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Race, Racism, Stress and Indigenous Health

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BSc MMedStats MPH

Submitted in total fulfilment of the requirements of the degree of Doctor of Philosophy

July, 2006

Department of Public Health

The University of Melbourne

This thesis is dedicated to my first child, Maya Batsheva Ileki Paradies, who was born on the 28th of April 2006 as I was completing this manuscript.

Abstract

This thesis is a transdisciplinary study aimed at exploring the role of race, racism and stress as determinants of health for indigenous populations and other oppressed ethnoracial groups. Commencing with an analysis of continuing racialisation in health research, it is shown that there is no consistent evidence that oppressed ethnoracial groups, who suffer disproportionately from type 2 diabetes, are especially genetically susceptible to this disease. Continued attribution of ethnoracial differences in health to genetics highlights the need to be attentive to both environmental *and* genetic risk factors operating within and between ethnoracial groups.

This exploration of racialisation is followed by a theoretical examination of racism as a health risk factor. This includes a comprehensive definition of racism, a diagrammatic representation of the aetiological relationship between racism and health and an examination of the dimensions across which perceived racism can be operationalised. An empirical review of 138 quantitative population-based studies of self-reported racism as a determinant of health reveals that self-reported racism is related to ill-health (particularly mental health) for oppressed ethnoracial groups after adjustment for a range of confounders. This review also highlights a number of limitations in this nascent field of research.

This thesis then attempts to clarify the plethora of conceptual approaches used in the study of stress and health as a first step towards comprehending how stress interacts with both racism and health. A review of the empirical association between stress and chronic disease for fourth world indigenous populations and African Americans was also conducted. This review, which located 65 studies, found that a range of chronic diseases (especially chronic mental conditions) were associated with psychosocial stress.

Utilising the conceptual work on operationalising racism discussed above, an instrument was developed to measure racism and its correlates as experienced by Indigenous Australians. This instrument demonstrated good face, content, psychometric and convergent validity in a pilot study involving 312 Indigenous Australians. The majority of participants in this study (70%) reported some experience of inter-personal racism, with this exposure most commonly reported in employment and public settings, from

service providers and from other Indigenous people. Strong and consistent associations were found between racism and chronic stress as well as between racism and depression (CES-D), poor/fair self-assessed health status/poor general mental health (SF-12) and a marker of CVD risk (homocysteine), respectively. There was also evidence that the association between inter-personal racism and poor mental health outcomes was mediated by somatic and inner-directed disempowered reactions to racism as well as by chronic stress and a range of psychosocial characteristics.

To conclude this thesis, an examination of approaches to addressing racism for Indigenous Australians is undertaken. The theoretical issues pertinent to the study of anti-racism are discussed along with empirical findings from social psychology on effective approaches to anti-racism. Recommendations for implementing these approaches through institutional and legal policies are also presented. Strategies for engendering political will to combat racism in the current neo-liberal capitalist climate are also briefly considered.

Declaration

This is to certify that

(i) *the thesis comprises only my original work towards the PhD except where indicated in the Preface;*

(ii) *due acknowledgement has been made in the text to all other material used;*

(iii) *the thesis is less than 100,000 words in length, exclusive of tables, maps, bibliographies and appendices.*

_____

Yin Paradies BSc MMedStats MPH

Preface

Several chapters of this thesis have been published during the course of my candidature:

- Chapter 2 as Paradies Y, Montoya MJ & Fullerton SM. (accepted). Racialized genetics and the study of complex diseases: The thrifty genotype revisited. *Perspectives in Biology and Medicine*;
- Chapter 3 as Paradies Y. (2006). Defining, conceptualising and characterising racism in health research. *Critical Public Health* 16(2): 143-157;
- Chapter 4 as Paradies Y. (2006) A systematic review of empirical research on self-reported racism and health. *International Journal of Epidemiology* 35(4): 888-901;
- Chapter 6 as Paradies Y. (2006). A review of psychosocial stress and chronic disease for 4th world Indigenous peoples and African Americans. *Ethnicity and Disease* 16(1): 295-308;
- and Chapter 10 as Paradies Y. (2005). Anti-racism and Indigenous Australians. *Analyses of Social Issues and Public Policy* 5: 1-28.

Chapter 2 was written in collaboration with Assistant Professors Michael Montoya and Malia Fullerton. I was responsible for the design and conception of this chapter, undertook the majority of the data collection and interpretation and wrote the first full draft. My two co-authors re-wrote and edited sections of this chapter as well as contributing to data collection and interpretation. In particular, Montoya and Fullerton were largely responsible for the anthropological and genetic aspects of this chapter, respectively.

During the course of my candidature, I also published several journal articles related but not central to this thesis. There are included in Appendix C.

Acknowledgements

Various aspects of this PhD have been greatly enhanced by the kind assistance of a range of colleagues and funding bodies. I am especially grateful to my wife, Dr Emma Kowal, for her support throughout my thesis and for erudite comment on drafts of various thesis chapters. Thanks are also due to my primary supervisor, A/Prof Joan Cunningham, for her assistance throughout my PhD research and to my secondary supervisor Prof Ian Anderson. I ws supported during work on this thesis by an Australian National Health and Medical Research Council (NHMRC) Training Scholarship for Indigenous Health Research (#193321) and a Cooperative Research Centre for Aboriginal Health (CRCAH) scholarship as well as by the CIPHER Program which is an Australian NHMRC Population Health Capacity-Building Grant (#236235) that is also an in-kind project of the CRCAH.

