first trimester exposed cohort. The first trimester declines are consistent with the predicted effects of stress exposure during gestation. Declines in the least-affected area do not appear consistent with fire-related stress. However, it is not possible to exclude the presence of another cause or stressor independently affecting this group.

In this dissertation, the predicted effects of disaster exposure on SSR and multiple birth rates appear to have occurred only inconsistently. They are most evident in response to the more severe disaster event, the Black Saturday fire. More detail on event severity and maternal stress is provided in Chapter Eight and Chapter Nine. Assuming that increases in early loss are due to an underlying evolutionary mechanism that sacrifices the current pregnancy in order to preserve future reproductive potential (a manifestation of the present-future trade-off), this mechanism should only operate in response to very significant (i.e. life threatening) stress. If triggerable by minor stresses, such a mechanism would needlessly sacrifice conceptions and would act to reduce, rather than increase, lifetime fertility. The occurrence of increased rates of early loss in response to only the more severe fire, and only in those women most directly exposed, supports the suggestion that early loss is triggered by extreme stress. Such a mechanism concurs with theories of ecologically sensitive reproduction mechanisms operating in modern humans.

Significantly, because fires inflict sporadic damage, the stress experienced by affected individuals will differ, even between people who live near to one another. Further, the stress experienced by any individual is cumulative, and thus fire stress can be expected to be mediated or exacerbated by maternal condition and pre-existing stressors. While previous studies which more reliably find stress to be a likely cause of declines in the SSR, the relevant stressors are generally chronic, such as economic decline (Catalano

2003; Catalano and Bruckner 2005; Jongbloet et al. 2001), or are related to disasters, which more uniformly and substantially damage large population areas, such as large earthquakes and tsunamis (Fukuda et al. 2013; Hamamatsu et al. 2014; Suzuki et al. 2015).

Potentially, the more variable quality of stress and disruption following bushfires results in more varied population responses. This suggests a very high degree of biological sensitivity to stress which has, appropriately, been finely tuned to ensure that pregnancies are not lost due to mild or transient stressors. In this case, the transient nature of fire stress may mean that it is a false indicator of overall environmental quality and has therefore not depressed the SSR except in areas where fire conditions were especially severe. Even in the Victorian sample, where a decline in SSR appears correlated with the most severe fire exposure, this variation is only significant when the year is analysed as a whole. Although a general trend is observable, the more patchy and transient nature of fire stress appears not to predicate a significant disruption to SSR.

## *5.9. Chapter summary*

In summary, this chapter finds no effects on secondary sex ratio in the Canberra population but does find patterns suggestive of an increase in early loss in the Victorian population reflected in both SSR and, to a lesser extent, in the rates of multiple births. Potentially, this difference may relate to the severity of the fires experienced and the strong action of selectivity in early pregnancy. The next chapter, Chapter Six, examines the effects of these two fires on fertility outcomes, examining the role of maternal stress in informing reproductive decisions.

# 6. EFFECTS OF FIRE EXPOSURE ON TOTAL FERTILITY RATE IN TWO POPULATIONS

## 6.1. Introduction

This chapter compares total fertility rates in affected areas to comparable but unaffected areas. In the stochastic environment created by an environmental disaster, such as the fires explored in this dissertation, both the quality-quantity trade-off and the present-future trade-off of life history theory should interact to inform reproductive outcomes. Where mortality threats are severe and cannot be overcome, they should be associated with increases in fecundity because children will face a high risk of not reaching adulthood. However, where mortality threats are lower, or could be overcome by parental effort, higher investment in fewer offspring might be a more effective reproductive strategy.

Importantly, the relationship between environmental threat and reproduction is complex and frequently non-linear. For example, the timing of threat exposure mediates reproductive response across the lifespan. Early life threat exposure shows the strongest effects on reproductive effort, despite the temporal dislocation from the time of reproduction (Placek and Quinlan 2012). Accordingly, the impact of current threats on reproduction is mediated by individual early life experience and, therefore, the effects of current threats become more difficult to predict.

The mortality threat posed by the fire exposure, and the accompanying endocrinological cascade that stress creates, is informed by prior experience and individual response to stress. Even where threat exposure is sufficient to manifest in an increased drive to reproduce, such pressure can be expected to interact with other contradictory factors. These include the downregulation of fertility in response to higher circulating cortisol or the effects of social changes, such as cramped living conditions following the loss of a home (e.g. moving in with family) that might also act to decrease the frequency of intercourse. The interaction of these factors is also complex, for example, Ellison (2001) finds that fertility and frequency of intercourse show a negligible relationship under stressful conditions.

Nevertheless, increases in birth rate have been detected in several populations following environmental disasters. This suggests that mortality threats arising from environmental disasters may fall into the first category (i.e. they are independent of parental care and pose a high mortality threat). As such, exposure to disaster appears to be a sufficient stressor to manifest in increases in reproductive effort and fecundity that can be detected using whole-of-population methods.

### 6.1.1. Research question

Using total fertility rate, this chapter examines the populations affected by the Canberra 2003 and Black Saturday 2009 fires. Total fertility rate is defined as the number of offspring a women would have over her lifetime if she achieved the age-specific population average number of children as she passed through each age bracket. Total fertility rate is calculated annually and thus reflects underlying changes in the crude fertility rate, creating annual variation. Specifically, this chapter considers the effect of fire exposure on total fertility rate in populations exposed to the Canberra and Black

Saturday bushfires.  These two bushfires could substantially increase mortality perception in a group of highly exposed individuals. Further, government policy during the period (the "Baby Bonus") may have created fertility effects that interact with fire effects. As such, the total fertility rate should have increased in accordance with the effects of mortality threats that could not be overcome by parental care.

## *6.2. Background*

Changes in the rate of marriages, births and divorces following an environmental disaster are known to occur based on a wide body of theory including (but not limited to) attachment theory and life history theory (LHT) (see Chapters Two and Three). The physical and psychological stress of disaster exposure, often coupled with significant social, financial and logistical challenges, means that relationships enter a significant period of flux. In terms of couple relationships, while the impact of stress suggests that separations should increase (as relationships struggle under greater strain), attachment theory suggests that people also experience enhanced desire to enter and remain in close relationships (Cohan and Cole 2002). Clearly, rates of separation and marriage or relationship formation are relevant to birth rates in most societies. Birth rates are also subject to logistical challenges following environmental disasters. Dislocation may mean that couples are living in circumstances not conducive to conception – either apart from each other or in excessively close-quarters with family, friends or strangers. Despite these challenges, several studied populations show a trend of increasing births. This accords with the predictions of LHT that stochastic environments with higher mortality risks and mortality perception should promote earlier and more frequent reproduction.

Portner (2001) argues that in developing nations, which tend to have less well-functioning insurance and social security provisions, children can act as insurance against future risk. Where risk is greater, and hence insurance needs higher, more children are born. In disasters where children have been killed, parents might also be expected to attempt to replace those children, particularly in contexts where children deliver economic benefits to households (Finlay 2009).

In fully-industrialised nations, however, a departure from this pattern may be expected to occur because individuals are able to hedge against risk in other ways (e.g. through purchasing fiscal insurance) and because children represent a cost to family budgets rather than an additional source of revenue. Access to contraception is also superior in most developed nations. As a result, couples in these circumstances can be expected to make conscious decisions to delay reproduction until a more favourable time. However, based on the limited data available, this does not appear to be the case. Birth rates following disasters in both the US and the UK have been shown to increase in the years following disasters, in accordance with the predictions of LHT and attachment theory.

In terms of non-environmental disasters and stressors, Raschky and Wang (2012) consider the influence of the Cuban missile crisis on reproduction in the US. They find that fertility increases in those states most exposed to risk (those nearest to Cuba and those with greater numbers of military installations). This suggests that, when facing mortality risks, "individuals might discount future at an extremely high rate and indulge in reproductive activities" (Raschky and Wang 2012 p.1). Similarly, Rodgers et al. (2005) find that birth rate increased significantly in Oklahoma counties in the four years after the 1995 Oklahoma bombing. For example, the overall gross fertility rate in

Oklahoma county increased by 0.32 ($p < 0.001$) over the four years following the bombing. This effect reached its maximum immediately after the bombing in the Oklahoma City area, but developed and persisted more broadly in large metropolitan areas in other Oklahoma counties (Rodgers et al. 2005). Additionally, Ruther (n.d.) reports similar effects following the September 11 terrorist attacks in New York in 2001, finding that fertility increased significantly in New York, with the greatest effects occurring closest to the World Trade Center site.

Research on environmental disasters includes analysis of the aftermath of hurricanes, earthquakes, and avalanches. Evans et al. (2010) analyse the effect of hurricane advisory warnings on fertility rates in US Atlantic and Gulf Coast states and find that low-risk storm warnings increase fertility while high-risk warnings do not. The impact of storm warning severity on fertility may be logistical: while low-risk warnings encourage people to remain at home, high-risk warnings encourage them to evacuate and thus move into environments less conducive to conception. Evans et al. (2010) also note that the bulk of the increase in birth rate comes from couples who already have one or more children, rather than primiparous couples, indicating that the opportunity costs of reproduction may be lower for multiparous couples.

Evans et al. (2010) suggest that hurricanes have a long-term depressive effect on fertility, but this effect is mild and requires further investigation. Examining hurricanes in Guatemala between 1880 and 1997, Portner (2008) finds that fertility changes relate to whether households owned land. Those households with land holdings increased their fertility in response to higher hurricane risk, possibly because they considered those land holdings to be a form of insurance or the children to be a way to help farm the familial land holding. Those households without land holdings decreased their fertility in response to risk, likely because the perceived benefits of children are reduced and the costs increase where families that cannot derive an income from their own land.

Cohan et al. (2002) study rates of births, divorces, and marriages in South Carolina (US) before and after Hurricane Hugo in 1989. Following the hurricane, rates of divorce, marriage, and birth all increased in the 24 counties that were disaster-declared when compared with the remaining 22 counties that were not. In terms of marriage rates, although the whole of South Carolina was experiencing a period of falling marriage rates, the increase in the disaster-affected areas was more than twice the magnitude of the rate of decline, showing a strong countertrend (Cohan and Cole 2002). Similarly, birth rates arrested a non-significant decline and grew state-wide with a net increase of 41 births per 100,000 population. This effect is most pronounced in disaster-affected areas and is greatest in the hardest hit areas (Cohan and Cole 2002). Divorce rates had been stable prior to the disaster, but increased in 1990 before returning to basal levels in 1991. Like marriages and births, this effect is most pronounced in the hardest hit regions. Although there was a small demographic peak in the age groups likely to marry, reproduce and divorce (here defined as individuals aged 18–45), the results do not alter substantially when these were controlled (Cohan and Cole 2002). Conversely, Nakonezny, Reddick and Rodgers (2004) find that divorces fell following the Oklahoma bombings compared to the trend over the prior decade. They attribute this in part to the functioning of attachment under conditions of stress and threat (Nakonezny et al. 2004).

Finlay (2009) finds that fertility increased significantly following three high mortality earthquakes: Gujarat, India, in 2001; North-West Frontier, Pakistan, in 2005; and Izmir, Turkey, in 1999. While numbers of children per women were in decline in all these countries (most likely due to modernisation), those declines are reduced in earthquake-exposed women. Consistent with replacement theory, women who had more children killed in the earthquakes had higher post-quake fertility (Finlay 2009).

Conversely, Hamamatsu et al. (2014) find that births and marriages (as an indicator of future births) have decreased following the March 2011 Tōhoku disaster (earthquake, tsunami and nuclear accident) in Fukushima, Japan. Notably, Japan's economic circumstances differ substantially from those of India, Pakistan and Turkey. In Japan, not only do children not contribute to family income, they place further strain on familial income in a developed economy that is experiencing long-term contraction. Nevertheless, more recent analysis of birth, divorce, and marriage rates following the Tōhoku disaster suggests that marriages may be increasing, but at a delayed rate throughout 2012 (O'Donnell and Behie 2014). While marriage rates fell in Fukushima prefecture itself (likely due to the major damage and permanent relocations caused by the nuclear disaster exclusion zone), marriage rates in the surrounding prefectures have increased, contrary to previous trends (O'Donnell and Behie 2014; Statistics Bureau of Japan 2013). Further analysis over the coming years will show whether the increase in marriages translates into an increase in births. In general, Japanese couples tend to have their first child within the first two years of marriage, so any extant trends should emerge relatively quickly.

In an older and smaller study, Williams and Parkes (1975) find that the birth rate in two Welsh mining villages, Aberfan and Merthyr Vale, rose following a 1966 avalanche which destroyed Aberfan Primary School and several houses, killing 116 children and 28 adults. Birth rates began to rise sharply in 1968 (increasing that year by 6.42 per cent) and remained higher than surrounding villages until 1972. Although some bereaved parents had further children, the substantial majority of new parents had not lost children in the disaster. The replacement effect did not, therefore, appear to be a major driver of this increase.

While theory suggests that birth rate should rise where people feel that they are exposed to high mortality risk, evidence is variable between nations and interacts with specific economic and social circumstances. Nevertheless, it does appear that fertility rises in response to increased environmental stress, albeit modified by local factors, including the impact of replacement effects and the financial impact of children.

This dissertation adds to these findings by examining the effects of fire as an environmental stressor on fertility in two modern human populations.

## *6.3. Methods*

### 6.3.1. Ethics approval

The ethical aspects of this dissertation were approved by The Australian National University Human Research Ethics Committee (ANU-HREC) on 23 September 2013 (project reference: 2013/143).

### 6.3.2. Limitations

This study uses total fertility rates as the unit of measurement due to its availability. Although commonly used as a proxy for cohort fertility (Sobotka and Wolfgang 2009), total fertility rates (TFRs) are a composite measure of lifetime fertility. The TFR is a derived measure which estimates the number of offspring a woman would have if she achieved the average birth rate in each age group over her lifetime. Because it is calculated annually, the TFR reflects changes in the crude and age-adjusted birth rates, allowing it to change over time. Age-specific fertility rates are the number of live births at each age of mothers per 1,000 of the female population of that age.

TFRs are not a cohort measure of fertility but are often used in this manner due to availability and ease of use. They are used here because of their availability at a consistent level of geographic classification over the time period studied. They are also able to account for the changing age profile in areas over time. Age-specific crude fertility rates at local geographical level were not available for this analysis.

Importantly, however, because TFRs are not a cohort fertility rate, they have a number of methodological shortcomings which should be considered when interpreting outcomes. Although frequently used – particularly by governments – to measure changes in fertility in response to acute influences (such as policy changes), TFRs are unable to account for tempo effects. This means that shortening of inter-birth intervals can appear as fecundity increases even while the number of births over a lifetime is actually stable. Therefore, the use of TFRs may overstate fertility effects. Some studies suggest, that unless carefully interpreted, TFRs can be misleading, particularly in the policy context (Sobotka and Wolfgang 2009).

This dissertation uses Australian Bureau of Statistics (ABS) TFRs (i.e. those calculated and published by the ABS). ABS fertility rates are based on birth registrations (rather than birth records) which means that a lag can occur between birth and registration of the birth; since parents are required to register a birth within 60 days, a birth at the end of one year may not be registered until the next. Data excludes still-born children. TFRs are also based on population estimates derived from the five-yearly Australian census. Because both fires fell between census dates, estimates do not account for post-disaster emigration. This will have the effect of understating fertility changes by overestimating population size.

For both fires, ABS statistical areas are limited in their specificity. This interacts with the complexity of classifying disaster-affected areas and the limitations of geographic definitions of disaster impact. This analysis does not include other factors likely to affect fertility rates, such as demographics and housing affordability. However, the analysis attempts to compare like areas so that the influence of these factors can be partially controlled. Because ABS fertility rates are not concordant with the samples used elsewhere in this dissertation, it is challenging to calculate effect sizes accurately. However, the limitations described in relation to the small samples available to this research (see section 4.5.2 for detail) are also reflected in this analysis.

### 6.3.3. Sampling strategy: Canberra fire 2003

The ABS uses the Australian Statistical Geography Standard to delineate geographical regions for statistics at four levels. Statistical level two (SA2), broadly equivalent to suburbs, is used in this analysis with the unaffected remainder of the ACT used as a comparison population.

A new variable indicating fire exposure (*firearea*) is constructed as follows. Areas in which houses were destroyed are classed as “heavily affected”, surrounding areas where other fire damage or direct threat occurred are classed as “moderately affected”, the remainder of the territory is classed as “least affected” (Murray 2003). Heavily and moderately affected areas are shown in Appendix B.

All variables are constructed using Microsoft Excel 2013.

### 6.3.4. Sampling strategy: Black Saturday 2009 (Victoria)

The Victorian total fertility rate data is divided into rural/peri-urban areas and suburban areas due to the much larger size of the sample and substantial social and geographical differences across Victoria. Inner metropolitan and inner-urban areas are removed from the sample to increase the consistency of the sample, as were non-residential districts (e.g. airports and military zones). The fires affected a smaller proportion of Victoria, due to the comparative size of the remainder of the state. The fires also affected a mix of suburban, peri-urban, and rural areas, which added complexity to constructing a comparative sample.

To create comparison groups, both the rural/pre-urban and suburban samples include nearby (mostly neighbouring) but largely unaffected areas. In the suburban sample, inner-urban, metropolitan, and western suburban regions, as well as Geelong and surrounds are removed as they are socially and geographically distant from the affected areas. In the rural/peri-urban sample, Mildura, Swan Hill and surrounds, Wilson's

Promontory and surrounds, and the peri-urban areas around Geelong and townships along the Great Ocean Road are removed for the same reason. The remaining areas were used as comparison areas, as they were not threatened but are also not substantially distant from the affected areas.

Fire affected SA2 areas in which homes were destroyed are classed as “heavily affected”, the surrounding SA2 areas are classed as “moderately affected”. In some cases, there were no habituated surrounding areas. The remainder of the state, except the areas noted above, are used as a comparison population. Areas are shown in Appendix B.

Similarly, the remainder of the suburban area, excluding the areas noted, are used as a comparison sample and termed “least affected”. All variables are constructed using Microsoft Excel 2013.

### 6.3.5. Collection methods

Total fertility rates for Victoria divisible at SA1 and SA2 between 2001 and 2012 were downloaded from the Australian Bureau of Statistics website on 1 March 2014. The variables included are: SA2 name (*firearea*); total fertility rate (*tfr*); and year (*year*).

Total fertility rates for the ACT divisible at SA1 and SA2 between 2003 and 2012 were downloaded from the Australian Bureau of Statistics website (1 March 2014). Fertility rates at SA1, SA2 or SA3 levels are not available for 2000–2003 but fertility rates for the ACT as a whole are available. The variables included are: SA2 name (*firearea*); total fertility rate (*tfr*); and year (*year*).

### 6.3.6. Data cleaning procedures

The SA2 region of Franklin in the ACT is excluded from the data. Franklin recorded extremely high fertility rates until 2006 (up to 22.33) owing to a high birth rate and very low population and was not reported on thereafter, as such, it disrupted data averages. Some cells of missing data reflect boundary changes or missing data. Otherwise, total fertility rate data is complete with no further cleaning required.

### 6.3.7. Analysis strategies

Total fertility rates for both the ACT and Victoria are graphed according to statistical area (SA). Groups are defined as outlined in section 6.3. Areas classed as heavily affected or moderately affected are compared with least affected areas using one-way and two-way Analyses of Variance (ANOVA). All analysis is conducted using Genstat 16th Edition and for all analysis alpha is set at 0.05.

## *6.4. Preliminary statistics – Canberra fire*

The total number of births increased in the ACT over the period 2000–2010, reflecting both increasing population and a moderate increase in the total fertility rate. Figure 6-1 shows the ACT fertility rate with the overall Australian fertility rate also shown for reference. The ACT fertility increases steadily until 2008 and then plateaus with some yearly fluctuation.

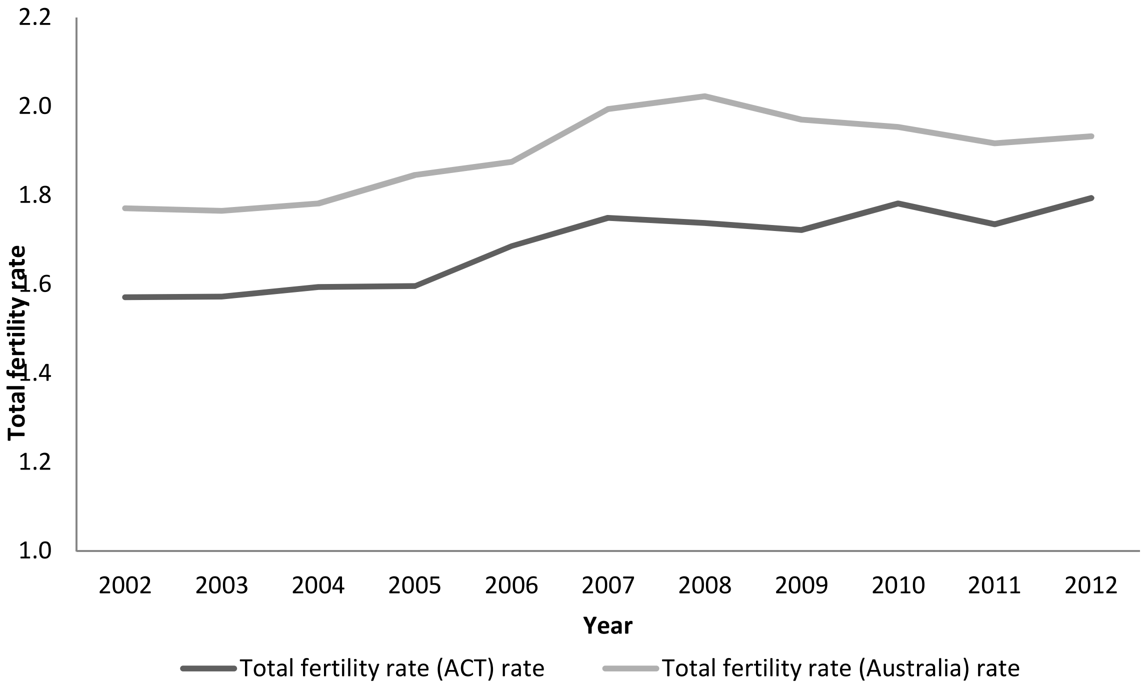


Figure 6-1: ACT mean fertility rate and Australian national mean fertility rate between 2001–2012

Figure 6-2 shows the total fertility rate for the seven Canberra subdivisions of interest on a truncated Y-axis, in order to show the patterns more clearly. Data at statistical geographical classification level two is unavailable for 2001 and 2002. The most substantially fire affected area, Weston, shows an accelerated period of growth for 2003–2009.

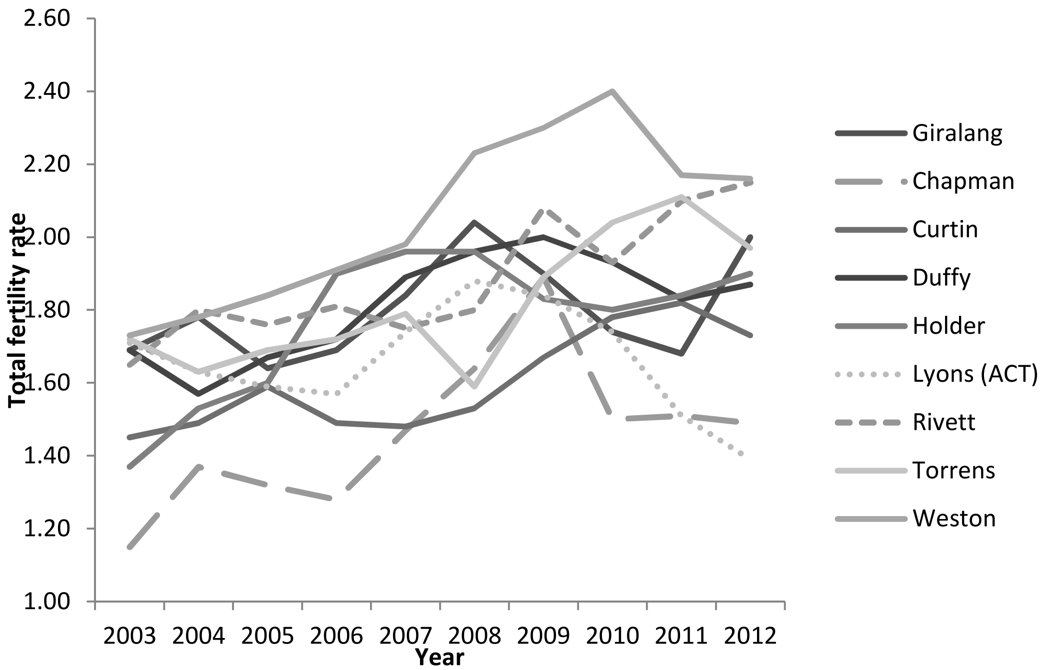


Figure 6-2: Total fertility rate for heavily fire affected ACT subdivisions 2003–2010 (shown with a truncated Y-axis for clarity).

## *6.5. Preliminary statistics – Black Saturday*

The total number of births increased in Victoria between 2006 and 2012, reflecting both an increase in population and an increase in the total fertility rate. The numbers of births in Victoria is growing, reflecting increases in both the population and increased in the fertility rate until 2009. The total fertility rate for Victoria and the Australian average over the period 2002–2012 is shown in Figure 6-3 and shows a decline between 2009 and 2011, consistent with national declines during the same period. Figure 6-4 shows fertility trends in the four suburban areas identified as fire affected in this analysis: Hampton Park–Lynbrook and Narre Warren North show a decline in fertility; Ferntree Gully shows a small decline followed by stabilisation; while Lynbrook-Lyndhurst shows substantial growth.

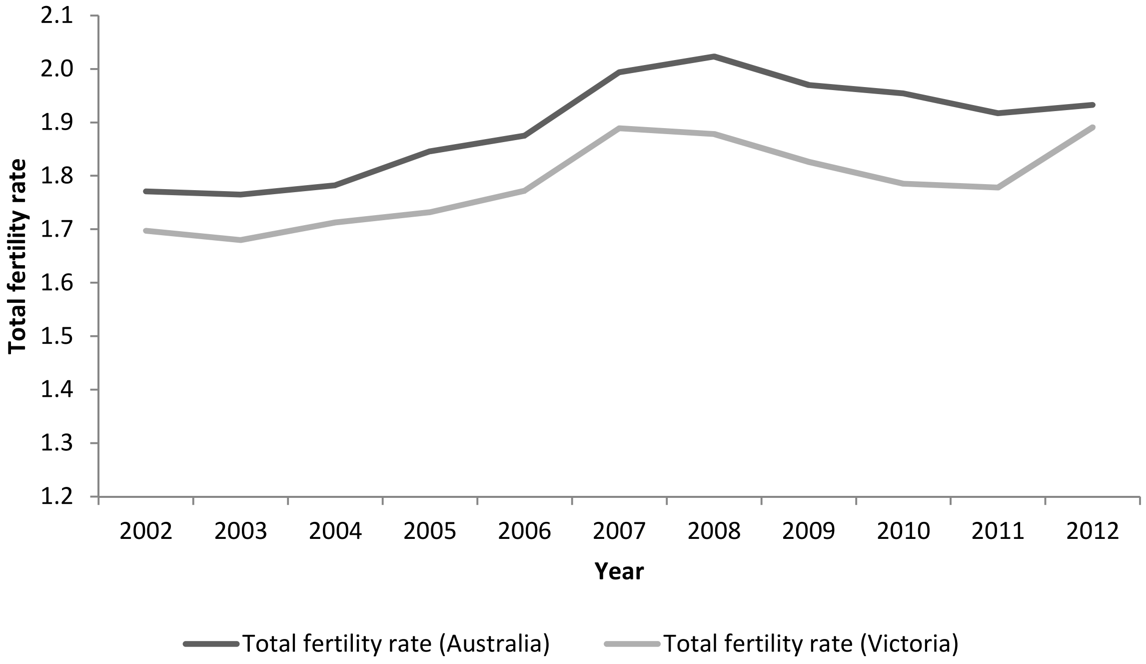


Figure 6-3: Total fertility rate for all of Victoria 2002–2012 (shown with a truncated Y-axis for clarity).

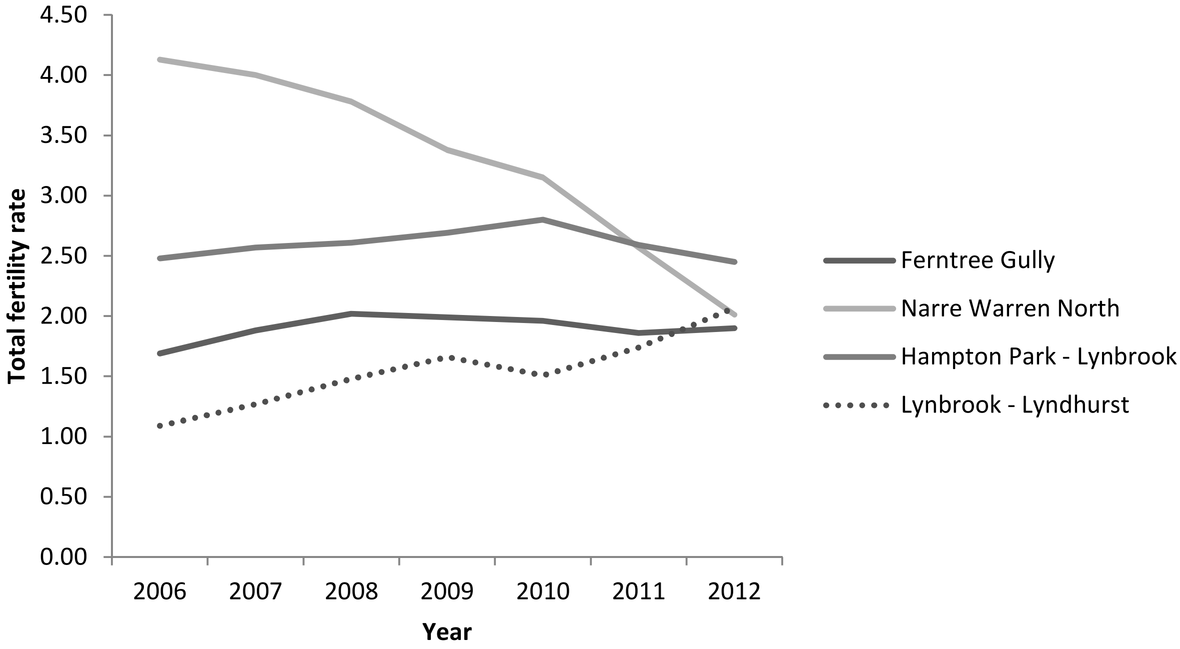


Figure 6-4: Total fertility rate in fire affected urban areas of Victoria 2002–2012.

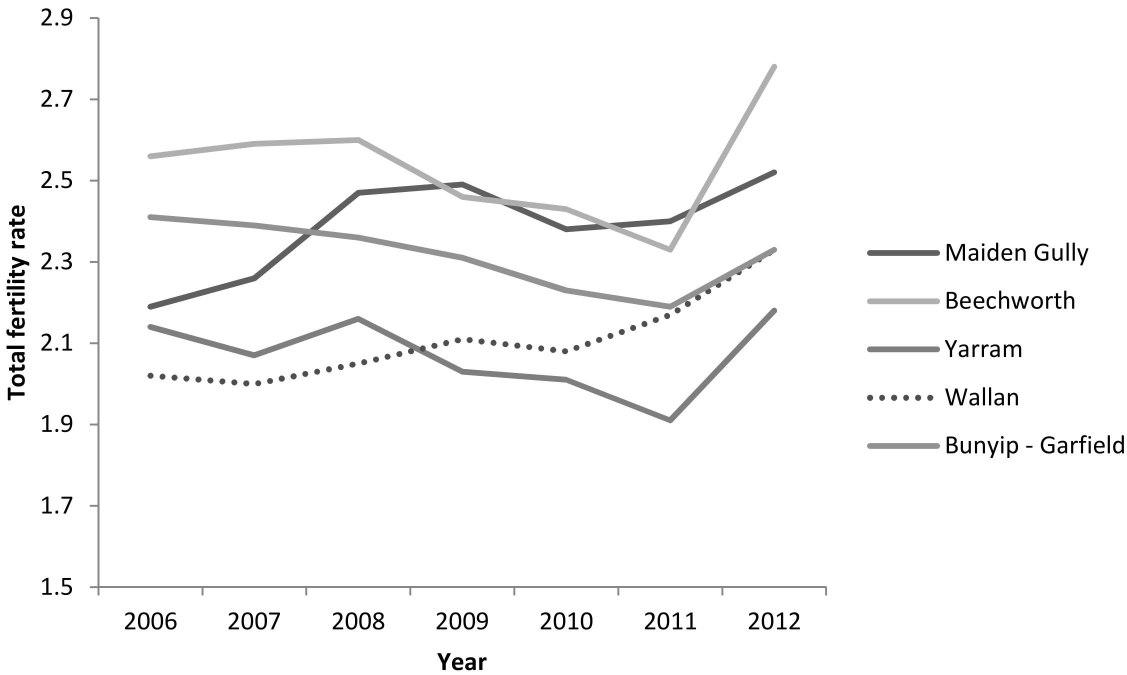


Figure 6-5: Total fertility rates for fire affected rural/peri-urban areas of Victoria where rates increased (only). Shown on a truncated axis for clarity.

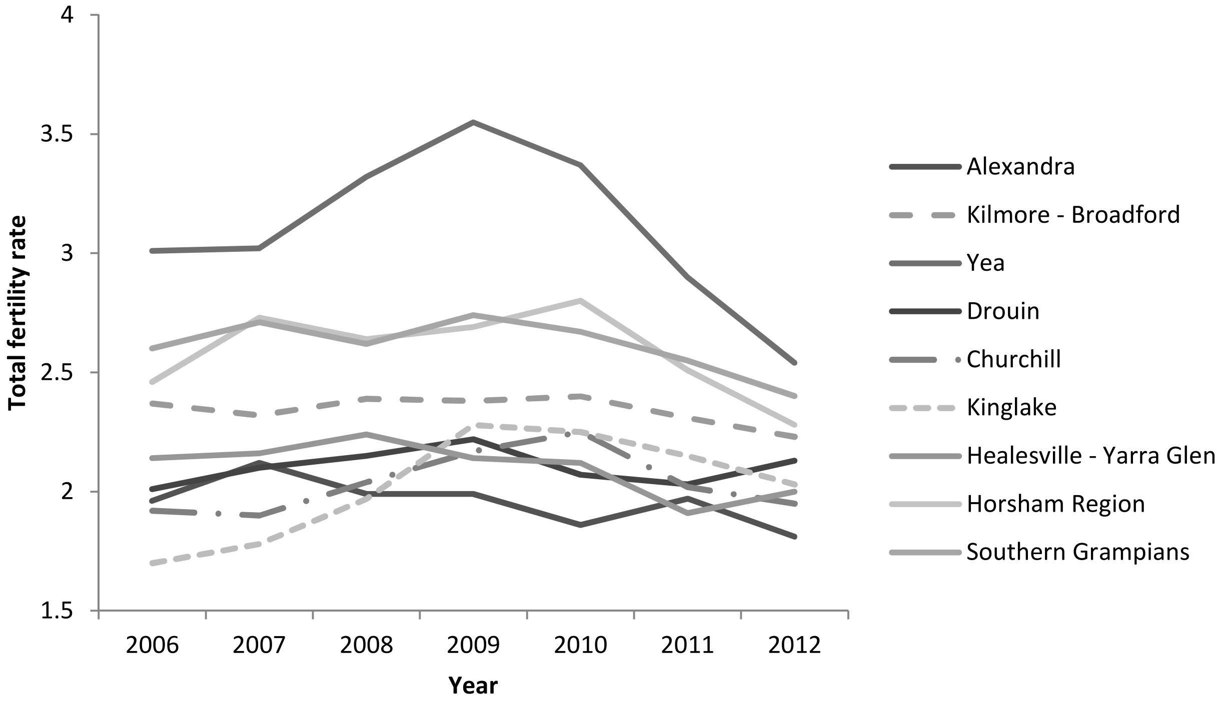


Figure 6-6: Total fertility rates 2006–2012 for the fire affected rural/peri-urban areas of Victoria where rates decreased only. Shown on a truncated axis for clarity.

Figure 6-5 shows total fertility rates for the areas included in the rural/peri-urban fire-affected areas in which fertility increased from 2009 onwards. Beechworth shows the most substantial increase. Figure 6-6 shows the patterns of change in fertility rates in the sampled rural areas in which fertility rates did not increase between 2009 and 2012. Yea shows the most dramatic decline. Although Healesville and Drouin show upward trends, their 2012 fertility rates remain lower than their 2009 fertility rates.

## *6.6. Results – Canberra fire*

Using the samples described in section 6.3, one-way ANOVAs are used to determine whether there were statistical differences between the unaffected, threatened and highly affected groups. Table 6-1 shows the analysis results and Figure 6-7 shows the fertility rates for the three levels of fire exposed areas between 2003 and 2012.

Table 6-1: ANOVA results for total fertility rates in the ACT in three levels of fire exposure by year.

| Year | Total fertility rates – "Least affected" | Total fertility rates – "Moderately affected" | Total fertility rates – "Heavily affected" | *p* |
|---|---|---|---|---|
| 2003 | 1.80 | 1.60 | 1.57 | 0.51 |
| 2004 | 1.98 | 1.63 | 1.62 | 0.33 |
| 2005 | 2.04 | 1.66 | 1.63 | 0.38 |
| 2006 | 1.82 | 1.71 | 1.68 | 0.75 |
| 2007 | 1.76 | 1.77 | 1.77 | 0.99 |
| 2008 | 1.81 | 1.82 | 1.85 | 0.98 |
| 2009 | 1.72 | 1.83 | 1.93 | 0.27 |
| 2010 | 1.67 | 1.87 | 1.87 | 0.11 |
| 2011 | 1.69 | 1.90 | 1.84 | 0.08 |
| 2012 | 1.76 | 1.98 | 1.85 | 0.17 |

All ANOVA models are shown in Appendix B.

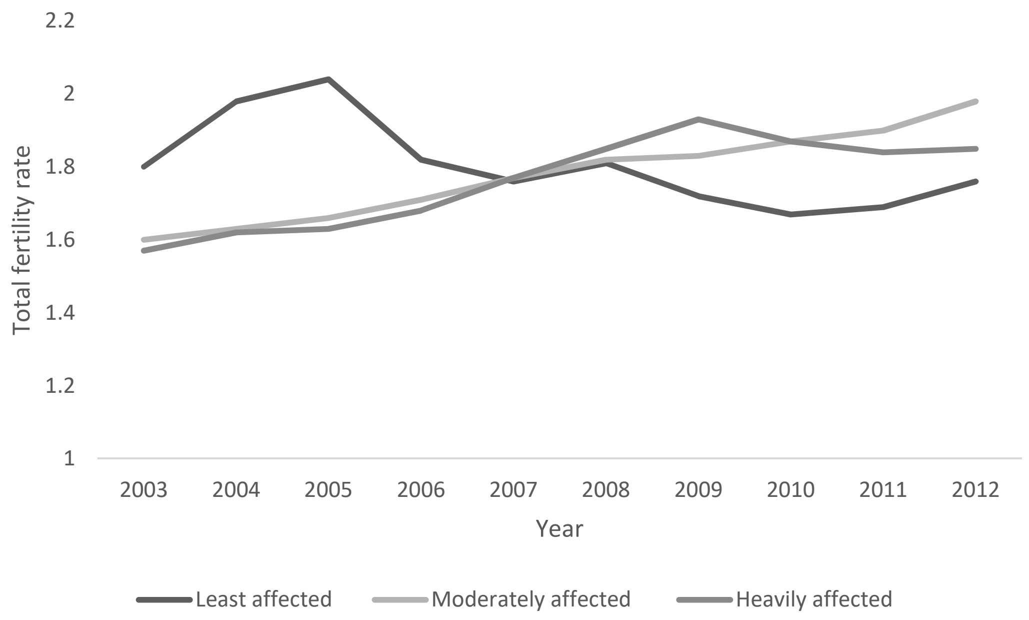


Figure 6-7: Fertility rate patterns at three levels of fire exposure in the ACT between 2003 and 2012. The Y-axis is truncated for clarity.

Over the longer-term, fertility rates in threatened and affected areas exceeded those in unaffected areas (see Figure 6-7). However, only unaffected areas showed an increase in the year immediately following the fire. The differences between the groups approached significance in 2011 ($p = 0.08$).

A two-way ANOVA is used to test the relationship between years over the full date range (2003–2012) with an interaction term between year and impact levels. The relationship is not significant at $p = 0.27$. As demonstrated in Figure 6-7, the moderately affected and the heavily affected area share a similar pattern in total fertility rates. Further analysis classifying the fire exposure simply into either “affected” (combining the moderately and heavily affected areas) or “unaffected” (the least affected area) shows significant change in the differences between the affected and unaffected areas across the time period ($p = 0.01$). Indeed, this affected group shows a steady increase in total fertility rate between 2003 and 2012, while the least affected group shows a sharp increase between 2003 and 2005 followed by an undulating decline. The ANOVA models are shown in Appendix B. The graph at Figure 6-8 shows the distinction between exposed and unexposed groups when analysed in binary terms.

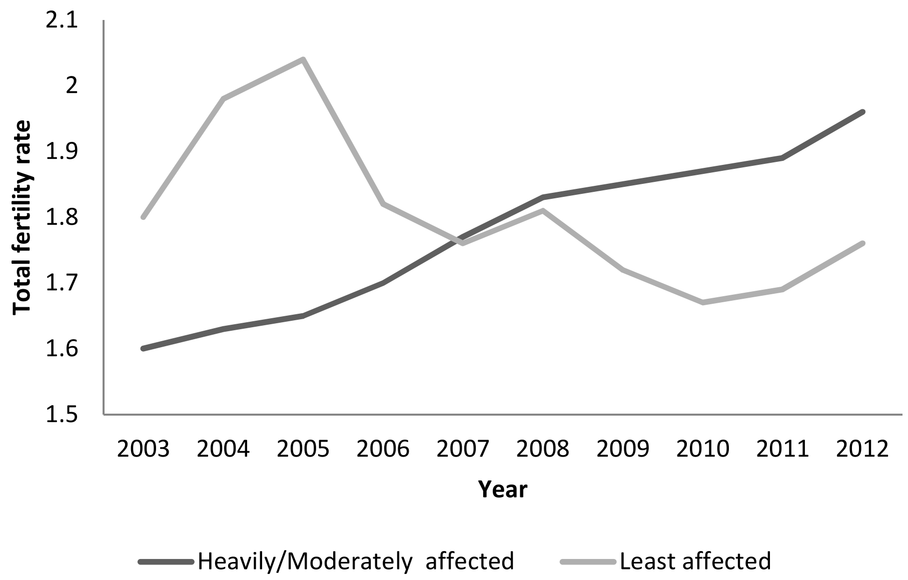


Figure 6-8: Average total fertility rates in the ACT between two levels of fire exposure between 2003 and 2012. The fire occurred in 2003, at the commencement of the analysis. The Y-axis is truncated for clarity.

## *6.7. Results – Black Saturday*

Using the samples described in section 6.3, one-way ANOVAs are used to determine whether there were statistical differences between the unaffected, threatened and highly affected groups in a given year.

Table 6-2: Table of means and p-values arising from ANOVA of fertility rates in rural areas in Victoria at three levels of fire exposure.

| Year | Total fertility rates – "Unaffected" | Total fertility rates – "Threatened" | Total fertility rates – "Highly affected" | *p* |
|---|---|---|---|---|
| 2006 | 2.17 | 2.07 | 2.25 | 0.30 |
| 2007 | 2.23 | 2.16 | 2.30 | 0.55 |
| 2008 | 2.31 | 2.22 | 2.36 | 0.42 |
| 2009 | 2.33 | 2.24 | 2.40 | 0.40 |
| 2010 | 2.29 | 2.19 | 2.35 | 0.36 |
| 2011 | 2.21 | 2.13 | 2.24 | 0.40 |
| 2012 | 2.19 | 2.12 | 2.25 | 0.17 |

Table 6-2 shows the results for the peri-urban/rural sample, with significant results marked by an asterisk. Figure 6-9 plots the total fertility rates for three exposure areas in the peri-urban/rural sample in Victoria from 2006 to 2012. There were no years where the difference in total fertility between groups were statistically significant.

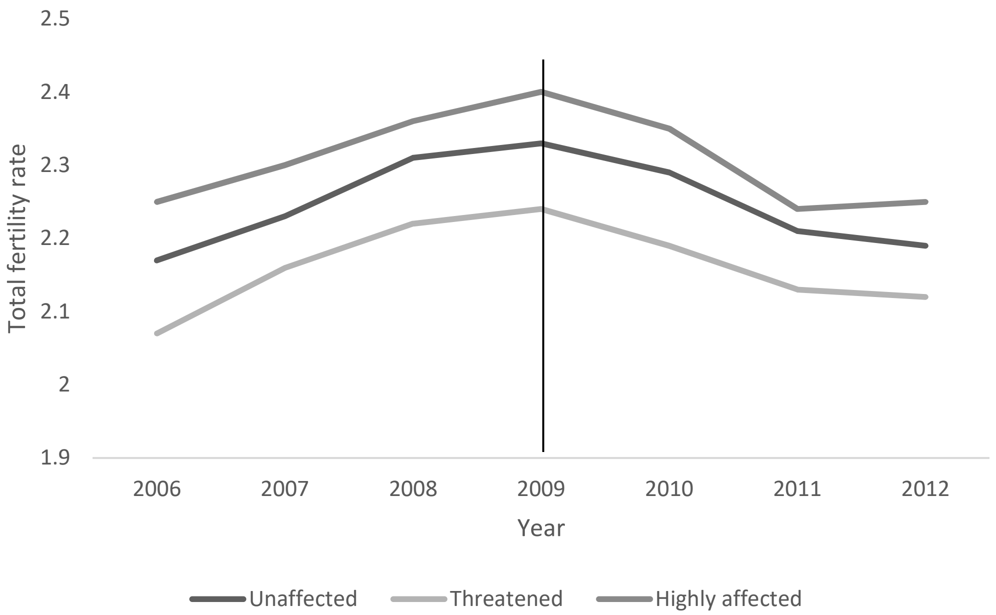


Figure 6-9: Mean total fertility rates in three exposure groups in the peri-urban/rural sample (Victoria). The vertical line indicates the time of the fire. The Y-axis is truncated for clarity.

Table 6-3 shows the results for the suburban sample, significant results are marked with an asterisk. The difference between groups was significant in all years, with threatened and highly affected areas recording significantly higher fertility rates before and after the 2009 fire. Figure 6-10 plots the analysis, showing substantial falls in the highly affected cohort.

Table 6-3: Table of means and p-values arising from ANOVA of fertility rates in suburban areas in Victoria at three levels of fire exposure. Significant results are shown with an asterisk.

| Year | Total fertility rate – “Unaffected” | Total fertility rate – “Threatened” | Total fertility rate – “Highly affected” | *p* |
|---|---|---|---|---|
| 2006 | 1.70 | 1.82 | 2.44 | <0.001* |
| 2007 | 1.77 | 1.88 | 2.48 | <0.001* |
| 2008 | 1.81 | 1.93 | 2.47 | <0.001* |
| 2009 | 1.81 | 1.95 | 2.39 | <0.001* |
| 2010 | 1.78 | 1.90 | 2.29 | 0.00* |
| 2011 | 1.74 | 1.87 | 2.12 | 0.00* |
| 2012 | 1.75 | 1.93 | 2.01 | <0.001* |

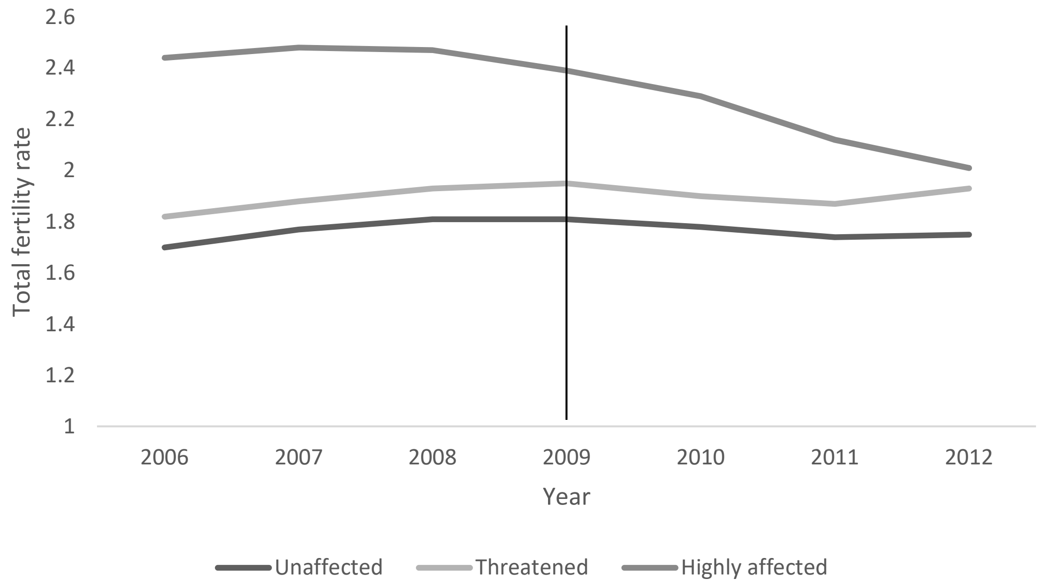


Figure 6-10: Mean total fertility rates in three exposure groups in the urban sample (Victoria). The vertical line indicates the time of the fire. The Y-axis is truncated for clarity.

A two-way ANOVA is used to test the relationship between years over the full date range (2006–2012) using an interaction term between year and impact levels in the suburban sample. The relationship is not significant at $p = 0.97$. All ANOVA models are shown in Appendix B.

## *6.8. Discussion*

While these results do not show convincing evidence that there was an effect of fire exposure on fertility rates in either the ACT or Victoria, and thus do not directly support the hypothesis, they do provide an indication of the influence of other environmental factors. The absence of a detectable fire effect may be due to the absence of a real effect in the population or a inappropriate method, including insufficient sample. However, another acute influence was detected, the change to maternity payments policy during the period studied suggesting that it was an appropriate means of looking for evidence of acute stressors.

Analysis of the Victorian rural-periurban total fertility rates shows no significant differences between affected, threatened and unaffected areas. In fact, there is remarkable consistency given the other social and economic differences expected to exist between different locations. The suburban sample shows significant differences between the three areas of fire impact, with the heavily affected area showing a higher total fertility rate. However, this area had a higher fertility rate before the 2009 fire and the rate declined thereafter, the inverse of the expected effect. It appears, therefore, that the fertility rate in the heavily affected area was influenced by non-fire factors. This is especially the case, given that fertility also declined in the remaining areas (threatened and unaffected) between 2009 and 2011.

Analysis of the ACT data shows a significant difference between fire affected and unaffected areas when analysed as a binary term, with total fertility rates growing in the affected areas from 2003 onwards. However, the absence of available data prior to 2003 means the possibility that this was part of a pre-existing trend cannot be excluded. Although the finding of a post-disaster increase in fertility is consistent with the influence of disaster reported elsewhere (Evans et al. 2010; Finlay 2009; Portner 2008; Raschky and Wang 2012; Rodgers et al. 2005; Ruther n.d.), it is not possible to conclude with confidence that the increase was the result of fire, given the available data. Because pre-fire data was not available, there are potentially other factors affecting localised fertility that might be driving the fertility increases observed, as in the Victorian suburban sample.

Previous research on how disasters affect fertility has found affects after major disasters with substantial impacts – for example a mining disaster which killed a high proportion of the affected communities (Finlay 2009; Williams and Parker 1975). Because the 2009 Black Saturday fire was the more severe event, there should be a more substantial effect. However, there is little evidence of fire-related patterns of fertility in Victoria. If the patterns of fertility in the ACT are associated with fire, it is unusual that they have only presented in response to the more minor disaster.

Fertility rates are subject to many influences, including strong social and economic ones not included in this analysis. One such influence is the Australian Government's introduction of an incentive welfare payment aimed at increasing the Australian birth rate. Known as the 'Baby Bonus', the Maternity Payment was introduced by the Australian Government in July 2004 and its value increased after 1 July 2006 (Gans and Leigh 2009). The Australian fertility rate shows a steady increase from 1.8 in 2004 to 2.0 in 2008.

Notably, although the payment was announced only seven weeks before its introduction in 2004, there was an excess of 1,169 births in July 2004 with the deficit occurring entirely in June 2004. The majority of these births were via induction or caesarean, indicating that women had shifted their births into the payment period. In 2006, when the rate increased, an excess of 687 births occurred in July 2006, again with the deficit occurring in June 2006 and the majority of births being via induction or caesarean (Gans and Leigh 2009). These temporal fluctuations indicate that births were planned around the timing of the Baby Bonus, although little more direct evidence exists.

In 2009, the Baby Bonus payment schedule was altered and means-testing was introduced. From January 2009 a $75,000 family income limit was applied to the payment and it was paid in 13 instalments rather than a single lump sum (Klapdor 2014). Mothers were able to select to receive either Government-funded Paid Parental Leave (which is additional to any maternity leave provided by their employers) or the Baby Bonus. Because Paid Parental Leave payment has a work-test applied (mothers must have been in employment prior to their maternity leave period), the Baby Bonus tends now to only be financially advantageous for unemployed, self-employed, or consistently underemployed women (Klapdor 2014). These changes to the statute means that the Baby Bonus was a much lower incentive for births from 2009 onwards which is reflected in the gentle decline in the national total fertility rate, falling to 1.93 by 2012.

The influence of this payment may be relevant to the upward trend in the ACT fertility rate, which was relatively flat between 2002 and 2004, before increasing steadily from 2004 (when payments commenced) to 2008. The rate shows a decline from 2009 (when payments were changed) onwards, with some evidence of stabilisation between 2010 and 2012. Victorian rates show the same pattern, as do total Australian rates (shown in Figure 6-11), indicating the influence of a national factor such as the Baby Bonus. The depressive effect of the 2009 changes to the payment coincides with the timing of the Black Saturday fire. If any effect of the fire is present, it is too small to detect in comparison to the downward influence of the changing Baby Bonus. Further, because changes to the Baby Bonus coincide with the 2008 economic downturn, a confluence of financial factors may be reflected in the depression in fertility.

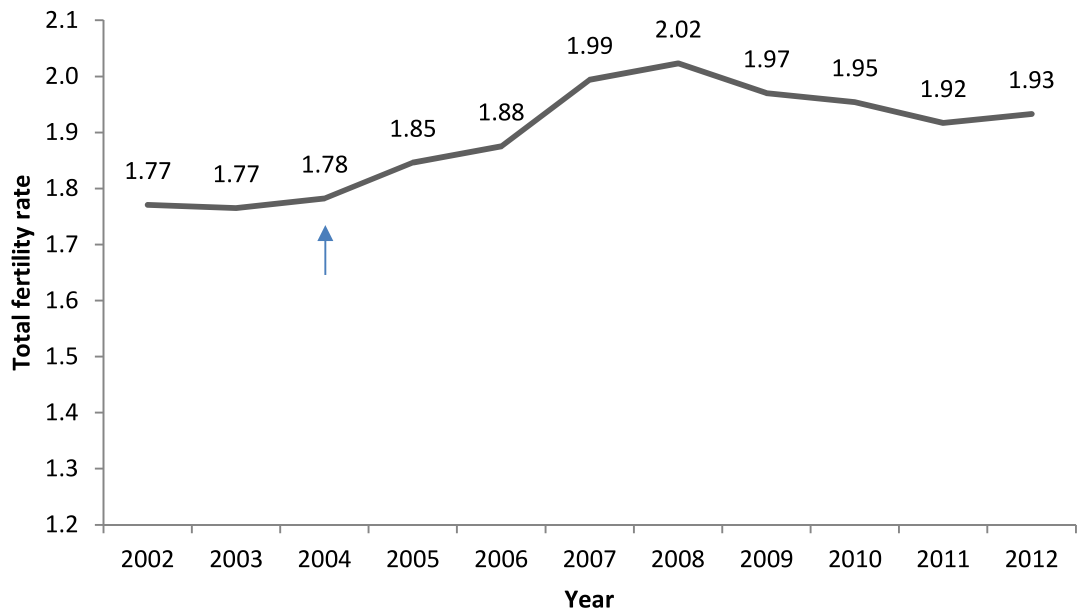


Figure 6-11: Total fertility rate in Australia between 2002 and 2012 showing the rate for each year. The Baby Bonus was introduced in 2004 (indicated by the arrow) and increased in 2006. In 2009 it was modified from a lump sum available to most mothers to a weekly payment available to fewer mothers. Y-axis is truncated for clarity.

In previous studies showing fertility increases after disaster, the disaster effects were more severe, more widely distributed in the population or involved substantial industrial and agricultural destruction. This means that people faced serious and immediate survival risks due to loss of livelihood (Finlay 2009; Williams and Parker 1975).

Further, the loss of children led to replacement effects (Finlay 2009; Portner 2001).While some people in Victoria and ACT faced immediate survival risks or lost their livelihoods, this was not the case for the bulk of the studied population. As such, any effect of peril present in the population would likely be too dilute to be visible. While 15 children were tragically killed in Black Saturday, this is not equivalent to the scale of the loss of children that occurred in incidences such as the Aberfan and Merthyr Vale coal-slurry avalanche, which buried a school killing 116 children (Williams and Parker 1975), or the three earthquakes examined by Finlay (2009), which collectively killed over 100,000 people.

While some evidence from industrialised nations finds fertility increases associated with disasters, about half of these examples are of the effects of national security threats: the Cuban missile crisis, the Oklahoma bombing and the September 11, 2001 terrorist attacks (Raschky and Wang 2012; Rodgers et al. 2005; Ruther n.d.). Such threats might have broader effects than most natural disasters, because there is a perception that national survival and wellbeing is threatened. Consequently, the fertility effects of these more existential threats may differ to those effects of bushfire. Bushfire, while traumatic and devastating to some individuals, does not threaten broad-scale populations and does not indicate long-term or exogenous threats to national wellbeing.

Of the more comparable previous studies, evidence for increases in fertility is mixed. Some find long-term depressive effects on fertility from hurricane warnings (Evans et al. 2010), while some find increases in fertility following Hurricane Hugo (Cohan and Cole 2002). Similarly, some find decreases in marriage (as a pre-fertility measure) following the East Japan Earthquake and Fukushima disaster (Hamamatsu et al. 2014) and while others find increases in marriage, for example following Hurricane Hugo (Cohan and Cole 2002) and the East Japan Earthquake and Fukushima disaster (O'Donnell and Behie 2014).

From an evolutionary perspective, it is difficult to determine from such studies where only reproductive output is measured whether there are changes to reproductive effort. These problems also highlight the difficulty in studying reproductive behaviour through populations that are not naturally fertile (i.e. without contraception). In contemporary populations where use of contraception is widespread, reproductive effort (i.e. sexual intercourse) need not lead to actual reproduction. Because reproductive behaviour and reproductive outcomes are disconnected, reproductive outcome (i.e. birth and fertility rates) becomes only a proxy measure of underlying reproductive behaviour.

It is not possible to determine in this study whether rates of sexual intercourse increased following the disasters. While broad-scale surveys of Australian sexual behaviour do exist (e.g. the Australian Study of Health and Relationships conducted in partnership between the University of New South Wales, the University of Sydney, La Trobe University and the University of Sussex in 2013, as well as The Great Australian Sex Census conducted by RedHotPie.com.au in 2012–13) they do not cover periods useful for this dissertation.

If detected, an increase in reproductive effort would provide evidence that an underlying psychological mechanism was functioning to increase reproduction. Another approach is to examine rates of coupling, as attempted by other studies cited earlier (Cohan and Cole 2002; Hamamatsu et al. 2014; O'Donnell and Behie 2014). However, in Australia, rates of couple formation are also becoming more complex to measure because of the rise of de-facto (common law) couples. Rates of de-facto couples are rising, most likely due to changing attitudes to acceptable living arrangements that make it more common for people to enter into de-facto relationships and more common for them to report that they are in a de-facto relationship during census data collection (Australian Bureau of Statistics 2012b). However, because reporting of de-facto relationships is less regulated than the reporting of marriages and divorces (because of the absence of legal processes), it is more difficult to measure changes in the rate of coupling. Further, while the census allows de-facto as a couple type, it is not possible to tell if the de-facto partner has remained the same person between census collection dates (Australian Bureau of Statistics 2012b). It is an interesting outcome of increasingly accepting social attitudes to sex, coupling, and child-bearing that research into secular trends is becoming more complex.

### *6.9. Chapter summary*

In summary, the absence of a clear fire effect in this study may stem from a number of causes. It is possible that the experience of mortality risk was either too rare to be detectable in a population level analysis or was too fleeting to affect reproductive decisions, or possibly both. Alternatively, actual increases in the fertility rate due to fire may have been masked by the depressive effects of Australian Government policy changes – the timing of these changes may help explain why an increase is observed in the ACT sample (where the timing of fire roughly coincided with the introduction of incentive payment for births) but not in Victoria (where the fire coincided with a remodelling of the payment which lessened its appeal, leading to widespread decreases in total fertility). It is also possible that there may have been underlying responsive changes in reproductive effort not visible in the fertility rate because the study population is not naturally fertile. Future work could usefully examine the gap between reproductive effort and outcome by seeking to use measures of coupling or intercourse instead of births, as well as seeking to repeat this analysis using a cohort measure of fertility. These issues are further discussed in Chapter Nine, where conclusions for this chapter are presented. The next chapter examines the influence on the Australian Alps fire (the fire complex including the Canberra fire) on child behaviour and development.

# 7. EFFECTS FIRE EXPOSURE ON CHILD BEHAVIOUR AND DEVELOPMENT

## *7.1. Introduction*

This chapter seeks to examine the effects of fire exposure on child development and behaviour. Accordingly, the chapter develops a cohort study using the Australian Government's Longitudinal Study of Australian Children (LSAC) dataset to examine potential relationships between *in utero* and early childhood stress and developmental changes in child temperament, child and maternal behaviour, and body weight. This follows the literature that suggests a possible negative effect of stress, both *in utero* and in early life, on these developmental domains. The dissertation examines a cohort of children included in the LSAC data set who were born to parents living in the fire area in the year following the 2003 Australian Alps fire (the complex which includes the Canberra fire). These children were exposed either to the fire or fire aftermath *in utero* and then also to the fire aftermath in their very early childhood. It then compares child and maternal behaviour, child emotional functioning, and child body weight of this exposed cohort to those of an unexposed cohort.

### 7.1.1. **Research question**

This chapter's central question concerns the effect of prenatal and early life bushfire exposure on developmental indicators in on populations exposed to the Canberra bushfire. It hypothesises that greater developmental comorbidity should be observed in children exposed to bushfires *in utero* or in early life. Disruptions to children's development related to stress will often affect the nature of their attachment to their parents (Chisholm 1999; Chisholm et al. 2005). For this reason, the analysis focused on social behaviours.

Environmental disasters are understood to cause stress to expectant mothers, potentially changing trajectory of their child's development due to exposure *in utero* to maternal stress; however, mothers who experience disaster while pregnant also have documented changes in health behaviours (Fuller 2013). It is thus the aim of this chapter to examine the impact of fire on maternal separation anxiety (i.e. the mother's reported anxiety on routine separation), attending prenatal health care, cigarette smoking, and alcohol consumption to examine whether exposure to disaster affects maternal health behaviour. Because some evidence shows that early life stress affects weight, the child's weight in kilograms at the Wave One interview is also assessed (Li et al. 2010).

## *7.2. Background*

Human behaviour and physiology is plastic across the lifespan, but that plasticity is greatest during periods of early growth, both *in utero* and in the first years of childhood. As children in this study will have been exposed to fire and fire aftermath–related stress, both *in utero* and in early life, this section focuses on the role of early life. An extensive discussion of *in utero* stress is located in Chapter Four.

Early life experience and resource availability emerge in both theory and research as principal influences on behavioural and phenotypic change, including those changes that affect the timing of life history traits (Chisholm 1999). In a biological sense, early life is defined as the period in which organisms can seek knowledge about their environment and have the ontogenic flexibility to respond to that knowledge. In research on humans, early life is generally defined as the first 1,000 days of life (Shonkoff 2011; Shonkoff and Garner 2012), although some authors extend this period until four years of age (Perry 2004).

Early life and the developmental origins of adult disorders is an area of increasing scientific and government focus. In developed nations, intervention in early life is increasingly viewed as a cost-effective method of improving adult outcomes and decreasing dependency on government-funded welfare and health services (Shonkoff 2011; Shonkoff and Garner 2012). Perry (2004 p.1) describes the opportunities presented by this early developmental period as a "biological gift". In this first 1,000 days, children are uniquely able to change and develop in ways which have lasting effects on their lifetime trajectories. For example, degree of exposure to vocabulary before three years of age is more robustly associated with adult literacy than access to school-based literacy programs (Hart and Risley 2003). For anthropologists, this susceptibility to lasting change in early life suggests a prolonged period of plasticity, beyond the natal plasticity discussed earlier, that may act to fine tune environmental adaptability.

### 7.2.1. Early life and attachment

While the effects of the prenatal environment might manifest through strictly biological mechanisms, such as altered methylation patterns, the effects of post-birth environmental instability are commonly understood to emerge as a result of evolved psychological mechanisms (EPMs). These EPMs encode decision rules which guide behavioural and physiological flexibility and are common to all humans (Gangestad and Simpson 2000). In particular, these EPMs are understood to function through childhood attachment, under the predictions of attachment theory. From evolutionary psychology, this theory suggests that, in early childhood, humans form bonds with their caregivers that are either secure or insecure and that these attachments form the basis of later life behaviour (Chisholm 1999). One possible role of responsive paternal care is to mitigate the action of the amygdala and HPA, as young children cannot calm themselves (emotional regulation is not believed to begin development until late toddlerhood). Attachment to care givers is also highly likely to be adaptive. Altricial human infants must receive intensive care to survive, so attachment to such a caregiver (and that caregivers' reciprocal attachment and investment) is necessary for survival (Chisholm 1999). Therapeutic applications of attachment theory view "secure" attachment as optimal and insecure attachment as pathological; however, applying life history theory (LHT) would not support that approach. In terms of LHT, there are no pathological forms of attachments, just variation in response to environmental pressure (Chisholm 1999).

### 7.2.2. Stress and development

In Shonkoff and Garner's (2012) extensive work on stress in childhood the stress response (collectively physical, psychological and behavioural), they provide a useful definition of the different kinds of stress experience by children. Stress is defined at three levels: "positive", "tolerable" and "toxic". This taxonomy reflects that fact that the stress response has an adaptive and positive role in promoting survival. Positive stress is defined as a normal part of development in which usual or everyday stressors results in a stress response, through which the child is supported (physically and psychologically) by its carers. This allows the child to develop understanding and tolerance of stressors and to understand that stress can be experienced and managed positively. The tolerable stress response is an exacerbation of the positive stress response in this case the stressor is more severe (e.g. a significant illness, death of a family member, divorce or environmental disaster) but the child is still supported through the response by carers and is able to return to homeostasis.

Shonkoff and Garner (2012) propose that these two first responses are unlikely to be harmful and that the positive stress response is necessary for normal psychological development. Conversely, the third stratum – the toxic stress response – is problematic. This finding is supported by others who also consider the effects of toxic-type stresses (Davis et al. 2011; Kishiyama et al. 2009; Kumar and Fonagy 2013; Perry 2009). The toxic stress response arises from severe, frequent or prolonged stress which cannot be moderated by carers (or carers are not available to moderate the stress). This kind of stress could be typified by children living in situations of abuse or chronic neglect (Kumar and Fonagy 2013). Under these conditions, the stress response becomes toxic because homeostasis cannot be restored, even over a prolonged time period and therefore the allostatic load is consistently high.

As discussed in Chapter Three, allostatic load in early life provides an indication of environmental stochasticity – it is a biological communication system. This load informs the developing body of the types of developmental adaptations that might act to aid survival and, ultimately, reproduction. For example, changes to the expression of genes which control neural development (Radtke et al. 2011) might act to assist a child in being more reactive to their environment, a useful trait in an environment which is threatening. Alternatively, early stress might accelerate sexual maturation to allow greater opportunities for reproduction (Chisholm et al. 2005), an adaptive shift where environmental dangers might mean that lifespan will be short and reproduction accordingly curtailed.

Exposure to stress *in utero* appears linked to a suite of developmental changes in the foetus, which are outlined extensively in Chapter Four. However, even after birth there appears to be sufficient neurological plasticity that stress experiences in early life also strongly influence developmental and behavioural outcomes. Indeed, in humans, it can be very difficult to determine the separate influence of late prenatal and earl post-natal stress exposure (Boersma et al. 2014). The role of early life stress, particularly in terms of behavioural outcomes, is likely due to the intensity of brain development in early childhood. The bulk of brain development occurs early in life, with around 90% of total brain growth usually achieved by four years of age (Perry 2004). During this period of rapid and important growth, both genetics and environment are highly relevant and intertwined. Sameroff (2010 p.234) describes this ongoing interplay between genetics and environment as “nature dancing with nurture over time”.

The brain’s development is a use-dependent process – those parts of the brain that are stimulated by the child’s environment grow and develop more rapidly and completely. Because brain development occurs as a “bottom-up” process, the timing of environmental disturbances changes the extent and nature of the developmental response. Therefore, the brain of an 18-month-old child will respond differently to that of a five year-old child to the same stimuli. Because each brain area functions on its own individual timetable, the differences are complex (Perry 2009). Additionally, changes to brain development are cumulative, so the child's first initial stress exposure will create a difference response to that of subsequent changes. Therefore, although it is widely accepted that timing is important in terms of brain development, there is debate on how these changes would manifest in terms of clinical impacts for individuals (Perry 2009).

In humans, exposure to stressors in childhood is shown to alter the size and architecture of the amygdala, hippocampus, and prefrontal cortex – all of which are central to stress management, mood control, and executive functions such as learning and memory (Shonkoff and Garner 2012). Perry (2004) finds that children who experienced extreme neglect before the age of four years have smaller brains and abnormally developed cortexes. Chronic stress is also associated with hypertrophy and hyperactivity in the amygdala and orbitofrontal cortex. The impact of stress on the amygdala is particularly interesting as the amygdala is central to instigating and mediating fear and anxiety and, therefore, is important in the future emotional functioning of individuals. Shonkoff and Garner (2012 p.236) conclude that hyper-responsivity in amygdala can result in a "chronically activated physiologic stress response, along with increased potential for fear and anxiety". There is concordant evidence that chronic stress in early life diminishes the capacity of the brain to moderate the stress response. For example, because cortisol can inhibit neurogenesis in the hippocampus, longer-term exposure to cortisol may therefore diminish functioning of the hippocampus and its capacity to regulate the stress response (Barrett and Swan 2015; Shonkoff and Garner 2012). Such neurogenic changes may act as a self-reinforcing cascade. As the development of the amygdala and frontal cortex is compromised, so is the child's ability to manage fear and arousal (Davis et al. 2011; Kumar and Fonagy 2013). This may lead to a deregulated stress response that increases the rate of change to neurogenesis – creating a self-reinforcing process of excessive stress response and altered neurogenesis (Perry 2004).

From an evolutionary perspective, alterations that enhance threat perception and the ability to manifest "flight or fight" responses have a clear survival dimension. In the environment of evolutionary adaptiveness (EEA), environmental signals of danger, through either maternal stress experienced *in utero* or direct experiences of stress, would support useful changes to increase threat responsivity since being easily aroused would act to preserve life (Gray 2010; Nepomnaschy and Flinn 2009). However, when lifelong increases in emotional responsivity are made in response to early life stress, this can lead to a problematic mismatch between modern environments and the adaptation (Radtke et al. 2011). Although being highly responsive may function usefully to increase short-term survival in the EEA, highly responsive children can struggle within modern environments, such as schools, to manage their impulsivity.

### 7.2.3. Early stress and life trajectory

Stressful early life experiences appear related to later life outcomes in ways that may create a problematic interaction between early life priming and the modern environment. Differences in life outcomes detected in adults who were exposed to high-stress childhood environments include: early initiation of drinking alcohol; greater risk of obesity; increased number of sexual partners; greater risk of problem gambling; and greater use of alcohol, tobacco, and other drugs (Putnam 2015). The increase in risk-taking behaviours in turn leads to greater risks of school drop-out, single parenthood, gang membership, unemployment and poverty, homelessness, violent crime, and incarceration (Shonkoff and Garner 2012). Unsurprisingly these factors are also linked to an increase in the prevalence of adult health problems. This is likely related to greater lifestyle risk factors, reduced quality and availability of preventative health care, and reduced access to appropriate medical treatment over the lifespan. Additionally, chronic stress exposure is understood to diminish immune function and increase somatic inflammatory markers, increasing both the risks and consequences of disease (Cole 2009). All these behaviours indicate underlying aspects of future discounting and high allostatic load that are consistent with the predictions of LHT.

Similarly, through the application of future-present and quality-quantity trade-offs, a predicted response to stochasticity is the front-loading of reproductive potential (Coall et al. 2016). In an environment which threatens survival, early reproduction (at whatever cost) is favoured because death might prevent later reproductive opportunities. Because life is unlikely to be long in such a stochastic environment, any later life costs of early reproduction are unlikely to be realised and therefore are not counted against the benefits of early reproduction. This theoretical prediction of stress driving earlier maturation appears supported by observation (Coall et al. 2016).. Across a number of studies, females with less secure upbringings demonstrate early menarche, younger age at first birth and greater lifetime fertility (Belsky 2012). Such patterns are particularly apparent in female children raised in households where the father is present compared with those where the father is absent. Girls raised in father-absent households demonstrate earlier menarche and younger age at first sex, as well as greater number of sexual partners (Chisholm 1999; Chisholm et al. 2005).

Responding to a potentially precarious environment by altering the biological cascade that triggers sexual maturation makes good adaptive sense in an environment where pursuing high-quality and low-quantity reproduction is less likely to result in a good payoff (Chisholm et al. 2005). Changes to biology can be observed through earlier puberty and menarche. In girls who have experienced early life stress, physiological maturity is correlated with early first sex and first birth (Chisholm et al. 2005). However, other studies of general populations of post-menopausal women do not consistently find an association between onset of puberty and timing of first birth, likely due to the use of contraception (Milne and Judge 2012). Such patterns may emerge in response to *in utero* stress: daughters of mothers who smoke during pregnancy, for example, experience earlier menarche (Behie and O'Donnell 2015). Notably, some of the resulting adaptations, such as early menarche (Milne et al. 2011), correlate with later life health risks, suggesting that long-term fitness has been sacrificed in order to benefit short-term survival.

While many studies show the links mentioned above, it is difficult empirically to demonstrate causation between physiological experiences of stress in early life and later life outcomes, due to the presence of numerous potentially confounding factors. For instance, evidence suggests that children who experience stress (e.g. children raised in poverty) have measurably altered functioning in the prefrontal cortex (Kishiyama et al. 2009). However, it is not possible to determine whether these changes occur *in utero* or in early life – that is, either as a direct outcome of the maternal stress response, or through experience of environmental deficits in early childhood, or, most probably, to a combination of both. Examples of early deficits include: insufficient parent-child interaction, insufficient nutrition, and insufficient access to the early childhood development activities. Nevertheless, associations between environments that are understood to be stressful and changes in neural development are widely found and it is possible to construct plausible evolutionary and biological explanations for the effects of early life stress experience on observed later life conditions.

## *7.3. Method*

### 7.3.1. **Ethical approval**

The ethical aspects of the study in this chapter were approved by The Australian National University Human Research Ethics Committee (ANU-HREC) on 23 September 2013 (project reference: 2013/143). Following approval by the ANU-HREC, access to the unconfidentialised dataset was requested from the Department of Social Services and access was approved on 18 November 2013.

### 7.1.1. **Collection methods**

The LSAC dataset is collected by the Australia Government. It is owned and funded by the Department of Social Services, with data is collected by the Australian Bureau of Statistics. LSAC comprises a stratified randomised nation-wide sample of 10,000 children across two cohorts of 5,000 each (named the *B* and *K* cohorts).

Much of the data in the LSAC survey is collected through face-to-face and computer assisted interviews with a parent, termed “Parent One”, who self identifies as the principal carer. This parent is very often the study child's mother, although some are fathers (n = 15) or grandmothers (n = 1). The remainder of the information, including anthropometrics, is collected by the interviewer directly from the child.

The study commenced in March 2004 when the B cohort was 3–19 months old and the K cohort was 4–5 years old. The B cohort represents children born between 1 March 2003 and 1 February 2004. This study uses only the B cohort, as they were either *in utero* at the time of the fire or were conceived in the fire aftermath. LSAC data was collected every two years from 2004 onwards, with each collection period termed a "wave" and number sequentially. Table 7-1 shows the LSAC data collection years and face-to-face data collection interview response rates.

Table 7-1: Data collection years for each wave of the LSAC

| Wave | Data collection year | Face-to-Face response rate |
|---|---|---|
| 1 | 2004 | 100% |
| 2 | 2006 | 90% |
| 3 | 2008 | 86% |
| 4 | 2010 | 82% |
| 5 | 2012 | 80% |

Participants in LSAC are the study child, their parents, carers and teachers. The study has maintained very high response rates over subsequent waves, including 100% response rates for the interview component.

### 7.1.2. **Sampling strategies**

The size of the sample is constrained by the LSAC sampling and power calculations which suggested that too few individuals are in the ACT sample. To rectify this, the affected area is extended to include the whole area affected by the Australian Alps fires which included areas of Victoria and New South Wales (NSW) along the 800 km fire line (the fire complex that culminated in the Canberra fire). The areas of Ovens-Murray (SA2 codes of 24505, 24510, and 24515) (n = 19), Snowy (SA2 codes of 14505 and 14510) (n = 12) and Upper Murray (SA2 codes of 15505, 15510, 15515 and 15520) (n = 45) are thus included as fire affected areas, as well as the same areas of Canberra determined as fire affected in Chapters Four, Five and Six: Belconnen (80510), Weston Creek-Stromlo (80520), Tuggeranong (80525) and Woden Valley (80515).

To create a comparison group, all other non-metropolitan areas of Victoria and NSW are included in the sample, as well as the remaining regions of the ACT (as outlined in Chapter Five). These populations are both geographically proximate and are not drawn from major metropolitan cities (the two nearest capital cities, Sydney and Melbourne, have populations more than 14 times larger than Canberra); therefore, they might be expected have broadly comparable social, political and geographic environments. This approach excludes children from the greater Sydney and Melbourne areas to reduce the degree of non-fire environmental variability.

Residential address at Wave One is used as a proxy for residential address at the time of the fire (a year earlier that the commencement of data collection). No information on movement patterns between the fire and date of data collection is available. HIC identification codes were used to merge subsequent waves of the dataset, so that participants are identified as fire exposed based on their Wave One residential address in the later waves of the dataset.

The number of participants in each category at each wave are shown in Table 7-2. Some natural attrition occurred over time. This sample is termed the Australian Alps LSAC

(AALSAC) sample. The confidence interval for the combined exposure sample size is shown in Table 7-2, where the confidence level is set at 95%, the proportion percentage at 0.05, and the population at 200,000 for all waves.

Table 7-2: Sample size for AALSAC sample and confidence interval

| Waves | *In utero* during fire | Conceived after fire | Exposure combined | Not exposed to fire | Total | Confidence interval |
|---|---|---|---|---|---|---|
| Wave 1 | 96 | 49 | 145 | 965 | 1110 | 0.08 |
| Wave 2 | 93 | 49 | 142 | 854 | 996 | 0.08 |
| Wave 3 | 93 | 46 | 139 | 809 | 948 | 0.08 |
| Wave 4 | 89 | 45 | 134 | 778 | 912 | 0.08 |
| Wave 5 | 84 | 43 | 127 | 751 | 878 | 0.09 |

### 7.1.3. **Data cleaning procedures**

In the wave three dataset, 108 cases did not have geographical codes, where these could not be matched to cases in the previous wave using HIC identification codes (this was possible in three cases) they were deleted. Because very few children were reported as either Torres Strait Islander or both Aboriginal and Torres Strait Islander, these categories are combined with 'Aboriginal' in later analyses. Table 7-3 shows an example of the combined categories in the Wave One dataset.

Table 7-3: Aboriginal or Torres Strait Islander (ATSI) status of AALSAC children in Wave One (0–1 years old).

| Old categories | Count | % | New categories | Count | % |
|---|---|---|---|---|---|
| No | 1042 | 93.9 | No | 1042 | 93.9 |
| Yes – Aboriginal | 61 | 5.5 | Yes | 68 | 6.1 |
| Yes – Both | 3 | 0.3 | | | |
| Yes – Torres Strait Islander | 4 | 0.4 | | | |

Similarly, plurality is combined into a binary variable with all multiple births included in a “multiple” category. The marital status of parents was similarly combined into coupled and single, with coupled including all forms of partnership (e.g. de-facto and same-sex unions). One case involves a child living with both parents who did not identify as partners. This single case was recoded as missing to exclude it from further analysis. Types of prenatal care used are shown in

Table 7-4. Because “other” and “shared care” had low counts, they were combined. Because only one parent reported “nobody” and this category was not suitable for combination with another care category, that data was recoded as missing. General Practitioners (GPs) are the most common source of prenatal care.

Table 7-4: Prenatal health care providers in AALSAC sample in Wave One (0–1 years old).

| Old categories | Count | % | New categories | Count | % |
|---|---|---|---|---|---|
| Nobody | 1 | 0.1 | GP | 451 | 40.6 |
| GP | 451 | 40.6 | Obstetrician | 395 | 35.6 |
| Obstetrician | 395 | 35.6 | Midwife/Nurse | 181 | 16.3 |
| Midwife/Nurse | 181 | 16.3 | Shared care/Other | 79 | 7.1 |
| Shared care | 56 | 5.0 | | | |
| Other | 23 | 2.1 | | | |
| Missing | 3 | 0.3 | Missing | 4 | 0.4 |

Almost all mothers attended prenatal care, with most having between seven and nine visits. Because mothers who had fewer than four visits were rare, they were regrouped into a single category and were excluded from further analysis. The change in prenatal care categories is shown in Table 7-5.

Table 7-5: Prenatal care visits in AALSAC sample in Wave One (0-1 years old).

| Old categories | Count | % | New categories | Count | % |
|---|---|---|---|---|---|
| None | 2 | 0.2 | Under 4 visits | 14 | 1.3 |
| 2 visits | 5 | 0.5 | 4–6 visits | 98 | 8.8 |

| | | | | | |
|---|---|---|---|---|---|
| 3 visits | 7 | 0.6 | 7–9 visits | 792 | 71.4 |
| 4–6 visits | 98 | 8.8 | 10 or more visits | 199 | 17.9 |
| 7–9 visits | 792 | 71.4 | | | |
| 10 or more visits | 199 | 17.9 | | | |
| Missing | 7 | 0.6 | Missing | 7 | 0.6 |

Parent Ones are asked to report their assessment of whether they have many problems and how well they are coping well with life generally. Most reported that they had “few problems or stresses” or “some problems or stresses”. Because few parents in each wave report having “very many problems or stresses”, they are combined with the “many problems or stresses” category for analysis. In Wave One, and in each subsequent wave, “not coping at all” is represented by very low counts. To manage this, “not coping at all” is combined with “coping a little” in each wave for analysis.

## 7.4. Data analysis

The analytic approach used was guided by the statistical adviser to this dissertation. Unbalanced Analyses of Variance (ANOVAs) are used to conduct an analysis of the influence of being in a fire area during gestation and other independent variables, these include temperament and behaviour domains and the child’s current body weight (at the time of data collection, in kilograms) for each wave of the AALSAC sample. The measures used at each wave are summarised in Table 7-6.

Table 7-6: Outcome measures used in the analysis of the AALSAC data Waves One to Five.

| Measures | Wave 1 (0-1 y.o.) | Wave 2 (2-3 y.o.) | Wave 3 (4-5 y.o.) | Wave 4 (6-7 y.o.) | Wave 5 (8-9 y.o.) |
|---|---|---|---|---|---|
| Maternal separation anxiety | ✓ | - | - | - | - |
| Child’s weight (as either kg or BMI) | ✓ | ✓ | ✓ | ✓ | ✓ |

| | | | | | |
|---|---|---|---|---|---|
| PEDS psychosocial health summary | - | ✓ | ✓ | ✓ | ✓ |
| PEDS emotional functioning | - | ✓ | ✓ | ✓ | ✓ |
| PEDS social functioning | - | ✓ | ✓ | ✓ | ✓ |

The influences of the following variables, which are understood to alter child wellbeing and behaviour, are tested for their presence in a fire area.

Most of these variables are indicated in previous work on the effects of social and economic disadvantage on children's development (Kishiyama et al. 2009; Perry 2004; Perry 2009). Some, such as Parent One's depression history, are included on the advice of the data manager regarding strong correlations in this dataset between parental and child mental health. Parental weight is included as a possible influence on behaviour, as high body weights are skewed in the Australian population toward disadvantaged groups (Australian Bureau of Statistics 2008).

Three measures of Parental Evaluation of Developmental Status (PEDS) social and emotional functioning were used as outcome variables: the PEDS psychosocial health summary scores, the PEDS emotional functioning scores and the PEDS social functioning scores. These are assessed from Wave Two onwards. They are all guided parental assessments of the children development level. The following independent variables were included in the analyses of the three PEDS scales:

- child's Aboriginal and/or Torres Strait Islander status;
- Parent One reporting any stressful life events in the past year;
- Parent One depression scales;
- Parent One's assessment of the difficulty of life at present;

- Parent One's assessment of their level of coping;
- Parent One's weight status by category;
- child's sex; and
- Parent One's assessment of the family's level of financial hardship.

For the analysis of children's weight, Parent One depression was removed and the following independent variables were added: birth weight; child's age in months; and breast feeding status. For analysis of maternal health behaviours, the following dependent variables were tested: alcohol consumption during pregnancy; smoking during pregnancy; average daily alcohol consumption; average daily cigarette smoking; number of prenatal care visits; and use of vitamin supplements. These are tested using the independent variables listed for the PEDS scales, in addition to prenatal care provider type.

Where interactions cannot be interpreted via the ANOVA, multiple linear regression was used. Additionally, the relationships between maternal health behaviours and fire exposure are tested by analysing any effect of being in a fire area at time of interview at Wave One on maternal cigarette, alcohol and prenatal vitamin consumption during pregnancy and number of prenatal care provider practitioner visits. All analysis is conducted using Genstat 16th Edition. For all analysis, significance is set at $p = 0.05$.

### 7.4.1. Children's characteristics

Descriptive analysis was conducted for relevant variables. Body Mass Index (BMI) scores for all AALSAC children at each wave are shown in Table 7-7, except for Wave One where BMI is not reported due to the children's age, weight in kilograms is reported instead. In each wave, Aboriginal and Torres Strait Islander children are intentionally oversampled compared to the general population. Table 7-7 shows the characteristics of children included in this study.

Table 7-7: Descriptive statistics for children in the AALSAC sample divided by fire exposure: comparison group (CG), FIU (fire experience *in utero*) or CDA (conceived during fire aftermath).

| Descriptive statistics - Child | | Wave 1 (0-1 y.o.) | | | Wave 2 (2-3 y.o.) | | | Wave 3 (4-5 y.o.) | | | Wave 4 (6-7 y.o.) | | | Wave 5 (8-9 y.o.) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exposure level | | CG | FIU | CDA | CG | FIU | CDA | CG | FIU | CDA | CG | FIU | CDA | CG | FIU | CDA |
| Birth weight (grams) | mean | 3430.96 | 3461.16 | 3504 | - | - | - | - | - | - | - | - | - | - | - | - |
| | s.d | 535.85 | 503.77 | 798.63 | - | - | - | - | - | - | - | - | - | - | - | - |
| Gestation (weeks) | mean | 39.30 | 39.4 | 39.48 | - | - | - | - | - | - | - | - | - | - | - | - |
| | s.d | 1.91 | 1.84 | 1.92 | - | - | - | - | - | - | - | - | - | - | - | - |
| Children’s age (months) | mean | 9.4 | 10.38 | 6.86 | 33.99 | 35.26 | 31.63 | 57.44 | 59.26 | 54.59 | 82.13 | 84.15 | 78.84 | 107.41 | 108.7 | 105.02 |
| | s.d | 2.59 | 1.91 | 1.73 | 2.98 | 2.7 | 2.56 | 2.83 | 2.45 | 1.57 | 3.42 | 3.28 | 2.35 | 3.34 | 2.97 | 2.04 |
| Children’s weight measure: | mean | 9.32 | 9.34 | 8.32 | 16.97 | 16.83 | 16.65 | 16.41 | 16.26 | 16.17 | 16.63 | 16.48 | 16.29 | 17.79 | 17.46 | 17.12 |
| | s.d | 1.55 | 1.38 | 1.31 | 1.63 | 1.45 | 1.99 | 1.72 | 1.61 | 2.13 | 2.22 | 1.88 | 1.93 | 3.08 | 2.74 | 3.02 |
| PEDS psychosocial health summary scores | mean | - | - | - | 81.58 | 80.35 | 82.90 | 80.29 | 79.37 | 80.62 | 78.56 | 75.71 | 78.28 | 76.94 | 77.28 | 78.55 |
| | s.d | - | - | - | 11.34 | 11.42 | 9.96 | 12.01 | 11.65 | 12.13 | 13.31 | 12.62 | 15.67 | 15 | 14.77 | 15.52 |
| PEDS emotional functioning scores | mean | - | - | - | 75.34 | 72.21 | 75 | 75.6 | 75 | 75.24 | 75.8 | 72.58 | 74.78 | 74.45 | 74 | 74.77 |
| | s.d. | - | - | - | 13.62 | 14.88 | 13.43 | 14.07 | 14.45 | 15.45 | 14.37 | 14.42 | 17.51 | 16.83 | 15.67 | 18.74 |
| PEDS social | mean | - | - | - | 88.05 | 89.14 | 90.97 | 85.04 | 83.85 | 85.75 | 81.35 | 79.11 | 81.72 | 79.45 | 80.86 | 82.33 |

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| functioning scores | s.d. | - | - | - | 12.59 | 12.09 | 10.47 | 14.22 | 12.57 | 13.28 | 16.69 | 15.73 | 16.95 | 17.68 | 17.46 | 17.19 |
| Percentage multiple births | | 2.9 | 1.04 | 2.04 | - | - | - | - | - | - | - | - | - | - | - | - |
| Percentage ATSI | | 6.84 | 2.08 | 0 | 5.97 | 2.15 | 0 | 5.44 | 2.15 | 0 | 3.73 | 2.1 | 2.04 | 4.01 | 2.38 | 2.63 |
| Percentage male | | 53.78 | 50 | 53.06 | 53.63 | 49.46 | 53.06 | 53.28 | 49.46 | 52.17 | 53.86 | 51.69 | 53.33 | 54.1 | 48.81 | 55.81 |

### 7.4.2. **Parents' characteristics**

Parent One characteristics are shown in Table 7-8. Maternal separation anxiety, the degree of anxiety experienced by mothers (only) on routine separation from their child (e.g. when leaving the child in the care of trusted others) was measured at Wave One only. Whether Parent One had experienced depression for two weeks or longer in the past two years is also recorded according to self-report (i.e. no diagnosis of depression is required).

Exposure to stressful life events is measured using an index measure at each wave. Stressful life events include: losing employment, unsuccessfully seeking employment, legal difficulties, financial crises, professional and work stresses, illness, assault or injury, pregnancy, ending a relationship, or the death of a friend or family member. Parent One was also asked about the family's financial position and this data is scaled to create a financial hardship score.

Parent One age and BMI at the time of interview for each wave are also recorded. Most parents in the sample are overweight (a BMI above 25) or close to overweight, reflecting the trend in the broader Australian population.

Table 7-8: Descriptive statistics for whole of AALSAC sample parental characteristics divided by fire exposure: either CG (comparison group), FIU (fire experience *in utero*), or CDA (conceived during fire aftermath).

| Descriptive statistics – Parent | | Wave 1 (child is 0-1 y.o.) | | | Wave 2 (child is 2-3 y.o.) | | | Wave 3 (child is 4-5 y.o.) | | | Wave 4 (child is 6-7 y.o.) | | | Wave 5 (child is 8-9 y.o.) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exposure level | | CG | FIU | CDA | CG | FIU | CDA | CG | FIU | CDA | CG | FIU | CDA | CG | FIU | CDA |
| Parent One age (years) | mean | 30.03 | 31.59 | 29.04 | 32.45 | 33.74 | 31.12 | 34.53 | 35.69 | 32.80 | 37.02 | 38.39 | 35.91 | 38.81 | 39.85 | 38.49 |
| | s.d | 5.64 | 5.34 | 5.46 | 5.56 | 5.51 | 5.45 | 5.21 | 5.48 | 5.56 | 5.28 | 5.61 | 5.81 | 5.3 | 5.44 | 6.29 |
| Parent One BMI | mean | 25.67 | 26.33 | 25.63 | 25.6 | 25.49 | 25.2 | 26.35 | 26.26 | 24.99 | 26.73 | 26.94 | 26.22 | 27.29 | 26.65 | 25.97 |
| | s.d | 5.41 | 6.18 | 5.11 | 5.33 | 4.47 | 4.93 | 5.63 | 5.75 | 5.21 | 6.1 | 6.1 | 5.78 | 6.42 | 6.07 | 5.64 |
| Parent One life stressors scale | mean | 1.44 | 1.26 | 1.14 | 1.19 | 1.45 | 1.27 | 1.08 | 0.91 | 0.92 | 2.95 | 2.56 | 2.24 | 2.63 | 2.38 | 2.88 |
| | s.d. | 1.61 | 1.40 | 1.32 | 1.3 | 1.16 | 0.96 | 1.36 | 1.21 | 1.2 | 2.57 | 2.04 | 2.24 | 2.52 | 1.89 | 2.56 |
| Maternal separation anxiety scores | mean | 2.60 | 2.81 | 2.44 | - | - | - | - | - | - | - | - | - | - | - | - |
| | s.d. | 0.87 | 0.79 | 0.92 | - | - | - | - | - | - | - | - | - | - | - | - |
| Parent One hardship scale | mean | 0.61 | 0.49 | 0.24 | 0.34 | 0.23 | 0.15 | 0.36 | 0.29 | 0.24 | 0.32 | 0.33 | 0.11 | 0.29 | 0.35 | 0.19 |
| | s.d. | 1.05 | 0.88 | 0.52 | 0.77 | 0.53 | 0.36 | 0.77 | 0.77 | 0.6 | 0.76 | 0.85 | 0.32 | 0.73 | 0.80 | 0.5 |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Percentage of Parent One identified as female | 98.65 | 98.96 | 100 | 98.71 | 97.85 | 100 | 98.39 | 98.92 | 97.83 | | | | | | |
| Percentage of parents coupled | 87.97 | 95.83 | 85.71 | 88.52 | 95.7 | 87.76 | 88.71 | 96.77 | 84.78 | 85.73 | 91.01 | 86.67 | 84.89 | 88.1 | 88.37 |
| Percentage Parent One reporting ‘many’ or ‘very many’ problems and stresses | 7.01 | 6.74 | 4.65 | 7.33 | 4.30 | 0 | 9.89 | 3.66 | 7.32 | 9.86 | 6.74 | 8.89 | 7.85 | 3.57 | 6.98 |
| Percentage Parent One reporting coping ‘fairly well’ or worse | 41.75 | 34.83 | 44.19 | 33.69 | 31.87 | 35.42 | 53.95 | 54.88 | 46.34 | 48.58 | 42.70 | 44.44 | 48.17 | 59.52 | 41.86 |
| Percentage Parent One reporting depression in past two weeks | 29.23 | 26.97 | 34.88 | 22.28 | 29.35 | 27.66 | 28.98 | 31.71 | 26.83 | 32.3 | 31.46 | 37.78 | 28 | 29.76 | 39.53 |
| Percentage of mothers reporting breastfeeding | 90.77 | 95.83 | 91.84 | 49.41 | 60.22 | 65.31 | 50.80 | 60.22 | 65.22 | - | - | - | - | - | - |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Percentage of mothers reporting drinking alcohol at any time during gestation | 21.76 | 17.78 | 20.93 | - | - | - | - | - | - | - | - | - | - | - | - |
| Percentage of mothers reporting smoking cigarettes at any time during gestation | 21.76 | 17.78 | 20.93 | | | | | | | | | | | | |
| Percentage of mothers reporting consuming prenatal vitamins at any time during gestation | 21.58 | 30.21 | 22.45 | - | - | - | - | - | - | - | - | - | - | - | - |
| Percentage of mothers reporting | 5.07 | 5.56 | 2.33 | - | - | - | - | - | - | - | - | - | - | - | - |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| gestational diabetes | | | | | | | | | | | | | | | |
| Percentage of mothers reporting pregnancy-related hypertension | 10.33 | 10 | 4.65 | - | - | - | - | - | - | - | - | - | - | - | - |

### 7.4.3. **Limitations**

Although the small sample size limits the generalizability and power of this analysis, the size is nevertheless sufficient to allow some analysis to be conducted. Limitations discussed in earlier chapters regarding inherent inaccuracies in identifying fire exposed individuals also apply to this analysis.

## *7.5. Results*

Results of ANOVA analysis for each wave of the AALSAC sample are presented below, followed by tables of means for significant results. Where appropriate, associations are further examined using linear regression. Four temperament and behaviour domains (maternal separation anxiety, PEDS psychosocial summary, PEDS emotional functioning, and PEDS social functioning) are also examined in relation to fire exposure. All models are shown in Appendix C.