My thanks also to two anonymous reviewers of Chapter 3, the editors of the International Journal of Epidemiology as well as two anonymous reviewers for valuable feedback on drafts of Chapter 4. I was partially supported during work on Chapters 4 and 9 by the Aboriginal and Torres Strait Islander Commission and Australian-American Fulbright Commission. Prof Ian Anderson, A/Prof Mark Daniel, Drs Tracy Westerman, Tarun Weeramanthri, and Jeannie Devitt as well as Ms Cheryl Furner commented on drafts of Chapters 5 and 6. Three anonymous reviewers provided feedback on Chapter 5 and members of the Menzies School of Health Research Writing Workshop group commented on drafts of Chapter 6. Work on Chapters 5-6 was partially funded by the Cooperative Research Centre for Aboriginal Health (CRCAH).

I also gratefully acknowledge the support of the Darwin Region Urban Indigenous Diabetes (DRUID) study participants, study staff, members of the Indigenous Steering Group, and partner organisations whose contributions made Chapters 7-9 possible. The DRUID Study was funded by the National Health and Medical Research Council (Project Grant #236207), with additional support from the Australian Government Department of Employment and Workplace Relations, the Clive and Vera Ramaciotti Foundation, the Vincent Fairfax Family Foundation, the AusDiab Partnership in Type 2 Diabetes, and Bayer HealthCare. My thanks also to Drs Jack Glaser, Nap Hosang and

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Ethical Clearance

Ethical clearance for the research conducted as part of this thesis was granted by the joint Menzies School of Health Research – Northern Territory Department of Health and Community Services Human Research Ethics Committee.

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Chapter 1. **Introduction**

Indigenous Australians[1] are those peoples who have maintained a relationship through descent, self-identification and community acceptance with pre-colonial populations in Australia. Indigenous people in Australia comprise approximately 2.4% of the Australian population with about 30% of Indigenous people living in major cities, 43% in regional areas and 26% in remote areas of Australia (Australian Bureau of Statistics & Australian Institute of Health and Welfare, 2005). Indigenous Australians are the most disadvantaged group in Australian society, suffering from high rates of unemployment and incarceration, low income, sub-standard housing and a high burden of ill-health and mortality, including a life expectancy that is approximately 17 years less than other Australians (Australian Bureau of Statistics & Australian Institute of Health and Welfare, 2005).

Such disadvantage is inextricably intertwined with the process of racialisation in which essentialised innate differences are ascribed to Indigenous people. The racialisation of Indigenous people in Australia has preoccupied the Australian popular imagination and nation-state from the earliest days of colonisation (Chesterman and Galligan, 1998; Gardiner-Garden, 2003; McCorquodale, 1997). Historical approaches to defining Indigeneity focused on the need to surveil and control the socialisation, mobility and biological reproduction of those with some descent from pre-colonial peoples of Australia (Dodson, 1994), while contemporary constructions of Indigeneity are primarily concerned with attributing the cause of Indigenous inequality to the deviant and 'othered' Indigene (Brough, 1999; Cunneen, 2001). Futhermore, public health research is deeply implicated in such racialising of Indigenous Australians, both historically (Anderson, 2002; Thomas, 2004) and in present-day research (Bond, 2005; Humphery, 2001; Smith, 1999).

[1] The term Indigenous is used here to refer to Australian Aboriginal and/or Torres Strait Islander people and is capitalised when denoting this specific social group rather than indigenous peoples more generally.

As a consequence of such racialising practices, Indigenous Australians are subject to racism in the political domain (Augoustinos et al., 1999), health (Henry et al., 2004) and education systems (Boughton 1999), sport (Gorman 2004), the legal and criminal justice systems (Cunneen 2001) and Australian civil society as a whole (Cowlishaw 1997; Department of Immigration and Multicultural Affairs 1997; Goodstone et al., 2001; Hollinsworth 1998; Human Rights and Equal Opportunity Commission 1991; Lattas, 2001; Mellor, 2003; Pedersen et al., 2000).

The continued racism that Indigenous people are subjected to is borne out by national survey research. Of 2,943 Indigenous respondents in a 1994 survey, 76% agreed that racism was a serious problem (Commonwealth Department of Human Services and Health 1995:19). A 2001 national survey of racism found that approximately 30% of Indigenous people report experiencing racism (i.e. discrimination due to ethnic origin), double the proportion of non-Indigenous Australians who reported racism in this survey (Dunn et al., 2003). The 2002-3 National Aboriginal and Torres Strait Island Social Survey (NATSISS) included 9400 Indigenous respondents aged 15 years and over, of whom 17.7% reported experiences of discrimination/racism (i.e. as a personal stressor experienced in the last 12 months) (Australian Bureau of Statistics, 2004).