### 7.5.1. **Child's body weight**

The influence of a range of factors on children's weight over time is shown at Table 7-9. Perinatal factors are significant in the short-term, while the influence of Parent One's own weight increases with time.

Table 7-9: Results for ANOVA of children's weight (Wave One only) and BMI (from Wave Two onwards) against listed variables. Significant effects are marked with an asterisk.

| Year | Wave 1 (0-1 y.o.) Kgs | Wave 2 (2-3 y.o.) BMI | Wave 3 (4-5 y.o.) BMI | Wave 4 (6-7 y.o.) BMI | Wave 5 (8-9 y.o.) BMI |
|---|---|---|---|---|---|
| ATSI | 0.91 | 0.27 | 0.08 | 0.93 | 0.93 |
| Birth weight | <.001* | 0.06 | - | -- | -- |
| Breastfed | <.001* | <.001* | 0.17 | -- | -- |
| Child's age | <.001* | 0.01* | 0.47 | 0.69 | 0.02 |
| Child's sex | <.001* | 0.00* | 0.10 | 0.25 | 0.06 |
| Parent One weight status | 0.87 | 0.61 | <.001* | <0.001* | <0.001* |
| Hardship | 0.03* | 0.81 | 0.57 | 0.23 | 0.20 |
| Stressful life events | 0.46 | 0.94 | 0.04* | 0.39 | 0.56 |
| Problems | 0.85 | 0.17 | 0.40 | 0.37 | 0.8 |
| Coping | 0.93 | 0.46 | 0.18 | 0.16 | 0.61 |
| Fire exposure | 0.06 | 0.27 | 0.36 | 0.69 | 0.70 |

Birth weight is associated with higher body weight only when children are under one year of age ($p < 0.001$). Breastfeeding was significantly associated with lower weight at less than one year-old ($p < 0.001$) and with lower BMI at between two and three years ($p < 0.001$). However, the association between breastfeeding and body weight dissipates by four to five years. The child's sex is influential with males being significantly heavier under one year ($p < 0.001$) and with higher BMIs at between two and three years ($p = 0.00$). However, this effect is not significant between four and seven years old but nears significance again ($p = 0.06$) once the children are eight to nine years old.

Parental weight status is not a significant influence on children's weights at less than one year-old but becomes significant after four years of age ($p < 0.001$). In all cases parental weight is positively correlated with children's BMIs.

When children are between four and five years old, Indigeneity approaches significance (*p* =0.08), with children who identified as Aboriginal, Torres Strait Islander or both significantly lighter on average than their peers who did not identify as Indigenous. However, this does not occur when the children are younger or older. Similarly, exposure to stressful life events is significant (*p* = 0.04) when children are between four and five years but not at any other age.

Fire exposure is nears significance when children are below one year (*p* = 0.06) but there was no significant trend thereafter. Children exposed to the fire *in utero* are lighter at one year (mean = 8.95kgs) than children conceived in the aftermath (mean = 9.18 kgs) and unexposed children (mean = 9.28kgs).

### 7.5.2. **PEDS psychosocial summary**

Levels of parental financial hardship, self-reported of levels of problems, and degree of coping all appears to have a significant effect on children's psychosocial functioning. Table 7-10 shows the results of an unbalanced ANOVA examining the PEDS psychosocial summary score.

Table 7-10: P-values arising from ANOVA for PEDS psychosocial summary scores against relevant variables. Significant effects are marked with an asterisk.

| Year | Wave 2 (2-3 y.o.) | Wave 3 (4-5 y.o.) | Wave 4 (6-7 y.o.) | Wave 5 (8-9 y.o.) |
|---|---|---|---|---|
| ATSI | 0.01 | 0.19 | 0.20 | 0.33 |
| Child's sex | 0.17 | 0.06 | 0.10 | 0.03* |
| Parent One weight status | 0.18 | 0.37 | 0.13 | <0.001* |
| Hardship scale | 0.38 | <0.001* | <0.001* | <0.001* |
| Stressful life events | 0.04* | 0.02* | <0.001* | 0.007* |
| Parent One depression | 0.002* | 0.00* | <0.001* | <0.001* |
| Problems | <0.001* | <0.001* | <0.001* | <0.001* |
| Coping | <0.03* | <0.001* | 0.07 | 0.06 |
| Fire exposure | 0.65 | 0.73 | 0.04* | 0.68 |

Hardship does not significantly affect children psychosocial functioning scores when they are between two and three years but emerges as significant from four to five years onwards. Identifying as Indigenous ($p = 0.01$) or having a principal parent who had experienced depression in the past year ($p < 0.00$) are significant when children were between two and three years but are not significant once over four years. Predicted means indicate that children in both these situations had lower psychosocial functioning than their peers. Parental self-reports of number of problems and level of coping were related to children's psychosocial functioning, with children of parents who experience many problems or with lower coping scores having lower average psychosocial functioning scores. The analysis shows an effect associated with being in a fire area at Wave Four only..

### 7.5.3. PEDS emotional functioning

Analysis of children's emotional functioning is consistent with the analysis of psychosocial functioning. An unbalanced ANOVA of relevant factors (see Table 7-11) indicates that identifying as Indigenous ($p = 0.01$) or having a principal parent who has had depression in the past year ($p < 0.001$) had a significant effect on children's emotional functioning at Wave One. Financial hardship emerges as significant ($p < 0.001$) at Wave Two. Levels of parental problems and degree of coping remained significant across all waves. Again, the analysis shows an effect associated with being in a fire area at Wave Four only.

Table 7-11: P-values arising from logistic regression for PEDS emotional functioning concern outcomes against relevant variables. Significant effects are marked with an asterisk.

| Year | Wave 2 (2-3 y.o.) | Wave 3 (4-5 y.o.) | Wave 4 (6-7 y.o.) | Wave 5 (8-9 y.o.) |
|---|---|---|---|---|
| ATSI | 0.004* | 0.39 | 0.59 | 0.72 |
| Child's sex | 0.22 | 0.75 | 0.25 | 0.10 |
| Parent One weight status | 0.42 | 0.38 | 0.27 | <0.001* |
| Hardship scale | 0.12 | <0.001* | <0.001* | 0.01* |
| Stressful life events | <0.001* | <0.001* | <0.001* | 0.01* |
| Parent One depression | 0.05* | <0.001* | <0.001* | <0.001* |
| Problems | <0.001* | <0.001* | <0.001* | <0.001* |
| Coping | 0.02* | 0.02 | 0.01* | 0.23 |
| Fire exposure | 0.12 | 0.96 | 0.02* | 0.89 |

Predicted means indicate that children identified as Indigenous or whose principal parent has lower psychosocial functioning than their peers. As with psychosocial functioning, the number of problems parents experience and their level of coping, is negatively related to children's emotional functioning, with children of parents who experience many problems or who were coping less well having lower average emotional functioning scores.

### 7.5.4. PEDS social functioning

Social functioning appears more robust than psychosocial functioning or emotional functioning. The level of parental problems is significant at Waves Two, Three and Five ($p < 0.00$), while degree of parental coping approaches significance in Wave Two and Five, and was significant at Wave Three and Four. The analysis shows no effect associated with being in a fire area.

Table 7-12: P-values arising from ANOVA for PEDS social functioning scores against relevant variables. Significant effects are marked with an asterisk.

| Year | Wave 2 (2-3 y.o.) | Wave 3 (4-5 y.o.) | Wave 4 (6-7 y.o.) | Wave 5 (8-9 y.o.) |
|---|---|---|---|---|
| ATSI | 0.31 | 0.17 | 0.12 | 0.06 |
| Child's sex | 0.2 | 0.006* | 0.10 | 0.05* |
| Parent One weight status | 0.35 | 0.63 | 0.29 | <0.001* |
| Hardship scale | 0.89 | 0.002* | <0.001* | <0.001* |
| Stressful life events | 0.3 | 0.78 | 0.07 | 0.04* |
| Parent One depression | 0.14 | 0.16 | <0.001* | <0.001* |
| Problems | <0.001* | 0.008* | <0.001* | 0.006* |
| Coping | 0.12 | 0.003* | 0.41 | 0.05* |
| Fire exposure | 0.74 | 0.64 | 0.32 | 0.46 |

In each measure, greater levels of problems and lower degrees of coping lead to decreases in children's average social functioning.

### 7.5.5. Maternal separation anxiety

Maternal anxiety is measured in the AALSAC with higher scores indicating greater anxiety. Maternal anxiety is only measured when the children were younger than one year. The unbalanced ANOVA analysis shows that identifying as Aboriginal, Torres Strait Islander or both ($p = 0.003$) and higher scores on the financial hardship scale ($p <$

0.001) are significantly related to changes in maternal anxiety. The mother's age also approaches significance at $p = 0.07$, with subsequent multiple linear regression showing that older mothers experience greater anxiety. The analysis shows no effect of being exposed to the fire.

Table 7-13: Table of predicted means for maternal separation anxiety scale values arising from an ANOVA of maternal separation anxiety, showing fire exposure when children were either in the comparison group and, hence, not exposed to fire, exposed to the fire while *in utero* (FIU) or conceived during the fire aftermath (CDA).

| Variable | Means for each category | | | Standard error |
|---|---|---|---|---|
| Fire | CG | FIU | CDA | |
| | 2.68 | 2.43 | 2.46 | 0.10 |

### 7.5.6. Maternal cigarette smoking

Maternal cigarette smoking during pregnancy is assessed to see if being present in a fire area affected cigarette smoking. Logistic regression is used to test the effect of being in a fire area on smoking as a bivariate variable (yes or no). The analysis shows no significant effect of being present in a fire area on maternal cigarette smoking ($p = 0.90$ for pre-fire conceptions and $p = 0.80$ for post-fire conceptions).

To examine the effect of being present in fire area on the amount of cigarettes smoked daily among mothers who did smoke, multiple linear regression is used. However, the analysis shows no significant effect of being in a fire area on the degree of maternal cigarette smoking.

In terms of smoking in general, maternal smoking was significantly and inversely related to financial hardship (OR = 0.58, $p < 0.001$); however, in terms of daily cigarette smoking (i.e. of those mothers who did smoke), financial hardship is positively associated with greater daily cigarette smoking ($p < 0.001$). Mothers with children who did not identify as Indigenous were more likely to smoke that those with children who did (OR = 0.25, $p < 0.001$).

Mothers who did not breastfeed were less likely to smoke than those who did (OR = 0.42, $p = 0.001$). Mothers who also consumed alcohol were 40% more likely to smoke than those who did not consume alcohol ($p = 0.04$). Increased daily cigarette smoking is also positively related to increased daily alcohol consumption ($p < 0.001$). Levels of problems and stresses and degree of coping are shown not to affect cigarette consumption.

### 7.5.7. **Maternal alcohol consumption**

Maternal alcohol consumption during pregnancy is assessed to ascertain if being present in a fire area affected alcohol consumption. Logistic regression is used to test the effect of being in a fire area on alcohol consumption as a bivariate variable (either “yes” or “no”). Maternal alcohol consumption in children who were exposed to the fire *in utero* trend toward a decline in consumption, approaching significance at $p = 0.06$, but there is no significant effect in children conceived after the fire shown.

Older mothers (OR = 0.96, $p = 0.002$) and mothers living with a partner (OR = 0.51, $p = 0.03$) are less likely to consume alcohol, while mothers whose children identified as Indigenous (OR = 2.7, $p = 0.02$) and mothers who were not breastfeeding at six months (OR = 1.8, $p = 0.04$) were more likely to consume alcohol. Again, the analysis shows a relationship between smoking and alcohol consumption, with mothers who smoked also being more likely to consume alcohol (OR = 1.47, $p = 0.04$).

Multiple linear regression is used to test for any effect of being present in a fire area on average daily alcohol consumption. The analysis shows no significant effect of being in a fire area on daily levels of alcohol consumption.

Among mothers who did consume alcohol, older mothers consumed a greater daily average amount ($p < 0.001$) and mothers who had higher daily rates of smoking also had higher average daily alcohol consumption ($p < 0.001$). Levels of problems and stresses, degree of financial hardship, and degree of coping are not shown to significantly affect alcohol consumption.

#### 7.5.8. Prenatal care provider visits

Because there are only 14 cases of prenatal visits below four visits, these were excluded from the analysis. Using an unbalanced ANOVA, the analysis shows that being present in a fire area was associated with fewer prenatal care visits ($p < 0.01$). Those attending midwife-led care had the fewest visits (2.51) while those attending GPs, Obstetricians and shared care had between 2.67 and 2.76 visits. Partnered mothers attended more visits (2.67 compared with 2.49 for single mothers). Significance levels for each variable are shown in Table 7-14.

Table 7-14: Results for ANOVA of prenatal care provider visits against analysed variables.

| Source | P |
|---|---|
| ATSI status | 0.992 |
| Mother's age | <0.001 |
| Partner status | 0.002 |
| Breastfed at 6 months | 0.404 |
| Parent One weight status | 0.067 |
| Hardship | 0.62 |
| Stressful life events | 0.029 |
| Parent One depressed in past two weeks | 0.121 |
| Prenatal provider type | 0.026 |
| Problems | 0.096 |
| Coping | 0.315 |
| Fire area | <0.001 |

### 7.5.9. **Prenatal vitamin supplements**

Logistic regression analysis is conducted to analyse the impact of being in a fire area on prenatal vitamin consumption. Being in a fire area is shown to have no influence on consumption of prenatal vitamins. Mothers who did not breastfeed (OR = 0.3, $p = 0.01$) were less likely to take vitamins. Mothers who reported having no problems or stresses were least likely to consume prenatal vitamins (OR = 2.7-4.6, $p < 0.00$), while mothers who were coping extremely well were more likely to take vitamins (OR = 4.4, $p = 0.03$). Mothers who were not depressed were less likely to take vitamins (OR = 0.62, $p = 0.006$). Mothers who principally saw midwives (OR = 1.97, $p = 0.002$) or obstetricians (OR = 1.4, $p = 0.04$) were more likely to take vitamins than those who saw GPs or shared care providers.

## 7.6. *Discussion*

Although the analysis shows few effects of fire exposure on children's health and wellbeing, but some potential effects on maternal behaviour emerge.

### 7.6.1. **Child health**

While perinatal factors, such as breastfeeding and birth weight are significant influences on children's weight under two years of age, a significant influence on BMI does not persist. The significance of early nutritional factors, such as breastfeeding, appear to reduce over time and become insignificant, while the influence of other factors, such as the weight status of the principal carer, increase with time and become significant by the time the children are four to five years old. This suggests that both social and genetic factors may be at play, with greater propensity to weight gain interacting with lifestyle factors that affect the body weight of both parent and child.

Notably, financial hardship is not related to higher body weight, contrary to other literature. This suggests that financial hardship in adulthood, particularly indebtedness, is associated with greater risk of obesity, although the direction of causality remains unclear (Averett and Smith 2014). Because young children are not able to control their own food supply, parental weight has an intuitive relationship with child weight, as the parent's diet and exercise choices and constraints will be reflected in the bodies of both parent and child.

Fire exposure is found to reduce weight for both exposed groups. Children exposed to the fire may have been exposed to a disrupted environment both during gestation and in early life. In this case, lower child body weight did not stem from lower birth weight. However, it may represent slower early growth or slower return to birth weight in the weeks immediately post-birth. Importantly, because children can express excessive catch-up growth following birth at a low weight, low birth weight shows an association with being overweight rather than underweight in later life (Ong et al. 2000).

There is good evidence that prenatal growth and weight in early life (the first 1000 days) are meaningful predictors of adult weight (Calkins and Devaskar 2011). Studies find that exposure to prenatal stress acts to increase body weight in childhood. For example, Dancause et al. (2015) find that maternal exposure to an Iowa flood and associated stresses in early gestation led to higher child BMI at 2.5 and 4 years, as well as higher total adiposity. Similarly, Li et al. (2010) find that prenatal stress arising from maternal bereavement during pregnancy, or in the year before conception, is associated with higher child bodyweight at 12 years of age. Further, von Kries et al. (2002) find that maternal smoking during pregnancy, a prenatal stressor, shows a dose-response relationship between the amount of smoking and odds of the child being overweight or obese at between five and seven years of age.

Greater childhood weight gain is consistent with ideas of stochasticity accelerating maturation, particularly in settings where weight is not moderated by food availability. In circumstances where food resources are constrained, childhood stress can be associated with stunting of growth (Reghupathy et al. 2012); however, such effects would be unlikely in the context of this dissertation and, in any case, are usually difficult to detect in analyses of BMI measures, as BMI is inflated in children who have short stature for age.

Further research is required to determine the nature of the opposing trend shown here, particularly in terms of eliminating potential effects of small sample size. Importantly, average BMIs reported in all groups are above the 50th percentile BMI for whole-of-population growth charts, indicating that the AALSAC population reflects the trend in the general Australian population toward increasing number of overweight children (Australian Bureau of Statistics 2009). Therefore, the lower average BMIs in the exposed population could be interpreted as an indicator of better, not worse, health.

### 7.6.2. **Child wellbeing**

Children's social, emotional, and psychosocial functioning are measured from two years of age and up. The analysis shows no consistent significant effect of being in a fire area during gestation on these factors at during the measured period, although it does find two significant effects on emotional and psychosocial functioning at Wave Four (only).

Consistent with the substantial and growing body of literature that examines the effect of early life on child development trajectories and adult outcomes (Belsky et al. 1991; Bronfenbrenner 1994; Kishiyama et al. 2009; Perry 2004; Putnam 2015; Shonkoff and Garner 2012), this analysis shows that children's social circumstances, including the mental health of their principal carer, exhibits great influence over their wellbeing. Children tended to report lower scores on measures of emotional, social and psychosocial functioning across all waves of data collection when their parents were subject to the following: financial hardship, problems that they could not manage, feelings of not coping or a high rate of stressful life events. The effect of these social factors appears to strengthen with time, potentially indicating a stronger effect as children become more cognisant of their parents' challenges and struggles.

The quality of the early childhood environment is increasingly recognised as an important factor in ultimate adult outcomes. For example, the Australian Government is investing in early childhood education and intervention programs because of the positive downstream effects on adult education attainment and employment (McClure 2015). Consistent with previous analysis (Hancock et al. 2013), this dissertation shows that the primary carers' mental health was a strong influence over child wellbeing. Parents who had been diagnosed with depression and had suffered a depressive episode in the past year were more likely to have children with lower psychosocial and emotional functioning scores across all waves and also had children with lower average social functioning scores after seven years of age.

There are several possible mechanisms through which parental depression might affect child wellbeing. Mothers who experience depression in the post-partum period may be less responsive to their children and thus foster less secure attachments, although this interaction is complex and depression only appears to meaningfully affect attachment where maternal sensitivity is also low when mothers are asymptomatic (Campbell et al. 2004). Alternatively, children may learn depressed behaviour and disposition from observing that of their parent or there may be a hereditary component (Hancock et al. 2013). Naturally, all three factors may interact to increase the likelihood of mental ill-health in children whose primary carer experiences chronic or recurrent depression. Nevertheless, relationships between disaster trauma and post-natal depression are found in the literature (Hibino et al. 2009; Xu et al. 2014; Yoshii et al. 2014). Because of the lasting effects of maternal mental ill-health on child wellbeing, additional screening for post-natal depression would be valuable following all major disasters.

This dissertation finds some effects of fire exposure on psychosocial and emotional functioning that were detected only when the children were between six and seven years of age. Because these associations occur outside a general pattern, a conservative interpretations is that they are associated with the small sample size available to this analysis, rather than actual fluctuations in child wellbeing.

Parental weight is not shown to have had an influence on the social, psychosocial or emotional functioning of younger children (i.e. during Waves Two to Four) but it does emerge as significant when children were eight to nine years. Interestingly, children of normal weight parents reported higher scores on all measures studied than the children of either under or overweight parents. The analysis of child body weight suggests a strong positive correlation between child and parent weight, as such parental weight may be acting as a proxy for child weight. As children age and are more exposed to social environments through childcare and schooling, being overweight or obese may have social costs which negatively affects their functioning scores across these three domains. Previous research finds that school-age children who are overweight or obese are more likely to be bullied and are also more to likely to bully others (Janssen et al. 2004). Both being bullied and being a bully could depress the measures of social functioning used in this analysis.

### 7.6.3. **Maternal health and behaviour**

The analysis shows that being in a fire area was associated with changes in alcohol consumption and prenatal care visits but not in maternal separation anxiety, maternal smoking rates or prenatal vitamin consumption.

Mothers who had significant financial hardship or who identified as Indigenous experienced less separation anxiety than other mothers. This measure in the LSAC dataset is used by other researchers as a proxy measure for overprotective parenting (Lucas et al. 2010). If accurate, this might suggest that Indigenous or poorer mothers are less overprotective than non-Indigenous or wealthier mothers. However, little research has been conducted on the validity of this measure when used in this manner.

Mothers exposed to the fire while pregnant showed a non-significant trend toward abstinence from alcohol. However, one of the strongest relationships in this analysis is unrelated to fire exposure. Maternal smoking and alcohol consumption interacted strongly. When viewed from one perspective, mothers who smoked were more likely to drink and drank more than non-smokers while, viewed from the other perspective, alcohol consumption increases the likelihood and amount of cigarette smoking.

Both alcohol and tobacco are teratogens which can cause foetal abnormalities and other pregnancy risks if consumed in sufficient quantities during pregnancy. Alcohol consumption during pregnancy is the only cause of Foetal Alcohol Spectrum Disorder (FASD), a group of mental and physical disorders arising from *in utero* alcohol exposure. The spectrum ranges from mild to severe cognitive impairment combined with facial dysmorphology (May et al. 2013). Although FASD symptoms appear in a dose-response relationship with maternal alcohol consumption, the dosages required to create symptoms vary between individuals and depend on drinking frequency and intensity, as well as with timing of exposure (May et al. 2013). Given that individual effects are so complex it is difficult, if not impossible, to establish a safe level of consumption.

Similarly, tobacco (and its component chemicals) is strongly associated with changes in foetal development and gestation. Effects in gestation are profound, raising the risk of stillbirth, miscarriage, preterm birth, growth restriction, and gestational diabetes (Hosler et al. 2011; Peacock et al. 1995; Rogers 2009; Shiverick and Salafia 1999). Further, there are developmental effects on the growing foetus. Maternal smoking during pregnancy is associated with a very wide range of birth defects, including cardiovascular defects, musculoskeletal defects, limb reduction defects, missing or additional digits, clubfoot, craniosynostosis, facial defects, eye defects, orofacial clefts, gastrointestinal defects, anal atresia, hernia, and undescended testes (Hackshaw et al. 2011). Foetal exposure to smoke is also associated with adult obesity (von Kries et al. 2002), with animal studies showing that nicotine exposure effects foetal brain development, particularly the regulation of catecholamines (Dwyer et al. 2009).

There are also later reproductive consequences for foetuses exposed to cigarette smoke. In males, testicular function and sperm production are negatively affected (Mamsen et al. 2010). In females, early onset of menarche is more likely (Behie and O'Donnell 2015; Ernst et al. 2012; Ferris et al. 2010; Shrestha et al. 2011; Windham et al. 2008). Early menarche is, in turn, associated with a greater risk of reproductive cancers (Milne et al. 2011).

The analysis does not show maternal vitamin consumption to be altered by fire exposure, consistent with a previous study (Badakhsh et al. 2010). Notably, only 22% of mothers reported consuming vitamins (including folate), despite widespread and long-standing advice on the importance of folate supplementation preceding and during pregnancy as a prophylactic measure against the development of foetal neural tube defects.

Finally, the analysis suggests that being in a fire area decreases the number of prenatal visits made by women and this effect is consistent across both pre- and post-fire conceptions. No other maternal variables (such as wellbeing indicators or financial hardship) appear to have a significant influence on the frequency of visits. Previous studies with similar findings ascribed these to logistical causes, such as disaster-related disruptions to transport (Fuller 2013). However, in this case the 2003 fire was not sufficient to disrupt care access for more than a few days (Dugdale and Guest 2003), although one participant had her induction delayed due to the fire (see Chapter Eight). In this case, a more widespread effect of fire on maternal care visitation is likely to be a result of maternal decision making rather than logistics.

In earlier work on the effects of fire on maternal behaviour, O'Donnell and Behie (2013) suggest that a cohort of women who gestated for longer than average after the Black Saturday fires might be indicative of mothers who were less willing to seek or receive intervention because the inevitable loss of control that occurs during disasters led to an expression of greater control over body and baby. Another study suggests that some women believed that it is easier and safer to have a baby *in utero* during a disaster and its aftermath rather than to manage a newborn (Badakhsh et al. 2010). This belief might encourage women to reduce their care visits as a method of reducing pressure to intervene in pregnancy. Further research is required to examine this relationship and its possible causes.

## *7.7. Chapter summary*

This chapter does not find substantive or systemic effects of fire exposure on behavioural or developmental outcomes in children exposed *in utero*. Potentially, the effects of this fire were insufficient to result in maturational changes previously associated with *in utero* stress exposure. However, some effects on maternal behaviour are detected, suggesting that even though foetal effects are not shown, mothers may have reacted behaviourally to the presence of the fire. Conclusions for this chapter are presented in Chapter Nine.

The next chapter is the final analytic chapter and presents a mixed-methods examination of the maternal experience of fire during pregnancy. Through surveys and interviews it aims to document the lived experience of fire during pregnancy.

# 8. MATERNAL EXPERIENCE OF FIRE EXPOSURE

## *8.1. Introduction*

The notion that bushfires are stressful for women who experience them while pregnant is a central assumption of this dissertation. While the physiology of the stress response is discussed in Chapter Three, this chapter explores the perception and experience of stress specifically in such women through a mixed-method approach combining survey, interview and observational data.

Since little work exists in this areas, this chapter seeks to test the assumption that bushfires are stressful for pregnant women and provide further understanding of that stress. It does this by examining predictors and mediators of maternal stress. The degree of perceived stress experienced in response to a consistent stressor often varies a great deal among individuals. Accordingly, as a way of understanding the sources of variability, this chapter introduces some of the challenges in measuring and researching perceived stress. It then briefly discusses the principal psychological theory behind the variability in individuals' perceptions of stress, before presenting the results. These combine quantitative analysis of survey responses along with commentary from survey and interview participants. The results are thematically grouped according to major issues arising from the analysis of interview transcripts. Finally, the discussion section follows the same thematic structure, drawing together themes and conclusions.

## *8.2. Research question*

The central question of this chapter concerns lived experience. In other words, what is the lived experience of bushfire exposure for women while pregnant? It focuses on the populations exposed to the Canberra and Black Saturday bushfires but includes some participants who experienced other fires. To assist in interpreting the results from earlier chapters, the research contrasts the experience of mothers between the Canberra and Black Saturday fires.

## *8.3. Background*

### 8.3.1. Defining and measuring stress

The stress response commences with a both a psychological assessment of threat and the resulting physiological response (outlined in Chapter Three). While Cartesian distinctions between body and mind (Descartes 1641) have long provided a useful way of structuring discussion of the stress response, the distinction between a psychological and physiological stress response risks simplifying the interplay between psychological and physiological functions. Modern neuropsychology, neurology, endocrinology, and epigenetics suggest a highly complex and connected relationship between psychological stress perception and physiological stress response. Although psychological perceptions inform the physiological stress response (e.g. the intrapsychic processes described by Appraisal Theory (King and Laplante 2015), physiology can also perpetuate the psychological perception of threat and stress (Radtke et al. 2011). In terms of psychological responses to threat (e.g. anxiety, fear, distress, panic, aggression, anger and withdrawal), cognition can both mediate and exacerbate the stress response. There is nevertheless a distinction between the methods used to measure psychological and physiological stress.

The interaction between the psychological and physiological makes measuring stress in humans complex. This complexity is furthered by the absence of a commonly agreed or entirely adequate definition of stress itself. Definitions of stress vary not only between disciplines, but also within them depending on context (James et al. 1989). Similarly, there are occasionally substantial differences between objective stressor exposure (e.g. type of disaster events experienced), subjective stress exposure (e.g. perception of those disaster events), objective stress response (e.g. measures of cortisol levels) and subjective stress response (e.g. self-reports of feeling stressed or anxious) (Graignic-Philippe et al. 2014).

To assist in creating a holistic description of mothers' stress, this chapter uses several methods of measurement, including objective exposure to a stressor, self-reporting and behavioural indicators of stress (physiological measures are not possible because of the time lag between the disasters and the research). The benefits of difficulties of these types of stress measurements are discussed below.

### 8.3.2. Measuring stressor exposure

Because of the difficulties in measuring perceived stress, some studies focus on measuring objective exposure to stressors as a proxy measure of stress (James et al. 1989). Traditionally, stressors are defined as noxious stimuli that compromise homeostasis and thus create arousal. Other stimuli that are considered pleasant and which create the same arousal reaction are, under this definition, not stressors (James et al. 1989). This definition means that stressors differ from individual to individual and within an individual according to context. Therefore, seeking to use stressors as a proxy for stress can be problematic.

This dissertation accepts such a definition but also seeks to investigate the gap between objective disaster exposure and perceived stress. The degree of stressor exposure and stress response do not always show a strong positive correlation. For example, in studies of prenatal stress, objective measures of disaster exposure do not act as reliable indicators of reported perceived stress (Boersma et al. 2014). Even when working with stressors widely perceived as noxious, such as major bushfires, there is likely to be some variation in the way individuals perceive that stressor.

In the literature on prenatal disaster stress, differences in stress perception also emerge between the effects of chronic and acute stress (Graignic-Philippe et al. 2014), with the duration of the stress response poorly linked to the duration of the stressor (Graignic-Philippe et al. 2014). While bushfires are a relatively acute stressor (generally lasting for days to weeks), they can lead to chronic stress, which is experienced for years. This is due to the lasting effects of the fire and subsequent stressors, such as homelessness or grief, or via the psychological sequelae of the event. For example, the development of Post-Traumatic Stress Disorder (PTSD) would create a chronic stressor. Ultimately, evidence for the impact of event characteristics (stressors) on the stress response and the subsequent development of psychopathology are highly varied (Grimm et al. 2012). While experiencing an injury does not appear to increase the immediate stress response, it does increase the likelihood of long-term stress effects. Witnessing fatalities, on the other hand, increases the immediate stress response but inconsistently increases the likelihood of long-term effects (Grimm et al. 2012).

This chapter attempts an objective assessment of stressor exposure, by asking participants to describe the number of disaster events (both acute and chronic) experienced. However, it is important to acknowledge that the relationship between an individual's degree of disaster exposure and the severity and duration of their stress is rarely straight forward. For example, in their study of maternal stress during the 1998 Quebec Ice Storm, King and Laplante (2015) find that there was no relationship between objective stress (measured by exposure to stressful events), subjective stress (measured by self-report), and salivary cortisol measurements in women exposed to the storm while pregnant. Consequently, measures of exposure to stressors should be treated cautiously if used as a proxy measure of perceived or physiological stress.

### 8.3.3. Physiological markers of stress

The physical components of the stress response are the most easily defined and measured. Physiological stress can be defined as arousal of the Sympathetic Adrenal Medullary (SAM) system or Hypothalamic Pituitary Adrenal (HPA) and thus measured via physiological indicators of SAM or HPA activation. This includes measurement of indicators such as blood pressure, heart rate, and hormonal changes (typically increases in cortisol and adrenaline), all of which are considered sound indicators that the body is responding to a stressor (Ice and James 2007).

Although measurements of cortisol are now often considered the "gold standard" in primate stress measurement, there remains debate as to the most reliable physical indicators, with salivary and blood cortisol, adrenaline, immune function, ambulatory blood pressure, and catecholamines all proposed as the most accurate physiological markers of stress (Ice and James 2007). Stress responses are now most commonly measured through salivary alpha-amylase measures of cortisol, which are predictive of plasma adrenaline levels, or through direct measures of plasma corticotropin-releasing Hormone (CRH), endorphin, or cortisol (Graignic-Philippe et al. 2014). Salivary measures of cortisol, however, are often favoured for reasons of convenience and ease of use, rather than accuracy. Measurement of hair samples are also used as these give an indication of cortisol levels over a longer period of time (Ice and James 2007)..

While these physiological measures can indicate that a person is experiencing stress, many can equally be indicators of exercise, sexual arousal, or other conditions that would not be commonly considered stress. Further, they do not give insight into the individual's perception or experience of that stress.

Importantly, physiological measures also require careful interpretation in light of underlying (non-stress) changes in stress markers. Both the SAM and HPA activation move in a circadian rhythm (e.g. the Cortisol Awakening Response); therefore, changes in activation of the SAM and HPA are part of homeostatic variation and diurnal change cannot be usefully be considered an indicator of stress. Cortisol is further complicated as a measure of stress during pregnancy because cortisol levels and maternal responsivity change during the normal course of gestation (Ellison 2009; Wadhwa et al. 1996). Great care should be taken, therefore, if ascribing maternal elevations to stress alone.

Documented relationships between maternal self-reporting of anxiety or stress and cortisol in pregnant women remain modest (Nepomnaschy and Flinn 2009; O'Donnell et al. 2009; Obel et al. 2005; Wadhwa et al. 1996). Conversely, Wadhwa et al. (1996) report that perceived stress is not significantly associated with maternal cortisol, only maternal ACTH levels. Due to the longer plasma half-life of cortisol, they suggest that it might be a better indicator of chronic stress than of acute stress.

Additionally, some forms of behavioural stress response, such as conservation-withdrawal, can create contradictory hormonal changes and result in reductions in markers such as cortisol (Menahem 1994). For example, following the September 11, 2001 terrorist attack on the US, some mothers with a PTSD diagnosis showed lower than normal average cortisol levels despite reporting a high degree of perceived stress (Yehuda et al. 2005).

Therefore, although physiological markers give a good indicator of bodily response, they remain imperfect indicators that require further interpretation. In terms of this dissertation, the time delay between disaster exposure and the commencement of research means that physiological measures are of little use. Although markers of chronic stress may remain in the population, it would not be possible to ascribe this stress directly and accurately to disaster exposure.

### 8.3.4. Self-report and behavioural measures of stress

A key challenge in defining stress is the variation in response to like circumstances. Thus, in much stress research, emphasis is placed on self-reporting, an acknowledgement that only individuals can determine whether they are experiencing stress or not.

Self-reporting is often made consistent through the use of stress scales, such as the Perceived Stress Scale. These scales most commonly ask participants to rate their own stress using a mixture of emotional cues, such as feeling stressed or anxious, and behavioural or physiological cues, such as a racing heart, dry mouth, or difficulty sleeping (Ice and James 2007). Many scales are currently in use, with validation information generally available for most of the commonly used instruments.

Behavioural markers of stress, such as sleep disruption, provide a useful mid-way point between the specificity of physiological markers and the amorphous nature of psychological markers. Behavioural responses likely represent responses synthesized from both psychological and physiological response processes. Changes in behaviour are influenced by hormonal release and other physical changes, but these are mediated through psychological appraisal and therefore are also informed by individuals' risk perceptions and appetites. Such responses can include sleep disturbance, increased use of alcohol, tobacco, caffeine, tranquillizers and other drugs, as well as changes in eating frequency and calorie intake (Harrison 1995). Monitoring behavioural responses offers more concrete indicators of stress because behaviours can be measured and recorded more exactly than emotions.

This dissertation uses self-reporting, including both behavioural and emotional questions. Because these are being used in a pregnant sub-population, careful interpretation is required and measures may be affected by the impact of pregnancy on sleep quality and duration, as well as diet and the use of alcohol and cigarettes.

### 8.3.5. Risk attitudes and perceptions

The high degree of individual variation can be explained by both physiological and psychological reasons. An individual's response to stressors can be conceptualised as a product of their risk attitude (or appetite) and their risk perception. Risk attitude is informed by risk content (i.e. how "bad" the worst outcome of the risk is judged to be), while risk perception is informed by the likelihood of the worst outcome of the risk (i.e. how likely it is that the individual will experience a negative outcome) (Pennings and Grossman 2008). The behavioural response that an individual will make to a threat (e.g. in the case of environmental disaster, whether they will evacuate) is informed by their risk attitude and perception. Higher threat perception, rather than objective degree of exposure, is also predictive of poorer psychological outcomes, including the development of PTSD (McDermott et al. 2005).

Risk perception ranges from high-risk to negligible. Risk perception appears to be a mostly conscious process involving a purposeful assessment of prior experience, the content of public communications, and the views of peers. Social Amplification of Risk theory proposes that the social interpretation and response to risk is central to individual perception (Kasperson et al. 1988). The social response to risk can either amplify or attenuate objective risk. The social response is informed by a range of factors including perceptions of controllability and the degree to which the risk is unfamiliar, as well as more pragmatic considerations of vested interests and threats to resources. Social amplification and attenuation can explain why common high-risk activities, such as driving, do not elicit individual concern (because they are perceived as prosaic and susceptible to the individual drivers' control), while lower-risk activities, such as flying, can elicit a strong stress response (Kasperson et al. 1988).

Some studies suggest that, for these reasons, environmental disasters are less stressful than technological or anthropogenic disasters because they are perceived as more natural and familiar, whereas technological or man-made disasters "should not have happened", even though some environmental disasters have anthropogenic causes (Grimm et al. 2012). Arguably, and particularly in the first world, communities believe that environmental disasters should not occur or that protective government responses should be more effective.

Similarly, risk attitudes can range from highly risk–adverse to highly risk–accepting. Risk attitude is informed by both experience and temperament. Individuals perceive risk differently depending on both cognition and neurology. As Ginsburg (1972 p.167) states the level of anxiety an individual experiences is a "matter of where the homeostat is set". This means that the basal function of the HPA axis directly influences the behaviour of individuals faced with risk in a complex interaction with their risk perception and attitude. The uterine environment is thought to be crucial in benchmarking basal HPA function (Perry 2009; Radtke et al. 2011). Therefore, an individual's risk attitude is reflective of more than their purposeful cognitive assessment of a situation. It is also informed by unconscious neuroendocrine processes that either stimulate or inhibit the stress response.

The interaction of neurological and endocrine factors with risk attitude and perception can also help explain variation in public responses to official warnings, particularly those responses that appear illogical. For instance, Riad et al. (1999) find that risk perception, social influence, and access to resources were not sufficient to explain peoples' evacuation behaviour in response to Hurricanes Hugo (in 1989) and Andrew (in 1992), both of which affected the Caribbean region and the south-eastern coastal US. While storm severity emerged as additional factors, much of the variance in behaviour could not be explained. Individual neuroendocrinal factors related to risk attitude may have been important in these cases.

Because of the role of attitude to risk, the overall risk assessment process is one which implicates both conscious and unconscious mechanisms, as well as both social and individual foundations. The division between risk perception and attitude is useful in examining how risk assessments moves above and below the level of consciousness.

### 8.3.6. Risk perception in pregnancy

In terms of risk perception and attitude, there is relatively little research as to how these alter during pregnancy. Badakhsh et al. (2010) consider mothers who were pregnant during Hurricane Katrina in 2005, which affected the US Gulf Coast. The women in the study were all displaced during the storm and experienced significant deprivation in terms of nutrition and living standards. Many did not receive adequate food for several days, although most were able to continue their prenatal vitamin supplements, and were also unable to retain their pre-existing relationships with health providers as the local hospital and clinics were destroyed.

The narratives of the women show that their perceptions and attitudes to risk were informed by their pregnancy predominantly in terms of their assessment of their future needs and the inability of the environment to provide for themselves and a newborn. For example, women were concerned at their lack of housing due to the impending need to house their infant, felt guilty about consuming limited food supplies (thus denying others, particularly elderly relatives), but felt that they must eat for their child's sake. They expressed relief that their child was not yet born as they perceived this to safer (Badakhsh et al. 2010). Women in this study commented that, although they knew that stress should be avoided in pregnancy, they felt powerless to reduce it.

Another study looking into pregnant women during Hurricane Isaac (in Louisiana, US in 2012) finds that the hurricane compounded pre-existing risks to mothers' wellbeing as well as an inverse relationship between degree of social support and development of post-natal depression (Arosemena et al. 2013). Because of the impact of pregnancy on individuals' assessments of their future needs, it is likely that disasters are more stressful for women while pregnant than they would be otherwise. This level of stress operates within the parameters of the woman's particular perceptions and attitude to risk, as well as their prior experience.

King and Laplante (2015), in their study of women who experienced the 1998 Quebec Ice Storm while pregnant, found no associations between measures of objective stress, perceived stress, and cortisol samples. This indicates considerable variation across women's feelings of stress, their experience of trauma or deprivation, and their physiological response. Because physiological response is related to the effects on the foetus, this suggests that a mother's sensation of stress may not be a good indicator of the environment experienced by the foetus.

### 8.3.7. Psychological stress and resilience

Much of the substantial individual variability in human behaviour following exposure to a stressor stems from variation in psychological responses to stressors (Koob et al. 1994; Sapolsky 1994). Pre-existing factors are known to contribute to the degree and duration of an individual's stress response. These include life experiences (e.g. prior insults or positive experiences) and genetic susceptibility (Benight and Harper 2002). Physiological responses to identical stressors can vary between individuals as a result of the psychological context. Unpredictability, perceived loss of control, perception of events worsening, a lack of social support, and a lack of outlets for frustration can increase the severity of the physiological response (Sapolsky 1994). Lack of control appears to be a powerful indicator of increased stress response and is highly likely to be experienced during an environmental disaster (Sapolsky 1994). The loss of social status and social dislocation that is common in the aftermath of environmental disasters may also act to increase stress in humans (Hoffman and Oliver-Smith 1999; Oliver-Smith and Hoffman 1999; Sapolsky 1994).

In some individuals, the stress response can progress into long-term psychological distress such as PTSD (as well as other anxiety, attachment, and adjustment disorders). A wide range of factors have been investigated as mediators of the severity of stress experienced and the translation of the stress response into long-term psychological distress. Those factors which appear to reduce the severity of the stress response include having a religious faith, being married, and having strong social affiliations (Fredman et al. 2010; Hoff et al. 2009; Hussain et al. 2011). Age also appears relevant, with younger people appearing to be less resilient to stressors (den Ouden et al. 2007; Scaramella et al. 2008; Soeteman et al. 2008). Factors that appear to exacerbate the stress response include being female, low socioeconomic level and having a history of previous trauma (Benight and Harper 2002).

Mental health prior to a stressful experience is a particularly relevant predictor of stress response. Those who experience a greater than average stress response tend to have: higher than average pre-existing stress level (den Ouden et al. 2007; Soeteman et al. 2008), a lower than average degree of coping self-efficacy, higher than average pre-disaster depression scores, low socioeconomic level, a history of child abuse (Benight and Harper 2002), or a pre-existing psychiatric diagnosis (Grimm et al. 2012). Thinking one's life is in danger and having a feeling of loss of control in the face of a traumatising event (Grimm et al. 2012) have also been considered predictive of PTSD development.

In order to help understand the role of risk and protective factors, this dissertation collects information on the degree of pre-disaster community connectedness and pre-disaster mental health via self-reporting, as well as asking whether participants felt a loss of control or feared for their lives. This chapter aims to describe and interpret the lived experience of bushfire exposure during pregnancy, using a mixed-methods approach. There is very limited research into the experiences of pregnant women during disasters and none which consider pregnant women during fires.

## *8.4. Method*

This chapter recognises women the expertise and experience of women and gives them women the opportunity to express their experiences and perceptions *verbatim.* Only then does it then seek to contextualise those experiences within the broader available literature. To ensure triangulation, three sources of data are collected: face-to-face interview transcripts, online survey responses (both scored and free text), and reflexive field notes.

### 8.4.1. Ethical aspects

Approval of the ethical aspects of the complete project was granted by The Australian National University Human Research Ethics Committee (ANU-HREC) on 23 September 2013 (project reference: 2013/143). Approval of a minor amendment to the survey text was granted on 25 November 2013. A key concern of the ANU-HREC was the management of participant distress during the interviews which form part of this study. A flow-chart documenting a distress management process was developed and used. The survey commenced with a statement regarding ethics approval, privacy, and consent, while the interviews were accompanied by a participant information sheet and consent form approved by ANU-HREC. Interviewees were assured anonymity. Because they were all residents of relatively small communities and shared detailed stories, every possible measure to ensure their privacy has been taken, including using pseudonyms and not reporting exact ages.

### 8.1.1. Procedures

The survey was delivered online using the Survey Monkey platform. It was hosted on the ANU website. Survey participants viewed a front page that contained information regarding consent, privacy information, psychosocial support information, and complaints procedures. Participants were asked to print or save that page for future reference. By participating in the survey, participants granted consent.

Interview participants were provided with an information sheet which included support information and contact details for myself, Dr Alison Behie (supervisor), Dr Christine Phillips (panel member and medical doctor with experience in working with traumatised populations), and the ANU-HREC. Participants were asked to sign a consent form covering their participation in the study and the use of a recording device (although the latter was not required for study participation). Interviews were held in individual's homes, workplaces, or in cafés, at the discretion of the participant. For all interviews held in cafés, beverage and food costs were covered by the project.

Interviews were semi-structured and included the 10-item version of the Perceived Stress Scale (which is discussed in more detail in section 8.1.3). They included prompt questions, such as "what was your experience of being pregnant during a fire?", "what support did you receive?" and "was there any support you wish you'd had?" Interviews were audio recorded and transcribed, and additional field notes were also collected. In one instance, the recording device failed and more detailed field notes (additional to contemporaneous notes and quotes) were added immediately following the interview. Interviews were conducted throughout 2014. Reflexive field notes were kept during and following all, in addition to photographs taken of streetscapes and reconstruction projects in that region.

### 8.1.2. Recruitment and sampling

Participants were sought via a wide range of means. The survey and interview invitations were advertised via Facebook, Twitter, and internet forums, State and Commonwealth public service notice boards, letters to the editor pages, and feature articles in local newspapers. Internet forums included Black Saturday and Beyond, Bub Hub Pregnancy and Parenting forums, Essential Baby forums, Essential Kids forums, Raising Children Network forums, and RiotACT forums. They were also advertised by posters in many local locations, including General Practitioners' offices, Maternal and Child Health Centres, supermarkets noticeboards and other community noticeboards. Outreach as carried out to maternal health professionals, schools and other Black Saturday support groups.

Unfortunately, the Victorian and ACT Departments of Education do not allow research to be conducted through schools unless it is education focused. Due to the extensive use of social media, some snowball sampling is likely to have occurred (e.g. the study information posts were "liked" on Facebook and re-tweeted, and word-of-mouth referrals took place).

Both survey and interview participants proved difficult to recruit. The target cohort is disparate and, due to the time between the event and the research, many potential participants no longer live in affected areas. Because participants were self-selecting, it is probable that there is a bias in the sample toward women who had a more memorable experience of fire and pregnancy. The data may therefore overestimate the degree of stress experienced in the general population and the impact of fire during pregnancy. To assist in interpreting the data accordingly, additional materials (including previous research and Government disaster payments data) have been collected and used to contextualise the results of the survey and interviews in the discussion of these results.

### 8.1.3. Data collection methods

*Survey*

The survey provides a wider view of the experience of participating women. Due to the specific and unusual nature of the research no existing survey instrument was available. Consistent with the literature regarding threat perceptions, the survey considers a range of factors relevant to maternal stress. These include: direct proximity to the threat as well as pre-disaster wellbeing; the individual's sense of control before; during and after the disaster; degree of social support; dislocation; perceived unpredictability of the disaster; and experiences of fear during the disaster. Consistent with the findings of Fuller (2013), the survey also considers alterations to women's health behaviours and access to prenatal care.

The survey asks maternal age and location and includes questions on the sex, birth weight, and gestational age of the child. It also covers questions on prenatal health and fire exposure. Concerning fire exposure, these include questions based on the assessment of impact conducted by Emergency Management Australia (the Australian Government's emergency management agency) in order to assess eligibility for the Australian Government Disaster Relief Payment (AGDRP). The AGDRP is a federal government cash transfer made available to affected people immediately following declared disasters (Shorten and Macklin 2009). The AGDRP was available to people who (1) were seriously injured; (2) were the immediate family member of someone killed or injured, or were the carer of a child adversely affected by the fires; (3) had their home damaged or destroyed; (4) were unable to return home for 24 hours or more; (5) had no connection to utilities for 48 hours or more; or (6) who had experienced psychological trauma.

Consistent with the literature, the survey includes questions on community connectedness and prenatal health behaviours (Fuller 2013). The survey asks participants a series of questions intended to allow an objective measure of their disaster exposure. This included: the extent to which their own families and properties had been affected, including deaths and injuries; the impact on their pets and stock, including deaths and losses; and the extent of damage and impact in their local communities. The degree of exposure according to these indicators is given a value (one for a positive response and zero for a negative response) to establish an exposure score. This score was not weighted according to event severity because of the difficulties of estimating a reliable and valid method of determining event severity for different individuals.

The survey includes free text sections for additional comments and recommendations for future practice. The survey text was piloted with the supervisory panel and two lay readers to test and improve content and comprehensibility. The full survey text is shown in Appendix D.

*Stress scale*

Recalled perceived stress has been measured in the survey using the 4-item version of the Perceived Stress Scale (PSS) survey (Cohen et al. 1983; Cohen and Williamson 1988). The PSS is a 4- or 10-item Likert-scale self-report instrument used to standardise recollections of perceived stress. The scale asks participants to rate the frequency with which they experience positive and negative stress and coping-related emotions over the previous month, from “never” to “always”. The PSS is designed and validated as a stress scale for recalling stress in the previous month but has not been validated as a retrospective instrument over longer recall periods (Cohen et al. 1983; Cohen and Williamson 1988). Given the lack of clear alternatives, however, it constitutes the most appropriate scale for this research. The PSS is validated among pregnant women (Solivan et al. 2015).

Participants were asked to recollect their stress in the month following the fire and a total stress score has then been calculated using the PSS scoring matrix. Importantly, the PSS scores are contextualised by the participants’ self-reporting regarding the level of damage and disruption they experienced and their scores on the measures of deprivation used by Emergency Management Australia, such as being unable to return home or utilities failing for more than a day.

### 8.1.4. Data cleaning procedures

Survey data has been cleaned to remove impossible responses (a gestational age at the time of fire of 66 weeks with a corresponding ultimate gestational age of 90 weeks was removed). Baby birth weights have been converted from imperial to metric measures as required. Two birth weight responses were recoded. One has been changed from ‘normal weight’ to 3370 grams (the Australian average). One has been changed from ‘just under 4 kilos’ to 3900 grams. One response to the question on maternal drinking was recoded to match the accompanying comment which specified that the reported drinking occurred after the completion of the pregnancy.

### 8.1.5. Analysis strategies

The survey data has been analysed using univariate and multivariate statistics. Survey metrics have been conducted using descriptive statistics, Cronbach's alpha and correlations. Stress scores were compared between fires using descriptive statistics and Student's t-tests. Indicators of trauma have been compared between fires using descriptive statistics and a chi-square test of independence. Evacuation behaviours have been examined using descriptive statistics and Student's t-tests. Maternal health behaviours have been examined using descriptive statistics and correlations. Sample size has restricted the power of the methods used. The analytic test used is specified alongside each result in the following results section with models provided in the appendix to this chapter. Analysis has been conducted in STATA version 13 and Genstat version 16. For all analysis significance is set at $p = 0.05$.

Interviews have been transcribed. Transcripts and free text survey responses were analysed using QDA Miner Lite version 1.2.2. Responses have been thematically coded and then grouped around the emergent central themes. These themes are reflected in the sub-headings presented in the results section. The listed ages of represent the estimated ages of participants at the time of the fire. They are grouped into five year ranges to protect participant anonymity.

## 8.5. Limitations

The small sample size limits the generalizability of the findings. This small sample is caused by the nature of the population, the time elapsed since the disasters, and the recruitment of participants through advertising (rather than, say, via a natal care clinic).

Similarly, the method of recruitment means that women with a particularly powerful or memorable experience are likely to be over-represented in the sample. Nevertheless, the views expressed by participants represent an authentic portion of the women affected. Further, their experiences interpreted and reported in the context of other broader indicators of population stress and bushfire experience.

## *8.6. Descriptive statistics*

### 8.6.1. Survey participants

The sample is made up of survey responses with 20 from Black Saturday day survivors and 17 from Canberra fire survivors. Five responses are from people who had experienced other fires and one from someone with experience of multiple fires. One respondent did not report which fire they experienced.

Participants have an average age at the time of the fire of 31.28 years (s.d. = 7.25), which is near to mean maternal age in the general ACT (~31 years in 2003) and Victorian (~30.5 years in 2009) populations. The mean gestational age of foetuses at the time of the fire of 23.83 weeks (s.d. = 11.35) (see Figure 8-1).

The mean birth weight for exposed children of surveyed mothers is 3427.66 grams (s.d. = 548.3), while the mean gestational age is 38.56 weeks (s.d. = 4.18), both also near population averages.


Figure 8-1: Length of gestation at the time of fire onset (n = 41)

### 8.6.2. Interview participants

A total of seven interviews have been conducted: three of Black Saturday survivors, and four of Canberra fire survivors. The ages of interviewees is shown in Figure 8-2.

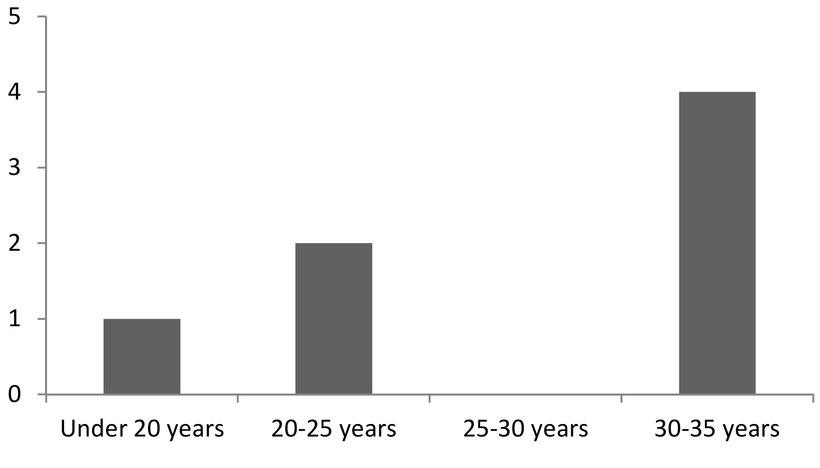


Figure 8-2: Age groups of interview participants at the time of the fire (n = 7)

At the time of the interview, interviewees remain living at or near the location of the fire they had experienced. All except one had subsequent children and all except one remained coupled with the same partner. All were in heterosexual relationships. Two were stay-at-home mothers, with the balance employed either full- or part-time. Those who were employed worked within their local community in service industries, public services or education services. All except one lived in suburbs or in townships, with the exception living on an acreage on the outer fringes of a small township.

### 8.6.3. Survey metrics

The mean stress score arising from the Perceived Stress Scale (PSS) is 11.27, out of a maximum possible of 16 (s.d. = 2.35). The mean exposure score is 3.82, out of a maximum possible of 16 (s.d. = 3.98). The PSS shows good internal consistency (Cronbach's alpha $\propto = 0.88$). An exposure score is derived from the items measuring objective exposure, such as exposure to deaths, injuries, and property damage. There is a moderate correlation ($r = 0.58$; $n = 36$) between participants' exposure and stress scores (see Figure 8-3).

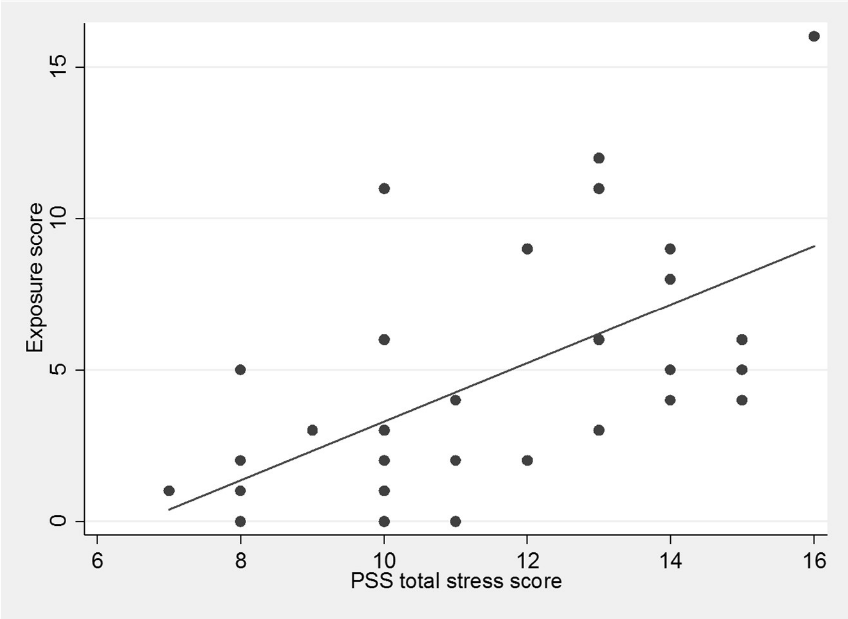


Figure 8-3: Correlation between exposure and stress scores (r = 0.58; n = 36) among survey participants with a linear trend line shown.

## *8.7. Results*

### 8.7.1. Stress

*Survey analysis*

Many participants have experienced events widely considered stressful, such as death and injury to family, friends, and community members, damage to property and damage to community properties (see Figure 8-4).

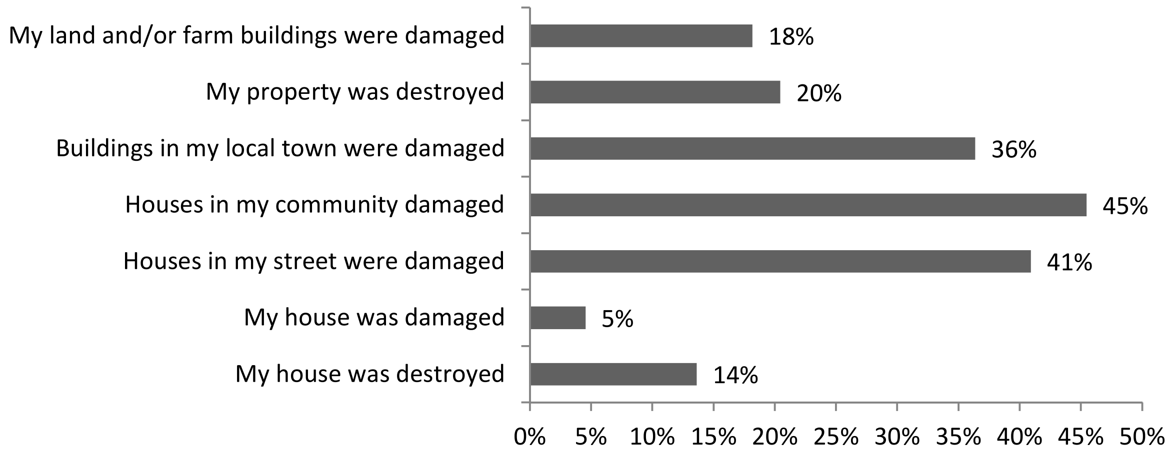


Figure 8-4: Property damage experienced by participants (n = 38)

Among participants, 45% could not return home for more than a week, including 11% who moved permanently following the fire. A total of 40% lost power for more than a day and one-fifth were evacuated. More participants indicated that their house was destroyed than damaged, indicating a high degree of exposure in the group (see Figure 8-5).

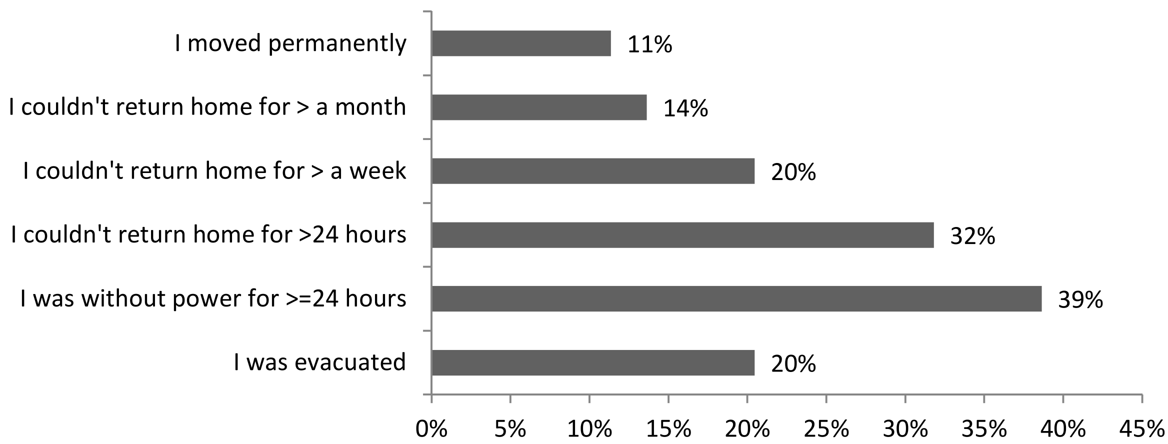


Figure 8-5: Dislocation and service disruption experienced by participants (n = 35)

A Student's t-test has been used to determine whether average stress scores were different between the Canberra fire and Black Saturday fire. Stress scores for other fires have been excluded due to small group size (n = 6). Stress scores reported by survivors of the Black Saturday fires (mean = 12.16) are significantly higher than those reported by survivors of the Canberra fires (mean = 10.14) at $p = 0.01$ (see Table 8-1).

Table 8-1: Results of Student's t-test on Perceived Stress Scores between the Black Saturday and Canberra fires (n = 32)

| Group | n | Mean | Std. Error | Std. Deviation | CI (95%) |
|---|---|---|---|---|---|
| Canberra | 14 | 10.14 | 0.645 | 1.978 | 11.183 – 13.15 |
| Black Saturday | 18 | 12.16 | 0.466 | 2.413 | 8.749 – 11.536 |

Maternal age is shown not to have been a significant influence on stress scores but is significantly associated with the number of fire events experienced, with older mothers experiencing a greater number of stressful events. In terms of mental health, too few women reported pre-existing mental health conditions to allow analysis.

### *Participants' commentary*

Participants described the stressful effects not only of the fire itself but also of ongoing vigilance as the fire threat persisted:

> [I was] at school [her workplace] … with embers falling and think this isn't safe for me, let alone … other people's kids. (Sarah, 30–35 years, Black Saturday)

> [For] days after the fire, it is this ‘unknowing’: is it coming back? I was packed. I mean, I was living at home but I had my bag packed. (Carmel, under 20 years, Canberra fires)

Others described the interaction of heat, pregnancy and fire:

> [J]ust being hot and being that pregnant would be stressful in itself, and then you have to deal with fires. (Amy, 30–35 years, Black Saturday)

Others mentioned the role of paperwork and interaction with government in increasing their stress in the immediate fire aftermath:

> With grants that were available I felt a real pressure to have the paper work done in time and a lot of case workers were not informed. (Survey participant, 35–*40* years, Black Saturday)

> I couldn't handle the insurance and all that … there were a few times when I'd just break down. The insurance people would call up and I was just like 'I can't do this - I can't do this!' (Suzanne, 30–35 years, Black Saturday)

Others stressed the length of recovery and persistence of their stress:

> Our home took five years to rebuild and it was a roller coaster ride. [I] feel that my children suffered as I wasn't in a happy place. (Survey participant, 25–30 years, Black Saturday)

Pregnancy had highly variable effects on stress. While some women described feeling more vulnerable, particularly physically, others described a:

> [A] kind of hormonal coping zone where I barely cared about what was going on around me… the baby was more important than the loss of a home. (Survey participant, 35–40 years, Black Saturday)

However, this participant also noted that her distress came "crashing back in" some months after the birth of her child. Survivors of the Black Saturday fires described not only their own stress but also that of their children:

> [H]e [the participant's eldest son] got a full stress reaction and came out in hives so we were recommended not to send him to school (Sarah, 30–35 years, Black Saturday)

Others found that their stress improved the bond they felt with the child they were carrying:

> Whilst the experience of having to evacuate my home due to a bushfire at 39 weeks pregnant was incredibly stressful, I do believe it made bonding with this child easier… I felt like I had already had my resolve tested and had to … protect her. I see this as an upside to an otherwise horrible experience. (Survey participant, 25–30 years, other fire)

The inability to help, in either fighting the fire or assisting in community recovery, was a common theme:

> I think the fact that I couldn't help was stressful. (Suzanne, (30–35 years), Black Saturday)

> My main issue following the fires was guilt that I had to focus on my new baby and two other children rather than being able to help other people … who had suffered terribly because of the fires.
>
> (Survey participant, 37 years, Black Saturday)

Similarly, the use of support networks differed between women. While some found that community groups and interaction was valuable, others found that one-on-one support was more effective:

> I wasn't at the stage where l wanted to go to playgroup or be around other women… we all need different things. It was lovely that someone [the maternal health nurse] could think outside the square and see that l needed a local friend and make the arrangement.
>
> (Survey participant, 35 years, Black Saturday)

Strains on interpersonal relationships were reflected in both the actual experience of participants and their reports of other people in their communities. There were no reports of domestic violence. One survey participant (Black Saturday) and one interviewee (Canberra fire) reported that their relationship with the baby's father had ended following the fires. Some effects on relationships were delayed, with tensions continuing to emerge in the years following the fire. Regarding a heavily affected Black Saturday region, one participant said:

> About two years after and that's when lots of families were breaking up… you get through survival mode, get into your new house, and then … (Sarah, 30-35 years, Black Saturday)

Several interviewees described tensions in their relationships which arose from the fires; these often seemed to reflect perceived or actual differences in emotional coping styles between men and women:

> It's very hard for the husband to understand, and I don't think that's going to change, I think that's just hardwired into them… they just deal with things differently. (Suzanne, 30-35 years, Black Saturday)

### 8.7.2. Trauma

*Survey analysis*

Although a subset of stress, issues of trauma (defined as the experience of deep distress) arise strongly through the interviews and surveys, particularly those regarding Black Saturday survivors. Among the survey respondents, some had been exposed to events likely to cause trauma. These included death of family (n = 1), friends (n = 5), or acquaintances in the community (n=11). They also included injuries to family (n = 4), friends (n = 10) or acquaintances (n = 16). Other trauma inducing events included the loss of stock (n = 2) and the death of pets (n = 6). Some participants experienced two or more of these events (see Figure 8-6).

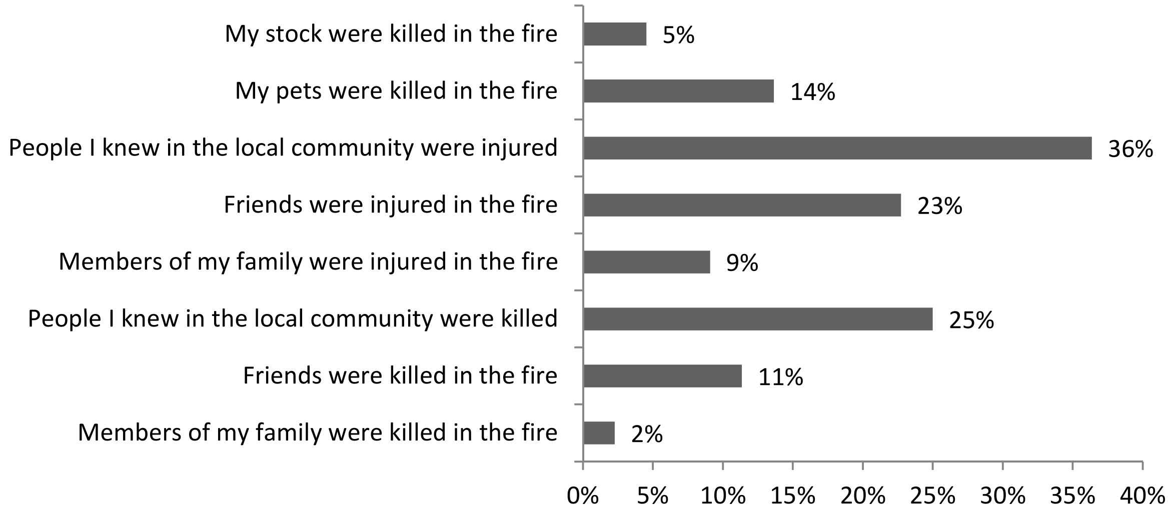


Figure 8-6: Exposure to traumatic events among survey participants (n = 38)

The respondents experienced predictors of traumatic stress, such as feeling scared or terrified (n = 22), being worried that they would die (n = 12), or feeling that events were uncontrollable (n = 27). Some participants showed indicators of traumatic stress, such as recurrent nightmares (n = 12) and disturbed sleep (n = 28). This excludes those who specified that their disturbed sleep was due to baby care alone, these were removed from the count (see Figure 8-7).

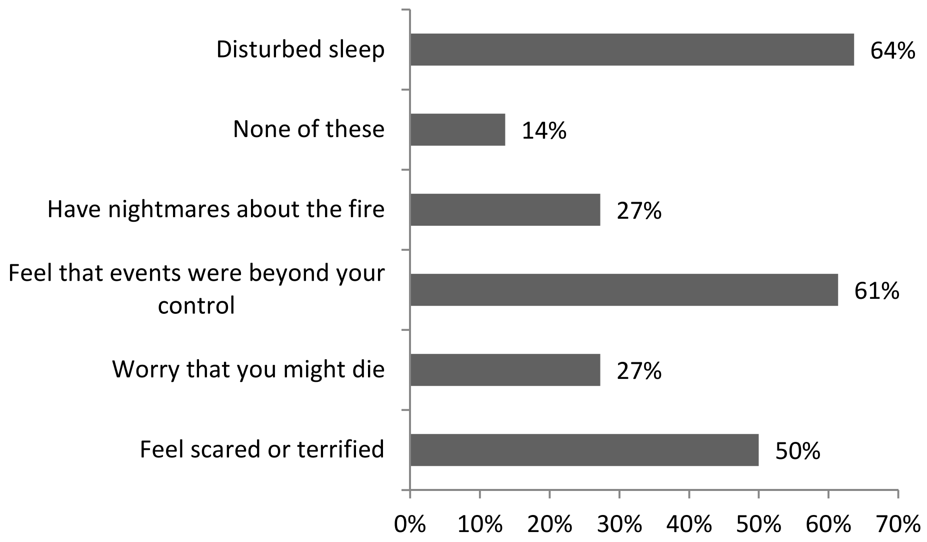


Figure 8-7: Predictors and indicators of traumatic stress among survey participants (n = 40)

Exposure to traumatic events is higher among the Black Saturday survivors, reflecting the greater severity of the fires. In particular, a greater proportion of Black Saturday survivors has experienced the death of family, friends, or acquaintances killed in the fire

*Participants' commentary*

Two interviewees from one of the hardest hit regions of the Black Saturday fire talked extensively about the degree of traumatic stress in their communities. This included children who had seen animals burning alive and people of all ages coping with extensive grief and loss.

They also discussed the role of vicarious trauma in the post-fire period. Because of such wide-spread exposure, trauma was sometimes increased by hearing accounts of the experiences of other people, many of whom had suffered severe trauma and loss. These two interviewees felt that more adequate counselling might have reduced the degree to which people relived their trauma with friends and neighbours:

> [Y]ou couldn't escape other people's stories… people would share the most horrendous stories in the supermarket aisle or in front of little kids. (Sarah, 30-35 years, Black Saturday)

Although some counselling was made available after the event, it seemed to be insufficient compared to the need and was largely delivered through the recovery centre at the local community hall, which did not provide a private environment. Counselling was also withdrawn in the years following the fire, although for some people the full force of traumatic stress was only being realised some time later. Even five years after the event, participants said:

> At that point [soon after the fire], I just wanted to talk to my friends, talk to people that knew me, but counselling would have been helpful afterward. (Amy, 30-35 years, Black Saturday)

> Everyone in the community needs counselling, to be honest... And maybe not right afterwards, because that might not be the time that it's actually needed. (Sarah, 30-35 years, Black Saturday)

A further two interviewees were diagnosed with post-natal depression following the fires and believed that there was a relationship between their fire experience and their depression:

> I was diagnosed with post-natal depression after the fires and it was thought that the stress after the fires contributed to this. (Survey participant, 34 years, Black Saturday)

### 8.7.3. Evacuation

*Survey analysis*

A large percentage of women (52%) opted to remain at their homes. Although only 9% were present at the fire front, a further 43% remained at home in a risk zone. A further 27% left shortly before the fire. Only 18% evacuated early, the manner which is defined by authorities as safe (see Chapter One). Altogether, 79% of women either did not evacuate or evacuated only shortly before the fire (see Figure 8-8).

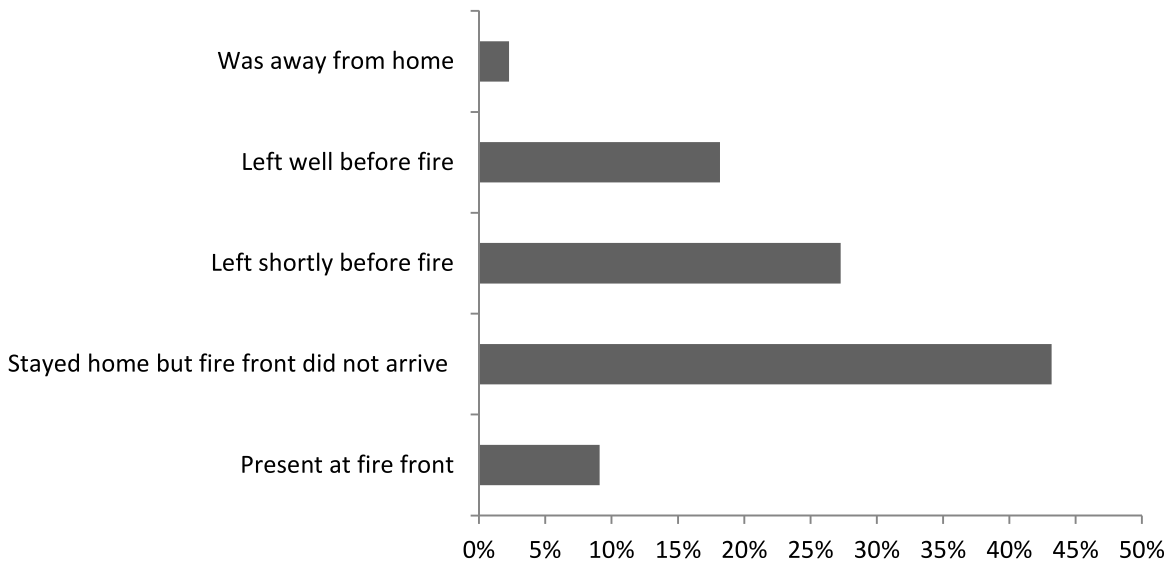


Figure 8-8: Evacuation decisions among survey participants (n= 39)

For those who did evacuate this did not necessarily correlate with lower recalled stress. Mean stress scores are very similar between the women who evacuated (mean = 11.42, n = 16) and those who remained at home (mean = 11.12, n = 15). Student's t-tests comparing the stress score of those who remained at home compared with those who evacuated (at any point) find no significant difference ($p$ = 0.63; model shown in

Appendix D). Similarly, Student’s t-tests show no differences between early evacuators and others ($p$ = 0.18; model shown in Appendix D) nor between late evacuators and others ($p$ = 0.70; model shown in Appendix D).

*Participants’ commentary*

Reasons for not evacuating varied between individuals. Some women expressed their strong desire to protect the home:

> I suppose my maternal instincts kicked in. (Carmel, under 20 years, Canberra fire)

Another reported attempting to drive toward the fire, accompanied by her young child (as well as being pregnant), in order to check and defend her home. In this instance, the participant was turned around by police at a road block.

Some remained because their escape options were not viable:

> [T]he houses on the end [of the road] had already gone up [meaning fire was blocking the road], so I couldn't really leave. (Suzanne, 30-35 years, Black Saturday)

One survey participant directly stated that the lack of information regarding local road closures made her reluctant to evacuate:

> [There was] no real information about, if you were to get out, what roads were closed and which one to take. That's why I was like: I'm just going to stay until the very last. (Cara, 20–25 years, Canberra fire)

Others discussed the impracticality of evacuating during a persistent bushfire threat. In contrast to some other environmental disasters, such as hurricanes, which pose a threat over a number of days and then either hit or pass, bushfires can pose a present and ongoing threat over months. As one participant said:

> We left three times … it lasted so damn long! (Amy, 30–35 years, Black Saturday)

The constant vigilance required took a clear toll. Women also reported that the information provided by authorities was insufficient and varied widely.

> the policeman would say "you're fine, you don't need to leave" and then the next morning they would be doorknocking houses waking people up telling people to leave. They were saying 'stay alert, keep on 774 [the Victorian emergency radio broadcast frequency], keep on the internet'. But how do you sleep?" (Sarah, 30–35 years, Black Saturday)

During the interviews, women said that the experience of evacuation was highly stressful in itself, particularly due to separation from their partners.

> That whole thing that happened – that shits me how it still happens – you know, that the men will stay. You take the kids… My husband doesn't do it anymore, he doesn't even bother anymore, but [among] lots of families I know it's a massive issue. (Sarah, 30-35 years, Black Saturday)

> I'm at my Mum's house and I can't be with my husband and that's terrifying me. (Kate, 20-25 years, Canberra fire)

> [Recalling a request made to her husband] come and be with me, be safe. (Amy, 30-35 years, Black Saturday)

Evacuation locations could also be problematic. Some women who were with friends or family reported a varied degree of support.

> I was in a house full of stressed people who kept telling me not to have the baby. (Katrina, 30–35 years, Canberra fire)

Another attended an evacuation centre. Although the centre staff made efforts to assist in making her more at ease by giving her a separate room away from the main hall (the centre was in a gymnasium), it was not a comfortable environment:

> They pulled out a whole bunch of, you know the crash mats, the thin crash mats, for me to sleep on. So that was as soft as it was getting. (Kate, 20–25 years, Canberra fire)

Some who evacuated also found that their refuge was soon under threat, leading to a subsequent evacuation, which further heightened their stress and sense that nowhere was safe.

Survey and interview respondents mentioned stock and pets, including evacuating stock. One woman led a frightened horse to safety when required to remove it from agistment by the property owners.

> I had no float [horse trailer]… so I had to lead him [the horse] and he jogged the whole way! (Cara, 20-25 years, Canberra fire)

Evacuation options for stock were generally limited. Public safe places (widely provided in the Canberra fires only) require that people remained with their animals, making those options problematic. The same interviewee kept her horse in her suburban backyard for several weeks:

> I didn't know what else to do. He [the horse] was pretty good: didn't get caught on the clothes line once and the dogs got on well with him! (Cara, 20–25 years, Canberra fire)

A strong theme among the women who evacuated concerned being cut off and unable to find information about their homes. For those whose partners had remained to defend the home, this was particularly distressing. In both fires, phone lines were either overwhelmed by call volume or phone towers were destroyed by the fire. This led to poor contact, particularly between family members, which raised stress.

> I felt completely out of control and at least if I was there [at home, in the fire line] I knew what was happening. (Kate, 20–25 years, Canberra fire)

Again many women in both fires reported that official warnings and broadcast information were inadequate and either out-of-date, hard to interpret, or insufficiently detailed.

> I think people completely misunderstand the 774 [emergency radio station] warnings. (Sarah, 30–35 years, Black Saturday)
>
> [T]hen the power went out … and that's when we found out the fire was coming through. (Kate, 20–25 years, Canberra fire)

> The warnings were too late, but they said the firestorm … was so unpredictable. (Carmel, under 20 years, Canberra fire)

> Still very angry about the complete incompetence that … emergency services showed. (Survey participant, 25–30 years, Canberra fire)

> More official information being available during the fire would have been better, Facebook was the best place to get information from others in the community. (Survey participant, 25–30 years, Black Saturday)

> “[Regarding CFA advice on safety in days following the initial fire] they said “it's just hot.” (Amy, 30–35 years, Black Saturday)

> [I] didn't hear any emergency broadcast until well after fire had started. Emergency services were non-existent in our street until 8pm and 3 houses had already burnt down. (Survey participant, 25–30 years, Canberra fire)

### 8.7.4. Maternal health behaviours

*Survey analysis*

Maternal alcohol intake and smoking following disasters are rarely studied. Fuller (2013) in a study concerning 20 years of environmental disaster exposure in North Carolina, US, finds that maternal cigarette smoking declined following disaster exposure, at least in part due to logistic factors such as shops being closed or cigarette stocks being destroyed in flooding. This analysis shows a sharp decrease in maternal smoking in Canberra in the year of the fire, although this decline is not limited to areas which were fire affected.

Three women (8% of those surveyed) began to smoke following the fire, while four (11%) smoked both before and after the fire. Of these, one survey response is difficult to interpret due to an impossible response on length of gestation at the time of the fire (66 weeks). It is possible that this respondent may have meant that she recommenced smoking after birth. There were too few mothers who smoked for further analysis.

Maternal alcohol consumption is not shown to have been significantly related to either exposure or stress scores. Five women increased their alcohol consumption while pregnant following the fire (14%) and five women reported drinking alcohol both before and after the fire (14%). Of these, one woman was post-term at the time of the fire and one provided an impossible gestation age (66 weeks) at the time of the fire. One mother who reported increasing her drinking following the fire reported very low consumption (i.e. having three standard drinks in the month following the fire).

Ten women (28%) reported an increased in prenatal care attendances following the fire. Although this may relate simply to a greater number of visits in the later stages of pregnancy, the degree of care use and progression of gestation at the time of the fire does not correlate strongly or positively ($r = 0.19$; $n = 35$). However, the correlation between total stress score and care use is positive and weak-moderate in strength ($r = 0.34$; $n = 35$). Only three women reported any change to their prenatal vitamin consumption, with two ceasing their intake and one commencing.

*Participants' commentary*

Some women interviewed, who did not drink, expressed that their desire to drink was greater after the fire:

> I often said if I wasn't pregnant, I'd be drinking right now! (Sarah, 30–35 years, Black Saturday)

Only one participant reported being provided directly with information on the possible risks of prenatal stress:

> My GP told me not to go to work. She said that we don't know how stress affects babies and she recommended that I use the certificate and stop going to work straight away. (Sarah, 30–35 years, Black Saturday)

Air quality during fires is an issue which has received greater attention in recent years, especially following the 2014 Hazelwood Mine Fire in Victoria, in which a coal mine burnt for some months shrouding nearby towns in persistent smoke. Some participants raised issues around air quality and concerns about its pre- and post-natal effects. None reported being able to find good information on possible effects during the fire.

> [A]fter the fires the air was quite smoky and my daughter now has asthma… I often wonder if the environment at the time was detrimental to her baby lungs. (Survey participant, 30–35 years, Black Saturday)

### 8.7.5. Support and practice recommendations

*Participants' commentary*

Many survey respondents praised the care that they received from both professionals and community members:

> Midwives were excellent. (Survey participant, 25–30 years, Canberra fires)

> I accessed mainly informal support through my usual networks, but also good support from the hospital and maternal and child health nurse. (Survey participant, 35–40 years, Black Saturday)

> I accessed free counselling and kinesiology … I wish I had gone sooner. (Survey participant, 35–40 years, Black Saturday)

> I did receive a lot of support from my community. (Survey participant, 35–40 years, Black Saturday)

> Mother-baby unit was very helpful. (Survey participant, 35–40 years, Black Saturday)

> Lots of support from a wonderful health centre nurse [and] also from GP after the fire. They [maternal health nurses] also visited more often than normal to keep an 'eye' on me and bub. (Survey participant, 30–35 years, Black Saturday)

> Help/refuge centres were great. (Survey participant, 35–40 years, Black Saturday)

> Met new friends in community. Maternal health nurse was great. Counselling was available and needed. (Survey participant, 30–35 years, Black Saturday)

> I had a wonderful health nurse who would hunt me down for check-ups. (Survey participant, 35–40 years, Black Saturday)

> I had support from friends. My obstetrician was marvellous. I was connected to a psychologist and received regular interpersonal therapy. (Survey participant, 35–40 years, Black Saturday)

> Red Cross visited and gave a package with information and contact details for assistance. (Survey participant, 25–30 years, other fire)

Fewer women reported that they did not receive sufficient support:

> I didn't get any assistance. My husband flew to Canada the day after the fire and I had to look after myself. (Survey Participant, 40–45 years, Canberra fires)

> No support. (Survey Participant, 25–30 years, Canberra fires)

Survey respondents were also asked for recommendations for future fires. These included more proactive support provision from health professionals:

> More active offers of or ideas for help from my GP would also have been useful. (Survey participant, 35–40 years, Black Saturday)

> More prenatal checks, ultrasound, stress relief. (Survey participant, 35–40 years, Black Saturday)

Another expressed a need for more home visits to reduce the incidental exposure of mothers to environmental toxins following fires:

> Wish there were house visits from your GP instead of going out in the smoke or soot for your check-ups. (Survey participant, 20–25 years, Black Saturday)

Another expressed the need for more lasting help and financial support. This comment was echoed incidentally by at least one community practitioner.

> More financial and ongoing support as the experience still affects me today. (Survey participant, 35–40 years, Black Saturday)

Being able to take an active role in community recovery was recommended by two survey participants:

> More support for mums with bubs to be able to contribute to helping recovery. (Survey participant, 35–40 years, Black Saturday)

> I helped form a support group for mothers with young children who had been displaced by the fire. I found this extremely helpful. (Survey participant, 35–40 years, Black Saturday)

Lastly, one interview participant recommended an information and preparation workbook for pregnant women. This would include information on staying safe and hydrated, knowing multiple routes to your hospital, contacting neighbours and what things to pack for an evacuation. The interviewee recommended that the workbook could be distributed through General Practitioners, Neighbourhood Watch groups, and schools. The pack could be designed as an all-hazards measure.

## *8.8. Discussion*

### 8.8.1. Stress

An association between environmental disasters and negative effects on the mental health of survivors is well-established in the literature (Bassilios et al. 2011; den Ouden et al. 2007; Hibino et al. 2009; Hussain et al. 2011; McFarlane 1987; McFarlane et al. 1997; McFarlane and Van Hooff 2009; Norris ; Roberts et al. 2009; Tucker and Pfefferbaum 2010; Weinstein et al. 2000), with female gender frequently identified as particular risk factor for the development of PTSD (Galea et al. 2005). From the survey and interviews, Black Saturday emerged as a substantially more stressful experience than the Canberra fires. While this was possibly influenced by the time elapsed between the fire event and the data collection, Black Saturday was also a demonstrably larger and more destructive fire (see Chapter One).

Gendered analysis suggests that the psychological effects of disaster can be greater for women, because they have a higher incidence of other risk factors. These include being the head of a sole parent household, having caring responsibilities (for either children or elderly relatives), having a low income and few resources, and living alone (particularly in older age) (Enarson 2012; Ollenburger and Tobin 1998). It is also likely that women may be socially able to exhibit their stress more readily than men (Ollenburger and

Tobin 1998). This may mean that men experience equivalent levels of stress but are less demonstrative. Reinsch (1998 p.153) describes women as the "symptom bearer of their family's health" in that they both absorb and reflect the wellbeing of their families". As such, female gender acts as a marker for a confluence of stress risk factors, both practical and psychological, and this results in a higher perceived disaster impact (Ollenburger and Tobin 1998).

Through the interviews, survey and other sources, objective measures of stress were found to be relatively high for many participants. Total stress scores (i.e. those measured using the PSS) were significantly higher for Black Saturday fire survivors than for Canberra fire survivors, likely reflecting both the greater objective severity of the event and its being more recent, which may affect recall. The survey data show a moderate positive correlation ($r = 0.58$; n = 36) between objective event exposure and total stress score. Previous work shows that there can be little relationship between objective and perceived stress among women who have experienced environmental disasters while pregnant (King and Laplante 2015). Because of this identified variation between exposure to stressful events and perception of stress, it is not possible to determine whether the moderate relationship between stress exposure and stress perception found in this dissertation is evidence of the PSS as a valid measure of recalled perceived stress.

Consistent with the literature suggesting that young people are less resilient to stress (den Ouden et al. 2007; Scaramella et al. 2008; Soeteman et al. 2008), even as exposure scores rise with age, perceived stress scores do not. This suggests that older mothers had no higher perceptions of stress even where objective exposure to stressful events was higher.

Through interviews and free text portions of the survey, participants identified a range of physical stressors, including heat, smoke, and the physical demands of fire vigilance including constant alertness and loss of sleep. Most participants also described psychological stressors, such as fear, pressure to complete recovery paperwork, and the guilt and frustration of not being able to aid family and neighbours. Notably, a few described the role of pregnancy as protective against some stressors because the baby provided a point of focus and a "hormonal buffer" against the post-fire environment.

Not being able to assist in community recovery activities emerged as a strong theme in both the interviews and surveys. This echoes a previous study of Australian women's experience of bushfire, which finds that caring for others is central to women's post-disaster psychological resilience (Cox 1998). Similarly in Reinsch's (1998 p.162) study of a Canadian agricultural disaster she directly links female empowerment to female action – as one participant states: "the harder I work, the better I feel".

For many pregnant women, the constraints on their ability to serve their community may negatively affect their own recovery. Some women who had their babies during the fire aftermath felt that they were not able to contribute to the community, because of the demands of caring for their newborn. It appears that forms of community service (i.e. those extending beyond service to their immediate family) are important in terms of women feeling that their contribution is valid and useful (Borrell and Boulet 2009; Tyler et al. 2012). Cox (1998) suggests that while men find a "pleasure in strength" during post-fire recovery, women perceive their roles as purely duty, with no associated pleasure or glory. Women who cannot participate in their perceived duty, due to their pregnancy or the needs of their newborn, may feel that they have neglected this important duty.  This may further the strong rural narrative described by other authors that bushfires are “men’s business” (Tyler and Fairbrother 2013).

Many participants reported that there was damage to homes in their community. Via the interviews, some participants reflected on the impact of living in damaged or burnt environments, identifying the damaged environment as an additional source of stress and despair. Bushfire-damaged environments certainly persist over the longer-term. For example, the Marysville township remains overshadowed by the skeletons of tall dead trees and many former house blocks remain vacant, even five years after the fire. It is possible that these tangible reminders of risk and destruction are influential in the well-being of people who remain in those communities. One interviewee reported that she knew her own depression was beginning to lift when she realised that there were green shoots in the bush around her home, not just “the black” of the burnt trees and ash (Suzanne, Black Saturday). For women living in heavily damaged communities, the daily physical reminders of the fire may be important factors in their own stress and recovery. Like the participant above who drew links between her own recovery and the regeneration of the bush, in a study of the Ash Wednesday fires, Cox (1998) finds that some women perceived the damage to the natural environment to be a loss as equally profound as loss to the loss of their homes.

Of the survey participants, more than 10% moved permanently after the fires. Some degree of dislocation was reported by 45% of survey respondents and is a well documented stressor (Enarson 2012). Some literature reports that women are more affected by post-fire recovery environments than men because they spend more time in their homes and communities, as a result of caring roles (Cox 1998). This is likely to be particularly the case for pregnant women and new mothers. They are likely to be spending more time in their homes and local communities due to maternity leave and their additional caring responsibilities.

Other literature notes that women in general are likely to spend more time interacting with their local communities. This occurs because women are less likely to undertake paid work outside the affected areas and are more likely to undertake tasks like shopping and school runs that expose them to the local community. Because of this increased exposure to the local community, women are more likely to experience the impact of a stressed and potentially fractured post-fire community (Hoffman 1998). They are also more likely to experience criticism if there is a perception that they have broken a social contract, such as receiving more recovery funds than is seen as fair. Certainly, some participants in this study expressed strongly that their interactions with the community could be highly stressful, as local people verbalised their fire experience to peers.

Conversely, some women described their connection to the local community as vital to their recovery. Because their connection to community may be heightened through this period, via prenatal classes, mother's groups, playgroups and more time spent locally while on maternity leave, pregnant women and new mothers are potentially more exposed to both the detrimental and restorative aspects of community involvement.

Disasters are well documented as placing strains on intimate relationships, and also as being strongly associated with an increase in domestic violence (Enarson 2012;

Parkinson 2011; Victorian Council of Social Services 2014). Although there were no reports of domestic violence recorded, there were reports of tensions in intimate relationships and of relationship breakdown. Most interviewees described changes in the allocation of roles in their families and the gendered divisions of labour that emerged following the disasters that had affected them. Some saw the tensions that had emerged in their relationships as rooted in fundamental differences between male and female responses to stress and trauma. Some research suggests that gender differences can emerge in the post-disaster environment. Hoffman (1998) describes post-fire environments as the "rubble of a social and cultural system" in which gender roles revert rapidly to "old, deeply rooted cultural patterns". Often this manifests as women returning to traditional home-centred caring roles and men taking on dominant and directive roles in recovery. For women who are pregnant or who have newborns, these effects are likely to be magnified by the intersection of the traditional gender roles that accompany pregnancy, birth, and childcare with those that may emerge during disasters and recovery.

### 8.8.2. Trauma

Traumatic stress is considered separately because of the risks associated with trauma exposure and the development of PTSD and also because it emerges as an important theme in interview and survey responses. Traumatic stress is understood to be the stress arising from a trauma or traumatic event. Traumatic events  are defined by Olff (2012) as an event involving "actual or threatened death, serious injury or threat to physical integrity" (p.1). Thus, much of the stress of immediate bushfire exposure can be considered traumatic stress, even though the threat to bodily harm is generally threatened rather than actually realised. Within this dissertation, actual or risk of bodily harm or death to relatives and friends is also included as a form of traumatic stress. By contrast, the social disruptions which follow fires represent a more chronic and general form of stress.

Following the 2009 Victorian Black Saturday bushfires, publicly funded mental health services were available for affected people. Over 190,000 people received some form of treatment (Bassilios et al., 2011). Service figures, however, are likely to be underestimates, as registrations were not required for all activities, and activities funded under Medicare (Australia's publically-funded medical services scheme) were not identifiable as bushfire-related. Following the Canberra fires, an Australian Psychological Society survey of private practitioners showed that 165 clients with bushfire-related conditions had presented, with conditions including PTSD, depression, acute stress responses, and relationship difficulties. In particular, children were reported as experiencing sleep disturbances, an increase in obsessive and anxious behaviours, and regressive behaviours such as bed wetting and nightmares (Bushfire Recovery Taskforce 2003).

Many interview and survey participants, had experienced events likely to be traumatic. These included threat of death or injury via exposure to the fire front and actual loss of family and friends in the fire. Such experiences produced feelings of terror, and fear of death. Feelings of loss of control are associated with worsened recovery (Benight and Harper 2002) and were experienced by two-thirds of the survey population and all interview participants. Other indicators of traumatic stress, such as disturbed sleep and nightmares (in addition to sleep disruption caused by caring for young babies) were widely experienced by survey participants.

A strong theme in the interviews was that of vicarious stress and trauma. Some women experienced this through evacuation, when crowded into houses with other stressed family members. Others experienced it during recovery, as people in the local community shared their stories of loss and survival. The phenomenon was best captured one participant who observed that people recounted their trauma by "blurting it out in aisle three" of the supermarket (Sarah, Black Saturday). Accordingly, these women's greater exposure to traumatic stories of other may have lengthened their own stress exposure (even as it may have assisted the person telling the story). For example, one interviewee became visibly (and understandably) distressed while recounting the experience of a child in the community who had watched his parent's cattle burning alive. Indeed, both interview and survey participants described their emotional responses (and those of the local community) as emerging over the long-term. Although the interviews with Black Saturday survivors took place almost five years after the disaster, all described their emotional response as ongoing – for themselves, their families and their communities.

Participants also described how their needs changed over time. Although counselling was offered in the immediate aftermath and during much of the following year. However, the case management service for the Black Saturday fires, one of the key linkages between people and services, closed in June 2010 (Grealy et al. 2010). Many people responded that they didn't want or accept intervention during this period but preferred the support of their families. However, over the longer-term they felt a greater need for professional assistance and would have accepted such services had they been offered later. Unfortunately, by this stage, many services had been withdrawn and support was much harder to access.

The lack of long-term support and the gap between community need and available services is noted in reviews of longer-term recovery (Borrell and Boulet 2009). Following the Black Saturday fires, the demand for practical support from Government (such as clothing and housing) declined but demand for emotional and social support increased for around 12 months before also beginning to slowly decline (Housing and Community Building Division 2010). However, due to the closure of many services, it is difficult to estimate the underlying unmet long-term demand.

Although the sample is not large enough to draw conclusions about prevalence, a small number of women in the study were diagnosed with Post-Natal Depression (PND). They felt that there was a connection between their fire experience and subsequent PND diagnosis. Potential links are supported by the literature (Apter et al. 2011; Harville et al. 2010), although there is a lack of diagnostic clarity due to overlapping symptomatology between PND and PTSD. Following the Black Saturday fires, there was a notable increase in demand for perinatal depression services in Victoria (Bassilios et al., 2011), which supports a potential relationship between fire exposure and PND.

### 8.8.3. Evacuation

There is a substantial body of literature on the sociology of evacuation (and disaster response more broadly) and in particular the gendered aspects of disaster behaviour. This literature consistently finds that women are more likely than men to evacuate and that women who are pregnant or caring for children are the group most likely to evacuate (Enarson 2012; Tyler 2013). These findings are supported to some degree by the findings of this dissertation, with women reporting that families often split – with the man staying to defend the property and the woman and children evacuating.

In contrast to the literature, a notable finding of this research concerns how few women evacuated. While this may be an artefact of sampling bias in the survey, some previous research also indicates relatively low fire preparation and evacuation rates (Strahan Research Pty Ltd 2009). Among survey participants, 52% remained at home, with almost 10% actually at home when the fire burnt through their property. A further 27% left when the fire was nearby. In total, 79% of women either did not to evacuate or did not make an early evacuation. In Australian bushfires, late evacuations are widely considered to pose the highest risk to safety (Haynes et al. 2010). Among a group that might be expected to consider themselves more at risk, the low rates of evacuation and relocation were an unexpected.