Furthermore, very recent Australian research has hinted at the relationships between exposure to racism and poor health. The 2004-5 National Aboriginal and Torres Strait Island Health Survey (NATSIHS) included 10, 439 Indigenous Australians aged 15 years and over (Australian Bureau of Statistics, 2006). Of respondents aged 18 years and over with self-reported diabetes, heart disease and cancer, 12.5%, 13.3% and 17.0%, respectively reported discrimination/racism in the last 12 months, compared to only 6.5% of respondents without a chronic condition (Australian Bureau of Statistics, 2006). Similarly, 21.5% of the 1, 073 Indigenous children aged 12- 17 years in the 2001-2 Western Australian Aboriginal Child Health Survey reported experiencing racism (i.e. being treated badly or refused service due to being Aboriginal, in the past 6 months) (Zubrick et al., 2005). After adjusting for age and sex, reported exposure to racism was associated with increased smoking, marijuana use and alcohol consumption in this survey (Zubrick et al., 2005). In addition, a preliminary report of a 2003 research project conducted in an isolated Western Australian rural town found that 35% of Aboriginal respondents said they were both emotionally upset and experienced physical

stress due to an experience of 'being treated differently on the basis of race' (Gilles et al., 2004).

Despite a call by the peak health and medical research body in Australia to study the effects of racism on Indigenous health (NHMRC, 2002:12), the data presented above represent the only quantification of either the extent of racism experienced by Indigenous Australians, or its impact on Indigenous health. Although the 2001 survey detailed above (Dunn et al., 2003) included a sample of only 94 Indigenous adults, this is the largest ever sample of Indigenous people in a national survey specifically focused on racism. Given that statistics are imbued with considerable authority in contemporary Australian society and are thus well placed to shape policy and practice (Walter, 2003), quantification by researchers of racism and its consequences is vitally important to addressing this divisive phenomenon.

This thesis is a transdisciplinary study (see Higginbotham et al., 2001) which aims to explore the role of race, racism and stress as determinants of health for indigenous populations and other oppressed ethnoracial groups, with a particular focus on Indigenous Australians.[2] The concept of racialisation is first explored through a critique of research on indigenous populations in relation to type 2 diabetes. Following this, both racism and the related phenomenon of stress are explored theoretically and quantitatively as they relate to the health of indigenous peoples and other oppressed ethnoracial groups, before concluding with an examination of anti-racism policies relevant to Indigenous Australians.

The related concepts of race, racism and stress were selected as topics of research for this thesis because the author, as an Aboriginal Australian, has a particular interest in how these phenomena contribute to the continuing poor health of Indigenous people. Furthermore, the topic of racism and anti-racism was selected because it represents one of the few areas of Indigenous health research which has the principal goal of reforming dominant culture and society rather than attempting to erase the 'deviance' of Indigenous people (Cowlishaw 2004:66; Kowal & Paradies, 2005).

[2] Following Montoya (2003), the terms 'ethnorace' and 'ethnoracial' are used to exemplify the ineluctably intertwined concepts of race and ethnicity as utilised in contemporary health research.

Chapter 2 of this thesis begins with a critique of 'race' as operationalised in genetic research on complex diseases through an examination of one of the oldest gene-based theories of chronic disease causation: the thrifty genotype hypothesis. This hypothesis is reviewed as a case study of the way in which genetic research attracts a high investment of scientific talent and resources while perpetuating folk concepts of human variation and contributing little to the capacity to understand or combat such diseases.

The examination of racism as a determinant of health is considered in Chapter 3 which highlights three areas in which conceptual clarity in this emerging area of research is lacking: the defining and theorising of racism; the aetiological relationship between racism and health; and the operationalisation of racism as health risk condition. Building upon the theoretical work in Chapter 3, the empirical evidence of a relationship between self-reported racism and health is reviewed in Chapter 4, which presents an analysis of the results from 138 empirical quantitative population-based studies that have been conducted in this nascent field of study.

Following the complex relationship identified between racism and stress in Chapter 4, this thesis turns, in Chapter 5, to a consideration of research on stress as a determinant of health. Although a plethora of research has been undertaken on the concept of stress, this research has been accompanied by a profusion (and confusion) of diverse conceptual approaches. In a similar vein to Chapter 3, this chapter attempts to bring some clarity to this diverse field by reviewing and synthesising the variety of concepts used in the study of psychosocial stress and health. Following the pattern for racism above, Chapter 6 entails a consideration of empirical evidence for an association between stress and health for fourth world indigenous populations,[3] for whom no previously published review on this topic has been conducted. This chapter focuses on chronic disease as the main driver of the health inequities that plague fourth world indigenous peoples, evaluating a total of 65 studies, including 15 intervention studies conducted in this area of research.

[3] Fourth world indigenous peoples are defined as minority indigenous populations living in first-world nations wherein institutionalised power is held by a colonising, subordinating majority (O'Neil, 1986a).

Following the broad theoretical and empirical reviews of racism and stress, Chapter 7 describes the development and validation of the Measure of Indigenous Racism Experiences (MIRE) questionnaire. This instrument was designed, using the conceptual work undertaken in Chapter 3, to assess experiences of racism as well as reactions/response to this racism and aspects of ethnoracial identity for Indigenous Australians. This instrument was piloted in a study conducted in Darwin, Australia in which a final sample of 312 Indigenous people aged 15 years completed at least some of the MIRE. The pattern and correlates of responses to the MIRE in this pilot study are detailed in Chapter 8 while Chapter 9 analyses these responses, along with data on stress, in relation to a range of mental and physical health outcomes measured in the pilot study. Preceding brief concluding remarks, the penultimate chapter of this thesis (Chapter 10) explores the implications of recognising racism as a significant determinant of health for indigenous populations by reviewing institutional and legal anti-racism policies which could be implemented in an attempt to both reduce racism against Indigenous Australians and ameliorate its ill-effects.