There are several possible reasons why women did not evacuate or did not evacuate sufficiently early. Firstly, many women in both fires reported that official warnings and broadcast information were inadequate due to being out-of-date, hard-to-interpret or insufficiently detailed. These comments echo the findings of the Royal Commission in the Black Saturday fires, which found that communication during the fire, both to the community and between emergency response workers, was inadequate (Teague et al. 2010b). Interview and survey participants reported that not only were public broadcasts unhelpful, but so too were directions given to communities via community meetings and police doorknocking. Some women did not have sufficiently advanced warning in order to leave, finding that the fire was already approaching or that all exit routes were already blocked. This applied to both fires, with some women in the Canberra fires also highly critical of the warning systems. In both fires, participants reported that warnings were either too late or difficult to interpret. This made their decisions to either "stay-or-go" largely uninformed.

Secondly, in the case of Black Saturday only, the fire threat was long-term, resulting in the need for multiple evacuations. Two interview participants reported evacuating multiple times with young children to an urban shopping centre around 30 minutes' drive from their community. This kind of evacuation is highly disruptive to work and schooling and it is difficult to consider it as feasible when high-risk warning days may occur several times per week for weeks on end. There is also no Government provision for overnight evacuations, limiting people's options unless families or friends are able to provide shelter or unless hotels are both available and affordable. Both these options are problematic for people with pets or stock, which includes a very large percentage of people living in rural areas.

Lastly, evacuation appears to be at least as stressful as remaining at home. Although women who evacuated late did report higher exposure scores, they did not report higher stress scores. Those who evacuated early (as recommended) reported stress scores which were not significantly different from those of women who evacuated late or who remained at home. This is suggestive of evacuation itself acting as a source of stress, consistent with the literature, creating relative parity between the groups (Enarson 2012).

Evacuation centres were uncomfortable and staying with family presented additional stresses (mostly related to overcrowding) for some women. Most importantly, because of poor public communication, evacuation acted to limit participants' access to fire information and caused increased stress through not knowing what was happening to spouses and homes left behind. Separation from partners and spouses was a considerable source of stress. This is consistent with literature on stress associated with separation from husbands or partners in both pregnant and non-pregnant women. Goodman and Proudley (2008) report that two women in their study with husbands away for long periods experienced chronic anxiety related to the threat of bushfire. An older study examining sources of psychological stress in pregnant women (through a sample of 67 American women) found that separation from husbands during pregnancy is rated as the 15th most stressful event. That is higher than the death of a close friend, a child leaving home against their parent's wishes, or the failure of a husband's business (Yamamoto and Kinney 1976).

Although this dissertation cannot exclude other differences between the groups other than evacuation status, it suggests that more consideration should be given to improving the experience of evacuation and encouraging partners to accompany evacuating pregnant women. Further research is required to determine whether evacuation can act as an explicit source of stress.

However, early evacuation is clearly safer in terms of survival, as demonstrated by the death of Ms Nicole Jefferson. Ms Jefferson was killed by the Black Saturday fires while eight and a half months pregnant and attempting a late evacuation. It remains unclear whether she was injured, overcome by smoke or both prior to her death. Ms Jefferson's partner, Mr James Bowker, remained to defend their property and was also killed. Ms Jefferson's body was found about 200 metres from her believed destination, a football oval where people were sheltering (Teague et al. 2010a). The people at the oval survived the fire and so it is likely that Ms Jefferson would also have survived had she arrived safely.

Ms Jefferson's neighbour (who brought this case to my attention), strongly believed that Ms Jefferson would not have died had she not been heavily pregnant. Victorian Police Forensic Services Centre's forensic officer Ms Karen Ireland, in testimony read to the Victorian Bushfires Royal Commission by Detective Superintendent Paul Hollowood (2009 p.10718), concurred, stating that "The deceased appeared to have decided to evacuate rather than defend the fire, but delayed the decision unduly. The decision to seek refuge on the oval may have been appropriate but the physical condition of the deceased may have affected her mobility" (p.10,718). While evacuation probably does not reduce the stress of fires, and in any case cannot mitigate stresses already experienced, early evacuation remains the most effective available tool to reduce outright mortality risk and perhaps prevent such tragedies.

### 8.8.4. Maternal health behaviours

The survey considers maternal cigarette smoking, alcohol consumption, use of prenatal vitamins and prenatal health visits in order to examine the effects of disaster on maternal health behaviours. The analysis of the ACT and LSAC data presented in Chapters Four and Seven respectively also considers indicators of prenatal health behaviours (cigarette smoking in the case of the ACT and all the above indicators in the case of LSAC).

Too few women surveyed smoked during pregnancy to allow further analysis; however, reports of smoking show no obvious relationship with stress levels or exposure. Notably, the ACT data shows a sudden decrease in maternal smoking following the fire, but this was not concentrated in the severely affected areas. Maternal smoking also rebounded in late 2003 and 2004. The timing of the decrease is suggestive of a possible relationship with the fire. Although statutory changes controlling smoking in public places were legislated in the ACT in 2003, they were not enacted until 2006 (Brown 2014). At the time of the fire there were public communications about air quality problems resulting from bushfires, which may have triggered some mothers to decrease their smoking, perhaps heeding advice to reduce exposure to other sources of air-borne pollutants (Brown 2014). In a study of 20 years of disaster data from North Carolina (US), Fuller (2013) finds that maternal cigarette smoking declined following disaster exposure. However, this was attributed largely to logistic factors, such as shops being closed or cigarette stocks being destroyed in flooding, which would not widely apply in this case.

Air quality following fire is an emerging issue in terms of effects on the foetus. Brunsma et al. (2007 p.6) term many modern disasters "natural-technological" because they represent an intersection of natural and technological risks. Bushfires are one such example of a natural-technological disaster, because they combine the natural risks of fire with technological risks such as the release of asbestos fibres from damaged homes and toxic pollutants in smoke and water (from fire run-off). Some of these risks, like polluted air and water, are transient, while others, such as asbestos fibres, can become persistent in the environment, creating psychological stress in the short-term and potential serious illness in the long-term.

Little advice on health or air quality released during bushfires is targeted at pregnant women. Neither the Victorian EPA nor the Department of Health mention pregnant women in their official advice on bushfires and air quality (Department of Health 2013; Environment Protection Authority 2014), despite evidence of risk to foetal wellbeing (Gehring et al. 2014; Sanders and Stoecker 2015). Further, despite the additional strain pregnancy puts on the maternal respiratory system, pregnant women are not identified for special advice, unlike children, older people, and people with cardiac or respiratory problems. During more recent fire responses, such as following the 2014 Morwell mine fire (in south-eastern Victoria), health authorities have been criticised by pregnant fire survivors for providing insufficient health warnings (Teague et al. 2014). Some women in the sample spontaneously expressed concerns about the effects of smoke and being required to go into the smoke in order to attend prenatal appointments. This suggests that timely and appropriate advice to pregnant women on the risks and management of smoke exposure remain insufficient through the official channels.

Although a small number of women reported increasing their drinking after the disaster, those who specified amounts indicated a very low level of consumption. Some women reported greater desire to consume alcohol, which aligns with the common social use of alcohol to mediate stress. Changes to the consumption of prenatal vitamin supplements were also minimal, indicating that access to supplies was not impeded by the fires.

Some research indicates that diet choices alter in pregnant women when they are stressed, with increased consumption of calorie-dense foods related to subjective stress (Hurley et al. 2005; Lobel et al. 2008). However, the effects of stress on diet were not examined in this research. Only one participant noted that her diet worsened, with an increase in the consumption of sweet foods.

Almost a third of women reported an increase in their use of prenatal care following the fire and this was not significantly related to the usual increases in care visits toward the end of gestation. However, increases in care use were significantly and positively related to stress scores. This suggests that women who were more stressed, and therefore potentially in need of more support, did in fact receive additional care. Although only one woman spontaneously reported that her GP directly discussed the potential impacts of maternal stress on foetal development, many women praised their prenatal care givers and felt that their support was important and valuable.

One unexpected health consideration arising from interviews was that of dehydration. Because of the metabolic demands of pregnancy, women have a higher-than-usual risk of dehydration under normal conditions (Timmons 2016). Under the conditions of bushfire – extreme heat, psychological stress and, potentially, exercise stress – this risk is heightened. In the case of one participant, her extreme dehydration became so severe that it triggered true contractions at 29 weeks gestation while she was in an evacuation centre. Under the care of the centre staff, including a medical doctor, she was able to rehydrate sufficiently that the contractions decreased in frequency and ultimately subsided. Had labour continued at that point, the fire would have impeded hospital access and the outcome could potentially have been tragic. This participant proposed that the self-care messages currently extended to older people and children should also include pregnant women as an at-risk group. In her case, the distraction of caring for her own young child and ageing parents meant that she neglected her own care.

### 8.8.5. Support access

A total of 5,506 people registered with the Victorian Bushfire Case Management Service until its closure in June 2010 (Grealy et al. 2010). Individuals were able to register on behalf of their whole family, so the number of individuals represented by this figure is higher. Following Black Saturday, peak service use occurred between March and July 2009. The most common reason for using the service was practical support (e.g. housing or financial assistance) with the second being emotional or social support (Grealy et al. 2010). In terms of practical assistance, money, clothing and housing were primary. For instance, 1,347 people registered for housing assistance following the fires. This assistance was provided through a mix of public housing, donated caravans, the Victorian Bushfire Response and Recovery Agency temporary villages (grouped demountable homes), and leased houses (Housing and Community Building Division 2010).

The ACT Government provided similar services following the Canberra fires. The onset of winter proved to be a particular difficulty for those Canberra families who were displaced in the fires. Canberra experiences very cold winters, increasing the strain on families living in temporary accommodation with limited donated clothing and bedding (Bushfire Recovery Taskforce 2003).

Most women involved in the study were happy with the support they received and praised the assistance, especially those services that were particularly relevant to their pregnancies, such as doctors, midwives, and mother-baby units or health centres. Although some participants took part in counselling, none reported attending targeted stress reduction programmes. These have been shown to be useful in reducing maternal stress (Khianman et al. 2012; Marc et al. 2011), although there is no evidence that mind-body stress reduction techniques effectively reduce the risk of premature labour (Khianman et al. 2012). One survey participant recommended some type of stress relief as an intervention for future fires.

The duration of support was raised as an issue by Black Saturday survivors. It is important to note that the Bushfire Case Management Service had 892 cases that were still “active” at the time it closed, with a further 419 still “transitioning to closure” (Grealy et al. 2010). This represents a large number of people and families who potentially had ongoing needs. Only a few women stated via the survey that they received no support and, due to the collection method, it was not possible to understand the reasons for this in detail. Among interviewees, nearly all received some form of support through either official recovery channels, community groups or through the maternal health system. For one interviewee who required care for her PND, this care was hard to access and was finally received only once she was already at a crisis point. For those women who received AGDRPs, the amounts they recalled receiving were lower that than those they would have been eligible for, although this may be an error of recall rather than assessment. Two women suggested that funding to leave the area for a break would have been the most effective way of reducing stress.

As mentioned above, the negative consequences of feeling unable to help in recovery were an important theme in these findings. This theme was echoed in women's accounts of what helped them after the fires and what they would like to see in future fire crises. One participant had been active in forming a local support group and felt that this activity was very helpful in her own recovery. Other participants, through both the survey and the interview, suggested that more active roles for pregnant women or new mothers to be able to take part in recovery would be valuable in future fires. This exclusion of pregnant women and new mothers from participatory community roles may be viewed through a gendered lens as an exacerbation of the broader exclusion of women from community recovery roles (Cox 1998; Tyler et al. 2012).

While in research following Hurricane Katrina, pregnant women spoke about their fears for the future needs of their children and their inability to find lasting safe housing, food, and medical care, these were not major themes in this research (Badakhsh et al. 2010). The absence of these worries might reflect the smaller scale of damage in both fires (when compared to that of Hurricane Katrina, particularly in New Orleans) and greater confidence in the ability of state support and infrastructure to provide for their future needs.

## *8.9. Chapter summary*

This chapter presented the method, results, and discussion of a mixed-methods study of the maternal experience of stress during bushfires. It finds that women experienced a considerable degree of stress when measured in terms of both perceived stress and objective exposure to stressful events. Women pinpointed aspects of their experience as particularly stressful, including being separated from partners, being unable to access good information, and being excluded from community recovery programs and other support mechanisms. Conclusions and recommendations for future research and practice are made in the next chapter.

# 9 DISCUSSION AND CONCLUSIONS

This dissertation asks four key research questions centred around the effects of bushfire exposure on reproduction. Firstly, what is the effect of prenatal bushfire exposure on birth weight, gestational age, plurality and secondary sex ratio (SSR)? Secondly, what is the effect of population bushfire exposure on fertility rate? Thirdly, what is the effect of prenatal bushfire exposure on developmental outcomes? And finally, what is the lived experience of bushfire exposure while pregnant?

Evidence to answer these questions was sought through five analyses: (1) a cohort study of birth records examining fire effects on birth weight and gestational age (Chapter Four); (2) a cohort study examining SSR and plurality rates (Chapter Five); (3) a cohort study of fertility rates (Chapter Six); (4) a cohort study of child developmental and behavioural indicators (Chapter Seven); and (5) a mixed-methods study of maternal experience of bushfires while pregnant (Chapter Eight).

Consistent with predictions of life history theory (LHT), the hypotheses were that exposure to bushfires, and attendant stressors, would have the following effects. Firstly, it would cause shortening of gestational length and declines in birth weight in exposed mother-child dyads. Secondly, it would lead to increases in the birth rate. Thirdly, it would cause declines in SSR and plurality rates in exposed cohorts. Fourthly, it would result in greater developmental comorbidity in children exposed to bushfires *in utero*. And lastly, it would cause pregnant women to experience high levels of stress.

This dissertation finds supporting evidence for hypothesis five (that bushfire exposure can be expected to cause high levels of stress) and, to some degree, hypothesis three (that bushfire exposure can be expected to cause declines in SSR and plurality rates). However, the results for the remaining hypotheses (one, two and four) are more complex. One the one hand, the dissertation finds supporting evidence for these hypotheses. On the other hand, however, it also finds evidence and trends running contrary to these hypotheses. This finding points to a range of complexities in adaptive response sensitivities, such as between mothers and foetuses, between male and female foetuses, and in response to differing levels of environmental threat. This chapter provides a discussion for the dissertation as a whole. Subsequently, in addressing these complexities, it also discusses each of five analyses in more detail, including exploring the interlinkages between the different analyses. Finally, the chapter concludes with recommendations for policymaking and future research.

## *9.1 Overall findings*

Overall, this dissertation finds patterns of biological and behavioural responses which indicate that even modern humans in first world environments are fundamentally influenced by their environments. The patterns of behaviour observed indicate the existence of underlying evolved psychological mechanisms (EMPs) that respond to stochasticity by altering investment in current reproduction. This evidence supports the dissertation's overarching argument that modern humans continue to react to environmental pressure in a manner consistent with evolved responses.

In this case, the experience of stress provides a biological indicator of environmental quality. The mechanisms appear to act in a manner consistent with evolutionary theories of reproductive investment. Specifically, the responses described are consistent with the future-versus-current reproductive trade-off and the offspring quality-versus-quantity reproductive trade-off. The evidence presented demonstrates the occurrence of fine adjustments to reproductive strategy in modern human populations – such as altering birth weight while maintaining an otherwise healthy pregnancy, which show a high degree of plasticity and environmental responsiveness. Taken as a whole, these findings suggest a high degree of fine-tuned sensitivity to the specific nature of the environmental threat, including its magnitude and duration.

This dissertation considered two fires as case studies: the Black Saturday and Canberra fires. Black Saturday was a substantially larger fire with exposed mothers reporting higher subjective and objective stress compared to mothers exposed to the Canberra fire. Where existential threats were greatest (Black Saturday), the dissertation shows that there was a possible increase in the number of lost foetuses. However, it does not show the occurrence of changes in birth weight or gestational age.

This dissertation demonstrates that changes in birth weight or gestational age did occur following the less severe fire (the Canberra fire). Yet it does not show changes to patterns of early loss. This suggests that, in response to the more extreme stress, mother-foetus dyads particularly sensitive to environmental changes may have experienced a pregnancy loss rather than an adaptive change to the level of investment during gestation – akin to the “culled cohort” hypothesis of Catalano and Bruckner (2006 p.1639). Those mother-foetuses who remained intact through early pregnancy may have been sufficiently robust (e.g. had lower maternal responsiveness or lowered foetal responsiveness) to the selective pressure created by the disaster. This may, in turn, have resulted in the absence of any detectable changes to gestation. Where environmental pressures were less extreme, there may have been insufficient pressure to trigger increased losses. Accordingly, the more sensitive cohort may have gone on to complete gestation and demonstrate adaptive responses.

The Canberra fires resulted in an increase in average male birth weight and in the number of macrosomic infants. Among female infants, there was no change in birth weight or gestation. This indicates an increase in maternal investment in male infants. However, it is not possible to determine whether the increased investment is a result of maternal or foetal action. Potentially, mothers increased their investment to create an environment buffer for those foetuses nearing birth, favouring the more fragile male foetuses. Alternatively, male foetuses may have responded to a change in maternal investment by acting to increase their resource access. Because of their smaller size, female foetuses may have been better equipped to manage reduced maternal investment without requiring a foetus-led response.

Similarly, although much literature finds behavioural and psychological sequelae associated with *in utero* disaster exposure (King et al. 2005; Kinney et al. 2008a; Laplante et al. 2008; St-Hilaire et al. 2015; Turcotte-Tremblay et al. 2014; Walder et al. 2014), the dissertation does not demonstrate behavioural responses in children exposed to the milder fire event. This again suggests that milder stressors do not trigger an adaptive response or trigger a minor response not detectable by the methods used here.

While neither fire seemed to trigger an increase in fertility rate, it is not possible to state definitively whether this is due to methodological limitations or to an absence of effect.

This finding of fine-tuned sensitivity to stress is consistent with LHT and the broader literature on environmental influences on reproductive strategy. To confer fitness benefits, any strategy that is responsive to environmental conditions must be responsive enough to be protective but not so responsive that costly over-reactions occur. Kuzawa (2005) makes such an argument in relation to nutrition during pregnancy, proposing that foetal growth responds to indicators of the quality of the mother's nutrition over her lifespan, rather than the nutrition exposure received directly *in utero*. Under normal conditions, the mother's lifetime nutrition provides a more genuine indication of a foetuses' likely future environment than fluctuations which might occur during pregnancy. Similarly, if changes in foetal growth occur in response to mild or transient maternal stressors, the costs of this level of plasticity could potentially outweigh benefits. Accordingly, a dose-response relationship between stress exposure and changes to foetal development is more likely to confer a fitness benefit while moderating the cost of plasticity.

In their extensive review of the neuroendocrinological evidence, Musazzi and Marrocco (2016) also find that the existence or degree of deleterious change associated with perinatal stress exposure is strongly moderated by when and how often stress occurs, as well as how intense the stress is, how the stress interacts with the individuals' genetic predisposition, and the quality of maternal care.

As an understanding of the fires was developed through analysis undertaken and the experience of female survivors, the two disasters emerged as characteristically different from one another. Because of the relative intensity of the two fires, as well as the quality of the recovery response, the disasters were quantitatively different in terms of the factors that Musazzi and Marrocco (2016) identify as important, such as degree and duration of change associated with the stressor and the intensity of the stressors themselves. Accordingly, it is possible to understand the events as examples of acute (Canberra fires) and chronic (Black Saturday fires) stressors for the bulk of the people affected – rather than two examples of similar or comparable stressors. Such a difference between the two fires is relevant in the application of LHT, which suggests that transient and acute stressors should be differentiated by the organism and responded to accordingly. The degree to which the different characters of the fire affected the outcomes measured is developed further in the sections below.

With this in mind, the relationship between the degree of deleterious effect and stress intensity suggests that if maternal stress can be effectively mediated, some of the negative effects of disaster exposure could be reduced. Although controlling the timing or frequency of disaster stress exposure is more difficult, if stress intensity is relevant, then stress management and support to perinatal maternal care become a viable management options. Chapter Eight and the policymaking recommendations presented later in this chapter provide guidance on how stress might be reduced during and after disasters, and how mothers might be supported post-disaster to give their infants a high-quality perinatal environment.

Because the actual effects of stress on the foetus and, later, on the child are variable, the impact of disaster exposure is perhaps best viewed as a vulnerability model. In this model maternal psychology, objective disaster exposure, post-natal care, foetal sex, and exposure timing are all considered when assessing the potential for deleterious stress effects. Overall, the findings of this dissertation are consistent with prior studies in so far as it finds an association between disaster exposure and health risks to both mother and child.

### *9.2 Effects of fire exposure on birth weight and gestational age*

With regard to the effects of fire exposure on birth weight and gestation age, the findings from this research do not support the hypothesis that exposure to stressful disaster events will reduce birth weight and gestational age.

Data from the Black Saturday fire suggests that severe disaster exposure is not associated with changes in birth weight when other foetal and maternal factors are accounted for. This contradicts other research examining the effects on birth weight of other type of environmental disasters, such as hurricanes, storms and earthquakes on birth weight (Auger et al. 2011; Auger et al. 2014; Dancause et al. 2011; Glynn et al. 2001; Torche and Kleinhaus 2012; Xiong et al. 2008).

Conversely, the ACT analysis shows a pattern of foetal macrosomia associated with disaster exposure. Yet, this pattern only presented in male foetuses. Three previous studies detect foetal macrosomia in response to stress, including the pilot to this dissertation (Margerison-Zilko et al. 2015; O'Donnell and Behie 2013; Oyarzo et al. 2012), although such findings are less common in the literature. Contrary to most previous studies, significant effects in this study occur only in those foetuses exposed late in gestation.

It is likely that maternal stress is the underlying cause for the macrosomia shown in the ACT sample. However, the effects of stress are likely mediated by evolutionary responses that create differing outcomes in response to different environmental conditions. Accordingly, the impact of the Canberra fire on a single community on a single day was different to that of the Black Saturday fire, which affected numerous communities over the course of many days.

In terms of the pattern of macrosomia found in the Canberra fire, this may be caused by the action of maternal stress in elevating blood glucose and thus accelerating foetal growth (O'Donnell and Behie 2015). Such glucose elevations may be subclinical and missed by routine screening. Male foetuses are known to have greater environmental responsivity (Stinson 1985) and create a higher risk of gestational diabetes for the mother (Bahado-Singh et al. 2012; Retnakaran et al. 2015). This sensitivity is demonstrated by the pattern of early loss of male foetuses outlined in Chapter Six. The combined effects of greater sensitivity and heightened susceptibility may have led to the expression of dysregulated foetal growth in males only.

Notably, these effects are only apparent in infants that were exposed later in gestation. As gestation progresses, maternal investment rises and the risk of foetal loss falls accordingly (due to the “sunk costs” of the pregnancy). Where gestation is substantially progressed and transient stress is encountered, responses which reduce maternal investment may not also confer the greatest fitness benefit. If investment is high and irretrievable (as in late pregnancy), mothers experiencing stress may opt for a “double or nothing” strategy in which maternal investment increases to boost the chance of neonatal survival and consequent return on investment (O'Donnell and Behie 2015).

While this strategy may disadvantage a small number of larger neonates by causing them to gain excessive body weight – and therefore have a higher risk of death during birth – for the majority of infants such a strategy would create an unproblematic increase in body weight that might provide a buffer against environmental stress (O'Donnell and Behie 2015). Such a strategy would act to promote the current reproductive event over future reproductive opportunities. It would, therefore, invest more heavily in the quality of the offspring rather than conserving effort to increase offspring quantity. Because the strategy does not conserve energy, it would only appear optimal where environmental stress remains manageable or is understood to be temporary (O'Donnell and Behie 2015).

Alternately, maternal stress may have triggered a decrease in investment, as theorised, which has been countered by male foetuses. When faced with decreasing maternal investment, male foetuses – who are more sensitive to environmental change and require greater maternal investment – may have responded by increasing allocation to placental growth (Coall and Chisholm 2003) in order to facilitate greater access to maternal resources. Larger placental size could result in higher levels of stimulating hormones, such as human placental lactogen (HPL), circulating in the maternal blood stream. Higher levels of HPL increase maternal insulin resistance and, if not countered by increased insulin production, result in higher circulating glucose and, ultimately, gestational diabetes (Haig 1993). Again, for a smaller cohort of already larger babies, this differential investment could result in excessive foetal growth. The effect of maternal stress on the placental-foetal weight ratio is thus an important area for future research.

According to LHT, where stress is extreme and natal survival is precarious, conservation of maternal energy for future investment and greater offspring numbers should become the predominant reproductive strategy. Although decreasing investment in foetal growth creates clear survival risks for the present foetus, as Coall and Chisholm (2003 p.1775) state, "when conditions are sufficiently risky… it can be evolutionarily rational to pay extreme reproductive costs". Although the risk that a viable pregnancy or infant (and the investment that represents) is lost is a high reproductive cost, the "decision" aims to conserve resources for later offspring and, hence, to maximise lifetime reproductive success.

The Black Saturday bushfires were a substantially larger event. Indeed, Chapter Eight finds a higher degree of objective and subjective maternal stress among Victorian survivors. Accordingly, decreases in gestational length and birth weight in the Black Saturday case should be expected. However, the Victorian sample does not show these effects. This is most likely due to the nature of the fire and it disparate effect on multiple communities. Because the Victorian fire affected different areas in different ways and over a long period of time, the effects of stress may have been insufficiently concentrated to be detected using a whole-of-population method. The Canberra fire is unusual in that, as an urban fire, it threatened large concentrations of people on a single day and in a more consistent manner.

Further, Victoria is also more subject to bigger and more frequent bushfires (Australian Emergency Management Institute 2012). This has perhaps resulted in a population which is more prepared for, or more resilient to, bushfire threats. Although it is unlikely that the population has been stable for long enough to show selective effects which have created greater resilience, it is possible that communities in high fire risk areas have become self-selected and contain a high proportion of people who are aware of bushfire risk. The intersections between familiarity with the stressor, threat awareness, appetite for risk, and the effect of those psychological factors on objective and subjective stress is an avenue for further work. Although Chapter Eight suggests that the Victorian participants experience greater objective and subjective stress, it is not possible to extrapolate these findings to the wider population.

Lastly, given the unusual effect in the Canberra sample, it appears that wildfire itself may create stressors which differ to those of other disaster types, perhaps because of the sporadic and geographically unpredictable nature of the damage they cause. Importantly, in any process which alters birth outcomes both the mother and infant are independent actors. Therefore, the foetus may act in its own interests when confronted with changes in maternal investment, either to increase its access to resources or to maximise its use of available resources, such as by developing the thrifty phenotype (Hales and Barker 2001). The role of the foetus in responding to maternal stress is an important area for future research.

## *9.3 Effects of fire on early foetal loss*

Both empirics and theory suggest that inherent male frailty should lead to a reduction in the secondary sex ratio (SSR) where mothers are stressed. Whether this occurs as a result of an altered primary sex ratio (PSR) (Fukuda et al. 1996) or as a result of a "culled cohort" (Catalano and Bruckner 2006) is an area of continued debate.

This research finds a lower SSR in the heavily affected Victorian population in 2009 (the year of the fire). This is consistent with previous studies of stress effects on SSR (Bruckner et al. 2010; Catalano 2003; Catalano and Bruckner 2005; Catalano and Bruckner 2006; Catalano et al. 2005; Catalano et al. 2013; Fukuda et al. 1998; Fukuda et al. 2013; Fukuda et al. 2014; Grech 2015; Grech and Scherb 2015). It is also consistent with biological studies of sex differentials in spontaneous abortions (Bellver et al. 2010; Byrne and Warburton 1987; Del Fabro et al. 2011; Hamamah et al. 1997) and theoretical predictions regarding sex selection (Ahlstrom 2011; Cameron and Dalerum 2009). The decline in SSR was accompanied by non-significant declines in multiple births, which is also indicative of a pattern of increased early loss. These results accord with those of a recent study of the effects of flooding on birth outcomes in Queensland (McFarlane 2015). McFarlane (2015) finds significant reductions to the number of plural births recorded in the most substantially flood-affected regions.

However, the analysis of the Canberra fires does not show a similar disruption. The SSR rose in the most heavily affected area among those exposed during early gestation. Although the SSR fell in the cohort exposed during the third trimester, this also occurred in the least affected area, indicating a non-fire-related cause. While plurality rates appear disrupted after the fire, they show both increases and declines in the most affected area. Again, a fall also occurs in the least affected area, suggesting another cause. Similarly, while rates of multiple births fell in the most affected area following the fire, they also fell in the least affected area. The correlation between SSR decreases and plurality rate decreases indicates the potential existence of a stressor, but the pattern of fire exposure does not support the fire being the principal cause of the observed disruption.

Potentially, there are underlying factors in Canberra's SSR which are affecting the overall trend. Canberra's SSR has risen steadily over recent decades, which is contrary to the rest of Australia and to international trends (Dickson & Parker, 1997; Dodds & Armson, 1997; Grech, Vassallo-Agius, & Savona-Ventura, 2003; Hamilton & Rasmussen, 2010; James, 1998; MacKenzie, Lockridge, & Keith, 2005). Economic conditions are one factor linked to SSR changes. For example, the East German economic collapse is significantly associated with SSR declines (Catalano, 2003), while economic growth has been linked to rising SSRs (Hamilton & Rasmussen, 2010). Other work shows that individuals of high social status and greater financial means have a higher proportion of sons (Cameron & Dalerum, 2009; Grant & Yang, 2003). Similarly, paternal occupation of managerial level or above increases offspring SSR compared to fathers in lower occupational classes (Magnuson, Bodin, & Montgomery, 2007). It is possible that the relative wealth, high education status and highly professionalised work of the Canberra population (as the national capital and centre of the nation's bureaucracy) (Australian Bureau of Statistics 2013), when compared with other Australian states, supports an unusually high and robust SSR. SSRs in such areas constitute a promising area for future research.

It is notable that the two fires should produce such different effects. The differences possibly indicate the presence of threshold effects under which only highly stressful events are sufficient to cause disruptions. As demonstrated by both objective measures of loss and the findings of Chapter Eight, the Canberra fire was a less significant stressor. This points to the existence of a protective threshold effect which prevents early loss in response to more minor stressors. While extreme situations may deserve extreme measures (Coall and Chisholm 2003), it would be highly costly to deploy such measures in response to mild or fleeting stressors. Therefore, an effective evolved mechanism would allow a high degree of responsivity to the magnitude and persistence of the stress experienced in order to moderate the risk of costly over-reactions. The differences between the effects of the two fires on SSR and plurality indicate a mechanism which is sufficiently sensitive to the magnitude of stress in order to avoid such an over-reaction.

Importantly, this dissertation indicates that foetal losses may have occurred in late pregnancy. They would therefore present as stillbirths rather than a spontaneous abortions, although the inaccuracies present in the timing variable mean that this should be interpreted cautiously. While much literature examines the role of early loss via spontaneous abortion (often prior to the pregnancy being clinically recognisable) (Bruckner et al. 2010; Catalano 2003; Catalano and Bruckner 2005; Catalano and Bruckner 2006; Fukuda et al. 1998; Fukuda et al. 2013; Fukuda et al. 2014), less work considers the role of stress, particularly acute stress, in increasing the risk of stillbirth (Stephansson et al. 2001; Wisborg et al. 2008).

Unlike very early pregnancy loss, of which parents may be unaware, stillbirth is a highly traumatic experience that can cause lasting harm to parents and other relatives. As such, understanding and preventing stillbirth is an avenue of research particularly worthy of further attention. Stillbirth is a relatively common cause of infant death: 0.7% of births are still-born compared to the 0.3% of infants who die of Sudden

Unexplained Death in Infancy, including Sudden Infant Death Syndrome. Yet causes of stillbirth are not well understood (Hilder et al. 2014; Linacre 2007). Further, the rates of stillbirths in Australia have plateaued over the past decade (even while neonatal and infant mortality rates have declined), suggesting the existence of important but unrecognised or unresolved risk factors for stillbirth. The role of acute maternal stress as a risk factor for stillbirth therefore represents an important, and potentially urgent, area for future work.

### *9.4 Effects of fire on fertility rate*

In terms of fire and fertility rates, LHT predicts that life threatening events should front-load reproduction and thereby create increases in the birth rate. The data outlined in this dissertation, however, does not support such a hypothesis. Although there was some fluctuation in fertility rate, in the ACT and Victoria, these could be more easily accounted for by alternative explanations, such as changes to government welfare payments. While the absence of an effect may simply reflect the limitations of the method employed, there are also potential theoretical explanations for the patterns observed.

Indeed, while these findings arguably indicate a gap between theory and observed behaviour there are good reasons why this is not the case. Firstly, the effects of stress on reproduction function through the individual perceiving a mortality risk and, hence, a threat to their future reproductive capacity. In both cases, only a few individuals experienced life threatening events compared to the overall size of the population. Therefore, it is possible that an insufficient number of individuals will have encountered direct mortality threats and responded accordingly. Findings from Chapter Eight indicate that around a third of respondents worried that they might die. However, owing to a likely bias in that analysis toward participants who experienced more extreme fire conditions or response, it is likely that less than a third of the exposed populations experienced a mortality threat. Where stressful events occur, but are not perceived to be sufficiently severe or persistent to threaten future reproduction, there is no requirement to accelerate reproduction in order to best resolve the future-present reproduction trade-off.

Secondly, in modern populations sexual intercourse and reproduction are often disconnected. Consequently, fertility rates are not necessarily good indicators of the degree of reproductive behaviour occurring in a population. Unfortunately, accurate population measures of intercourse frequency are rare. Moreover, although broad-scale surveys of sexual behaviour exist, they have not been conducted in these populations during the necessary time period. As such, it is not possible to accurately determine whether an underlying change in reproductive behaviour occurred following the fires. A good avenue for future research will be to examine post-fire marriage rates as a proxy for reproductive behaviour, as has been shown elsewhere (O'Donnell and Behie 2014).

Further, the analysis does support the proposal that reproductive decisions are responsive to general environmental conditions; however, the most influential environment in this research is economic rather than natural. Fertility rates did respond to public policy changes that reduced the resources available to support children. Because this change was not coupled with widespread threats to wellbeing, parents appear to have resolved the quality-quantity trade-off by investing their available resources in fewer children, rather than distributing a shrinking resource pool across more children to enhance survival. Such a response accords well with the predictions of LHT.

## 9.5 Effects of fire exposure on behaviour and development

Although a substantial body of work finds that stressful events *in utero* and in early childhood affect maturation and behaviour (Behie and O'Donnell 2015; Chisholm 1999; Chisholm et al. 2005; Shonkoff 2011; Shonkoff and Garner 2012), this research finds few significant effects associated with early exposure to the Australian Alps fire (the fire complex which includes the Canberra fire). LHT, particularly as applied by Chisholm (1999) and Belsky (2012), suggests that early life stress should lead to front-loaded maturation that manifests in behavioural changes, such as younger age at first intercourse (among a suite of social and biological responses). However, this analysis finds no consistent significant effects on the social, emotional or psychosocial functioning of exposed children when compared to their unexposed peers. Although some significant associations were detected, they appear more likely to be artefacts of small sample size than indications of actual changes in functioning.

In Chapter Seven, the dissertation does find that the body weight of exposed children is lower in early childhood. However, this difference does not persist beyond the first two years of life. Potentially, this finding is an artefact of the small sample size. Even if true, its short duration means that this effect seems unlikely to be a substantial contributor to later life heath or wellbeing. The weight of a child's main carer emerges in this analysis as a powerful and durable influence on the child's weight, with the weights of the child and main caregiver being strongly and positively associated throughout childhood. Although the results also controlled for the influence of the weight of the principal carer, in terms of public policy aimed at reducing the risks of childhood obesity, the weight of the main carer is an important area of focus.

This research also examines the behaviour of primary carers of exposed children, predominantly their mothers, to look for alterations in maternal health behaviours, as found one previous study suggests (Fuller 2013). It finds that mothers exposed to the Canberra fire received significantly fewer prenatal health services, which is contrary to other findings (see Chapter Eight). It also shows a non-significant trend toward lower alcohol consumption, which is contrary to small increases in alcohol consumption found elsewhere (see Chapter Eight). Notably, however, although women sometimes expressed a heightened desire to consume alcohol they also reported not actually increasing their consumption.

Although some work finds an increased risk of psychological disorder in children exposed to disaster *in utero* (Armstrong 2009; Kinney et al. 2008a; Kinney et al. 2008b; Walder et al. 2014), other work presents contradictory findings (Rai et al. 2012). More broadly, the bulk of work on the effects of *in utero* and childhood stress on adult wellbeing assesses the impact of chronic stressors, such as neglect and domestic violence (Chisholm 1999; Chisholm et al. 2005; Kishiyama et al. 2009; Moffitt et al. 1992; Perry 2004; Perry 2009; Shonkoff 2011; Shonkoff and Garner 2012). It is appears that any stress resulting from the Canberra fire may (fortunately) have been too transient to create lasting effects in the exposed population, due to the sensitivity of adaptations under LHT, or may have been fully mitigated by parental care received during and after the fire (Musazzi and Marrocco 2016).

As with the analysis on early foetal loss, these findings indicate that there may be threshold effects which govern the role of stress in pregnancy and early life and its impacts on children's social, psychological, and emotional development. Similar to the loss of pregnancy, accelerated maturation is a strategy that carries physical and social costs. For example, front-loaded maturation should result in earlier reproduction. Early parenting can act to reduce the degree of investment in offspring and can imposes social and economic costs on parents, such as greater difficulty in completing education and gaining employment (Chisholm 1999).

In resolving the "uncertain futures problem", it is vital that any organism is protected from over-reaction to transient environmental fluctuations. The absence of any effect on the maturation of children in this dissertation may indicate that the cohort's assessment of its environment remained consistent in the face of transient fluctuations in maternal stress. This meant that unnecessary and potentially costly adaptations were avoided.

## *9.6 Maternal experience of fire exposure*

What is crucial to the resolution of the uncertain futures problem – and, consequently, the choice of reproductive strategy – is the manner in which an organism assesses and responds to its environment in terms of quality and stability. How people "know" their environment is informed by their own early life, their temperament, and their attitudes to risk. Perception of risk is therefore a complex construction informed by both the person's stable traits (as products of early environment) and current state. As such, perceptions of risk should vary over time and change with circumstance but only within relatively stable parameters. The way in which a woman knows her environment should change with pregnancy, as this constitutes a significant shift in a woman's current state. This is an area in which very little research exists. However, it is important because a better understanding of pregnant women's stress as a result of experiencing natural disasters is crucial to developing effective public policy to reduce or eliminate the damaging effects of stress.

This research demonstrates that pregnant women do experience considerable stress during bushfires, in terms of both objective exposure to stressors and self-reported perceptions of stress. It shows, moreover, that in the two main fires examined – Black Saturday and the Canberra fires –both objective and subjective forms of stress were higher in women who experienced Black Saturday. This likely reflects the more extreme nature of the Black Saturday fire and its greater recency. Women expressed fears common to people exposed to bushfires, such as fear of being killed by the fire or losing property, and well as fears more specific to pregnant women, such as concerns over the effect of stress and air pollution on the foetus or concern over changes in the frequency of foetal movements.

Surprisingly, few women acted in a manner which obviously appeared consistent with a heightened perception of risk to themselves and their foetuses. Few women evacuated "safely" (i.e. well in advance of the fire front, as recommended by authorities) or at all, and some stayed to fight the fires or actively tried to return to the fire front. Arguably, these actions show a revealed preference that does not indicate heightened feelings of self-preservation or protectiveness. However, there is good evidence that maternal cortisol responsivity reduces in later pregnancy (Obel et al. 2005), potentially leading to a dampened stress response and, accordingly, a lesser perception of threat. Work in sheep models also finds that ewes show decreased fear reactions while pregnant, although this may also be due to older average age of pregnant ewes (compared to non-pregnant ewes) as fear reaction also declines with age (Vierin 2002). Declining fear in late pregnancy appears, *prima facie*, to be maladaptive – potentially exposing both mother and foetus to danger. However, there is evidence that maternal production of enzymes which convert cortisol and corticosterone into inactive products (11β-hydroxysteriod dehydrogenase) decreases when mothers experience stress during pregnancy, with the effect of increasing foetal glucocorticoid exposure (Musazzi and Marrocco 2016). This means that maternal stress may still act as a reliable indicator of external stressors despite decreasing maternal responsiveness. As such, it is possible that women in this study would still have experienced perceived stress despite their lower cortisol responsivity during pregnancy.

Potentially, decreased cortisol responsiveness occurs as an accidental consequence of rising oxytocin levels in late pregnancy or as an excessively compensatory mechanism for late pregnancy cortisol rises. It is necessary that steroidal hormones rise in later pregnancy to trigger foetal maturation and, ultimately, parturition. However, if cortisol responsiveness remained stable throughout pregnancy, women may suffer high perceived stress as cortisol rises in late pregnancy. Such heightened stress levels may also impede oxytocin secretion and thus act to increase maternal pain during parturition, decrease maternal bonding, and compromise lactation (Musazzi and Marrocco 2016; National Childbirth Trust 2015; Olff 2012). Declines in cortisol responsivity therefore may act to protect the mother against excessive stress in later pregnancy (and the risks that confers), as well as against extreme pain, poor bonding during and after birth, and delayed or absent onset of lactation. However, an accidental consequence of these endocrine changes may be that fear decreases in late pregnancy, leading to greater risk-taking.

Alternatively, it is also possible to interpret these actions as evidence of a stronger than usual desire to protect the home. Arguably, this could be viewed as "nesting" behaviour, as when a mother's "instincts kicked in" (Carmel, under 20 years, Canberra). Nesting behaviour is common in mammals that bear altricial young, so its presence in human behaviour is also plausible. Nesting in pregnant women is widely recognised anecdotally. For example, the "nesting instinct", usually typified by an unusual desire to clean or organise, is commonly discussed by pregnancy guidebooks and websites. Still, there has been little scholarly investigation of the phenomenon. One study (Anderson and Rutherford 2013) however, creates an index of nesting behaviours and measured their frequency in pregnant and non-pregnant women. It finds that pregnant women show a significant increase in some aspects of nesting behaviour during the first and third trimesters, particularly social selectivity (e.g. novelty aversion). Anderson and Rutherford (2013) suggest that avoiding new people and places may be protective against pathogen exposure in the first trimester and against infanticide and other attacks in the third trimester. If novelty aversion behaviours are common in pregnant women, they might present under fire conditions as a reluctance to evacuate or an increased desire to stay within the home.

Given that this group might be expected to consider themselves more at risk, the low rates of evacuation and relocation represent an unexpected finding. In Victoria, public awareness campaigns since Black Saturday have stressed evacuation as a primary fire response, which may alter this pattern of behaviour in future fires.

Notably, in this study, women reported that evacuating was at least as stressful as remaining at home. Although explicitly safer, evacuation does not appear to be an effective method of reducing maternal stress. A range of factors increase maternal stress during evacuation such as lack of appropriate facilities and separation from partners. Evacuation provisions remain limited in Victoria; therefore, it is likely that the evacuation experience may remain stressful in the future. However, a principal source of stress during concerns access to good information (e.g. about the fire's movement). The recommendations of the 2009 Victorian Bushfires Royal Commission give detailed directions for improvements to fire communication, some of which have now been enacted. As uncertainty and feelings of loss of control are strong predictors of stress, improved information about fire impact and movement should assist in reducing stress by helping people to make proactive decisions.

Age is also a factor. Consistent with work that suggests that young people are less resilient to stressors (den Ouden et al. 2007; Scaramella et al. 2008; Soeteman et al. 2008), objective stress exposure scores rose with maternal age even as subjective stress self-report scores did not. This suggests that older mothers are better able to manage their stress and perceived less stress relative to their exposure to stressful events. For very young mothers who are already at higher risk of poor birth outcomes, lower resilience to stress could compound the effects of stress on natal development. Services that seek to support and reduce stress in pregnant women exposed to extreme events should triage young pregnant women and girls accordingly.

Similar to the study of the Quebec Ice Storm in Canada in 1998 by Cao-Lei et al. (2014), this dissertation investigates the association between objective stress exposure and perceived stress. In contrast to the Cao-Lei et al. (2014) study, however, it finds a stronger relationship with objective stress being moderately and positively correlated with subjective stress. Cao-Lei et al. (2014) concludes that objective rather than subjective maternal stress is the most accurate predictor of epigenetic change in the foetus when measured in affected children at 13 years of age. Further, in later work, Cao-Lei et al. (2015) find that the mother's cognitive appraisal of disaster events as either positive or negative is associated with significant differences between methylation levels of 2872 C-G sites in the same cohort.

In this research, women who reported higher stress levels also reported more frequent attendance at prenatal care, suggesting that they sought and received additional support. However, the provision of information to women remained insufficient, particularly with regard to air quality. A common theme regarding antenatal care is that care should have been more proactively offered. Consistent with theories of social behaviour following disasters (Oliver-Smith 1996), few women appear willing to annex resources for themselves, instead feeling that other recipients are more worthy. Active offers of help could assist in overcoming this reticence. In the Australian policy context, it would be possible to make it a condition of Australian Government Disaster Payment receipt that individuals speak with a disaster assistance office (e.g. via a disaster hotline). This would mean that mothers and pregnant women could be directly and personally encouraged to take up relevant service offers. All the women in this study who received support felt that it had aided their recovery.

In terms of assisting recovery, some women felt that they would have recovered from the fire more readily had they been able to be more involved in broader community recovery activities. Because of the practical restrictions of caring for an infant, women became inadvertently excluded from many such activities. Previous work in this field identifies acts of community service as valuable parts of recovery which can inadvertently exclude women (Cox 1998; Enarson 2012; Hoffman 1998), and this appears to be particularly so for pregnant women or those caring for newborns.

## *9.7 Recommendations for practice*

This dissertation demonstrates that experiencing disaster stress while pregnant can have negative effects on foetal wellbeing, with indications that survival, healthy birth weight, and appropriate gestational age can be compromised. Because of these effects, interventions that focus on supporting pregnant women during and after disasters are of particular importance. Unfortunately, there are few standard measures or practices in the disaster management literature for assisting pregnant women.

Finding and identifying pregnant women is a particular difficulty in post-disaster management, with the challenge being especially acute in terms of those women whose pregnancy is in the first trimester who may not have established contact with their usual health care provider (or, indeed, be aware of their pregnancy). Across all types of preventative care delivery in pregnancy, a major challenge is that the most vulnerable period for foetal development occurs early in pregnancy, before most women attend prenatal service and, in some cases, before the pregnancy is recognised. Because of the risk to women in early pregnancy, warnings currently given (e.g. via the Australian Broadcasting Commission Local Radio services, which act as the emergency broadcaster Australia-wide) for at-risk individuals (older people and children) should be extended to include specific mention of pregnant women and women who believe they may be pregnant.

Horney et al. (2012) trial a cluster referral method for identifying pregnant women in disaster-affected communities. They contact a sample of households in areas where demographic data show that women of reproductive age are present. They then ask each household to refer any pregnant women of their acquaintance. This method doubles the number of contactable pregnant women at two trial sites and increases referrals by around 20 times a third trial site.

Such an approach would likely need to be modified in the Australian setting to manage privacy requirements. For example, requests to pass on contact details to any known pregnant women could be added to the application process for disaster assistance instead or outreach services could be extended through General Practitioners and other antenatal care providers. Alternatively, disaster information packs could be provided as a routine service to women through antenatal services, so that women have access to information prior to a disaster.

Once women are contacted, their degree of risk must be assessed. While there are a range of clinical tools used to score the risk of preterm delivery or low weight infants, none of these tools have been evaluated via clinical trials (Davey et al. 2011). Zotti et al. (2014) develop a set of post-disaster health indicators for pregnant and post-partum women; these now require further testing in disaster settings. Such indicators were developed via professional consultation at the 2011 Maternal and Child Health Epidemiology Conference and the 2012 Association of Maternal and Child Health Programs Conference. The 25 indicators include service access, disaster exposure, existing health problems, and psychosocial issues, such as substance abuse and exposure to domestic violence. Those indicators could be usefully trialled in Australian disaster settings. Because this research demonstrates that younger mothers manage stress less well, they should be prioritised when seeking disaster assistance.

In terms of direct intervention, there is little evidence that customary stress management techniques, such as meditation, massage, yoga, breathing exercises, or visualisation exercises, prevent preterm birth (Khianman et al. 2012). Moreover, there is only limited evidence that these types of interventions reduce stress in pregnant women (Marc et al. 2011). Because of the paucity of evidence, the provision of these services post-disaster should not be a priority.

A major finding of this dissertation is the positive correlation between disaster exposure and stress scores. This suggests that limiting exposure to disaster events by encouraging early evacuation would be useful. However, the dissertation also finds that evacuation is at least as stressful as remaining at home. Therefore, in order to effectively reduce stress, the evacuation experience for pregnant women would have to be substantially improved. Key suggestions for improvement are to provide pregnant women with timely and accurate information about the fire’s path and impact, adequate and comfortable accommodation, and reminders to remain well hydrated.

One aspect of bushfire evacuation which differs from other disaster types relates to the recurrent threat that bushfires can pose over weeks and months. This recurrent threat may, in turn, require multiple evacuations or prolonged relocations. Financial assistance to relocate for longer periods (i.e. to relocate for a week rather than three times within a week) could constitute a useful stress reduction intervention. Importantly, Australian Government Disaster Recovery Payments cannot be received until after an area is declared a disaster zone, meaning that such payments cannot be accessed in the stressful pre-disaster period when towns are recurrently threatened by fire. Consideration should be given to a bushfire threat relocation payment for people at high risk of harm, including pregnant women and women immediately post-partum.

This research also shows how fire affected women link the experience of stress to lack of information. It would be highly beneficial, therefore, if better information on the location and direction of the fire and the impact of attendant environmental conditions (such as heat and smoke) during pregnancy were provided. Improving communication accuracy and frequency is a key focus of the recommendations for the 2009 Victorian Bushfires Royal Commission (Teague et al. 2010d). Improved safety messages could also include sending reminders for self-care activities, such as ensuring adequate hydration, to a range of vulnerable people, including older people, young children, and people with existing health conditions, as well as pregnant women.

Both this research and previous work identify women's inability to take part in community recovery activities as problematic. Community recovery and resilience policy should therefore recommend outreach to pregnant women and new mothers by recovery workers to ensure that they are offered opportunities for practicable involvement, supported by services such as childcare where needed.

Because the deleterious effects of adverse prenatal experience appear, for children, to be moderated by positive post-natal experience (Musazzi and Marrocco 2016), all mothers affected by disasters should be encouraged to regularly access maternal support programs to assist them in maximising the quality of their infant care. Maternal warmth, responsiveness, and reliability appear central to providing a quality post-natal environment. Because women affected by disasters may be particularly at risk of conditions that might compromise their care, such as PND, it is important that they are regularly in contact with health professionals. Accordingly, outreach services should be provided to assist women experiencing logistical or emotional challenges in accessing care. These services could be delivered through existing care networks, such as Maternal and Child Health Centres. During post-disaster recovery, additional resources could be allocated through national disaster recovery funding in order to assist local Maternal and Child Health Centres to implement and maintain outreach services for the duration of the disaster and recovery period (i.e. several years post-disaster).

## *9.8 Future research*

### *Effects of placental growth*

Recent research indicates that epigenetic changes in the placenta relate to the expression of stress-linked alterations to foetal development (Janssen et al. 2016) and that these alterations might be sex-specific (Torche and Kleinhaus 2012). Additionally, these changes act as a record of stress exposure over the length of the pregnancy. In earlier work, Coall and Chisholm (2003) also examine the relationship between placental weight and pregnancy outcomes. While most work to date on the effects of stress in pregnancy focuses on measuring those effects by examining the resulting infant, future work might usefully examine placental effects to develop greater insights into the mechanics of change and potential for therapeutic interventions.

*Effects on stillbirth and miscarriage*

To develop a more accurate investigation into the effects of stress on pregnancy loss, future research might also examine rates of stillbirth and reported miscarriage in disaster-affected populations. By using disasters as a “natural experiment” such analysis would help illuminate the role of maternal stress as a potentially causal element in stillbirth and miscarriage. The identification of additional risk factors for miscarriage and stillbirth could, in turn, help identify women who are at risk of experiencing pregnancy loss or neonatal death.

*Effects on marriage rates*

Because reproductive behaviour may not be well represented by fertility rate, future research could also examine post-disaster marriage rates as a proxy indicator of reproductive intent. Such work might be effectively undertaken in countries where few births occur out of wedlock and disaster frequency is high, such as Japan. Such work would provide empirical information to improve understanding of how theories such as LHT could be applied to modern human populations.

*Causes of rising SSR*

The rising SSR in the ACT found in this dissertation is contrary to national and international trends. Examining potential underlying causes for the local elevation of SSR, including the effects of maternal wealth and education and the effects of echo-booms and other sources of natural variation, offers an additional avenue for future research. As with the examination of the effects of disaster on marriage rates, such work would provide important validation for the application of LHT to modern humans. Similarly, because the significant findings in the Victorian analysis do not account well for underlying variation in the SSR, further investigation of this finding is important to clarify the actual effects in the Victorian population.

*Causes of low birth weight in younger and single mothers*

The analysis of birth weight presented in Chapter 4 affirms patterns consistently found in the literature regarding the depressive effects of younger maternal age and single couple status on birth weight. While these effects are normally considered to be social, behavioural or financial in origin (particularly within the health policy, health promotion and medical literature), relatively little work places these biological patterns within an evolutionary context. However, LHT could be usefully applied to patterns of lowered investment among mothers with reduced resource access. Such a viewpoint may provide novel insights into both the physiology and sociology of young or single mothers, a cohort usually considered vulnerable within the health system.

### *9.9 Final summary*

This dissertation has sought to analyse reproductive responses to environmental challenges in a modern human population. Reproduction is the engine of natural selection. Like all organisms who inhabit a variety of changing habitats, humans need to be able to adapt to their environment. While the macro process of this adaptation takes place over many generations, micro processes can be expressed within generations. The mechanism for these micro adjustments appears to occur substantially in response to *in utero* conditions. A key method of triggering an epigenetic or developmental change is through the effects of the maternal experience of stress on the foetus. For these reasons, the dissertation has examined the effects of the maternal experience of stress on foetal development (including perinatal measures, such as birth weight and sex ratio, along with measures of childhood wellbeing) maternal fertility, as well as the maternal experience of stress itself.

Using maternal stress as a trigger for epigenetic change can be both adaptive and maladaptive – for individuals there are dangers as well as advantages to phenotypic plasticity. While change may prove adaptive in later life, *in utero* conditions are not a perfectly reliable indicator of the conditions the individual will experience in adulthood (the uncertain futures problem). Therefore, some responses to *in utero* conditions are mismatched to adult conditions and result in deleterious outcomes. However, whether outcomes of prenatal stress will be positive, negative, or neutral is difficult to predict. These outcomes appear to be strongly moderated by the timing, intensity and duration of the stressor, as well as by both the mother and the foetuses' genetic predispositions and the parental care the foetus receives after birth.

This dissertation demonstrates that there is sufficient evidence regarding the negative effects of severe and prolonged stress exposure to recommend that stress should be avoided by pregnant women wherever possible. Further, it shows that bushfires constitute an important stressor and may result in problematic immediate effects, such as pregnancy loss, as well as long-run effects such as increases in macrosomia and its attendant health impacts. However, it also finds that deleterious effects may potentially be mediated by moderating exposure severity and duration, as well as by attending to pre-existing maternal risk factors. Accordingly, the dissertation proposes that foetal susceptibility to maternal stress is most accurately understood through a vulnerability model that encompasses the influence of both internal and external factors. Because the negative effects of stress on foetal development may be moderated, there is a pressing need for a stronger public health response supporting women experiencing any severe or prolonged stress during pregnancy. This dissertation finds that, in the case of environmental disasters, pregnant women report insufficient assistance. Because the prenatal period is an extraordinary time of both opportunity and vulnerability, greater support for pregnant women would fulfil a substantial need while delivering immense benefit.

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# CONTENTS OF APPENDICES

# APPENDIX A: ANALYSES FROM CHAPTER FOUR

Table 0-1: Classification of areas in the Victorian analysis

| Heavily affected | Affected | Surrounding | | | |
|---|---|---|---|---|---|
| Alexandra | Alberton | Abbeyard | Ferny Creek | Merriang | Warragul |
| Arthurs Creek | Alberton West | Arapiles | Floral Hill | South | West |
| Buxton | Balook | Barwidgee | Flynns | Miga Lake | Wartook |
| Eildon | Beechworth | Bayles | Creek | Millgrove | West |
| Flowerdale | Boolarra | Bendigo | Gapstead | Mitre | Bendigo |
| Glenburn | Bruarong | Bendigo South | Glenfyne | Mockinya | Whipstick |
| Hazeldene | Bunyip | Benloch | Golden | Morwell | White Hills |
| Healesville | Calrossie | Berwick | Gully | Mount | Wallan |
| Humevale | Churchhill | Beveridge | Golden | Tassie | Womdelano |
| Kinglake | Coleraine | Big Hill | Square | Mudgegonga | Wonnangatta |
| Kinglake Central | Dargo | Blackwarry | Goldie | Murra Warra | Wonwondah |
| Kinglake West | Dederang | Bona Vista | Grand Ridge | Myrtleford | Woodvale |
| Marysville | Devon North | Boronia | Grass Flat | Naroghid | Yannathan |
| Murrindindi | Drouin West | Brandy Creek | Greenhill | Narre | Yarrambat |
| Narbethong | Garfield North | Bravington | Guim | Warren | Yering |
| Reedy Creek | Gelliondale | Buffalo River | Hamilton | Natimuk | Yinnar |
| Rubicon | Gembrook | Bullaharre | Harkaway | Nilma | South |
| St Andrews | Gippsland Mail Centre | Buln Buln | Havilah | Nilma North | Zumsteins |
| Steels Creek | Hazelwood | Buln Buln East | Hazelwood | Noradjuha | |
| Strath Creek | Hiawatha | Byaduk North | Hazelwood | North | |
| Strathewen | Horsham | California | North | Bendigo | |
| Taggerty | Hunterston | Gully | Hazelwood | Nug | |
| Toolangi | Jack River | Callignee | South | Nug | |
| Wandong | Jindivick | Callignee | Heath Hill | Nulla Vale | |
| Whittlesea | Kilmore | North | High Camp | Quarry Hill | |
| Woodstock | Kilmore East | Callignee | Horsham | Rokeby | |
| Yarck | Koornalla | South | Ironbark | Romsey | |
| Yarra Glen | Labertouche | Campbells | Jackass Flat | Rosewhite | |
| Yea | Langsborough | Forest | Myers Flat | Sailors Gully | |
| | Macks Creek | Carrajung | Jancourt | Sandhurst | |
| | Madalya | Carrajung | Jancourt | East | |
| | Maiden Gully | Lower | East | Seaview | |
| | Manns Beach | Carrajung | Jeeralang | Sebastian | |
| | Neerim Junction | South | Jeeralang | Selwyn | |
| | Noojee | Casterton | Junction | Seymour | |
| | Port Albert | Catani | Jilpanger | Shady Creek | |
| | Redesdale | Cherokee | Kangaroo | Simpson | |
| | Robertsons Beach | Chintin | Flat | Spring Gully | |
| | Staceys Bridge | Clear Lake | Kangaroo | Springfield | |
| | Stanley | Cobden | Ground | Strath Creek | |
| | Tarra Valley | Cobrico | Kennington | Strathdale | |
| | Tarraville | Corndale | Kerrie | Tetoora | |
| | Tonimbuk | Crossover | Knoxfield | Road | |
| | Won Wron | Dalmore | Koo Wee | Tooan | |
| | Woodside | Dandongadale | Rup | Torwood | |
| | Yarram | Daraweit | Koo Wee | Traralgon | |
| | | Diamond Creek | Rup North | Traralgon | |
| | | Don Valley | Koornalla | East | |
| | | Douglas | Lancefield | Traralgon | |
| | | Driffield | Lansell | South | |
| | | Duchembegarra | Plaza | Tylers | |
| | | Eaglehawk | Lardner | Tynong | |
| | | Eaglehawk | Lillico | Tynong | |
| | | North | Long Gully | North | |
| | | East Bendigo | Longerenong | Upper Plenty | |
| | | Elingamite | Loy Yang | Upper Plenty | |
| | | Elingamite | Lynn | Upwey | |
| | | North | Maryvale | Warragul | |
| | | Ellinbank | Mernda | Warragul | |
| | | Ferndale | Merriang | South | |

### *ACT analysis*

Table 0-2: Results of unbalanced ANOVA for birth weight for both sexes in 2000 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| Indigenous status | 1 | 936077 | 936077 | 2.83 | 0.093 |
| Marital status | 1 | 4649254 | 4649254 | 14.03 | <.001 |
| Maternal age | 1 | 34205 | 34205 | 0.1 | 0.748 |
| Baby's sex | 1 | 16543797 | 16543797 | 49.93 | <.001 |
| Plurality of birth | 1 | 154880773 | 154880773 | 467.44 | <.001 |
| Previous pregnancy status | 1 | 4025899 | 4025899 | 12.15 | <.001 |
| Smoking during pregnancy | 1 | 37077333 | 37077333 | 111.9 | <.001 |
| Fire exposed | 2 | 278663 | 139331 | 0.42 | 0.657 |
| Residual | 3942 | 1306135562 | 331338 | | |
| Total | 3951 | 1524561563 | 385867 | | |

Table 0-3: Results of unbalanced ANOVA for birth weight for both sexes in 2001 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 3801871 | 3801871 | 11.99 | <.001 |
| Marital status | 1 | 11581755 | 11581755 | 36.53 | <.001 |
| Maternal age | 1 | 89610 | 89610 | 0.28 | 0.595 |
| Baby's sex | 1 | 12449626 | 12449626 | 39.27 | <.001 |
| Plurality of birth | 1 | 159384933 | 159384933 | 502.69 | <.001 |
| Previous pregnancy status | 1 | 6909414 | 6909414 | 21.79 | <.001 |
| Smoking during pregnancy | 1 | 22379559 | 22379559 | 70.58 | <.001 |
| Fire exposed | 2 | 541346 | 270673 | 0.85 | 0.426 |
| Residual | 3733 | 1183591204 | 317062 | | |
| Total | 3742 | 1400729316. | 374326. | | |

Table 0-4: Results of unbalanced ANOVA for birth weight for both sexes in 2002 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 853595 | 853595 | 2.58 | 0.108 |
| Marital status | 1 | 9469718 | 9469718 | 28.61 | <.001 |
| Maternal age | 1 | 1326745 | 1326745 | 4.01 | 0.045 |
| Baby's sex | 1 | 14439119 | 14439119 | 43.63 | <.001 |
| Plurality of birth | 1 | 122706053 | 122706053 | 370.78 | <.001 |
| Previous pregnancy status | 1 | 12729399 | 12729399 | 38.46 | <.001 |
| Smoking during pregnancy | 1 | 24414052 | 24414052 | 73.77 | <.001 |
| Fire exposed | 2 | 719145 | 359572 | 1.09 | 0.337 |
| Residual | 3904 | 1291986560 | 330939 | | |
| Total | 3913 | 1478644387. | 377880. | | |

Table 0-5: Results of unbalanced ANOVA for birth weight for both sexes in 2003 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 1004451 | 1004451 | 3.01 | 0.083 |
| Marital status | 1 | 4674449 | 4674449 | 14 | <.001 |
| Maternal age | 1 | 1311309 | 1311309 | 3.93 | 0.048 |
| Baby's sex | 1 | 13985712 | 13985712 | 41.9 | <.001 |
| Plurality of birth | 1 | 131566496 | 131566496 | 394.13 | <.001 |
| Previous pregnancy status | 1 | 7592171 | 7592171 | 22.74 | <.001 |
| Smoking during pregnancy | 1 | 37600669 | 37600669 | 112.64 | <.001 |
| Fire exposed | 2 | 2919433 | 1459716 | 4.37 | 0.013 |
| Residual | 4073 | 1359616518 | 333812 | | |
| Total | 4082 | 1560271209. | 382232. | | |

Table 0-6: Results of unbalanced ANOVA for birth weight for both sexes in 2004 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 9346234 | 9346234 | 27.92 | <.001 |
| Marital status | 1 | 11047073 | 11047073 | 33.01 | <.001 |
| Maternal age | 1 | 38998 | 38998 | 0.12 | 0.733 |
| Baby's sex | 1 | 10967830 | 10967830 | 32.77 | <.001 |
| Plurality of birth | 1 | 211219399 | 211219399 | 631.07 | <.001 |
| Previous pregnancy status | 1 | 11585980 | 11585980 | 34.62 | <.001 |
| Smoking during pregnancy | 1 | 30515430 | 30515430 | 91.17 | <.001 |
| Fire exposed | 2 | 832083 | 416042 | 1.24 | 0.289 |
| Residual | 4046 | 1354200109 | 334701 | | |
| Total | 4055 | 1639753137. | 404378 | | |

Table 0-7: Results of unbalanced ANOVA for birth weight for both sexes in 2005 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 3708092 | 3708092 | 10.5 | 0.001 |
| Marital status | 1 | 10121240 | 10121240 | 28.67 | <.001 |
| Maternal age | 1 | 38045 | 38045 | 0.11 | 0.743 |
| Baby's sex | 1 | 13863229 | 13863229 | 39.27 | <.001 |
| Plurality of birth | 1 | 123531404 | 123531404 | 349.93 | <.001 |
| Previous pregnancy status | 1 | 6053820 | 6053820 | 17.15 | <.001 |
| Smoking during pregnancy | 1 | 33292576 | 33292576 | 94.31 | <.001 |
| Fire exposed | 2 | 1261778 | 630889 | 1.79 | 0.168 |
| Residual | 4264 | 1505264268 | 353017 | | |
| Total | 4273 | 1697134453. | 397176. | | |

Table 0-8: Results of unbalanced ANOVA for birth weight for both sexes in 2006 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 7047685 | 7047685 | 22.54 | <.001 |
| Marital status | 1 | 3715010 | 3715010 | 11.88 | <.001 |
| Maternal age | 1 | 252655 | 252655 | 0.81 | 0.369 |
| Baby's sex | 1 | 13519046 | 13519046 | 43.24 | <.001 |
| Plurality of birth | 1 | 171467333 | 171467333 | 548.47 | <.001 |
| Previous pregnancy status | 1 | 7492262 | 7492262 | 23.97 | <.001 |
| Smoking during pregnancy | 1 | 35378288 | 35378288 | 113.16 | <.001 |
| Fire exposed | 2 | 230620 | 115310 | 0.37 | 0.692 |
| Residual | 4542 | 1419965143 | 312630 | | |
| Total | 4551 | 1659068043. | 364550 | | |

Table 0-9: Results of unbalanced ANOVA for birth weight for both sexes in 2007 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 2056554 | 2056554 | 6.19 | 0.013 |
| Marital status | 1 | 4089304 | 4089304 | 12.31 | <.001 |
| Maternal age | 1 | 196844 | 196844 | 0.59 | 0.441 |
| Baby's sex | 1 | 12437463 | 12437463 | 37.45 | <.001 |
| Plurality of birth | 1 | 126623133 | 126623133 | 381.32 | <.001 |
| Previous pregnancy status | 1 | 5350054 | 5350054 | 16.11 | <.001 |
| Smoking during pregnancy | 1 | 24974280 | 24974280 | 75.21 | <.001 |
| Fire exposed | 2 | 601855 | 300928 | 0.91 | 0.404 |
| Residual | 4596 | 1526173397 | 332066 | | |
| Total | 4605 | 1702502885. | 369707 | | |

Table 0-10: Results of unbalanced ANOVA for birth weight for both sexes in 2008 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 3051096 | 3051096 | 9.52 | 0.002 |
| Marital status | 1 | 1691627 | 1691627 | 5.28 | 0.022 |
| Maternal age | 1 | 2639431 | 2639431 | 8.24 | 0.004 |
| Baby's sex | 1 | 8363259 | 8363259 | 26.11 | <.001 |
| Plurality of birth | 1 | 150326002 | 150326002 | 469.27 | <.001 |
| Previous pregnancy status | 1 | 1481809 | 1481809 | 4.63 | 0.032 |
| Smoking during pregnancy | 1 | 16765148 | 16765148 | 52.34 | <.001 |
| Fire exposed | 2 | 4553704 | 2276852 | 7.11 | <.001 |
| Residual | 4722 | 1512633248 | 320337 | | |
| Total | 4731 | 1701505323. | 359650. | | |

Table 0-11: Results of unbalanced ANOVA for birth weight for both sexes in 2009 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 7512231 | 7512231 | 23.91 | <.001 |
| Marital status | 1 | 8185764 | 8185764 | 26.05 | <.001 |
| Maternal age | 1 | 122496 | 122496 | 0.39 | 0.532 |
| Baby's sex | 1 | 12603426 | 12603426 | 40.11 | <.001 |
| Plurality of birth | 1 | 136185282 | 136185282 | 433.43 | <.001 |
| Previous pregnancy status | 1 | 6285047 | 6285047 | 20 | <.001 |
| Smoking during pregnancy | 1 | 23371547 | 23371547 | 74.38 | <.001 |
| Fire exposed | 2 | 2781080 | 1390540 | 4.43 | 0.012 |
| Residual | 4807 | 1510394258 | 314207 | | |
| Total | 4816 | 1707441131. | 354535 | | |

Table 0-12: Results of unbalanced ANOVA for birth weight for both sexes in 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 1262155 | 1262155 | 3.88 | 0.049 |
| Marital status | 1 | 4082627 | 4082627 | 12.56 | <.001 |
| Maternal age | 1 | 456864 | 456864 | 1.41 | 0.236 |
| Baby's sex | 1 | 14083374 | 14083374 | 43.33 | <.001 |
| Plurality of birth | 1 | 125251618 | 125251618 | 385.33 | <.001 |
| Previous pregnancy status | 1 | 5283713 | 5283713 | 16.26 | <.001 |
| Smoking during pregnancy | 1 | 13834563 | 13834563 | 42.56 | <.001 |
| Fire exposed | 2 | 2260389 | 1130195 | 3.48 | 0.031 |
| Residual | 4942 | 1606382717 | 325047 | | |
| Total | 4951 | 1772898020 | 358089 | | |

Table 0-13: Results of unbalanced ANOVA for gestational age for both sexes in 2000 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.104 | 0.104 | 0.02 | 0.879 |
| Marital status | 1 | 2.711 | 2.711 | 0.6 | 0.439 |
| Maternal age | 1 | 66.027 | 66.027 | 14.6 | <.001 |
| Baby's sex | 1 | 0.972 | 0.972 | 0.22 | 0.643 |
| Plurality of birth | 1 | 2175.018 | 2175.018 | 480.98 | <.001 |
| Previous pregnancy status | 1 | 21.351 | 21.351 | 4.72 | 0.03 |
| Smoking during pregnancy | 1 | 71.04 | 71.04 | 15.71 | <.001 |
| Fire exposed | 2 | 6.691 | 3.345 | 0.74 | 0.477 |
| Residual | 3941 | 17821.451 | 4.522 | | |
| Total | 3950 | 20165.367 | 5.105 | | |

Table 0-14: Results of unbalanced ANOVA for gestational age for both sexes in 2001 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 141.068 | 141.068 | 32.18 | <.001 |
| Marital status | 1 | 10.41 | 10.41 | 2.37 | 0.123 |
| Maternal age | 1 | 95.676 | 95.676 | 21.82 | <.001 |
| Baby's sex | 1 | 9.109 | 9.109 | 2.08 | 0.15 |
| Plurality of birth | 1 | 2344.734 | 2344.734 | 534.83 | <.001 |
| Previous pregnancy status | 1 | 0.993 | 0.993 | 0.23 | 0.634 |
| Smoking during pregnancy | 1 | 22.999 | 22.999 | 5.25 | 0.022 |
| Fire exposed | 2 | 6.922 | 3.461 | 0.79 | 0.454 |
| Residual | 3735 | 16374.641 | 4.384 | | |
| Total | 3744 | 19006.551 | 5.077 | | |

Table 0-15: Results of unbalanced ANOVA for gestational age for both sexes in 2002 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 1.708 | 1.708 | 0.39 | 0.532 |
| Marital status | 1 | 4.679 | 4.679 | 1.07 | 0.301 |
| Maternal age | 1 | 0.92 | 0.92 | 0.21 | 0.647 |
| Baby's sex | 1 | 1.055 | 1.055 | 0.24 | 0.623 |
| Plurality of birth | 1 | 1759.241 | 1759.241 | 402.35 | <.001 |
| Previous pregnancy status | 1 | 0.048 | 0.048 | 0.01 | 0.917 |
| Smoking during pregnancy | 1 | 23.22 | 23.22 | 5.31 | 0.021 |
| Fire exposed | 2 | 4.284 | 2.142 | 0.49 | 0.613 |
| Residual | 3904 | 17070.004 | 4.372 | | |
| Total | 3913 | 18865.159 | 4.821 | | |

Table 0-16: Results of unbalanced ANOVA for gestational age for both sexes in 2003 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 2.776 | 2.776 | 0.57 | 0.449 |
| Marital status | 1 | 1.013 | 1.013 | 0.21 | 0.648 |
| Maternal age | 1 | 33.565 | 33.565 | 6.92 | 0.009 |
| Baby's sex | 1 | 5.107 | 5.107 | 1.05 | 0.305 |
| Plurality of birth | 1 | 1623.845 | 1623.845 | 334.8 | <.001 |
| Previous pregnancy status | 1 | 0.095 | 0.095 | 0.02 | 0.889 |
| Smoking during pregnancy | 1 | 97.166 | 97.166 | 20.03 | <.001 |
| Fire exposed | 2 | 14.227 | 7.114 | 1.47 | 0.231 |
| Residual | 4073 | 19755.003 | 4.85 | | |
| Total | 4082 | 21532.798 | 5.275 | | |

Table 0-17: Results of unbalanced ANOVA for gestational age for both sexes in 2004 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 113.128 | 113.128 | 25.81 | <.001 |
| Marital status | 1 | 8.929 | 8.929 | 2.04 | 0.154 |
| Maternal age | 1 | 89.203 | 89.203 | 20.35 | <.001 |
| Baby's sex | 1 | 9.986 | 9.986 | 2.28 | 0.131 |
| Plurality of birth | 1 | 2816.679 | 2816.679 | 642.51 | <.001 |
| Previous pregnancy status | 1 | 1.879 | 1.879 | 0.43 | 0.513 |
| Smoking during pregnancy | 1 | 62.536 | 62.536 | 14.26 | <.001 |
| Fire exposed | 2 | 0.837 | 0.419 | 0.1 | 0.909 |
| Residual | 4045 | 17732.826 | 4.384 | | |
| Total | 4054 | 20836.002 | 5.140 | | |

Table 0-18: Results of unbalanced ANOVA for gestational age for both sexes in 2005 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 21.274 | 21.274 | 4.5 | 0.034 |
| Marital status | 1 | 35.357 | 35.357 | 7.48 | 0.006 |
| Maternal age | 1 | 57.852 | 57.852 | 12.24 | <.001 |
| Baby's sex | 1 | 8.768 | 8.768 | 1.85 | 0.173 |
| Plurality of birth | 1 | 1393.969 | 1393.969 | 294.91 | <.001 |
| Previous pregnancy status | 1 | 14.576 | 14.576 | 3.08 | 0.079 |
| Smoking during pregnancy | 1 | 62.862 | 62.862 | 13.3 | <.001 |
| Fire exposed | 2 | 3.277 | 1.638 | 0.35 | 0.707 |
| Residual | 4264 | 20155.025 | 4.727 | | |
| Total | 4273 | 21752.959 | 5.091 | | |

Table 0-19: Results of unbalanced ANOVA for gestational age for both sexes in 2006 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 60.335 | 60.335 | 12.92 | <.001 |
| Marital status | 1 | 0.069 | 0.069 | 0.01 | 0.903 |
| Maternal age | 1 | 34.49 | 34.49 | 7.38 | 0.007 |
| Baby's sex | 1 | 25.814 | 25.814 | 5.53 | 0.019 |
| Plurality of birth | 1 | 2448.619 | 2448.619 | 524.15 | <.001 |
| Previous pregnancy status | 1 | 17.083 | 17.083 | 3.66 | 0.056 |
| Smoking during pregnancy | 1 | 41.59 | 41.59 | 8.9 | 0.003 |
| Fire exposed | 2 | 10.094 | 5.047 | 1.08 | 0.34 |
| Residual | 4542 | 21218.415 | 4.672 | | |
| Total | 4551 | 23856.508 | 5.242 | | |

Table 0-20: Results of unbalanced ANOVA for gestational age for both sexes in 2007 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 19.313 | 19.313 | 4.3 | 0.038 |
| Marital status | 1 | 3.392 | 3.392 | 0.76 | 0.385 |
| Maternal age | 1 | 113.547 | 113.547 | 25.31 | <.001 |
| Baby's sex | 1 | 14.549 | 14.549 | 3.24 | 0.072 |
| Plurality of birth | 1 | 1574.97 | 1574.97 | 351.05 | <.001 |
| Previous pregnancy status | 1 | 13.76 | 13.76 | 3.07 | 0.08 |
| Smoking during pregnancy | 1 | 26.225 | 26.225 | 5.85 | 0.016 |
| Fire exposed | 2 | 0.058 | 0.029 | 0.01 | 0.994 |
| Residual | 4593 | 20606.186 | 4.486 | | |
| Total | 4602 | 22371.999 | 4.861 | | |

Table 0-21: Results of unbalanced ANOVA for gestational age for both sexes in 2008 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 30.692 | 30.692 | 6.7 | 0.01 |
| Marital status | 1 | 0.408 | 0.408 | 0.09 | 0.765 |
| Maternal age | 1 | 8.615 | 8.615 | 1.88 | 0.17 |
| Baby's sex | 1 | 55.432 | 55.432 | 12.11 | <.001 |
| Plurality of birth | 1 | 1988.845 | 1988.845 | 434.33 | <.001 |
| Previous pregnancy status | 1 | 19.415 | 19.415 | 4.24 | 0.04 |
| Smoking during pregnancy | 1 | 62.841 | 62.841 | 13.72 | <.001 |
| Fire exposed | 2 | 16.48 | 8.24 | 1.8 | 0.166 |
| Residual | 4722 | 21622.767 | 4.579 | | |
| Total | 4731 | 23805.495 | 5.032 | | |

Table 0-22: Results of unbalanced ANOVA for gestational age for both sexes in 2009 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 65.755 | 65.755 | 15.81 | <.001 |
| Marital status | 1 | 7.904 | 7.904 | 1.9 | 0.168 |
| Maternal age | 1 | 79.742 | 79.742 | 19.18 | <.001 |
| Baby's sex | 1 | 20.057 | 20.057 | 4.82 | 0.028 |
| Plurality of birth | 1 | 1842.552 | 1842.552 | 443.14 | <.001 |
| Previous pregnancy status | 1 | 13.357 | 13.357 | 3.21 | 0.073 |
| Smoking during pregnancy | 1 | 23.679 | 23.679 | 5.69 | 0.017 |
| Fire exposed | 2 | 9.002 | 4.501 | 1.08 | 0.339 |
| Residual | 4807 | 19987.455 | 4.158 | | |
| Total | 4816 | 22049.503 | 4.578 | | |

Table 0-23: Results of unbalanced ANOVA for gestational age for both sexes in 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 15.356 | 15.356 | 3.34 | 0.068 |
| Marital status | 1 | 0.602 | 0.602 | 0.13 | 0.717 |
| Maternal age | 1 | 19.804 | 19.804 | 4.31 | 0.038 |
| Baby's sex | 1 | 19.586 | 19.586 | 4.26 | 0.039 |
| Plurality of birth | 1 | 1822.295 | 1822.295 | 396.58 | <.001 |
| Previous pregnancy status | 1 | 15.633 | 15.633 | 3.4 | 0.065 |
| Smoking during pregnancy | 1 | 6.434 | 6.434 | 1.4 | 0.237 |
| Fire exposed | 2 | 3.374 | 1.687 | 0.37 | 0.693 |
| Residual | 4942 | 22708.877 | 4.595 | | |
| Total | 4951 | 24611.96 | 4.971 | | |

Table 0-24: Results of unbalanced ANOVA for birth weight for males in 2000 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 284813 | 284813 | 0.85 | 0.357 |
| Marital status | 1 | 1736347 | 1736347 | 5.17 | 0.023 |
| Maternal age | 1 | 8532 | 8532 | 0.03 | 0.873 |
| Plurality of birth | 1 | 87649241 | 87649241 | 261.2 | <.001 |
| Previous pregnancy status | 1 | 2292968 | 2292968 | 6.83 | 0.009 |
| Smoking during pregnancy | 1 | 29144400 | 29144400 | 86.85 | <.001 |
| Fire exposed | 2 | 1539800 | 769900 | 2.29 | 0.101 |
| Residual | 2021 | 6.78E+08 | 335563 | | |
| Total | 2029 | 8.01E+08 | 394691 | | |

Table 0-25: Results of unbalanced ANOVA for birth weight for males in 2001 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 7308177 | 7308177 | 21.59 | <.001 |
| Marital status | 1 | 3752944 | 3752944 | 11.09 | <.001 |
| Maternal age | 1 | 390119 | 390119 | 1.15 | 0.283 |
| Plurality of birth | 1 | 70668416 | 70668416 | 208.77 | <.001 |
| Previous pregnancy status | 1 | 3014370 | 3014370 | 8.91 | 0.003 |
| Smoking during pregnancy | 1 | 7082449 | 7082449 | 20.92 | <.001 |
| Fire exposed | 2 | 549294 | 274647 | 0.81 | 0.444 |
| Residual | 1908 | 645847264 | 338494 | | |
| Total | 1916 | 738613033 | 385497 | | |

Table 0-26: Results of unbalanced ANOVA for birth weight for males in 2002 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 120185 | 120185 | 0.34 | 0.559 |
| Marital status | 1 | 5002994 | 5002994 | 14.19 | <.001 |
| Maternal age | 1 | 349594 | 349594 | 0.99 | 0.319 |
| Plurality of birth | 1 | 51293085 | 51293085 | 145.48 | <.001 |
| Previous pregnancy status | 1 | 9690814 | 9690814 | 27.48 | <.001 |
| Smoking during pregnancy | 1 | 13696943 | 13696943 | 38.85 | <.001 |
| Fire exposed | 2 | 529207 | 264604 | 0.75 | 0.472 |
| Residual | 1993 | 702711509 | 352590 | | |
| Total | 2001 | 783394332 | 391501 | | |

Table 0-27: Results of unbalanced ANOVA for birth weight for males in 2003 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 25425 | 25425 | 0.07 | 0.788 |
| Marital status | 1 | 2631505 | 2631505 | 7.47 | 0.006 |
| Maternal age | 1 | 2222229 | 2222229 | 6.3 | 0.012 |
| Plurality of birth | 1 | 54168980 | 54168980 | 153.68 | <0.001 |
| Previous pregnancy status | 1 | 5277697 | 5277697 | 14.97 | <0.001 |
| Smoking during pregnancy | 1 | 17536545 | 17536545 | 49.75 | <0.001 |
| Fire exposed | 2 | 4039079 | 2019539 | 5.73 | 0.003 |
| Residual | 2108 | 743021600 | 352477 | | |
| Total | 2116 | 828923060 | 391741 | | |

Table 0-28: Results of unbalanced ANOVA for birth weight for males in 2004 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 6284482 | 6284482 | 16.91 | <.001 |
| Marital status | 1 | 7861928 | 7861928 | 21.15 | <.001 |
| Maternal age | 1 | 97893 | 97893 | 0.26 | 0.608 |
| Plurality of birth | 1 | 110349255 | 110349255 | 296.84 | <.001 |
| Previous pregnancy status | 1 | 4427068 | 4427068 | 11.91 | <.001 |
| Smoking during pregnancy | 1 | 21786726 | 21786726 | 58.61 | <.001 |
| Fire exposed | 2 | 332197 | 166099 | 0.45 | 0.64 |
| Residual | 2098 | 779925735 | 371747 | | |
| Total | 2106 | 931065284 | 442101 | | |

Table 0-29: Results of unbalanced ANOVA for birth weight for males in 2005 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 2359238 | 2359238 | 6.14 | 0.013 |
| Marital status | 1 | 5141033 | 5141033 | 13.38 | <.001 |
| Maternal age | 1 | 62097 | 62097 | 0.16 | 0.688 |
| Plurality of birth | 1 | 76614204 | 76614204 | 199.42 | <.001 |
| Previous pregnancy status | 1 | 4730226 | 4730226 | 12.31 | <.001 |
| Smoking during pregnancy | 1 | 15068444 | 15068444 | 39.22 | <.001 |
| Fire exposed | 2 | 2350535 | 1175268 | 3.06 | 0.047 |
| Residual | 2144 | 823692541 | 384185 | | |
| Total | 2152 | 930018318 | 432165 | | |

Table 0-30: Results of unbalanced ANOVA for birth weight for males in 2006 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 4743723 | 4743723 | 13.6 | <.001 |
| Marital status | 1 | 1569858 | 1569858 | 4.5 | 0.034 |
| Maternal age | 1 | 95341 | 95341 | 0.27 | 0.601 |
| Plurality of birth | 1 | 75935668 | 75935668 | 217.66 | <.001 |
| Previous pregnancy status | 1 | 5871424 | 5871424 | 16.83 | <.001 |
| Smoking during pregnancy | 1 | 13583901 | 13583901 | 38.94 | <.001 |
| Fire exposed | 2 | 325331 | 162666 | 0.47 | 0.627 |
| Residual | 2350 | 819845351 | 348870 | | |
| Total | 2358 | 921970597 | 390997 | | |

Table 0-31: Results of unbalanced ANOVA for birth weight for males in 2007 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 2620407 | 2620407 | 7.33 | 0.007 |
| Marital status | 1 | 198529 | 198529 | 0.56 | 0.456 |
| Maternal age | 1 | 1043839 | 1043839 | 2.92 | 0.088 |
| Plurality of birth | 1 | 51234983 | 51234983 | 143.36 | <.001 |
| Previous pregnancy status | 1 | 4054925 | 4054925 | 11.35 | <.001 |
| Smoking during pregnancy | 1 | 14571171 | 14571171 | 40.77 | <.001 |
| Fire exposed | 2 | 667972 | 333986 | 0.93 | 0.393 |
| Residual | 2340 | 836300356 | 357393 | | |
| Total | 2348 | 910692182 | 387859 | | |

Table 0-32: Results of unbalanced ANOVA for birth weight for males in 2008 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 1314429 | 1314429 | 3.86 | 0.05 |
| Marital status | 1 | 1516824 | 1516824 | 4.45 | 0.035 |
| Maternal age | 1 | 1826762 | 1826762 | 5.36 | 0.021 |
| Plurality of birth | 1 | 87536123 | 87536123 | 256.87 | <.001 |
| Previous pregnancy status | 1 | 1803119 | 1803119 | 5.29 | 0.022 |
| Smoking during pregnancy | 1 | 18024859 | 18024859 | 52.89 | <.001 |
| Fire exposed | 2 | 2176271 | 1088135 | 3.19 | 0.041 |
| Residual | 2456 | 836940267 | 340774 | | |
| Total | 2464 | 951138654 | 386014 | | |

Table 0-33: Results of unbalanced ANOVA for birth weight for males in 2009 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 4174960 | 4174960 | 12.5 | <.001 |
| Marital status | 1 | 9083269 | 9083269 | 27.19 | <.001 |
| Maternal age | 1 | 491372 | 491372 | 1.47 | 0.225 |
| Plurality of birth | 1 | 62643794 | 62643794 | 187.53 | <.001 |
| Previous pregnancy status | 1 | 3308364 | 3308364 | 9.9 | 0.002 |
| Smoking during pregnancy | 1 | 14610836 | 14610836 | 43.74 | <.001 |
| Fire exposed | 2 | 1237343 | 618671 | 1.85 | 0.157 |
| Residual | 2566 | 857178149 | 334052 | | |
| Total | 2574 | 952728086 | 370135 | | |

Table 0-34: Results of unbalanced ANOVA for birth weight for males in 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 2072607 | 2072607 | 5.89 | 0.015 |
| Marital status | 1 | 2252702 | 2252702 | 6.4 | 0.011 |
| Maternal age | 1 | 61784 | 61784 | 0.18 | 0.675 |
| Plurality of birth | 1 | 68255495 | 68255495 | 193.97 | <.001 |
| Previous pregnancy status | 1 | 2739207 | 2739207 | 7.78 | 0.005 |
| Smoking during pregnancy | 1 | 2974090 | 2974090 | 8.45 | 0.004 |
| Fire exposed | 2 | 2479638 | 1239819 | 3.52 | 0.03 |
| Residual | 2517 | 885693833 | 351885 | | |
| Total | 2525 | 966529355 | 382784 | | |

Table 0-35: Results of unbalanced ANOVA for gestational age for males in 2000 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.197 | 0.197 | 0.04 | 0.836 |
| Marital status | 1 | 5.829 | 5.829 | 1.27 | 0.26 |
| Maternal age | 1 | 58.088 | 58.088 | 12.66 | <.001 |
| Plurality of birth | 1 | 1093.748 | 1093.748 | 238.43 | <.001 |
| Previous pregnancy status | 1 | 19.744 | 19.744 | 4.3 | 0.038 |
| Smoking during pregnancy | 1 | 71.695 | 71.695 | 15.63 | <.001 |
| Fire exposed | 2 | 3.336 | 1.668 | 0.36 | 0.695 |
| Residual | 2020 | 9266.146 | 4.587 | | |
| Total | 2028 | 10518.78 | 5.187 | | |

Table 0-36: Results of unbalanced ANOVA for gestational age for males in 2001 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 213.594 | 213.594 | 46.61 | <.001 |
| Marital status | 1 | 0.537 | 0.537 | 0.12 | 0.732 |
| Maternal age | 1 | 50.603 | 50.603 | 11.04 | <.001 |
| Plurality of birth | 1 | 994.547 | 994.547 | 217.05 | <.001 |
| Previous pregnancy status | 1 | 0.782 | 0.782 | 0.17 | 0.68 |
| Smoking during pregnancy | 1 | 3.915 | 3.915 | 0.85 | 0.355 |
| Fire exposed | 2 | 5.341 | 2.671 | 0.58 | 0.558 |
| Residual | 1909 | 8747.257 | 4.582 | | |
| Total | 1917 | 10016.576 | 5.225 | | |

Table 0-37: Results of unbalanced ANOVA for gestational age for males in 2002 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 16.026 | 16.026 | 3.08 | 0.079 |
| Marital status | 1 | 0.425 | 0.425 | 0.08 | 0.775 |
| Maternal age | 1 | 16.28 | 16.28 | 3.13 | 0.077 |
| Plurality of birth | 1 | 958.829 | 958.829 | 184.34 | <.001 |
| Previous pregnancy status | 1 | 9.667 | 9.667 | 1.86 | 0.173 |
| Smoking during pregnancy | 1 | 0.315 | 0.315 | 0.06 | 0.806 |
| Fire exposed | 2 | 24.258 | 12.129 | 2.33 | 0.097 |
| Residual | 2517 | 13092.172 | 5.201 | | |
| Total | 2525 | 14117.973 | 5.591 | | |

Table 0-38: Results of unbalanced ANOVA for gestational age for males in 2003 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.065 | 0.065 | 0.01 | 0.911 |
| Marital status | 1 | 1.349 | 1.349 | 0.26 | 0.612 |
| Maternal age | 1 | 0.512 | 0.512 | 0.1 | 0.755 |
| Plurality of birth | 1 | 573.076 | 573.076 | 109.38 | <.001 |
| Previous pregnancy status | 1 | 0.011 | 0.011 | 0 | 0.963 |
| Smoking during pregnancy | 1 | 72.382 | 72.382 | 13.81 | <.001 |
| Fire exposed | 2 | 22.458 | 11.229 | 2.14 | 0.118 |
| Residual | 2108 | 11044.897 | 5.24 | | |
| Total | 2116 | 11714.75 | 5.536 | | |

Table 0-39: Results of unbalanced ANOVA for gestational age for males in 2004 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 64.288 | 64.288 | 12.92 | <.001 |
| Marital status | 1 | 7.453 | 7.453 | 1.5 | 0.221 |
| Maternal age | 1 | 73.709 | 73.709 | 14.81 | <.001 |
| Plurality of birth | 1 | 1354.086 | 1354.086 | 272.13 | <.001 |
| Previous pregnancy status | 1 | 1.191 | 1.191 | 0.24 | 0.625 |
| Smoking during pregnancy | 1 | 54.078 | 54.078 | 10.87 | <.001 |
| Fire exposed | 2 | 4.6 | 2.3 | 0.46 | 0.63 |
| Residual | 2098 | 10439.339 | 4.976 | | |
| Total | 2106 | 11998.744 | 5.697 | | |

Table 0-40: Results of unbalanced ANOVA for gestational age for males in 2005 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 6.857 | 6.857 | 1.38 | 0.241 |
| Marital status | 1 | 30.758 | 30.758 | 6.18 | 0.013 |
| Maternal age | 1 | 27.928 | 27.928 | 5.61 | 0.018 |
| Plurality of birth | 1 | 788.505 | 788.505 | 158.36 | <.001 |
| Previous pregnancy status | 1 | 4.109 | 4.109 | 0.83 | 0.364 |
| Smoking during pregnancy | 1 | 56.89 | 56.89 | 11.43 | <.001 |
| Fire exposed | 2 | 3.556 | 1.778 | 0.36 | 0.7 |
| Residual | 2144 | 10675.22 | 4.979 | | |
| Total | 2152 | 11593.83 | 5.387 | | |

Table 0-41: Results of unbalanced ANOVA for gestational age for males in 2006 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 49.36 | 49.36 | 9.34 | 0.002 |
| Marital status | 1 | 2.946 | 2.946 | 0.56 | 0.455 |
| Maternal age | 1 | 25.911 | 25.911 | 4.9 | 0.027 |
| Plurality of birth | 1 | 979.256 | 979.256 | 185.29 | <.001 |
| Previous pregnancy status | 1 | 9.808 | 9.808 | 1.86 | 0.173 |
| Smoking during pregnancy | 1 | 13.554 | 13.554 | 2.56 | 0.109 |
| Fire exposed | 2 | 16.742 | 8.371 | 1.58 | 0.205 |
| Residual | 2350 | 12419.56 | 5.285 | | |
| Total | 2358 | 13517.14 | 5.732 | | |

Table 0-42: Results of unbalanced ANOVA for gestational age for males in 2007 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 18.152 | 18.152 | 3.74 | 0.053 |
| Marital status | 1 | 0.223 | 0.223 | 0.05 | 0.83 |
| Maternal age | 1 | 67.76 | 67.76 | 13.98 | <.001 |
| Plurality of birth | 1 | 644.788 | 644.788 | 133.03 | <.001 |
| Previous pregnancy status | 1 | 3.132 | 3.132 | 0.65 | 0.422 |
| Smoking during pregnancy | 1 | 14.022 | 14.022 | 2.89 | 0.089 |
| Fire exposed | 2 | 1.144 | 0.572 | 0.12 | 0.889 |
| Residual | 2338 | 11332.24 | 4.847 | | |
| Total | 2346 | 12081.46 | 5.15 | | |

Table 0-43: Results of unbalanced ANOVA for gestational age for males in 2008 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 2.747 | 2.747 | 0.53 | 0.467 |
| Marital status | 1 | 0.163 | 0.163 | 0.03 | 0.859 |
| Maternal age | 1 | 2.941 | 2.941 | 0.57 | 0.452 |
| Plurality of birth | 1 | 1341.71 | 1341.71 | 258.72 | <.001 |
| Previous pregnancy status | 1 | 1.547 | 1.547 | 0.3 | 0.585 |
| Smoking during pregnancy | 1 | 104.886 | 104.886 | 20.22 | <.001 |
| Fire exposed | 2 | 10.316 | 5.158 | 0.99 | 0.37 |
| Residual | 2456 | 12736.81 | 5.186 | | |
| Total | 2464 | 14201.12 | 5.763 | | |

Table 0-44: Results of unbalanced ANOVA for gestational age for males in 2009 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 46.025 | 46.025 | 10.31 | 0.001 |
| Marital status | 1 | 14.981 | 14.981 | 3.36 | 0.067 |
| Maternal age | 1 | 45.282 | 45.282 | 10.15 | 0.001 |
| Plurality of birth | 1 | 753.495 | 753.495 | 168.82 | <.001 |
| Previous pregnancy status | 1 | 5.458 | 5.458 | 1.22 | 0.269 |
| Smoking during pregnancy | 1 | 12.013 | 12.013 | 2.69 | 0.101 |
| Fire exposed | 2 | 14.87 | 7.435 | 1.67 | 0.189 |
| Residual | 2566 | 11453.03 | 4.463 | | |
| Total | 2574 | 12345.15 | 4.796 | | |

Table 0-45: Results of unbalanced ANOVA for gestational age for males in 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 16.026 | 16.026 | 3.08 | 0.079 |
| Marital status | 1 | 0.425 | 0.425 | 0.08 | 0.775 |
| Maternal age | 1 | 16.28 | 16.28 | 3.13 | 0.077 |
| Plurality of birth | 1 | 958.829 | 958.829 | 184.34 | <.001 |
| Previous pregnancy status | 1 | 9.667 | 9.667 | 1.86 | 0.173 |
| Smoking during pregnancy | 1 | 0.315 | 0.315 | 0.06 | 0.806 |
| Fire exposed | 2 | 24.258 | 12.129 | 2.33 | 0.097 |
| Residual | 2517 | 13092.17 | 5.201 | | |
| Total | 2525 | 14117.97 | 5.591 | | |