Chapter 2. ***Racialised genetics and the study of complex diseases: The thrifty genotype revisited***[4]

2.1 Introduction

This millennium has seen a resurgence of debate on the use of ethnorace in genetics research (e.g. Bamshad, 2005; Berg et al., 2005; Burchard et al., 2003, Cooper et al., 2003; Risch, 2002; Schwartz, 2001; Tang, 2006). The complex topic of genetics and its relation to ethnoracial identity is best elucidated through specific case studies in association with more abstract debate (Shields et al., 2005) that incorporates transdisciplinary perspectives (Fee, 2006). As such, this chapter seeks to examine the continued misuse of ethnorace in human genetics, and the implications of this use for public health, through a discussion of the genetic, epidemiological and sociocultural aspects of the Thrifty Genotype (TG) hypothesis. The TG hypothesis, which posits an ancient biological (genetic) adaptation to cycles of feast and famine, has been employed repeatedly in research seeking to explain why Type 2 Diabetes Mellitus (T2DM) disproportionately afflicts a range of disadvantaged ethnoracial groups. This chapter begins by outlining a transdisciplinary position on human variation in order to situate the critique of the TG hypothesis that follows. An overview of the epidemiology of T2DM is then followed by a detailed description of the TG hypothesis, a critical review of its central premises, and the epidemiological and genetic evidence which has been collected in its wake. The chapter concludes by examining the prospects for rehabilitating the genetic study of health and disease in light of the concerns identified below.

2.2 Ethnorace, Racialisation and Health

Human beings are remarkably genetically similar, sharing 99.9% of their genomes in common (Li & Sadler, 1991; Stephens et al., 2001). Of this relatively small intra-

[4] This chapter is taken from Paradies Y, Montoya MJ & Fullerton SM. (accepted). Racialized genetics and the study of complex diseases: The thrifty genotype revisited. *Perspectives in Biology and Medicine*.

species diversity, approximately 85-90% occurs within, rather than between, groups of individuals classified as belonging to different ethnoraces (Barbujani et al., 1997; Lewontin, 1972; Nei & Roychoudhury, 1982; Romualdi et al., 2002; Rosenberg et al., 2002). A currently unknown, but likely quite small, proportion of such differences have identifiable effects on physiology or disease susceptibility (Bamshad, 2005). Thus, a very small proportion of total genetic variation is even potentially relevant to disease prevalence, or other health-related differences, between groups of individuals classed as ethnoraces.

Nevertheless, ethnoracial concepts have a currency and relevance in everyday discourse that are independent of any definable genetic distinction (Krieger, 2005a; Ossorio & Duster, 2005). Ethnoracial identity is best understood as a marker of social relationship rather than as an unalterable individual trait (Zuberi, 2001). In contemporary society, as in the past, however, the attribution of ethnorace to individuals often depends on the presence of particular morphological characteristics, such as skin colour, as well as known or assumed lines of descent from ancestors with particular morphological characteristics or defined geographic ancestry (Berg et al., 2005). The continuously varying character of such features in human populations, combined with the historically and culturally contingent nature of attaching social meaning to specific morphologies, explains the marked variation in the number, nature, and type of ethnoraces identified over both time and space (Brodkin, 2000; Marshall, 1993).

Despite the clear social character of ethnoracial identity, and what is best recognised as a largely arbitrary system of classification laid atop complex geographic patterns of population genetic variation, recent research has demonstrated that it is possible to assign individuals to likely ethnoracial (or, as they are often designated, ancestral) backgrounds via a cumulative examination of differences in the frequencies of common genetic variants as well as more geographically restricted (i.e. population-specific) polymorphisms (Rosenberg et al., 2002; Tang et al., 2005). Contrary to the fashion in which such research has been interpreted, these efforts do not demonstrate that naturally aggregating clusters delimit ethnoraces. Instead, they underline that individuals sampled from geographically disparate regions will often differ to a small degree genetically, and in a manner predicted by standard isolation-by-distance models (Weiss & Fullerton, 2005). Importantly, while human populations can be readily differentiated into groups of more or less genetically related individuals, it is not at all clear whether the genetic differences which distinguish such groups have health relevance (Bamshad, 2005;

Ioannidis et al., 2004). Although single gene variants with clear health consequences, such as the point mutation that leads, in homozygous dose, to sickle cell anemia, are known to vary across ethnoracial populations (Kittles & Weiss, 2003), such genetic diseases account for a very small percentage of the disparities in health across ethnoraces.

These well-established pitfalls of conflating ethnorace and genetics have been overlooked or misunderstood in many quarters and such categories continue to be utilised by scientists, government, media and lay persons under the misplaced assumption that individuals with different ethnoracial identities are genetically distinct and that this has relevance to ethnoracial inequalities in health outcomes (Braun, 2002; Stevens, 2003:1056-7). For instance, the National Library of Medicine in the U.S. lists genetic research as the top investigative priority for health disparities (including ethnoracial disparities) (Sankar et al., 2004), ahead of research into the environmental and social determinants of ethnoracial inequalities in health.

The two key fallacies that drive the continued overemphasis on genetics in explaining ethnoracial health disparities are (1) that genetic variation explains much or all of the health differentials among human populations and (2) that there is more genetic variation between, rather than within, ethnoracial population groups. While the former fallacy is a form of geneticisation (i.e. the notion that genes are the primary determinants of biology and behaviour), the latter represents a form of racialisation, which in this case, entails the exaggerated salience of ethnorace in genetics (Goodman, 2000).

Geneticisation is the process by which priority is given to differences between individuals based on their genetic distinctiveness (Lippman, 1993:178). Geneticisation occurs: (1) conceptually, when genetic terminology is used to define problems; (2) institutionally, when genetic expertise is enlisted to deal with specific problems; (3) culturally, when genetic knowledge and technology lead to changing individual and social attitudes towards, for instance, the prevention and control of disease; and (4) philosophically, when genetic imagery produces particular views on human identity, interpersonal relationships and individual responsibility (ten Have, 2001).

Extreme forms of geneticisation become a kind of 'genetic fatalism' in which genes and genetic variants are considered to be both completely determining and unalterable (Alper & Beckwith, 1995). Geneticisation appeals to dominant ideas of individuality and related notions of personal responsibility for health. Through geneticisation, the public health gaze is misfocused onto individual biophysical risk factors and away from social, environmental and ecological factors (Ellison & Jones, 2002; Lee et al., 2001:60; Nelkin & Lindee, 1995; Shostak, 2003). Despite the complexity involved in attributing causality to specific genetic determinants, genes are routinely accorded exaggerated etiological significance (Ellison & Jones, 2002) with scientists themselves, as well as the media, routinely overstate the certainty and implications of genetic research (Sankar et al., 2004; Shields et al., 2005).

The incredible popularity of genetic knowledge would be more understandable if its allure was based upon evidence of sustained and incontrovertible public health benefit. Rather, to date, genetics has been sold largely on its incredible *potential* for benefit (Stevens, 2003:1039), with genetic explanations being customarily accepted in the absence of therapies based upon genetic insights (Conrad & Gabe, 1999). Despite many millions of dollars and years of time spent searching for genes that contribute to common complex diseases (Cruickshank et al., 2001), very few convincingly replicable findings have been reported (Altmuller et al., 2001; Hirshchhorn et al., 2002). To date, such null findings are explained largely as a function of inadequate methodology or sample size (Buchanan et al., in press) and this unshakable faith in the genetic paradigm appears to go hand in hand with the use of ethnorace as a marker of genetic difference.

Racialisation is a cultural process wherein population descriptors are used in a fashion that ascribes, to the human groups labeled, a somatic essentialised innate difference (Miles & Brown, 2003) based on phenotype, ancestry and/or culture. Such population descriptors have been shown time and again to be historically and situationally determined, unreliable, and imprecise proxies for human genetic differences. Nevertheless, as detailed below, racialisation continues to occur when, for example, geneticists studying T2DM use population descriptors as de facto markers of biological differences between human groups. When inequalities of morbidity and mortality are explained or hypothesised as a condition of innate and fixed genetic differences, the existence of sociopolitical inequalities between human groups are effectively denied and

embodied health outcomes are attributed to these putative biological differences.

Although most health-related genetic research has been conducted on populations of European ancestry (Ioannidis et al, 2004), it is ethnoracial groups in third and fourth world contexts who are often considered to be genetically susceptible to a plethora of chronic diseases (Cooper et al., 2003). For such ethnoracial groups, genetic explanations for ill-health are proposed when no such data are available, on the basis of non-significant findings, or even when genetic variation is unrelated to the disease under study. The association between unsubstantiated genetic attributions and the social status of an ethnoracial group (Cooper et al., 2003), demonstrates the potential harm that may result from the continuing (and increasing) use of ethnoracial categories in genetic research (Ellison & Jones, 2002). The presentation of genetic findings using folk taxonomies of ethnorace reinforces the fallacy that ethnoraces are meaningful genetic categories. This chapter aims to demonstrate how this fallacy operates on groups disproportionately affected by T2DM, leading to the misinterpretation of ethnoracial health disparities as geneticised and hence natural in origin, rather than recognising such disparites as being due, largely or solely, to social disadvantage (Condit et al., 2004; Reanne, 2001).

2.3 The Epidemiology of Type 2 Diabetes Mellitus

Currently there are several different recognised types of diabetes, including: Type 1, Type 2, gestational diabetes and Mature Onset Diabetes in Youth (MODY). Type 1 diabetes is characterised by absolute insulin deficiency while other types of diabetes are characterised by insulin resistance and/or abnormal insulin secretion (Zimmet et al., 2001). By far the most common type of diabetes is T2DM which accounts for over 90% of cases worldwide (Zimmet et al., 2001). T2DM is typically experienced as a late-onset chronic disease (although a younger age of onset is becoming increasingly common) and unlike the autoimmune-related Type 1 diabetes, is associated with risk factors such as increased obesity, dietary fat intake, smoking and low physical activity (Quinn, 2003). Additional risk factors such as low socio-economic status (Cruickshank et al., 2001; Everson et al., 2002; Green et al., 2003; Maty et al., 2002;), stress (Black, 2003; Rosmond, 2003; Surwit et al., 1992) and racism (Piette, 2006; Tull & Chambers, 2001) have also been associated with T2DM. Low birth weight (Barker, 1999; Benyshek et al.,

2001; Dyck et al., 2001; Hales & Barker, 1992; Hales & Barker, 2001; Harding, 2001; McCance et al., 1994) and a mother who experienced gestational diabetes (Carter et al., 1996) have also been associated with T2DM in later life.