Table 0-46: Results of unbalanced ANOVA for birth weight for females in 2000 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 640817 | 640817 | 1.96 | 0.161 |
| Marital status | 1 | 2965306 | 2965306 | 9.09 | 0.003 |
| Maternal age | 1 | 114229 | 114229 | 0.35 | 0.554 |
| Plurality of birth | 1 | 67261577 | 67261577 | 206.25 | <.001 |
| Previous pregnancy status | 1 | 1751377 | 1751377 | 5.37 | 0.021 |
| Smoking during pregnancy | 1 | 10150344 | 10150344 | 31.12 | <.001 |
| Fire exposed | 2 | 346545 | 173272 | 0.53 | 0.588 |
| Residual | 1913 | 623876123 | 326124 | | |
| Total | 1921 | 707106317 | 368093 | | |

Table 0-47: Results of unbalanced ANOVA for birth weight for females in 2001 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 1133 | 1133 | 0 | 0.95 |
| Marital status | 1 | 7234010 | 7234010 | 24.66 | <.001 |
| Maternal age | 1 | 27463 | 27463 | 0.09 | 0.76 |
| Plurality of birth | 1 | 89082525 | 89082525 | 303.69 | <.001 |
| Previous pregnancy status | 1 | 3948005 | 3948005 | 13.46 | <.001 |
| Smoking during pregnancy | 1 | 15757262 | 15757262 | 53.72 | <.001 |
| Fire exposed | 2 | 83526 | 41763 | 0.14 | 0.867 |
| Residual | 1817 | 532980293 | 293330 | | |
| Total | 1825 | 649114217 | 355679 | | |

Table 0-48: Results of unbalanced ANOVA for birth weight for females in 2002 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 614466 | 614466 | 1.99 | 0.159 |
| Marital status | 1 | 4179597 | 4179597 | 13.52 | <.001 |
| Maternal age | 1 | 1030765 | 1030765 | 3.33 | 0.068 |
| Plurality of birth | 1 | 71377222 | 71377222 | 230.81 | <.001 |
| Previous pregnancy status | 1 | 3699233 | 3699233 | 11.96 | <.001 |
| Smoking during pregnancy | 1 | 10741178 | 10741178 | 34.73 | <.001 |
| Fire exposed | 2 | 183389 | 91694 | 0.3 | 0.743 |
| Residual | 1903 | 588493527 | 309245 | | |
| Total | 1911 | 680319378 | 356002 | | |

Table 0-49: Results of unbalanced ANOVA for birth weight for females in 2003 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 1180438 | 1180438 | 3.76 | 0.052 |
| Marital status | 1 | 2216919 | 2216919 | 7.07 | 0.008 |
| Maternal age | 1 | 12069 | 12069 | 0.04 | 0.844 |
| Plurality of birth | 1 | 77252560 | 77252560 | 246.39 | <.001 |
| Previous pregnancy status | 1 | 2358954 | 2358954 | 7.52 | 0.006 |
| Smoking during pregnancy | 1 | 20484671 | 20484671 | 65.33 | <.001 |
| Fire exposed | 2 | 125943 | 62971 | 0.2 | 0.818 |
| Residual | 1957 | 613595956 | 313539 | | |
| Total | 1965 | 717227512 | 365001 | | |

Table 0-50: Results of unbalanced ANOVA for birth weight for females in 2004 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 3405019 | 3405019 | 11.56 | <.001 |
| Marital status | 1 | 3347865 | 3347865 | 11.37 | <.001 |
| Maternal age | 1 | 7515 | 7515 | 0.03 | 0.873 |
| Plurality of birth | 1 | 101135007 | 101135007 | 343.39 | <.001 |
| Previous pregnancy status | 1 | 7424749 | 7424749 | 25.21 | <.001 |
| Smoking during pregnancy | 1 | 9839415 | 9839415 | 33.41 | <.001 |
| Fire exposed | 2 | 1145059 | 572530 | 1.94 | 0.143 |
| Residual | 1940 | 571369472 | 294520 | | |
| Total | 1948 | 697674100 | 358149 | | |

Table 0-51: Results of unbalanced ANOVA for birth weight for females in 2005 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 1139557 | 1139557 | 3.56 | 0.059 |
| Marital status | 1 | 4918065 | 4918065 | 15.37 | <.001 |
| Maternal age | 1 | 298862 | 298862 | 0.93 | 0.334 |
| Plurality of birth | 1 | 49328369 | 49328369 | 154.12 | <.001 |
| Previous pregnancy status | 1 | 1559012 | 1559012 | 4.87 | 0.027 |
| Smoking during pregnancy | 1 | 18451268 | 18451268 | 57.65 | <.001 |
| Fire exposed | 2 | 963188 | 481594 | 1.5 | 0.222 |
| Residual | 2112 | 675995672 | 320074 | | |
| Total | 2120 | 752653994 | 355025 | | |

Table 0-52: Results of unbalanced ANOVA for birth weight for females in 2006 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 2723405 | 2723405 | 9.98 | 0.002 |
| Marital status | 1 | 2190845 | 2190845 | 8.03 | 0.005 |
| Maternal age | 1 | 252240 | 252240 | 0.92 | 0.336 |
| Plurality of birth | 1 | 98675380 | 98675380 | 361.6 | <.001 |
| Previous pregnancy status | 1 | 2026909 | 2026909 | 7.43 | 0.006 |
| Smoking during pregnancy | 1 | 22157925 | 22157925 | 81.2 | <.001 |
| Fire exposed | 2 | 56719 | 28360 | 0.1 | 0.901 |
| Residual | 2184 | 595975096 | 272882 | | |
| Total | 2192 | 724058519 | 330319 | | |

Table 0-53: Results of unbalanced ANOVA for birth weight for females in 2007 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 157882 | 157882 | 0.52 | 0.472 |
| Marital status | 1 | 6383926 | 6383926 | 20.95 | <.001 |
| Maternal age | 1 | 161455 | 161455 | 0.53 | 0.467 |
| Plurality of birth | 1 | 75918111 | 75918111 | 249.14 | <.001 |
| Previous pregnancy status | 1 | 1476828 | 1476828 | 4.85 | 0.028 |
| Smoking during pregnancy | 1 | 10505877 | 10505877 | 34.48 | <.001 |
| Fire exposed | 2 | 65826 | 32913 | 0.11 | 0.898 |
| Residual | 2248 | 685015248 | 304722 | | |
| Total | 2256 | 779685152 | 345605 | | |

Table 0-54: Results of unbalanced ANOVA for birth weight for females in 2008 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 2006687 | 2006687 | 6.76 | 0.009 |
| Marital status | 1 | 325121 | 325121 | 1.1 | 0.295 |
| Maternal age | 1 | 664084 | 664084 | 2.24 | 0.135 |
| Plurality of birth | 1 | 62829923 | 62829923 | 211.62 | <.001 |
| Previous pregnancy status | 1 | 124592 | 124592 | 0.42 | 0.517 |
| Smoking during pregnancy | 1 | 2148426 | 2148426 | 7.24 | 0.007 |
| Fire exposed | 2 | 3389815 | 1694908 | 5.71 | 0.003 |
| Residual | 2258 | 670403024 | 296901 | | |
| Total | 2266 | 741891671 | 327401 | | |

Table 0-55: Results of unbalanced ANOVA for birth weight for females in 2009 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 3570722 | 3570722 | 12.25 | <.001 |
| Marital status | 1 | 936695 | 936695 | 3.21 | 0.073 |
| Maternal age | 1 | 44732 | 44732 | 0.15 | 0.695 |
| Plurality of birth | 1 | 73841784 | 73841784 | 253.43 | <.001 |
| Previous pregnancy status | 1 | 3069823 | 3069823 | 10.54 | 0.001 |
| Smoking during pregnancy | 1 | 8381525 | 8381525 | 28.77 | <.001 |
| Fire exposed | 2 | 2021028 | 1010514 | 3.47 | 0.031 |
| Residual | 2233 | 650636462 | 291373 | | |
| Total | 2241 | 742502770 | 331327 | | |

Table 0-56: Results of unbalanced ANOVA for birth weight for females in 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 17719 | 17719 | 0.06 | 0.807 |
| Marital status | 1 | 1570349 | 1570349 | 5.31 | 0.021 |
| Maternal age | 1 | 513909 | 513909 | 1.74 | 0.187 |
| Plurality of birth | 1 | 57002325 | 57002325 | 192.84 | <.001 |
| Previous pregnancy status | 1 | 2552513 | 2552513 | 8.64 | 0.003 |
| Smoking during pregnancy | 1 | 12838253 | 12838253 | 43.43 | <.001 |
| Fire exposed | 2 | 3345326 | 1672663 | 5.66 | 0.004 |
| Residual | 2417 | 714434803 | 295587 | | |
| Total | 2425 | 792275197 | 326711 | | |

Table 0-57: Results of unbalanced ANOVA for gestational age for females in 2000 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.792 | 0.792 | 0.18 | 0.673 |
| Marital status | 1 | 0.014 | 0.014 | 0 | 0.956 |
| Maternal age | 1 | 15.616 | 15.616 | 3.51 | 0.061 |
| Plurality of birth | 1 | 1103.676 | 1103.676 | 248.27 | <.001 |
| Previous pregnancy status | 1 | 4.057 | 4.057 | 0.91 | 0.34 |
| Smoking during pregnancy | 1 | 11.147 | 11.147 | 2.51 | 0.113 |
| Fire exposed | 2 | 6.233 | 3.116 | 0.7 | 0.496 |
| Residual | 1913 | 8504.051 | 4.445 | | |
| Total | 1921 | 9645.586 | 5.021 | | |

Table 0-58: Results of unbalanced ANOVA for gestational age for females in 2001 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 2.407 | 2.407 | 0.58 | 0.446 |
| Marital status | 1 | 14.05 | 14.05 | 3.4 | 0.065 |
| Maternal age | 1 | 44.311 | 44.311 | 10.72 | 0.001 |
| Plurality of birth | 1 | 1358.648 | 1358.648 | 328.57 | <.001 |
| Previous pregnancy status | 1 | 0.28 | 0.28 | 0.07 | 0.795 |
| Smoking during pregnancy | 1 | 21.783 | 21.783 | 5.27 | 0.022 |
| Fire exposed | 2 | 22.295 | 11.147 | 2.7 | 0.068 |
| Residual | 1818 | 7517.546 | 4.135 | | |
| Total | 1826 | 8981.318 | 4.919 | | |

Table 0-59: Results of unbalanced ANOVA for gestational age for females in 2002 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.065 | 0.065 | 0.02 | 0.9 |
| Marital status | 1 | 5.587 | 5.587 | 1.36 | 0.244 |
| Maternal age | 1 | 1.33 | 1.33 | 0.32 | 0.569 |
| Plurality of birth | 1 | 958.603 | 958.603 | 233.5 | <.001 |
| Previous pregnancy status | 1 | 1.926 | 1.926 | 0.47 | 0.494 |
| Smoking during pregnancy | 1 | 17.329 | 17.329 | 4.22 | 0.04 |
| Fire exposed | 2 | 0.475 | 0.238 | 0.06 | 0.944 |
| Residual | 1903 | 7812.601 | 4.105 | | |
| Total | 1911 | 8797.916 | 4.604 | | |

Table 0-60: Results of unbalanced ANOVA for gestational age for females in 2003 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 4.209 | 4.209 | 0.95 | 0.329 |
| Marital status | 1 | 7.327 | 7.327 | 1.66 | 0.198 |
| Maternal age | 1 | 57.548 | 57.548 | 13.01 | <.001 |
| Plurality of birth | 1 | 1059.545 | 1059.545 | 239.6 | <.001 |
| Previous pregnancy status | 1 | 0.427 | 0.427 | 0.1 | 0.756 |
| Smoking during pregnancy | 1 | 28.502 | 28.502 | 6.45 | 0.011 |
| Fire exposed | 2 | 1.167 | 0.584 | 0.13 | 0.876 |
| Residual | 1957 | 8654.254 | 4.422 | | |
| Total | 1965 | 9812.979 | 4.994 | | |

Table 0-61: Results of unbalanced ANOVA for gestational age for females in 2004 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 47.917 | 47.917 | 12.78 | <.001 |
| Marital status | 1 | 2.431 | 2.431 | 0.65 | 0.421 |
| Maternal age | 1 | 23.12 | 23.12 | 6.16 | 0.013 |
| Plurality of birth | 1 | 1464.143 | 1464.143 | 390.38 | <.001 |
| Previous pregnancy status | 1 | 0.683 | 0.683 | 0.18 | 0.67 |
| Smoking during pregnancy | 1 | 13.15 | 13.15 | 3.51 | 0.061 |
| Fire exposed | 2 | 3.576 | 1.788 | 0.48 | 0.621 |
| Residual | 1939 | 7272.312 | 3.751 | | |
| Total | 1947 | 8827.332 | 4.534 | | |

Table 0-62: Results of unbalanced ANOVA for gestational age for females in 2005 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 15.547 | 15.547 | 3.48 | 0.062 |
| Marital status | 1 | 8.315 | 8.315 | 1.86 | 0.173 |
| Maternal age | 1 | 30.335 | 30.335 | 6.78 | 0.009 |
| Plurality of birth | 1 | 619.845 | 619.845 | 138.63 | <.001 |
| Previous pregnancy status | 1 | 11.832 | 11.832 | 2.65 | 0.104 |
| Smoking during pregnancy | 1 | 13.114 | 13.114 | 2.93 | 0.087 |
| Fire exposed | 2 | 9.027 | 4.513 | 1.01 | 0.365 |
| Residual | 2112 | 9443.44 | 4.471 | | |
| Total | 2120 | 10151.46 | 4.788 | | |

Table 0-63: Results of unbalanced ANOVA for gestational age for females in 2006 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 12.404 | 12.404 | 3.12 | 0.078 |
| Marital status | 1 | 4.881 | 4.881 | 1.23 | 0.268 |
| Maternal age | 1 | 11.268 | 11.268 | 2.83 | 0.093 |
| Plurality of birth | 1 | 1554.247 | 1554.247 | 390.59 | <.001 |
| Previous pregnancy status | 1 | 7.404 | 7.404 | 1.86 | 0.173 |
| Smoking during pregnancy | 1 | 27.233 | 27.233 | 6.84 | 0.009 |
| Fire exposed | 2 | 5.066 | 2.533 | 0.64 | 0.529 |
| Residual | 2184 | 8690.565 | 3.979 | | |
| Total | 2192 | 10313.07 | 4.705 | | |

Table 0-64: Results of unbalanced ANOVA for gestational age for females in 2007 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 3.665 | 3.665 | 0.89 | 0.345 |
| Marital status | 1 | 8.699 | 8.699 | 2.11 | 0.146 |
| Maternal age | 1 | 45.636 | 45.636 | 11.09 | <.001 |
| Plurality of birth | 1 | 943.665 | 943.665 | 229.34 | <.001 |
| Previous pregnancy status | 1 | 13.096 | 13.096 | 3.18 | 0.075 |
| Smoking during pregnancy | 1 | 12.707 | 12.707 | 3.09 | 0.079 |
| Fire exposed | 2 | 2.132 | 1.066 | 0.26 | 0.772 |
| Residual | 2247 | 9245.793 | 4.115 | | |
| Total | 2255 | 10275.39 | 4.557 | | |

Table 0-65: Results of unbalanced ANOVA for gestational age for females in 2008 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 41.278 | 41.278 | 10.63 | 0.001 |
| Marital status | 1 | 1.692 | 1.692 | 0.44 | 0.509 |
| Maternal age | 1 | 33.028 | 33.028 | 8.51 | 0.004 |
| Plurality of birth | 1 | 664.538 | 664.538 | 171.18 | <.001 |
| Previous pregnancy status | 1 | 28.144 | 28.144 | 7.25 | 0.007 |
| Smoking during pregnancy | 1 | 0.449 | 0.449 | 0.12 | 0.734 |
| Fire exposed | 2 | 11.778 | 5.889 | 1.52 | 0.22 |
| Residual | 2258 | 8765.764 | 3.882 | | |
| Total | 2266 | 9546.671 | 4.213 | | |

Table 0-66: Results of unbalanced ANOVA for gestational age for females in 2009 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 19.881 | 19.881 | 5.23 | 0.022 |
| Marital status | 1 | 0.025 | 0.025 | 0.01 | 0.936 |
| Maternal age | 1 | 35.477 | 35.477 | 9.33 | 0.002 |
| Plurality of birth | 1 | 1108.488 | 1108.488 | 291.41 | <.001 |
| Previous pregnancy status | 1 | 7.383 | 7.383 | 1.94 | 0.164 |
| Smoking during pregnancy | 1 | 10.479 | 10.479 | 2.75 | 0.097 |
| Fire exposed | 2 | 7.748 | 3.874 | 1.02 | 0.361 |
| Residual | 2233 | 8493.991 | 3.804 | | |
| Total | 2241 | 9683.472 | 4.321 | | |

Table 0-67: Results of unbalanced ANOVA for gestational age for females in 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | p |
|---|---|---|---|---|---|
| ATSI status | 1 | 1.335 | 1.335 | 0.34 | 0.562 |
| Marital status | 1 | 2.889 | 2.889 | 0.73 | 0.393 |
| Maternal age | 1 | 5.097 | 5.097 | 1.29 | 0.257 |
| Plurality of birth | 1 | 862.595 | 862.595 | 217.84 | <.001 |
| Previous pregnancy status | 1 | 6.16 | 6.16 | 1.56 | 0.212 |
| Smoking during pregnancy | 1 | 18.079 | 18.079 | 4.57 | 0.033 |
| Fire exposed | 2 | 7.017 | 3.509 | 0.89 | 0.412 |
| Residual | 2417 | 9570.605 | 3.96 | | |
| Total | 2425 | 10473.78 | 4.319 | | |

Table 0-68: Results of unbalanced ANOVA for birth weight for females between 2000 - 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 11487617 | 11487617 | 38.08 | <.001 |
| Marital status | 1 | 32350317 | 32350317 | 107.24 | <.001 |
| Maternal age | 1 | 1743556 | 1743556 | 5.78 | 0.016 |
| Plurality of birth | 1 | 813853629 | 813853629 | 2698 | <.001 |
| Previous pregnancy status | 1 | 25892639 | 25892639 | 85.84 | <.001 |
| Smoking during pregnancy | 1 | 132191391 | 132191391 | 438.23 | <.001 |
| Birth timing | 16 | 10573776 | 660861 | 2.19 | 0.004 |
| Fire exposed | 2 | 3299309 | 1649655 | 5.47 | 0.004 |
| Birth timing.Fire exposed interaction term | 32 | 12599330 | 393729 | 1.31 | 0.116 |
| Residual | 23024 | 6945205310 | 301651 | | |
| Total | 23080 | 7989196876 | 346152 | | |

Table 0-69: Results of unbalanced ANOVA for birth weight for males between 2000 - 2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 25820850 | 25820850 | 73.62 | <0.001 |
| Marital status | 1 | 33805113 | 16902556 | 48.19 | <0.001 |
| Maternal age | 1 | 185005 | 185005 | 0.53 | 0.03 |
| Plurality of birth | 1 | 815350352 | 407675176 | 1162.41 | <0.001 |
| Previous pregnancy status | 1 | 45059058 | 45059058 | 128.48 | <0.001 |
| Smoking during pregnancy | 1 | 160520440 | 160520440 | 457.69 | <0.001 |
| Birth timing | 16 | 12218913 | 763682 | 2.18 | <0.001 |
| Fire exposed | 2 | 4171961 | 2085980 | 5.95 | <0.001 |
| Birth timing.Fire exposed interaction term | 32 | 20598973 | 643718 | 1.84 | 0.04 |
| Residual | 2108 | 8606925884 | 350716 | | |
| Total | 2116 | 9724656549 | 395327 | | |

Table 0-70: Results of unbalanced ANOVA for gestational age for females between 2000 -2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 103.061 | 103.061 | 25.17 | <.001 |
| Marital status | 1 | 33.315 | 33.315 | 8.14 | 0.004 |
| Maternal age | 1 | 238.022 | 238.022 | 58.14 | <.001 |
| Plurality of birth | 1 | 11501.009 | 11501.009 | 2809.24 | <.001 |

| | | | | | |
|---|---|---|---|---|---|
| Previous pregnancy status | 1 | 54.959 | 54.959 | 13.42 | <.001 |
| Smoking during pregnancy | 1 | 161.334 | 161.334 | 39.41 | <.001 |
| Birth timing | 16 | 82.444 | 5.153 | 1.26 | 0.214 |
| Fire exposed | 2 | 14.391 | 7.196 | 1.76 | 0.172 |
| Birth timing.Fire exposed interaction term | 32 | 109.878 | 3.434 | 0.84 | 0.726 |
| Residual | 23023 | 94255.91 | 4.094 | | |
| Total | 23079 | 106554.324 | 4.617 | | |

Table 0-71: Results of unbalanced ANOVA for gestational age for males between 2000 -2010 (showing fire exposed at three levels: heavily, moderately or least affected).

| | Degrees of freedom | Sums of squares | Mean square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 243.408 | 243.408 | 49.41 | <.001 |
| Marital status | 1 | 15.845 | 15.845 | 3.22 | 0.073 |
| Maternal age | 1 | 277.084 | 277.084 | 56.25 | <.001 |
| Plurality of birth | 1 | 10232.653 | 10232.65 | 2077.31 | <.001 |
| Previous pregnancy status | 1 | 24.092 | 24.092 | 4.89 | 0.027 |
| Smoking during pregnancy | 1 | 309.414 | 309.414 | 62.81 | <.001 |
| Birth timing | 16 | 54.89 | 3.431 | 0.7 | 0.801 |
| Fire exposed | 2 | 5.077 | 2.539 | 0.52 | 0.597 |
| Birth timing.Fire exposed interaction term | 32 | 182.832 | 5.714 | 1.16 | 0.245 |
| Residual | 24540 | 120881.815 | 4.926 | | |
| Total | 24596 | 132227.111 | 5.376 | | |

*Victorian analysis*

Table 0-72: Multiple linear regression for gestational age for both sexes in 2009

| | Coef. | Std. Err. | t | P>t | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| | | | | | | |
| ATSI | -0.271 | 0.072 | -3.770 | 0.000 | -0.410 | -0.130 |
| | | | | | | |
| Gravidity (1) | | | | | | |
| 2 | -0.039 | 0.023 | -1.710 | 0.086 | -0.080 | 0.006 |
| 3 | -0.163 | 0.023 | -7.150 | 0.000 | -0.210 | -0.118 |
| | | | | | | |
| Male | -0.102 | 0.018 | -5.710 | 0.000 | -0.140 | -0.067 |
| Plurality | -3.655 | 0.051 | -71.950 | 0.000 | -3.750 | -3.555 |
| Maternal age | -0.014 | 0.002 | -7.930 | 0.000 | -0.020 | -0.011 |
| Non-smoker | 0.249 | 0.032 | 7.900 | 0.000 | 0.190 | 0.311 |
| Coupled marital status | 0.346 | 0.029 | 11.810 | 0.000 | 0.290 | 0.403 |
| | | | | | | |
| Fire area | | | | | | |
| Affected | 0.442 | 0.728 | 0.610 | 0.544 | -0.990 | 1.869 |
| Surrounding | 0.373 | 0.632 | 0.590 | 0.555 | -0.870 | 1.612 |
| Unaffected | 0.389 | 0.604 | 0.640 | 0.519 | -0.790 | 1.572 |
| | | | | | | |
| Trimester | | | | | | |
| 1 | -0.995 | 0.634 | -1.570 | 0.117 | -2.240 | 0.248 |
| 2 | -0.783 | 0.645 | -1.210 | 0.225 | -2.050 | 0.480 |
| 3 | -0.509 | 0.628 | -0.810 | 0.417 | -1.740 | 0.722 |
| 4 | -1.142 | 0.655 | -1.740 | 0.081 | -2.420 | 0.141 |
| | | | | | | |
| Trimester#Firearea | | | | | | |
| 1#Affected | -0.553 | 0.766 | -0.720 | 0.471 | -2.050 | 0.949 |
| 1#Surrounding | -0.049 | 0.667 | -0.070 | 0.941 | -1.360 | 1.258 |
| 1#Unaffected | -0.203 | 0.637 | -0.320 | 0.749 | -1.450 | 1.045 |
| 2#Affected | -0.420 | 0.778 | -0.540 | 0.589 | -1.950 | 1.104 |
| 2#Surrounding | -0.460 | 0.678 | -0.680 | 0.497 | -1.790 | 0.868 |
| 2#Unaffected | -0.451 | 0.648 | -0.700 | 0.486 | -1.720 | 0.818 |
| 3#Affected | -0.453 | 0.759 | -0.600 | 0.551 | -1.940 | 1.035 |
| 3#Surrounding | -0.405 | 0.660 | -0.610 | 0.539 | -1.700 | 0.889 |
| 3#Unaffected | -0.485 | 0.631 | -0.770 | 0.442 | -1.720 | 0.751 |
| 4#Affected | -0.740 | 0.790 | -0.940 | 0.349 | -2.290 | 0.808 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 4#Surrounding | -0.610 | 0.688 | -0.890 | 0.375 | -1.960 | 0.739 |
| 4#Unaffected | -0.769 | 0.658 | -1.170 | 0.242 | -2.060 | 0.520 |
| | | | | | | |
| _cons | 39.724 | 0.603 | 65.830 | 0.000 | 38.540 | 40.906 |

Table 0-73: Multiple linear regression for birthweight for both sexes in 2009

| | Coef. | Std. Err. | t | P>t | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| | | | | | | |
| ATSI | -91.274 | 18.512 | -4.930 | 0.000 | -127.560 | -54.991 |
| | | | | | | |
| Gravidity (1) | | | | | | |
| 2.000 | 99.693 | 5.850 | 17.040 | 0.000 | 88.230 | 111.159 |
| 3.000 | 119.510 | 5.863 | 20.380 | 0.000 | 108.020 | 131.002 |
| | | | | | | |
| Male | 108.679 | 4.596 | 23.640 | 0.000 | 99.670 | 117.688 |
| Plurality | -1009.979 | 13.071 | -77.270 | 0.000 | -1035.600 | -984.359 |
| Maternal age | -0.574 | 0.464 | -1.240 | 0.216 | -1.480 | 0.335 |
| Non-smoker | 221.573 | 8.130 | 27.250 | 0.000 | 205.640 | 237.508 |
| Coupled marital status | 86.112 | 7.546 | 11.410 | 0.000 | 71.320 | 100.902 |
| | | | | | | |
| Fire area | | | | | | |
| Affected | -82.108 | 189.286 | -0.430 | 0.664 | -453.110 | 288.893 |
| Surrounding | -138.799 | 162.617 | -0.850 | 0.393 | -457.530 | 179.930 |
| Unaffected | -121.246 | 155.274 | -0.780 | 0.435 | -425.580 | 183.091 |
| | | | | | | |
| Trimester | | | | | | |
| 1.000 | -369.633 | 163.164 | -2.270 | 0.023 | -689.440 | -49.831 |
| 2.000 | -222.067 | 165.846 | -1.340 | 0.181 | -547.130 | 102.992 |
| 3.000 | -256.524 | 161.591 | -1.590 | 0.112 | -573.240 | 60.194 |
| 4.000 | -320.169 | 168.421 | -1.900 | 0.057 | -650.270 | 9.936 |
| | | | | | | |
| Trimester#Firearea | | | | | | |
| 1#Affected | 160.025 | 199.033 | 0.800 | 0.421 | -230.080 | 550.128 |
| 1#Surrounding | 245.235 | 171.583 | 1.430 | 0.153 | -91.070 | 581.537 |
| 1#Unaffected | 187.642 | 163.916 | 1.140 | 0.252 | -133.630 | 508.917 |
| 2#Affected | 56.640 | 202.016 | 0.280 | 0.779 | -339.310 | 452.591 |
| 2#Surrounding | 53.745 | 174.325 | 0.310 | 0.758 | -287.930 | 395.421 |
| 2#Unaffected | 31.000 | 166.604 | 0.190 | 0.852 | -295.540 | 357.545 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 3#Affected | 138.726 | 197.137 | 0.700 | 0.482 | -247.660 | 525.114 |
| 3#Surrounding | 169.225 | 169.878 | 1.000 | 0.319 | -163.740 | 502.184 |
| 3#Unaffected | 100.918 | 162.334 | 0.620 | 0.534 | -217.260 | 419.093 |
| 4#Affected | 71.261 | 205.144 | 0.350 | 0.728 | -330.820 | 473.344 |
| 4#Surrounding | 76.577 | 177.114 | 0.430 | 0.665 | -270.570 | 423.721 |
| 4#Unaffected | 2.465 | 169.195 | 0.010 | 0.988 | -329.160 | 334.087 |
| | | | | | | |
| _cons | 3306.533 | 155.258 | 21.300 | 0.000 | 3002.230 | 3610.838 |

Table 0-74: Multiple Linear Regression for males (only) of gestation age in 2009

| | Coef. | Std. Err. | t | P>t | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| ASTI | -0.209 | 0.103 | -2.030 | 0.042 | -0.411 | -0.007 |
| | | | | | | |
| Gravidity (1) | | | | | | |
| 2.000 | -0.004 | 0.033 | -0.120 | 0.901 | -0.068 | 0.060 |
| 3.000 | -0.118 | 0.033 | -3.620 | 0.000 | -0.182 | -0.054 |
| | | | | | | |
| Plurality | -3.678 | 0.072 | -50.880 | 0.000 | -3.819 | -3.536 |
| Maternal age | -0.012 | 0.003 | -4.730 | 0.000 | -0.017 | -0.007 |
| | | | | | | |
| Smoking | | | | | | |
| Unstated | 0.216 | 0.114 | 1.890 | 0.059 | -0.008 | 0.439 |
| Smoker | -0.243 | 0.045 | -5.350 | 0.000 | -0.332 | -0.154 |
| | | | | | | |
| Coupled marital status | 0.354 | 0.042 | 8.400 | 0.000 | 0.272 | 0.437 |
| | | | | | | |
| Fire area | | | | | | |
| Affected | 0.546 | 1.046 | 0.520 | 0.601 | -1.503 | 2.596 |
| Surrounding | 0.508 | 0.887 | 0.570 | 0.566 | -1.229 | 2.246 |
| Unaffected | 0.551 | 0.849 | 0.650 | 0.516 | -1.112 | 2.215 |
| | | | | | | |
| Trimester | | | | | | |
| 1.000 | -0.526 | 0.897 | -0.590 | 0.558 | -2.284 | 1.232 |
| 2.000 | -0.580 | 0.916 | -0.630 | 0.527 | -2.376 | 1.216 |
| 3.000 | -0.550 | 0.889 | -0.620 | 0.536 | -2.294 | 1.193 |
| 4.000 | -0.876 | 0.927 | -0.940 | 0.345 | -2.693 | 0.941 |
| | | | | | | |
| Trimester#Firearea | | | | | | |
| 1#Affected | -0.930 | 1.103 | -0.840 | 0.399 | -3.093 | 1.233 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 1#Surrounding | -0.591 | 0.941 | -0.630 | 0.530 | -2.436 | 1.254 |
| 1#Unaffected | -0.731 | 0.902 | -0.810 | 0.417 | -2.499 | 1.036 |
| 2#Affected | -0.593 | 1.122 | -0.530 | 0.597 | -2.791 | 1.606 |
| 2#Surrounding | -0.715 | 0.961 | -0.740 | 0.457 | -2.598 | 1.168 |
| 2#Unaffected | -0.724 | 0.921 | -0.790 | 0.431 | -2.529 | 1.080 |
| 3#Affected | -0.442 | 1.094 | -0.400 | 0.686 | -2.585 | 1.702 |
| 3#Surrounding | -0.419 | 0.933 | -0.450 | 0.653 | -2.247 | 1.409 |
| 3#Unaffected | -0.507 | 0.894 | -0.570 | 0.571 | -2.259 | 1.245 |
| 4#Affected | -1.054 | 1.137 | -0.930 | 0.354 | -3.283 | 1.174 |
| 4#Surrounding | -1.069 | 0.972 | -1.100 | 0.272 | -2.974 | 0.837 |
| 4#Unaffected | -1.126 | 0.932 | -1.210 | 0.227 | -2.952 | 0.699 |
| | | | | | | |
| _cons | 39.676 | 0.848 | 46.810 | 0.000 | 38.015 | 41.338 |
| | | | | | | |

Table 0-75: Multiple Linear Regression for females (only) of gestation age in 2009

| | Coef. | Std. Err. | t | P>t | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| ASTI | -0.332 | 0.100 | -3.320 | 0.001 | -0.528 | -0.136 |
| | | | | | | |
| Gravidity (1) | | | | | | |
| 2.000 | -0.075 | 0.032 | -2.380 | 0.017 | -0.138 | -0.013 |
| 3.000 | -0.208 | 0.032 | -6.580 | 0.000 | -0.271 | -0.146 |
| | | | | | | |
| Plurality | -3.628 | 0.071 | -50.920 | 0.000 | -3.768 | -3.489 |
| Maternal age | -0.016 | 0.002 | -6.560 | 0.000 | -0.021 | -0.011 |
| | | | | | | |
| Smoking | | | | | | |
| Unstated | 0.211 | 0.110 | 1.920 | 0.055 | -0.005 | 0.427 |
| Smoker | -0.248 | 0.044 | -5.650 | 0.000 | -0.334 | -0.162 |
| | | | | | | |
| Coupled marital status | 0.342 | 0.041 | 8.400 | 0.000 | 0.262 | 0.421 |
| | | | | | | |
| Fire area | | | | | | |
| Affected | 0.326 | 1.015 | 0.320 | 0.748 | -1.663 | 2.316 |
| Surrounding | 0.206 | 0.901 | 0.230 | 0.819 | -1.561 | 1.972 |
| Unaffected | 0.195 | 0.858 | 0.230 | 0.820 | -1.487 | 1.876 |
| | | | | | | |
| Trimester | | | | | | |
| 1.000 | -1.461 | 0.897 | -1.630 | 0.103 | -3.218 | 0.297 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 2.000 | -1.000 | 0.908 | -1.100 | 0.271 | -2.779 | 0.780 |
| 3.000 | -0.524 | 0.888 | -0.590 | 0.555 | -2.263 | 1.216 |
| 4.000 | -1.435 | 0.924 | -1.550 | 0.121 | -3.247 | 0.377 |
| | | | | | | |
| Trimester#Firearea | | | | | | |
| 1#Affected | -0.208 | 1.065 | -0.200 | 0.845 | -2.296 | 1.880 |
| 1#Surrounding | 0.489 | 0.945 | 0.520 | 0.605 | -1.364 | 2.342 |
| 1#Unaffected | 0.321 | 0.900 | 0.360 | 0.721 | -1.443 | 2.086 |
| 2#Affected | -0.266 | 1.081 | -0.250 | 0.806 | -2.385 | 1.853 |
| 2#Surrounding | -0.193 | 0.957 | -0.200 | 0.840 | -2.069 | 1.683 |
| 2#Unaffected | -0.165 | 0.912 | -0.180 | 0.856 | -1.952 | 1.622 |
| 3#Affected | -0.428 | 1.054 | -0.410 | 0.685 | -2.495 | 1.639 |
| 3#Surrounding | -0.335 | 0.936 | -0.360 | 0.721 | -2.169 | 1.500 |
| 3#Unaffected | -0.409 | 0.891 | -0.460 | 0.647 | -2.156 | 1.338 |
| 4#Affected | -0.411 | 1.099 | -0.370 | 0.709 | -2.565 | 1.744 |
| 4#Surrounding | -0.089 | 0.975 | -0.090 | 0.927 | -2.000 | 1.822 |
| 4#Unaffected | -0.385 | 0.928 | -0.420 | 0.678 | -2.205 | 1.434 |
| | | | | | | |
| _cons | 40.194 | 0.858 | 46.850 | 0.000 | 38.513 | 41.876 |

Table 0-76: Multiple Linear Regression for males (only) of birth weight in 2009

| | Coef. | Std. Err. | t | P>t | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| ASTI | -83.472 | 26.602 | -3.140 | 0.002 | -135.613 | -31.331 |
| | | | | | | |
| Gravidity (1) | | | | | | |
| 2.000 | 112.011 | 8.400 | 13.330 | 0.000 | 95.547 | 128.475 |
| 3.000 | 135.740 | 8.434 | 16.090 | 0.000 | 119.209 | 152.271 |
| | | | | | | |
| Plurality | -1015.840 | 18.653 | -54.460 | 0.000 | -1052.400 | -979.275 |
| Maternal age | -0.718 | 0.669 | -1.070 | 0.283 | -2.028 | 0.593 |
| | | | | | | |
| Smoking | | | | | | |
| Unstated | 56.108 | 29.499 | 1.900 | 0.057 | -1.711 | 113.927 |
| Smoker | -219.390 | 11.714 | -18.730 | 0.000 | -242.350 | -196.430 |
| | | | | | | |
| Coupled marital status | 85.473 | 10.890 | 7.850 | 0.000 | 64.127 | 106.818 |
| | | | | | | |
| Fire area | | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| Affected | 53.677 | 273.009 | 0.200 | 0.844 | -481.429 | 588.782 |
| Surrounding | -28.305 | 228.661 | -0.120 | 0.901 | -476.489 | 419.878 |
| Unaffected | -30.601 | 218.898 | -0.140 | 0.889 | -459.649 | 398.446 |
| | | | | | | |
| Trimester | | | | | | |
| 1.000 | -212.027 | 231.407 | -0.920 | 0.360 | -665.592 | 241.538 |
| 2.000 | -97.206 | 236.350 | -0.410 | 0.681 | -560.460 | 366.048 |
| 3.000 | -242.229 | 229.404 | -1.060 | 0.291 | -691.869 | 207.411 |
| 4.000 | -181.546 | 239.091 | -0.760 | 0.448 | -650.173 | 287.081 |
| | | | | | | |
| Trimester#Firearea | | | | | | |
| 1#Affected | -52.395 | 287.776 | -0.180 | 0.856 | -616.445 | 511.656 |
| 1#Surrounding | 24.590 | 242.795 | 0.100 | 0.919 | -451.295 | 500.475 |
| 1#Unaffected | 4.320 | 232.559 | 0.020 | 0.985 | -451.503 | 460.142 |
| 2#Affected | -150.279 | 292.461 | -0.510 | 0.607 | -723.512 | 422.955 |
| 2#Surrounding | -92.867 | 247.755 | -0.370 | 0.708 | -578.476 | 392.741 |
| 2#Unaffected | -116.658 | 237.504 | -0.490 | 0.623 | -582.173 | 348.857 |
| 3#Affected | 37.545 | 285.338 | 0.130 | 0.895 | -521.726 | 596.816 |
| 3#Surrounding | 111.676 | 240.549 | 0.460 | 0.642 | -359.808 | 583.160 |
| 3#Unaffected | 61.657 | 230.545 | 0.270 | 0.789 | -390.219 | 513.533 |
| 4#Affected | -185.075 | 296.340 | -0.620 | 0.532 | -765.910 | 395.760 |
| 4#Surrounding | -139.681 | 250.767 | -0.560 | 0.578 | -631.192 | 351.830 |
| 4#Unaffected | -171.528 | 240.273 | -0.710 | 0.475 | -642.470 | 299.415 |
| | | | | | | |
| _cons | 3566.574 | 218.626 | 16.310 | 0.000 | 3138.060 | 3995.089 |

Table 0-77: Multiple Linear Regression for females (only) of birth weight in 2009

| | Coef. | Std. Err. | t | P>t | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| ASTI | -97.570 | 25.698 | -3.800 | 0.000 | -147.940 | -47.201 |
| | | | | | | |
| Gravidity (1) | | | | | | |
| 2.000 | 86.733 | 8.126 | 10.670 | 0.000 | 70.805 | 102.661 |
| 3.000 | 102.906 | 8.132 | 12.650 | 0.000 | 86.967 | 118.846 |
| | | | | | | |
| Plurality | -1003.030 | 18.277 | -54.880 | 0.000 | -1038.850 | -967.203 |
| Maternal age | -0.414 | 0.641 | -0.650 | 0.518 | -1.670 | 0.842 |
| | | | | | | |
| Smoking | | | | | | |
| Unstated | 66.334 | 28.361 | 2.340 | 0.019 | 10.745 | 121.923 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Smoker | -221.803 | 11.274 | -19.670 | 0.000 | -243.901 | -199.705 |
| | | | | | | |
| Coupled marital status | 88.157 | 10.437 | 8.450 | 0.000 | 67.700 | 108.615 |
| | | | | | | |
| Fire area | | | | | | |
| Affected | -215.787 | 262.680 | -0.820 | 0.411 | -730.650 | 299.075 |
| Surrounding | -270.918 | 231.131 | -1.170 | 0.241 | -723.943 | 182.106 |
| Unaffected | -223.459 | 220.023 | -1.020 | 0.310 | -654.712 | 207.794 |
| | | | | | | |
| Trimester | | | | | | |
| 1.000 | -532.231 | 229.946 | -2.310 | 0.021 | -982.933 | -81.530 |
| 2.000 | -348.310 | 232.856 | -1.500 | 0.135 | -804.716 | 108.096 |
| 3.000 | -292.472 | 227.638 | -1.280 | 0.199 | -738.650 | 153.706 |
| 4.000 | -469.794 | 237.066 | -1.980 | 0.048 | -934.451 | -5.137 |
| | | | | | | |
| Trimester#Firearea | | | | | | |
| 1#Affected | 361.647 | 275.498 | 1.310 | 0.189 | -178.339 | 901.632 |
| 1#Surrounding | 480.840 | 242.469 | 1.980 | 0.047 | 5.591 | 956.089 |
| 1#Unaffected | 375.306 | 230.925 | 1.630 | 0.104 | -77.314 | 827.926 |
| 2#Affected | 255.324 | 279.539 | 0.910 | 0.361 | -292.582 | 803.230 |
| 2#Surrounding | 208.408 | 245.517 | 0.850 | 0.396 | -272.814 | 689.630 |
| 2#Unaffected | 179.093 | 233.848 | 0.770 | 0.444 | -279.259 | 637.444 |
| 3#Affected | 247.404 | 272.689 | 0.910 | 0.364 | -287.077 | 781.885 |
| 3#Surrounding | 257.111 | 240.028 | 1.070 | 0.284 | -213.351 | 727.574 |
| 3#Unaffected | 161.084 | 228.604 | 0.700 | 0.481 | -286.988 | 609.155 |
| 4#Affected | 331.193 | 284.149 | 1.170 | 0.244 | -225.750 | 888.135 |
| 4#Surrounding | 319.780 | 250.091 | 1.280 | 0.201 | -170.407 | 809.967 |
| 4#Unaffected | 186.780 | 238.077 | 0.780 | 0.433 | -279.860 | 653.420 |
| | | | | | | |
| _cons | 3607.582 | 220.050 | 16.390 | 0.000 | 3176.277 | 4038.887 |

**APPENDIX B: ANALYSES FROM CHAPTER SIX**

Table 0-1: The ACT sample areas were classed as shown below.

| Heavily affected | Moderately affected | | |
|---|---|---|---|
| Chapman | Aranda | Gilmore | Melba |
| Curtin | Banks | Gooromon | Molonglo |
| Duffy | Belconnen | Gordon (ACT) | Monash |
| Giralang | Bonython | Gowrie (ACT) | Mount Taylor |
| Holder | Bruce | Greenway | Namadgi |
| Lyons | Calwell | Hawker | O'Malley |
| Rivett | Charnwood | Higgins | Oxley (ACT) |
| Torrens | Chifley | Holt | Page |
| Weston | Chisholm | Hughes | Pearce |
| | Conder | Isaacs | Phillip |
| | Cook | Isabella Plains | Richardson |
| | Dunlop | Kaleen | Scullin |
| | Evatt | Kambah | Spence |
| | Fadden | Latham | Stirling |
| | Farrer | Lawson | Theodore |
| | Fisher | Macarthur | Tuggeranong |
| | Florey | Macgregor (ACT) | Wanniassa |
| | Flynn (ACT) | Macquarie | Waramanga |
| | Fraser | Mawson | Weetangera |
| | Garran | McKellar | |

Table 0-2: The Victorian rural-perurban sample areas were classed as shown below.

| Fire | Heavily affected | Moderately affected |
|---|---|---|
| Beechworth Fire | Beechworth | Bright - Mount Beauty |
| | Healesville - Yarra Glen | Chiltern - Indigo Valley |
| | | Myrtleford |
| | | Belgrave - Selby |
| | | Chirnside Park |
| | | Kilsyth |
| | | Lilydale - Coldstream |
| | | Monbulk - Silvan |
| | | Montrose |
| | | Mooroolbark |
| | | Mount Dandenong - Olinda |
| | | Mount Evelyn |
| | | Upwey - Tecoma |
| | | Wandin - Seville |
| | | Yarra Valley |

| Fire | Heavily affected | Moderately affected |
|---|---|---|
| Bendigo Fire | Maiden Gully | Bendigo<br>California Gully - Eaglehawk<br>East Bendigo - Kennington<br>Flora Hill - Spring Gully<br>Kangaroo Flat - Golden Square<br>Strathfieldsaye<br>White Hills – Ascot |
| Bunyip Fire | Bunyip – Garfield<br>Drouin | Mount Baw Baw region<br>Trafalgar<br>Warragul<br>Beaconsfield - Officer<br>Emerald - Cockatoo<br>Koo Wee Rup<br>Pakenham - North<br>Pakenham - South |
| Coleraine Fire | Southern Grampians | - |
| Churchill Fire | Churchill<br>Yarram | Moe - Newborough<br>Morwell<br>Traralgon<br>Yallourn North – Glengarry<br>Longford - Loch Sport<br>Maffra<br>Rosedale<br>Sale |
| Horsham Fire | Horsham Region | Ararat<br>Ararat Region<br>Horsham<br>Nhill Region<br>St Arnaud<br>Stawell<br>West Wimmera<br>Glenelg<br>Hamilton<br>Portland |
| Kilmore Fire | Kilmore – Broadford<br>Wallan | Euroa<br>Mansfield<br>Nagambie<br>Seymour<br>Seymour Region<br>Upper Yarra Valley |
| Kinglake Fire | Kinglake<br>Yea | Eltham<br>Hurstbridge<br>Panton Hill - St Andrews<br>Plenty - Yarrambat<br>Research - North Warrandyte<br>Wattle Glen - Diamond Creek<br>South Morang<br>Whittlesea |
| Murrindindi Fire | Alexandra | - |

Table 0-3: The Victorian suburban sample areas were classed as shown below.

| Heavily affected | Moderately affected | |
|---|---|---|
| Ferntree Gully | Bayswater | Mitcham |
| Hampton Park - Lynbrook | Boronia - The Basin | Narre Warren |
| Lynbrook - Lyndhurst | Clarinda - Oakleigh South | Narre Warren South |
| Narre Warren North | Clayton South | Noble Park |
| | Cranbourne East | Noble Park North |
| | Cranbourne North | Nunawading |
| | Cranbourne South | Rowville - Central |
| | Cranbourne West | Rowville - North |
| | Dandenong | Rowville - South |
| | Dandenong North | Springvale |
| | Dingley Village | Springvale South |
| | Donvale - Park Orchards | Vermont |
| | Forest Hill | Vermont South |
| | Keysborough | Wantirna |
| | Knoxfield - Scoresby | Wantirna South |
| | Lysterfield | |

Table 0-4: Results of ANOVA of Total Fertility Rate in the ACT in 2003 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.362 | 0.181 | 0.3 | 0.738 |
| Residual | 85 | 50.535 | 0.595 | | |
| Total | 87 | 50.898 | | | |

Table 0-5: Results of ANOVA of Total Fertility Rate in the ACT in 2004 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 7.775 | 3.888 | 2.25 | 0.111 |
| Residual | 85 | 150.037 | 1.725 | | |
| Total | 87 | 157.813 | | | |

Table 0-6: Results of ANOVA of Total Fertility Rate in the ACT in 2005 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 14.69 | 7.345 | 2.19 | 0.119 |
| Residual | 85 | 282.011 | 3.357 | | |
| Total | 87 | 296.701 | | | |

Table 0-7: Results of ANOVA of Total Fertility Rate in the ACT in 2006 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 15.017 | 7.508 | 1.45 | 0.241 |
| Residual | 85 | 441.005 | 5.188 | | |
| Total | 87 | 456.022 | | | |

Table 0-8: A Results of ANOVA of Total Fertility Rate in the ACT in 2007 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.0024 | 0.0012 | 0 | 0.996 |
| Residual | 85 | 26.9763 | 0.3174 | | |
| Total | 87 | 26.9787 | | | |

Table 0-9: Results of ANOVA of Total Fertility Rate in the ACT in 2008 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.013 | 0.0065 | 0.02 | 0.978 |
| Residual | 86 | 25.4419 | 0.2958 | | |
| Total | 88 | 25.4549 | | | |

Table 0-10: Results of ANOVA of Total Fertility Rate in the ACT in 2009 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.63 | 0.3129 | 1.99 | 0.14 |
| Residual | 86 | 13.5 | 0.157 | | |
| Total | 88 | 14.13 | | | |

Table 0-11: Results of ANOVA of Total Fertility Rate in the ACT in 2010 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.951 | 0.4755 | 2.66 | 0.076 |
| Residual | 90 | 16.0953 | 0.1788 | | |
| Total | 92 | 17.0464 | | | |

Table 0-12: Results of ANOVA of Total Fertility Rate in the ACT in 2011 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.8571 | 0.4285 | 2.52 | 0.086 |
| Residual | 92 | 15.6569 | 0.1702 | | |
| Total | 94 | 16.5139 | | | |

Table 0-13: Results of ANOVA of Total Fertility Rate in the ACT in 2012 divided by fire area (heavily, moderately or least affected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.8448 | 0.4224 | 1.62 | 0.203 |
| Residual | 91 | 23.722 | 0.2607 | | |
| Total | 93 | 24.5668 | | | |

Table 0-14: Results of ANOVA of Total Fertility Rate in the ACT for the full date range (2003-2012) with interaction term between fire area (heavily, moderately or least affected) and year.