Several different sources of evidence also suggest that genetic factors may play a role in T2DM susceptibility. Studies of siblings have found greater concordance for T2DM status among identical (monozygotic) as compared to non-identical (dizygotic) twins (Kaprio et al., 1992; Newman et al., 1987; Matsuda & Kuzuya, 1994). Evidence of familial aggregation (Gottlieb, 1980) combined with studies demonstrating impaired glucose tolerance in first-degree relatives of T2DM patients (Eriksson et al., 1989), also support the importance of inherited susceptibility. While a handful of segregation studies have hinted at the influence of one or more loci of major effect (e.g. Schumacher et al., 1992), the analysis of patterns of inheritance in families has been largely inconclusive (McIntyre & Walker, 2002); suggesting a more complex, polygenic mode of inheritance instead (Rich, 1990). Many commentators also cite, without justification, evidence of pronounced prevalence differences among distinct populations (e.g. Knowler et al., 1990) as support for a presumed genetic influence on disease etiology.

The prevalence of T2DM in adults aged 20 years and over is on the increase worldwide, with a projected rise from 4.0% in 1995 to 5.4% in 2025. Over this time period the prevalence is expected to increase from 6.0% to 7.6% in developed nations and from 3.3% to 4.9% in developing nations (King et al., 1998). It is unlikely that such large changes in prevalence over the course of a single generation are related solely to differences in genetic susceptibility. Indeed, it has become clear that both obesity and T2DM are a consistent response to the voluntary or involuntary adoption of a high consumption lifestyle. This is particularly clear from studies that document a sharp increase in the incidence of T2DM for migrants moving from countries with low prevalence to those with a high prevalence of T2DM (Lev-Ran, 1999:11; Motala et al., 2003; van Dam, 2003), from studies of urbanisation (Aschner, 2002; Lev-Ran, 1999:11; Motala et al., 2003; Taufa & Benjamin, 2004), and from transnational comparative studies (Ravussin et al., 1994).

Since Kelly West's seminal paper (1974), it has been known that T2DM is a disease that disproportionately affects fourth world indigenous populations around the world. In 1997 the crude prevalence of diabetes (all types) for Native Canadian men and women

was 3.6 and 5.3 times higher, respectively, than the diabetes prevalence in the general Canadian population (Young et al., 2000). The age-standardised prevalence of self-reported diabetes among Indigenous Australians in 2004-5 was three times that of the non-Indigenous population (Australian Bureau of Statistics, 2006). In the U.S., the prevalence of T2DM for non-Hispanic blacks, Hispanic/Latino Americans and American Indians/Alaskan Native (who receive care from the Indian Health Service) aged 20 years or older was 1.6 times, 1.5 times and 2.2 times the rate for Non-Hispanic Whites in 2002 (National Center for Chronic Disease Prevention and Health Promotion, 2006). Similarly, in 2000, the prevalence of diabetes for Maori and Pacific Islanders in New Zealand was nearly three times that of White New Zealanders (New Zealand Ministry of Health, 2000).

Although Inuit populations have historically had one of the lowest rates of diabetes in the world (Ebbesson et al., 1998), this is now changing with the overall age-adjusted prevalence among Alaskan Inuits increasing from 8.8 per 1,000 people in 1985 to 12.1 per 1,000 people in 1993, with a prevalence of 6.6% reported among three Inuit groups in 1994 (Ebbesson et al., 1998). In the Pacific, T2DM has gone from being virtually non-existent to a major health problem in recent decades (Foliaki & Pearce, 2003).

Overall, the epidemiology of T2DM suggests a complex etiological picture, with evidence from migrant studies and secular trends clearly implicating social and environmental risk factors while twin studies and patterns of familial aggregation of disease are simultaneously consistent with a plausible genetic component. While genetic predispositions may, in part, explain *inter-individual* variation in T2DM, the racialised form of the TG hypothesis proposes that genetic predispositions also explain prevalence differences *between populations* – a contention which will be considered below.

2.4 The Thrifty Genotype Hypothesis

The thrifty genotype was first proposed by James Neel in 1962 as an explanation for the much greater prevalence of obesity, and by extension diabetes, in contemporary society. Neel argued that during 99% of human existence we existed as hunter-gatherers who experienced frequent feast or famine cycles. As a result we developed a genotype exceptionally efficient in the absorption and/or utilisation of nutrients, which has now

become maladaptive in a first-world context of sustained energy surplus.[5] It is important to note that the initial form of the TG hypothesis was not racialised but was, instead, an attempt to explain the sudden rise in diabetes throughout the modern world (Neel, 1962).

One of the earliest references to the TG hypothesis was in an article published in 1964 which suggested that a high prevalence of T2DM among the Alabama-Coushatta Indians was due to a TG that was associated with T2DM in all humans, but that this gene was much more common among Alabama-Coushatta Indians (Johnson & McNutt, 1964). Another early study, published in 1969, suggested that the high prevalence of T2DM among Seneca Indians from Western New York was "compatible with the presence of an inherited susceptibility to diabetes in this ethnic group" (Doeblin et al., 1969:625). In 1976, the TG hypothesis was proposed as an explanation for hyperglycemia that was more common in "full blood obese compared with part blood obese Aborigines" in Australia (Wise et al., 1976:194). However, this paper also noted that the environment was still likely to be the "dominant…causative factor" in T2DM prevalence (Wise et al., 1976:195).