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.1905 | 0.0952 | 0.2 | 0.815 |
| Year | 9 | 2.9558 | 0.3284 | 0.71 | 0.704 |
| Fire area.Year | 18 | 9.8177 | 0.5454 | 1.17 | 0.277 |
| Residual | 863 | 401.5062 | 0.4652 | | |
| Total | 892 | 413.7303 | | | |

Table 0-15: Results of ANOVA of Total Fertility Rate in the ACT for the full date range (2003-2012) with interaction term between two fire area levels only (heavily and moderately affected combined, and least affected) and year.

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 1 | 0.1708 | 0.1708 | 0.37 | 0.543 |
| Year | 9 | 2.9569 | 0.3285 | 0.71 | 0.696 |
| Fire area.Year | 9 | 9.589 | 1.0654 | 2.32 | 0.014 |
| Residual | 873 | 401.7549 | 0.4602 | | |
| Total | 892 | 413.7303 | | | |

Table 0-16: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2006 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.5529 | 0.2764 | 1.21 | 0.299 |
| Residual | 172 | 39.1399 | 0.2276 | | |
| Total | 174 | 39.6928 | | | |

Table 0-17: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2007 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.2652 | 0.1326 | 0.6 | 0.551 |
| Residual | 172 | 38.1558 | 0.2218 | | |
| Total | 174 | 38.421 | | | |

Table 0-18: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2008 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.39 | 0.195 | 0.86 | 0.423 |
| Residual | 172 | 38.8027 | 0.2256 | | |
| Total | 174 | 39.1927 | | | |

Table 0-19: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2009 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.3889 | 0.1944 | 0.92 | 0.401 |
| Residual | 172 | 36.4 | 0.2116 | | |
| Total | 174 | 36.7889 | | | |

Table 0-20: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2010 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.5271 | 0.2636 | 1.03 | 0.358 |
| Residual | 172 | 43.8759 | 0.2551 | | |
| Total | 174 | 44.4031 | | | |

Table 0-21: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2011 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.2624 | 0.1312 | 0.93 | 0.398 |
| Residual | 172 | 24.355 | 0.1416 | | |
| Total | 174 | 24.6173 | | | |

Table 0-22: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample in 2012 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.26294 | 0.13147 | 1.79 | 0.17 |
| Residual | 172 | 12.61264 | 0.07333 | | |
| Total | 174 | 12.87557 | | | |

Table 0-23: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample for the full date range (2006-2012) with interaction term between fire area (highly affected, threatened or unaffected) and year.

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 2.5699 | 1.285 | 6.63 | 0.001 |
| Year | 6 | 3.9121 | 0.652 | 3.36 | 0.003 |
| Fire area.Year | 12 | 0.0794 | 0.0066 | 0.03 | 1 |
| Residual | 1204 | 233.3419 | 0.1938 | | |
| Total | 1224 | 239.9034 | | | |

Table 0-24: Results of ANOVA of Total Fertility Rate in the Victorian rural and peri-urban sample for the full date range (2006-2012) with interaction term between fire area (highly affected and threatened combined, and unaffected) and year.

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 1 | 10.3981 | 10.3981 | 104.01 | <.001 |
| Year | 6 | 1.10164 | 0.18361 | 1.84 | 0.089 |
| Fire area.Year | 6 | 0.07678 | 0.0128 | 0.13 | 0.993 |
| Residual | 819 | 81.88076 | 0.09998 | | |
| Total | 832 | 93.45727 | 0.11233 | | |

Table 0-25: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2006 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 2.1952 | 1.0976 | 7.47 | <.001 |
| Residual | 116 | 17.0343 | 0.1468 | | |
| Total | 118 | 19.2295 | | | |

Table 0-26: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2007 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 2.0571 | 1.0286 | 7.8 | <.001 |
| Residual | 116 | 15.2937 | 0.1318 | | |
| Total | 118 | 17.3508 | | | |

Table 0-27: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2008 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 1.8199 | 0.9099 | 7.89 | <.001 |
| Residual | 116 | 13.3738 | 0.1153 | | |
| Total | 118 | 15.1937 | | | |

Table 0-28: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2009 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 1.51913 | 0.75957 | 7.68 | <.001 |
| Residual | 116 | 11.47286 | 0.0989 | | |
| Total | 118 | 12.99199 | | | |

Table 0-29: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2010 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 1.1816 | 0.5908 | 5.81 | 0.004 |
| Residual | 116 | 11.7925 | 0.1017 | | |
| Total | 118 | 12.9741 | | | |

Table 0-30: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2011 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.781 | 0.3905 | 6.4 | 0.002 |
| Residual | 116 | 7.07771 | 0.06101 | | |
| Total | 118 | 7.85871 | | | |

Table 0-31: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample in 2012 divided by fire area (highly affected, threatened or unaffected).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 0.80034 | 0.40017 | 7.79 | <.001 |
| Residual | 116 | 5.95646 | 0.05135 | | |
| Total | 118 | 6.7568 | | | |

Table 0-32: Results of ANOVA of Total Fertility Rate in the Victorian suburban sample for the full date range (2006-2012) with interaction term between fire area (highly affected and threatened combined, and unaffected) and year.

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| Fire area | 2 | 14.26955 | 7.13477 | 74.62 | <.001 |
| Year | 6 | 1.10164 | 0.18361 | 1.92 | 0.075 |
| Fire area.Year | 12 | 0.4477 | 0.03731 | 0.39 | 0.967 |
| Residual | 812 | 77.63839 | 0.09561 | | |
| Total | 832 | 93.45727 | 0.11233 | | |

**APPENDIX C: ANALYSES FROM CHAPTER SEVEN**

Table 0-1: ANOVA of Children’s' weight in kilograms against listed variables in Wave One (AALSAC sample) showing predicted means for fire exposure status (either fire exposed in utero, conceived during aftermath or comparison group).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.014 | 0.014 | 0.01 | 0.908 |
| Birth weight | 1 | 110.525 | 110.525 | 103.36 | <.001 |
| Breastfed | 1 | 24.81 | 24.81 | 23.2 | <.001 |
| Study child sex | 1 | 78.453 | 78.453 | 73.37 | <.001 |
| Study child age | 1 | 609.918 | 609.918 | 570.39 | <.001 |
| Parent one weight | 3 | 0.747 | 0.249 | 0.23 | 0.874 |
| Hardship | 1 | 5.208 | 5.208 | 4.87 | 0.028 |
| Stressful life events | 1 | 0.596 | 0.596 | 0.56 | 0.456 |
| Problems | 3 | 0.847 | 0.282 | 0.26 | 0.851 |
| Coping | 2 | 0.145 | 0.073 | 0.07 | 0.934 |
| Fire exposure | 2 | 5.918 | 2.959 | 2.77 | 0.064 |
| Residual | 659 | 704.668 | 1.069 | | |
| Total | 676 | 1541.849 | 2.281 | | |

| | |
|---|---|
| | Prediction |
| Fire exposure | |
| Comparison group | 9.28 |
| Exposed in utero | 8.945 |
| Conceived in aftermath | 9.178 |
| | |
| | |
| Minimum standard error of difference | 0.1402 |
| Average standard error of difference | 0.1965 |
| Maximum standard error of difference | 0.2434 |

Table 0-2: Results of unbalanced ANOVA of Children’s' Body Mass Index against listed variables in Wave Two (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 3.016 | 3.016 | 1.21 | 0.271 |
| Breastfed | 1 | 8.904 | 8.904 | 3.58 | 0.059 |
| Study child age | 1 | 31.532 | 31.532 | 12.69 | <.001 |
| Study child sex | 1 | 19.778 | 19.778 | 7.96 | 0.005 |
| Parent one weight status | 3 | 38.536 | 12.845 | 5.17 | 0.002 |
| Hardship | 1 | 0.635 | 0.635 | 0.26 | 0.613 |
| Stressful life events | 1 | 0.152 | 0.152 | 0.06 | 0.805 |
| Problems | 3 | 1.06 | 0.353 | 0.14 | 0.935 |
| Coping | 2 | 8.725 | 4.363 | 1.76 | 0.174 |
| Fire exposure | 2 | 3.849 | 1.925 | 0.77 | 0.461 |
| Residual | 657 | 1633.092 | 2.486 | | |
| Total | 673 | 1749.28 | 2.599 | | |

Table 0-3: Results of unbalanced ANOVA of Children’s' Body Mass Index against listed variables in Wave Three (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 7.727 | 7.727 | 3.01 | 0.083 |
| Breastfed | 1 | 4.807 | 4.807 | 1.87 | 0.172 |
| Study child age | 1 | 1.349 | 1.349 | 0.53 | 0.469 |
| Study child sex | 1 | 6.932 | 6.932 | 2.7 | 0.101 |
| Parent one weight status | 3 | 107.113 | 35.704 | 13.91 | <.001 |
| Hardship | 1 | 0.832 | 0.832 | 0.32 | 0.569 |
| Stressful life events | 1 | 10.623 | 10.623 | 4.14 | 0.042 |
| Problems | 3 | 7.595 | 2.532 | 0.99 | 0.399 |
| Coping | 2 | 8.957 | 4.479 | 1.74 | 0.175 |
| Fire exposure | 2 | 5.32 | 2.66 | 1.04 | 0.355 |
| Residual | 721 | 1850.739 | 2.567 | | |
| Total | 737 | 2011.996 | 2.73 | | |

Table 0-4: Results of unbalanced ANOVA of Children's' Body Mass Index against listed variables in Wave Four (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 0.03 | 0.03 | 0.01 | 0.933 |
| Study child age | 1 | 0.669 | 0.669 | 0.16 | 0.693 |
| Study child sex | 1 | 5.629 | 5.629 | 1.32 | 0.252 |
| Parent one weight status | 3 | 312.787 | 104.262 | 24.36 | <.001 |
| Hardship | 1 | 6.177 | 6.177 | 1.44 | 0.23 |
| Stressful life events | 1 | 3.143 | 3.143 | 0.73 | 0.392 |
| Problems | 3 | 13.533 | 4.511 | 1.05 | 0.368 |
| Coping | 3 | 22.319 | 7.44 | 1.74 | 0.158 |
| Fire exposure | 2 | 3.172 | 1.586 | 0.37 | 0.69 |
| Residual | 831 | 3556.693 | 4.28 | | |
| Total | 847 | 3924.152 | 4.633 | | |

Table 0-5: Results of unbalanced ANOVA of Children's' Body Mass Index against listed variables in Wave Five (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 10.795 | 10.795 | 1.32 | 0.25 |
| Study child age | 1 | 44.67 | 44.67 | 5.48 | 0.019 |
| Study child sex | 1 | 29.363 | 29.363 | 3.6 | 0.058 |
| Parent one weight status | 3 | 814.639 | 271.546 | 33.31 | <.001 |
| Hardship | 1 | 13.542 | 13.542 | 1.66 | 0.198 |
| Stressful life events | 1 | 2.724 | 2.724 | 0.33 | 0.563 |
| Problems | 3 | 8.196 | 2.732 | 0.34 | 0.8 |
| Coping | 3 | 14.8 | 4.933 | 0.61 | 0.612 |
| Fire exposure | 2 | 5.773 | 2.886 | 0.35 | 0.702 |
| Residual | 805 | 6563.247 | 8.153 | | |
| Total | 821 | 7507.75 | 9.145 | | |

Table 0-6: Results of unbalanced ANOVA of Parents' Evaluation of Developmental Status Psychosocial score against listed variables in Wave Two (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 708.3 | 708.3 | 6.22 | 0.013 |
| Study child sex | 1 | 216.4 | 216.4 | 1.9 | 0.169 |
| Parent one weight status | 3 | 553.1 | 184.4 | 1.62 | 0.184 |
| Hardship | 1 | 87.9 | 87.9 | 0.77 | 0.38 |
| Parent one depressed in past two weeks | 1 | 1144.1 | 1144.1 | 10.05 | 0.002 |
| Stressful life events | 1 | 496.3 | 496.3 | 4.36 | 0.037 |
| Problems | 3 | 4873.7 | 1624.6 | 14.27 | <.001 |
| Coping | 2 | 857.6 | 428.8 | 3.77 | 0.024 |
| Fire exposure | 2 | 98.2 | 49.1 | 0.43 | 0.65 |
| Residual | 664 | 75612.5 | 113.9 | | |
| Total | 679 | 84648 | 124.7 | | |

Table 0-7: Results of unbalanced ANOVA of Parents' Evaluation of Developmental Status Psychosocial score against listed variables in Wave Three (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 218.4 | 218.4 | 1.72 | 0.19 |
| Study child sex | 1 | 449.1 | 449.1 | 3.53 | 0.061 |
| Parent one weight status | 3 | 401.6 | 133.9 | 1.05 | 0.369 |
| Hardship | 1 | 2299.7 | 2299.7 | 18.08 | <.001 |
| Stressful life events | 1 | 687.4 | 687.4 | 5.4 | 0.02 |
| Parent one depressed in past two weeks | 1 | 1189.9 | 1189.9 | 9.36 | 0.002 |
| Problems | 3 | 3679.4 | 1226.5 | 9.64 | <.001 |
| Coping | 2 | 1805.3 | 902.7 | 7.1 | <.001 |
| Fire exposure | 2 | 81.7 | 40.9 | 0.32 | 0.725 |
| Residual | 721 | 91697.2 | 127.2 | | |
| Total | 736 | 102509.6 | 139.3 | | |

Table 0-8: Results of unbalanced ANOVA of Parents' Evaluation of Developmental Status Psychosocial score against listed variables in Wave Four (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 250.4 | 250.4 | 1.62 | 0.204 |
| Study child sex | 1 | 422.8 | 422.8 | 2.73 | 0.099 |
| Parent one weight status | 3 | 879.1 | 293 | 1.89 | 0.129 |
| Hardship | 1 | 7261 | 7261 | 46.95 | <.001 |
| Stressful life events | 1 | 1737.7 | 1737.7 | 11.24 | <.001 |
| Parent one depressed in past two weeks | 1 | 4478.3 | 4478.3 | 28.96 | <.001 |
| Problems | 3 | 5721.9 | 1907.3 | 12.33 | <.001 |
| Coping | 2 | 833.8 | 416.9 | 2.7 | 0.068 |
| Fire exposure | 2 | 1020.6 | 510.3 | 3.3 | 0.037 |
| Residual | 842 | 130207.1 | 154.6 | | |
| Total | 857 | 152812.7 | 178.3 | | |

Table 0-9: ANOVA of Parents' Evaluation of Developmental Status Psychosocial score against listed variables in Wave Five (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 191.5 | 191.5 | 0.95 | 0.331 |
| Study child sex | 1 | 946.8 | 946.8 | 4.68 | 0.031 |
| Parent one weight status | 3 | 3393.8 | 1131.3 | 5.59 | <.001 |
| Hardship | 1 | 5059.6 | 5059.6 | 24.99 | <.001 |
| Stressful life events | 1 | 1479.6 | 1479.6 | 7.31 | 0.007 |
| Parent one depressed in past two weeks | 1 | 3961.4 | 3961.4 | 19.56 | <.001 |
| Problems | 3 | 4701.6 | 1567.2 | 7.74 | <.001 |
| Coping | 3 | 1541.4 | 513.8 | 2.54 | 0.056 |
| Fire exposure | 2 | 154.7 | 77.4 | 0.38 | 0.683 |
| Residual | 814 | 164829.2 | 202.5 | | |
| Total | 830 | 186259.7 | 224.4 | | |

Table 0-10: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Two (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 1386.1 | 1386.1 | 8.26 | 0.004 |
| Study child sex | 1 | 254.6 | 254.6 | 1.52 | 0.219 |
| Parent one weight status | 3 | 473.4 | 157.8 | 0.94 | 0.421 |
| Hardship | 1 | 408 | 408 | 2.43 | 0.119 |
| Stressful life events | 1 | 2648.1 | 2648.1 | 15.77 | <.001 |
| Parent one depressed in past two weeks | 1 | 675.1 | 675.1 | 4.02 | 0.045 |
| Problems | 3 | 5956 | 1985.3 | 11.83 | <.001 |
| Coping | 2 | 1360.3 | 680.2 | 4.05 | 0.018 |
| Fire exposure | 2 | 714.4 | 357.2 | 2.13 | 0.12 |
| Residual | 662 | 111143.7 | 167.9 | | |
| Total | 677 | 125019.6 | 184.7 | | |

Table 0-11: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Three (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 129.1 | 129.1 | 0.74 | 0.39 |
| Study child sex | 1 | 17.8 | 17.8 | 0.1 | 0.75 |
| Parent one weight status | 3 | 533.4 | 177.8 | 1.02 | 0.383 |
| Hardship | 1 | 3059.6 | 3059.6 | 17.54 | <.001 |
| Stressful life events | 1 | 2238.4 | 2238.4 | 12.83 | <.001 |
| Parent one depressed in past two weeks | 1 | 2491.1 | 2491.1 | 14.28 | <.001 |
| Problems | 3 | 5601.1 | 1867 | 10.7 | <.001 |
| Coping | 2 | 1433.3 | 716.6 | 4.11 | 0.017 |
| Fire exposure | 2 | 14.5 | 7.2 | 0.04 | 0.959 |
| Residual | 721 | 125752.6 | 174.4 | | |
| Total | 736 | 141270.8 | 191.9 | | |

Table 0-12: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Four (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 52.5 | 52.5 | 0.29 | 0.592 |
| Study child sex | 1 | 242.6 | 242.6 | 1.33 | 0.249 |
| Parent one weight status | 3 | 710.2 | 236.7 | 1.3 | 0.274 |
| Hardship | 1 | 8530.4 | 8530.4 | 46.72 | <.001 |
| Stressful life events | 1 | 2939.4 | 2939.4 | 16.1 | <.001 |
| Parent one depressed in past two weeks | 1 | 4453.2 | 4453.2 | 24.39 | <.001 |
| Problems | 3 | 7738.2 | 2579.4 | 14.13 | <.001 |
| Coping | 2 | 1556 | 778 | 4.26 | 0.014 |
| Fire exposure | 2 | 1387.9 | 694 | 3.8 | 0.023 |
| Residual | 842 | 153727.5 | 182.6 | | |
| Total | 857 | 181337.7 | 211.6 | | |

Table 0-13: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Five (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 32.7 | 32.7 | 0.12 | 0.724 |
| Study child sex | 1 | 709.7 | 709.7 | 2.7 | 0.101 |
| Parent one weight status | 3 | 2421.2 | 807.1 | 3.07 | 0.027 |
| Hardship | 1 | 2945.8 | 2945.8 | 11.22 | <.001 |
| Stressful life events | 1 | 1736.2 | 1736.2 | 6.61 | 0.01 |
| Parent one depressed in past two weeks | 1 | 5145.6 | 5145.6 | 19.59 | <.001 |
| Problems | 3 | 6727.7 | 2242.6 | 8.54 | <.001 |
| Coping | 3 | 1140 | 380 | 1.45 | 0.228 |
| Fire exposure | 2 | 60.4 | 30.2 | 0.12 | 0.891 |
| Residual | 814 | 213766.7 | 262.6 | | |
| Total | 830 | 234686.1 | 282.8 | | |

Table 0-14: ANOVA of Parents' Evaluation of Developmental Status Social functioning score against listed variables in Wave Two (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 155.2 | 155.2 | 1.05 | 0.307 |
| Study child sex | 1 | 248.1 | 248.1 | 1.67 | 0.196 |
| Parent one weight status | 3 | 486.9 | 162.3 | 1.1 | 0.35 |
| Hardship | 1 | 2.9 | 2.9 | 0.02 | 0.89 |
| Stressful life events | 1 | 162 | 162 | 1.09 | 0.296 |
| Parent one depressed in past two weeks | 1 | 327.5 | 327.5 | 2.21 | 0.138 |
| Problems | 3 | 3856.6 | 1285.5 | 8.67 | <.001 |
| Coping | 2 | 623.3 | 311.6 | 2.1 | 0.123 |
| Fire exposure | 2 | 88.4 | 44.2 | 0.3 | 0.742 |
| Residual | 657 | 97367.3 | 148.2 | | |
| Total | 672 | 103318.2 | 153.7 | | |

Table 0-15: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Three (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 358.1 | 358.1 | 1.9 | 0.168 |
| Study child sex | 1 | 1443.3 | 1443.3 | 7.68 | 0.006 |
| Parent one weight status | 3 | 328.6 | 109.5 | 0.58 | 0.626 |
| Hardship | 1 | 1828.7 | 1828.7 | 9.73 | 0.002 |
| Stressful life events | 1 | 14.6 | 14.6 | 0.08 | 0.781 |
| Parent one depressed in past two weeks | 1 | 375.9 | 375.9 | 2 | 0.158 |
| Problems | 3 | 2239.8 | 746.6 | 3.97 | 0.008 |
| Coping | 2 | 2182 | 1091 | 5.8 | 0.003 |
| Fire exposure | 2 | 169.5 | 84.8 | 0.45 | 0.637 |
| Residual | 718 | 134951.2 | 188 | | |
| Total | 733 | 143891.8 | 196.3 | | |

Table 0-16: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Four (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 620.4 | 620.4 | 2.45 | 0.118 |
| Study child sex | 1 | 679.3 | 679.3 | 2.68 | 0.102 |
| Parent one weight status | 3 | 956.1 | 318.7 | 1.26 | 0.287 |
| Hardship | 1 | 6230.6 | 6230.6 | 24.61 | <.001 |
| Stressful life events | 1 | 860.8 | 860.8 | 3.4 | 0.066 |
| Parent one depressed in past two weeks | 1 | 4676.4 | 4676.4 | 18.47 | <.001 |
| Problems | 3 | 4321.9 | 1440.6 | 5.69 | <.001 |
| Coping | 2 | 456.9 | 228.4 | 0.9 | 0.406 |
| Fire exposure | 2 | 584.8 | 292.4 | 1.16 | 0.316 |
| Residual | 840 | 212636.2 | 253.1 | | |
| Total | 855 | 232023.4 | 271.4 | | |

Table 0-17: ANOVA of Parents' Evaluation of Developmental Status Emotional functioning score against listed variables in Wave Five (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 1056.4 | 1056.4 | 3.71 | 0.055 |
| Study child sex | 1 | 1153.4 | 1153.4 | 4.05 | 0.045 |
| Parent one weight status | 3 | 5535 | 1845 | 6.48 | <.001 |
| Hardship | 1 | 7482.9 | 7482.9 | 26.27 | <.001 |
| Stressful life events | 1 | 1196.9 | 1196.9 | 4.2 | 0.041 |
| Parent one depressed in past two weeks | 1 | 3120.1 | 3120.1 | 10.95 | <.001 |
| Problems | 3 | 3535.4 | 1178.5 | 4.14 | 0.006 |
| Coping | 3 | 2194.7 | 731.6 | 2.57 | 0.053 |
| Fire exposure | 2 | 442.8 | 221.4 | 0.78 | 0.46 |
| Residual | 812 | 231333.4 | 284.9 | | |
| Total | 828 | 257050.9 | 310.4 | | |

Table 0-18: ANOVA of maternal separation anxiety against listed variables in Wave One (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 6.2578 | 6.2578 | 8.85 | 0.003 |
| Mother's age | 1 | 2.6049 | 2.6049 | 3.69 | 0.055 |
| Breastfed at 6 months | 1 | 0.1672 | 0.1672 | 0.24 | 0.627 |
| Study child age | 1 | 8.2528 | 8.2528 | 11.68 | <.001 |
| Hardship | 1 | 8.915 | 8.915 | 12.61 | <.001 |
| Stressful life events | 1 | 0.5909 | 0.5909 | 0.84 | 0.361 |
| Parent one depressed in past two weeks | 1 | 1.395 | 1.395 | 1.97 | 0.16 |
| Problems | 3 | 4.0278 | 1.3426 | 1.9 | 0.128 |
| Coping | 2 | 0.5012 | 0.2506 | 0.35 | 0.702 |
| Fire exposure | 2 | 1.441 | 0.7205 | 1.02 | 0.361 |
| Residual | 883 | 624.0712 | 0.7068 | | |
| Total | 897 | 658.2249 | 0.7338 | | |

Table 0-19: Logistic regression of any maternal alcohol consumption during gestation controlling for listed variables in Wave One (AALSAC sample).

| Source | d.f. | deviance | mean deviance | deviance ratio | approx. chi pr |
|---|---|---|---|---|---|
| Regression | 13.000 | 47.000 | 3.643 | 3.640 | <.001 |
| Residual | 904.000 | 1154.000 | 1.277 | | |
| Total | 917.000 | 1201.000 | 1.310 | | |
| | | | | | |
| **Parameter** | **estimate** | **s.e.** | **t(*)** | **t pr.** | **OR** |
| Constant | 2.328 | 0.586 | 3.970 | <.001 | 10.250 |
| Child ATSI status: Yes | 0.993 | 0.435 | 2.290 | 0.022 | 2.700 |
| Mother's age | -0.044 | 0.014 | -3.080 | 0.002 | 0.957 |
| Partner status: Partnered | -0.680 | 0.306 | -2.220 | 0.026 | 0.507 |
| Breastfed at 6 months: No | 0.605 | 0.294 | 2.060 | 0.040 | 1.832 |
| Smoked during gestation | 0.382 | 0.187 | 2.040 | 0.041 | 1.466 |
| Hardship scale | 0.020 | 0.085 | 0.230 | 0.814 | 1.020 |
| Problems: Few problems or stresses | -0.174 | 0.250 | -0.700 | 0.485 | 0.840 |
| Problems: Some problems and stresses | -0.128 | 0.283 | -0.450 | 0.651 | 0.880 |
| Problems: Many problems and stresses | 0.345 | 0.408 | 0.840 | 0.398 | 1.411 |
| Coping: Very well | -0.024 | 0.170 | -0.140 | 0.886 | 0.976 |
| Coping: Extremely well | 0.055 | 0.258 | 0.210 | 0.830 | 1.057 |
| Fire exposure – In utero during fire | -0.437 | 0.231 | -1.890 | 0.058 | 0.646 |
| Fire exposure – Conceived in aftermath | -0.532 | 0.324 | -1.640 | 0.101 | 0.588 |
| | | | | | |
| Parameters for factors are differences compared with the reference level: | | | | | |
| | Factor | Reference level | | | |
| | Child ATSI status | No | | | |
| | Partner status | One parent only | | | |
| | Breastfed at 6 months | Yes | | | |
| | Problems | No problems or stress | | | |
| | Coping | Fairly well | | | |
| | Fire exposure | Comparison group | | | |

Table 0-20: Multiple linear regression of daily rate of maternal alcohol consumption during gestation controlling for listed variables in Wave One (AALSAC sample).

| Source | d.f. | s.s. | m.s. | v.r. | F pr. |
|---|---|---|---|---|---|
| Regression | 13.000 | 13.200 | 1.017 | 3.410 | <.001 |
| Residual | 865.000 | 258.000 | 0.298 | | |
| Total | 878.000 | 271.300 | 0.309 | | |
| | | | | | |
| **Parameter** | | **estimate** | **s.e.** | **t(865)** | **t pr.** |
| Constant | | -0.288 | 0.142 | -2.030 | 0.043 |
| Child ATSI status: Yes | | -0.167 | 0.090 | -1.850 | 0.065 |
| Mothers' age | | 0.015 | 0.004 | 4.030 | <.001 |
| Partner status: Partnered | | 0.092 | 0.068 | 1.370 | 0.172 |
| Breastfed at 6 months: No | | -0.113 | 0.069 | -1.640 | 0.101 |
| Average cigarettes per day in gestation | | 0.018 | 0.005 | 3.720 | <.001 |
| Hardship scale | | -0.022 | 0.021 | -1.030 | 0.304 |
| Problems: Few problems or stresses | | 0.036 | 0.065 | 0.550 | 0.583 |
| Problems: Some problems and stresses | | 0.034 | 0.073 | 0.460 | 0.645 |
| Problems: Many problems and stresses | | -0.057 | 0.102 | -0.560 | 0.576 |
| Coping: Very well | | -0.009 | 0.044 | -0.210 | 0.836 |
| Coping: Extremely well | | -0.069 | 0.067 | -1.030 | 0.303 |
| Fire exposure: In utero | | -0.025 | 0.063 | -0.400 | 0.688 |
| Fire exposure: Conceived in aftermath | | -0.009 | 0.087 | -0.100 | 0.919 |
| | | | | | |
| Parameters for factors are differences compared with the reference level: | | | | | |
| | Factor | Reference level | | | |
| | Child ATSI status | No | | | |
| | Partner status | One parent only | | | |
| | Breastfed status | Yes | | | |
| | Problems | No problems or stress | | | |
| | Coping | Fairly well | | | |
| | Fire exposure | Comparison group | | | |

Table 0-21: Logistic regression of any maternal cigarette smoking during gestation controlling for listed variables in Wave One (AALSAC sample).

| Source | d.f. | deviance | mean deviance | deviance ratio | approx. chi pr |
|---|---|---|---|---|---|
| Regression | 13 | 134.3 | 10.3271 | 10.33 | <.001 |
| Residual | 904 | 817.8 | 0.9047 | | |
| Total | 917 | 952.1 | 1.0383 | | |
| | | | | | |
| **Parameter** | **estimate** | **s.e.** | **t(*)** | **t pr.** | **OR** |
| Constant | -0.961 | 0.679 | -1.42 | 0.157 | 0.383 |
| Child ATSI status: Yes | -1.406 | 0.356 | -3.95 | <.001 | 0.245 |
| Mother's age | 0.0651 | 0.0173 | 3.77 | <.001 | 1.067 |
| Partner status: Partnered | 0.697 | 0.273 | 2.56 | 0.011 | 2.008 |
| Breastfed at 6 months: No | -0.879 | 0.274 | -3.21 | 0.001 | 0.415 |
| Alcohol during gestation: Yes | 0.382 | 0.188 | 2.03 | 0.043 | 1.465 |
| Hardship scale | -0.5391 | 0.0896 | -6.01 | <.001 | 0.583 |
| Problems: Few problems or stresses | -0.093 | 0.335 | -0.28 | 0.781 | 0.911 |
| Problems: Some problems and stresses | 0.016 | 0.371 | 0.04 | 0.965 | 1.016 |
| Problems: Many problems and stresses | 0.128 | 0.491 | 0.26 | 0.795 | 1.136 |
| Coping: Very well | 0.028 | 0.209 | 0.13 | 0.895 | 1.028 |
| Coping: Extremely well | 0.256 | 0.326 | 0.79 | 0.432 | 1.292 |
| Fire exposure – Gestation during fire | 0.039 | 0.313 | 0.12 | 0.901 | 1.04 |
| Fire exposure – Conceived in aftermath | -0.103 | 0.411 | -0.25 | 0.802 | 0.902 |
| | | | | | |
| Parameters for factors are differences compared with the reference level: | | | | | |
| | Factor | Reference level | | | |
| | Child ATSI status | No | | | |
| | Partner status | One parent only | | | |
| | Breastfed at 6 months | Yes | | | |
| | Problems | No problems or stress | | | |
| | Coping | Fairly well | | | |
| | Fire exposure | Comparison group | | | |

Table 0-22: Multiple linear regression of daily rate of maternal cigarette smoking during gestation controlling for listed variables in Wave One (AALSAC sample).

| Source | d.f. | s.s. | m.s. | v.r. | F pr. |
|---|---|---|---|---|---|
| Regression | 13 | 2237 | 172.07 | 12.37 | <.001 |
| Residual | 864 | 12015 | 13.91 | | |
| Total | 877 | 14252 | 16.25 | | |
| | | | | | |
| **Parameter** | | **estimate** | **s.e.** | **t(864)** | **t pr.** |
| Constant | | 2.739 | 0.965 | 2.84 | 0.005 |
| Child ATSI status: Yes | | 3.808 | 0.602 | 6.33 | <.001 |
| Mother's age | | -0.0566 | 0.0248 | -2.28 | 0.023 |
| Partner status: Partnered | | -0.955 | 0.461 | -2.07 | 0.038 |
| Breastfed at 6 months: No | | 1.81 | 0.465 | 3.89 | <.001 |
| Average daily alcoholic drinks during gestation | | 3.99 | 1.01 | 3.93 | <.001 |
| Hardship scale | | 0.716 | 0.143 | 4.99 | <.001 |
| Problems: Few problems or stresses | | 0.694 | 0.443 | 1.57 | 0.117 |
| Problems: Some problems and stresses | | 0.482 | 0.498 | 0.97 | 0.334 |
| Problems: Many problems and stresses | | 0.894 | 0.694 | 1.29 | 0.198 |
| Coping: Very well | | -0.291 | 0.303 | -0.96 | 0.338 |
| Coping: Extremely well | | -0.236 | 0.458 | -0.52 | 0.607 |
| Fire exposure – Gestation during fire | | 0.345 | 0.431 | 0.8 | 0.424 |
| Fire exposure – Conceived in aftermath | | -0.051 | 0.595 | -0.09 | 0.932 |
| | | | | | |
| Parameters for factors are differences compared with the reference level: | | | | | |
| | Factor | Reference level | | | |
| | Child ATSI status | No | | | |
| | Partner status | One parent only | | | |
| | Breastfed at 6 months | Yes | | | |
| | Problems | No problems or stress | | | |
| | Coping | Fairly well | | | |
| | Fire exposure | Comparison group | | | |

Table 0-23: ANOVA of number of prenatal care visits during gestation controlling for listed variables in Wave One (AALSAC sample).

| Source | Degrees of freedom | Sum of squares | Mean of square | Variance ratio | *p* |
|---|---|---|---|---|---|
| ATSI status | 1 | 0 | 0 | 0 | 0.992 |
| Mother's age | 1 | 6.105 | 6.105 | 14.26 | <.001 |
| Partner status | 1 | 4.309 | 4.309 | 10.06 | 0.002 |
| Breastfed at 6 months | 1 | 0.298 | 0.298 | 0.7 | 0.404 |
| Parent one weight status | 3 | 3.072 | 1.024 | 2.39 | 0.067 |
| Hardship | 1 | 0.105 | 0.105 | 0.25 | 0.62 |
| Stressful life events | 1 | 2.038 | 2.038 | 4.76 | 0.029 |
| Parent one depressed in past two weeks | 1 | 1.031 | 1.031 | 2.41 | 0.121 |
| Prenatal provider type | 3 | 3.988 | 1.329 | 3.1 | 0.026 |
| Problems | 3 | 2.724 | 0.908 | 2.12 | 0.096 |
| Coping | 3 | 1.521 | 0.507 | 1.18 | 0.315 |
| Fire exposure | 2 | 6.343 | 3.172 | 7.41 | <.001 |
| Residual | 826 | 353.711 | 0.428 | | |
| Total | 847 | 385.245 | 0.455 | | |

Table 0-24: Logistic regression of any maternal vitamin supplement consumption during gestation controlling for listed variables in Wave One (AALSAC sample).

| Source | d.f. | deviance | mean deviance | deviance ratio | approx chi pr |
|---|---|---|---|---|---|
| Regression | 21.000 | 68.400 | 3.255 | 3.260 | <.001 |
| Residual | 826.000 | 620.700 | 0.752 | | |
| Total | 847.000 | 689.100 | 0.814 | | |
| | | | | | |
| **Parameter** | **estimate** | **s.e.** | **t(*)** | **t pr.** | **antilog of estimate** |
| Constant | -5.358 | 0.981 | -5.460 | <.001 | 0.005 |
| Child ATSI status: Yes | -0.460 | 0.537 | -0.860 | 0.391 | 0.631 |
| Mother's age | 0.029 | 0.016 | 1.870 | 0.062 | 1.030 |
| Partner status: Partnered | 0.895 | 0.441 | 2.030 | 0.043 | 2.446 |
| Breastfed at 6 months: No | -1.194 | 0.463 | -2.580 | 0.010 | 0.303 |
| Parent 1 weight: Obese | -0.201 | 0.218 | -0.920 | 0.356 | 0.818 |
| Parent 1 weight: Overweight | -0.111 | 0.189 | -0.590 | 0.558 | 0.895 |
| Parent 1 weight: Underweight | 0.171 | 0.236 | 0.720 | 0.469 | 1.187 |
| Hardship scale | -0.130 | 0.108 | -1.210 | 0.228 | 0.878 |
| Stressful life events scale | -0.073 | 0.060 | -1.220 | 0.222 | 0.930 |
| Parent 1 depressed in past two weeks: No | -0.479 | 0.174 | -2.750 | 0.006 | 0.620 |
| Prenatal provider: Midwife or nurse | 0.676 | 0.221 | 3.060 | 0.002 | 1.966 |
| Prenatal provider: Obstetrician | 0.366 | 0.179 | 2.050 | 0.041 | 1.443 |
| Prenatal provider: Shared care/Other | 0.374 | 0.308 | 1.210 | 0.225 | 1.454 |
| Problems: Few problems or stresses | 1.068 | 0.331 | 3.230 | 0.001 | 2.910 |
| Problems: Some problems and stresses | 1.009 | 0.367 | 2.750 | 0.006 | 2.743 |
| Problems: Many problems and stresses | 1.526 | 0.465 | 3.280 | 0.001 | 4.602 |
| Coping Fairly well | 0.743 | 0.623 | 1.190 | 0.233 | 2.103 |
| Coping Very well | 1.046 | 0.633 | 1.650 | 0.099 | 2.847 |
| Coping Extremely well | 1.476 | 0.670 | 2.200 | 0.028 | 4.374 |

| | | | | | |
|---|---|---|---|---|---|
| Fire exposure – Gestation during fire | 0.064 | 0.239 | 0.270 | 0.787 | 1.067 |
| Fire exposure – Conceived in aftermath | -0.215 | 0.374 | -0.580 | 0.565 | 0.806 |
| | | | | | |
| Parameters for factors are differences compared with the reference level: | | | | | |
| | Factor | Reference level | | | |
| | Study child ATSI Status | No | | | |
| | Partner status | One parent only | | | |
| | Breastfed until 6 months | Yes | | | |
| | Parent one weight group | Normal weight | | | |
| | Parent one depressed in past two weeks | Yes | | | |
| | Prenatal provider | General Practitioner (GP) | | | |
| | Problems | No problems or stress | | | |
| | Coping | A little | | | |
| | Fire exposure | Comparison group | | | |

**APPENDIX D: ANALYSES FROM CHAPTER EIGHT**

Table 0-1: Students t-test of PSS total stress scores by the fire (Canberra or Black Saturday) to which participants were exposed $p$ = 0.007 (n = 32).

| Group | n | Mean | Std. Error | Std. Deviation | CI (95%) |
|---|---|---|---|---|---|
| Canberra | 14 | 10.14 | 0.645 | 1.978 | 11.183 – 13.15 |
| Black Saturday | 18 | 12.16 | 0.466 | 2.413 | 8.749 – 11.536 |

Table 0-2: Students' t-test comparing Perceived Stress Scale total stress score against evacuation decision (p = 0.63).

| Group | n | Mean | Std. Error | Std. Deviation | CI (95%) |
|---|---|---|---|---|---|
| Did not evacuate | 16 | 11.125 | 0.569 | 2.277 | 9.912 – 12.338 |
| Evacuated | 20 | 11.4 | 0.550 | 2.460 | 10.250 – 12.550 |

Table 0-3: Students' t-test comparing PSS total stress score of late evacuators against other evacuation decisions (p = 0.18).

| Group | n | Mean | Std. Error | Std. Deviation | CI (95%) |
|---|---|---|---|---|---|
| All other categories | 26 | 11.5 | 0.497 | 2.534 | 10.477 – 12.523 |
| Late evacuation | 10 | 10.7 | 0.559 | 1.767 | 9.436 – 11.964 |

Table 0-4: Table 6: Students' t-test comparing PSS total stress score of early evacuators against other evacuation decisions (p = 0.70).

| Group | n | Mean | Std. Error | Std. Deviation | CI (95%) |
|---|---|---|---|---|---|
| All other categories | 29 | 11.172 | 0.415 | 2.237 | 10.321 – 12.023 |
| Early evacuators | 7 | 11.714 | 1.107 | 2.928 | 9.007 – 14.422 |

Table 0-5: Students' t-test of PSS total stress scores by the fire to which participants were exposed (Black Saturday or Canberra) $p$ = 0.007 (n = 32).

| Group | Observations | Mean | Std. Error | Std. Deviation | CI (95%) |
|---|---|---|---|---|---|
| Canberra | 14 | 10.14 | 0.645 | 1.978 | 11.183 – 13.15 |
| Black Saturday | 18 | 12.16 | 0.466 | 2.413 | 8.749 – 11.536 |

*Survey Text*

Thank you for your interest in this study. If you are a woman who was pregnant during a bushfire, you are invited to take this survey about your experience.

Why are we doing this research?

This survey is part of a larger research project that aims to help understand how bushfires affect people. Some research suggests that high-stress experiences during pregnancy may influence birth outcomes, such as gestational age and birth weight. To help us quantify the stress experienced during bushfires, this survey asks about the experience of bushfires for pregnant women.

About the survey:

The survey should take less than 10 minutes to complete. The survey is anonymous, voluntary and you may skip any question that you don't wish to answer. Questions cover your experience during the fire and the impact on your health and wellbeing following the fire. These questions may be distressing for some people. If you don't wish to complete the survey – simply close the window.

It can be difficult answering questions about frightening events that have had a major impact on you and your family, even when these events are in the past. If you're feeling more distressed after this survey, the following resources may be useful:

- Your own GP. Your GP can be an excellent resource and is generally aware of the referral services in your region that can support you.

- Lifeline offers telephone and online counselling, as well as information on other services. Contact 13 11 14 or at www.lifeline.org.au (please copy and paste into your browser).

- Beyond Blue. Further information about Post Traumatic Stress Disorder, and responses, including online communities, can be accessed here: www.beyondblue.org.au/the-facts/anxiety/types-of-anxiety/ptsd (please copy and paste into your browser).

Please print this page so you can refer to it later.

Ethical approval:

The ethical aspects of this research have been approved by the ANU Human Research Ethics Committee. If you have any concerns or complaints about how this research has been conducted, please contact:

Ethics Manager
The ANU Human Research Ethics Committee
The Australian National University
Telephone: +61 2 6125 3427
Email: Human.Ethics.Officer@anu.edu.au

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This section asks some questions about where you lived and what age you were at the time of the fire.

1. Which bushfire were you exposed to?

2009 Black Saturday

2003 Canberra Fires

Another fire (please specify)

2. How many weeks pregnant were you when the fire started?

3. What suburb or council area did you live in during the fire?

4. What was your age at the time of the fire?

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Pregnancy during bushfires survey

Fire Exposure

This section asks questions about what happened to you during and after the fire.

5. Before the fire, did you feel well connected to the local community?

Very much,

Somewhat,

Not very much,

Not at all

6. Before the fire did you have any diagnosis of a mental illness?

No,

Yes (please specify)

7. What was your direct experience of the fire? Please tick as many boxes as apply.

I was present when the fire front passed (for instance, if you were sheltering in the home when the fire passed over)

I remained in the area but the fire front did not reach our home

I left the area shortly before the fire arrived

I left the area well before the fire arrived

Other (please specify)

8. What losses did you suffer in the fire? Please tick as many boxes as apply.

Houses in my community were burnt or damaged

My land and/or farm buildings were damaged or destroyed

I was unable to return home for more than a month

I was unable to return home for more than a week

I was unable to return home for more than 24 hours

Members of my family were injured in the fire

Friends were injured in the fire

Houses in my street were burnt or damaged

People I knew in the local community were killed

My house was destroyed (couldn't be lived in)

Buildings in my local town were damaged or destroyed

My house was damaged (couldn't be lived in) but not destroyed

My property (such as cars, farm equipment and caravans) was destroyed

My pets were killed in the fire

I was without power for 24 hours or more

My stock were killed in the fire

People I knew in the local community were injured

My house was damaged but could still be lived in

Members of my family were killed in the fire

Friends were killed in the fire

I was evacuated

I moved permanently from the area following the fires

Other (please specify)

9. For this question please tick as many boxes as apply. During and after the fire, did you::

Feel scared or terrified

Worry that you might die

Feel that events were beyond your control

Have nightmares about the fire

None of these

Other (please specify)

**Fire Exposure**

This section asks about how you felt during the fire.

10. In the months following the fire, how often did you feel that you were unable to control the important things in your life?

Never

Almost never

Sometimes

Fairly often

Very often

11. In the months following the fire, how often did you feel confident about your ability to handle your personal problems?

Never

Almost never

Sometimes

Fairly often

Very often

12. In the months following the fire, how often did you feel that things were going your way?

Never

Almost never

Sometimes

Fairly often

Very often

13. In the months following the fire, how often did you feel that you had difficulties piling up so high that you could not overcome them?

Never

Almost never

Sometimes

Fairly often

Very often

14. In the year following the fire, did you have disrupted sleep?

Yes, following the fire but it’s now resolved

Yes, I continue to have disrupted sleep

Yes, I had disrupted sleep before and after the fire

No

**Health behaviours**

This section asks about whether your health behaviours changed following the fire.

15. Did your use of prenatal care (i.e. your GP, maternal health nurse or other prenatal health services) change after the fire but while you were still pregnant?

No, my access to prenatal care remained about the same

Yes, I used less prenatal care

Yes, I used more prenatal care

Other (please specify)

16. Did your consumption of prenatal vitamins change after the fire but while you were still pregnant?

No, I didn’t take prenatal vitamins before the fire

No, my consumption of prenatal vitamins remained the same

Yes, I began taking prenatal vitamins

Yes, I stopped taking prenatal vitamins

Other (please specify)

17. Did your smoking behaviour change following the fire?

No, I didn’t smoke before the fire

No, my smoking remained the same

Yes, I increased my cigarette consumption

Yes, I decreased my cigarette consumption

Other (please specify)

18. Did your alcohol consumption change following the fire?

No, I didn’t drink alcohol before the fire

No, my alcohol consumption remained the same

Yes, I increased my alcohol consumption

Yes, I decreased my alcohol consumption

Other (please specify)

**Life course section**

This section asks about the baby that you were carrying during the fire.

19. At how many weeks did you give birth to the child you were carrying during the fire? Please refer to your ‘baby book’ if you are unsure.

20. What was the birth weight of the child you were carrying during the fire? Please refer to your ‘baby book’ if you are unsure.

21. What is the sex of the child you were carrying during the fire?

What is the sex of the child you were carrying during the fire?

Male

Female

Unknown/Undetermined

22. In terms of your own birth, were you born early or at a low birth weight? If you're unsure, choose the answer which you are most certain about or simply select 'Can't recall/Unsure'.

'Can't recall/Unsure'.

Yes, at both low birth weight and pre-term

Yes, pre-term only

Yes, at low weight only

No

Can't recall/Unsure

**Assistance and support**

This section asks about any support you received during and following the fire.

23. What support or assistance was helpful to you during and following the fire, and what support do you wish had been available?

24. Do you have any other comments?

Thank you

Thank you for completing this survey. You have assisted important research into the effects of bushfires on pregnant women.

If you wish to find further help or support, please consider contacting one or more of the following:

- Your own GP. Your GP can be an excellent resource and is generally aware of the referral services in your region that can support you. You may wish to discuss with your GP having a Mental Health Plan, which could help you access subsidised psychological support.

- Lifeline offers telephone and online counselling, as well as information on other services. Contact 13 11 14 or at www.lifeline.org.au (please copy the link into your browser).

- Beyond Blue. Further information about Post Traumatic Stress Disorder, and responses, including online communities, can be accessed here: www.beyondblue.org.au/the-facts/anxiety/types-of-anxiety/ptsd (please copy the link into your browser).

Interviews with participants are also being held as part of this project. The interview will cover your experience during and following the fire in more detail and will take around 45 minutes at a location of your choice or on the telephone. Interviews are confidential and you can also bring a support person to the interview. You can also change your mind and withdraw at any time.

If you're interested in taking part in an interview about your experiences, please follow the instructions on the page that appears after you've pressed "Done".

If you would like any further information or if you would like to forward information about this survey to someone else, please follow the instructions on the page that appears after you've pressed "Done".