The TG hypothesis was utilised by Zimmet (1979) to suggest that "Micronesians and Polynesians have a hereditary susceptibility to diabetes (i.e. diabetic genotype)" (Zimmet, 1979:147) and that "[t]he genotype for [T2DM] may render an individual susceptible to such factors as obesity, dietary abuse, stress, certain viruses or other as yet undefined environmental factors" (Zimmet, 1979:147). In a study of young Aboriginal men in northeastern Arnhem Land, O'Dea (1982) suggested that evidence of insulin resistance in Aboriginal Australians, despite their extreme leanness, regular physical activity, and traditional diet, may indicate a susceptibility to T2DM if they were to Westernise further.

Neel's second article on the TG was published in 1982. In this article he explicitly discussed the high prevalence of diabetes in a range of ethnoracial groups, suggesting

[5] It has also been suggested that a lack of carbohydrates through a primarily carnivorous diet over the last 2 million years of human history – rather than feast and famine cycles per se – lead to increased insulin resistance in order to ensure a regular carbohydrate supply to the brain, fetus and mammary glands (Brand et al., 1994).

that this could be seen "as a 'telescoped' example of the type of genotype-environmental interaction which populations of Western European extraction had sustained earlier" (Neel, 1982). In this article, Neel discusses diabetes as "a genetic disease," (Neel, 1982:284) suggesting that the heterogeneity of diabetes requires "not one but several different thrifty genotypes" (Neel, 1982:285) The hypothesis, as presented by Neel, involved a first-time discussion of ethnorace as a factor in the TG, but still did not involve racialised genetics as no particular susceptibility was proposed for one ethnoracial group over another but only differences in the timing of environmental changes between ethnoraces (Neel, 1982).

In the same article Neel proposed two physiological pathways through which this evolutionary effect might operate. First, over-responsive beta cells of the pancreas might produce excess insulin (a "quick insulin trigger" Neel, 1982:288) which would eventually reduce insulin receptors to subnormal numbers, creating insulin resistance (a risk factor for T2DM). Second, there might be a genetically-determined decrease in the number of insulin receptors or an excessive down-regulation in response to a given level of insulin, also leading to insulin resistance (Neel, 1982). A more recent variation of this hypothesis involves the development of skeletal muscle cells that are particularly insulin resistant (Lev-Ran, 1999:14).

Neel suggested that there could be as many types of thrifty genes as types of T2DM, but that if there turned out to be too many (say 100 or more) then this could be explained by simple mutation pressure and "the need for the thrifty genotype would disappear" (Neel, 1982:290). In a mix of cautious science and flamboyant intellectualism he concluded his second article on the TG as follows:

> All these speculations may be utterly demolished the moment the precise etiologies of [T2DM] become known. Until that time, however, devising fanciful hypotheses based on evolutionary principles offers an intellectual sweepstakes in which I invite you all to join (Neel, 1982:290).

Over the coming years, many did indeed join this "intellectual sweepstake" (Neel, 1982:290). For example, McGarvey suggested that, for Polynesians, evolutionary pressure to produce a TG occurred through the hardship of inter-island colonisation in the Pacific (McGarvey, 1994), likely difficulties in establishing agricultural crops

(McGarvey, 1994) upon arrival, and the destruction of such crops after cyclones (McGarvey, 1994). Wendorf hypothesised that the TG was selected for during the colonisation of North America, Australia, and some Pacific Islands due to the common experience of: 1) an adaptation to unfamiliar biotic systems; (2) a reliance on foods that became extinct or were severely depleted; and (3) frequent food shortages that continued for several generations. He suggested that this putative genotype did not occur in Inuits and some South American tribes because these groups utilised a range of nutritional sources and did not have to suffer sever environmental changes or food sources that became extinct (Wendorf, 1992).

These speculations notwithstanding, in his last written statement on the TG hypothesis in 1999,[6] Neel wrote that he found "no support to the notion that high frequency of [T2DM] in reservation Amerindians might be due simply to an ethnic predisposition – rather, it must predominantly reflect lifestyle changes" (Neel, 1999:S3). He went on to discuss how the original TG hypothesis "presented an overly simplistic view of the physiological adjustments involved in the transition from the lifestyle of our ancestors to life in the high-tech fast lane" (Neel, 1999:S4) and highlighted the role of phenotypic (i.e. developmental) rather than genotypic changes implicated in the much higher contemporary prevalence of diabetes (Neel, 1999).

However, despite Neel himself refuting many aspects of the TG hypothesis, the hypothesis continues to outlive its progenitor in so far as it is reiterated and researched in relation to Indigenous Australians (Bambrick, 2003; Busfield et al., 2002; Irvine et al., 2003; O'Dea, 1997; Rowley & O'Dea, 2001; Zimmet, 2003), Pacific Islanders (Dowse et al., 1991; McGarvey, 1994), Native Americans (Boullu-Sanchis et al., 1999; Fox et al., 1998; Ravussin et. al., 1994; Schulz & Weidensee, 1995; Williams R.C. et al., 2000; Wolford et al., 2001) and First Nation Canadians (Hegele et al., 2000a). In addition to Indigenous peoples, the TG hypothesis has also been applied to such diverse populations as African-Americans (Cowie et al., 1993; Grim & Robinson, 2003; Matsutani et al., 1990a; Osei, 1999; Wilson & Grim, 1991), Mexican-Americans (Duggirala et al., 1999; Haffner et al., 1991; Horikawa et al., 2000; Ravussin et. al., 1994; Schulz & Weidensee, 1993; Stern et al., 1992), Indians (Swai et al., 1990), Asians (Fujimoto, 1992) and Africans (Osei, 1999).

[6] James V. Neel died on February 1st 2000 at the age of 84.

The TG hypothesis also retains currency in research fields as diverse as epidemiology (Williams R.C. et al., 2000), anthropology (Allen & Cheer, 1996; Bindon & Baker, 1997) and psychology (Cacioppo et al., 2000) and is the focus of ongoing biological and genetic research (Busfield et al., 2002; Clark et al., 2005; Fullerton et al., 2002; Poulsen et al., 2003; Rotimi et al., 2004; Ruiz-Narvaez, 2005; Vander Molen et al., 2005).[7] In Australia, a proposed Centre for Aboriginal Genetic Epidemiology will focus on the genetic epidemiology of T2DM among Indigenous Australians. The founding director of this Centre cited the interplay between a genetic propensity for T2DM among Indigenous people due to their history as hunter-gathers and modern lifestyle changes (i.e. the TG hypothesis) as the reason for such a focus (Blackwell 2005). This persistent recourse to the TG hypothesis is perplexing, not only in the light of Neel's subsequent disavowal but also because of the many conceptual limitations of the theory.

2.5 Limitations of the Thrifty Genotype Hypothesis

Modern humans are believed to have evolved from a shared ancestor no earlier than 200,000 years ago which is, in an evolutionary context, a relatively short period of time compared to the four million years that hominids have walked erect on the Earth (Kittles & Weiss, 2003). The primary occupation of our forebears during this vast period of time has been hunting and gathering. It is therefore likely that metabolic efficiency and energy balance would have been a selective advantage in most human populations during the last million years of subsistence (McGarvey, 1994). Hence, under the TG hypothesis, there is a need for an intense environmental impetus to reverse this selective pressure for Europeans first (or only) to explain why this ethnoracial group does not now suffer from T2DM at the same rate as other ethnoracial groups. It has been suggested that agriculture, which became a dominant mode of subsistence 10,000 years ago for some human societies (Jackson, 1991), could be this mechanism.

[7] There have also been several variant hypotheses proposed with the salt-sensitive slavery hypothesis being arguably the most prominent. Although beyond the scope of this paper, a detailed critique of this hypothesis can be found in Kaufman & Hall (2003).

One of the underlying assumptions of this proposed mechanism is that European populations had access to an abundant food supply (De Courten et al., 1997; Turner et al., 1993), perhaps due to the advent of agriculture. However, it is not at all clear that the ethnoracial groups who now suffer from T2DM underwent feast and famine cycles in historic times *or* that agricultural societies were free of such cycles. Furthermore, it has been suggested that, in any case, famines are not capable of producing the differential mortality required to select for a TG (Speakman, 2006).

Available data suggests that nutrient instability was not as high as previously thought for hunter-gather societies (Winterhalder & Smith, 1981). For instance, Nauruans and other Pacific populations who suffer from a very high prevalence of T2DM formerly lived in thinly populated tropical islands which boasted a generous food supply all year round (Baschetti, 1999). Furthermore, it is clear that the advent of agriculture did not preclude major fluctuations in food supply (Turner et al., 1993). In fact, it is likely that agricultural societies were much more susceptible to feast and famine cycles than were hunter gatherers (Benyshek et al., 2001:34; Speakman, 2006), with undernourishment rampant in the Greco-Roman empire and Europe both before and during the Industrial Revolution (Allen & Cheer, 1996). Even during the 20th century, famine was common during the First World War, the Great Depression, and the Second World War (Rock, 2003:165).

Thus, the overabundance of food associated with chronic disease, which disproportionately affects those from disadvantaged ethnoracial groups, is very much a phenomenon restricted to First World nations in the second half of the 20th century. To complicate this simple evolutionary story further, it is also clear that a number of disadvantaged racialised groups, who are thought to now have the putative TG, have been using agriculture for many thousands of years. The Pima Indians, the leitmotif of the TG story for example, have been farming intensively for over 2,000 years (Benyshek et al., 2001:34; Schulz & Weidensee, 1993). This historical perspective also reveals limitations in the hypotheses of McGarvey (1994) and Wendorf (1992) which fail to describe how the particular nutritional hardships suffered by indigenous peoples differed from those suffered by colonists of various ethnoraces throughout history.

A further implicit assumption of the TG hypothesis is the notion that a disease such as T2DM could affect either survival to reproductive age or fertility during reproductive years of life (Dowse & Zimmet, 1993; Lev-Ran, 1999:15). This assumption is

problematic given the post-reproductive age of onset of T2DM in the context of the relatively short lifespan of Europeans (< 50 years) during agricultural periods of the last millennium (Weiss & Terwilliger, 2000). Furthermore, the suggestion that such an effect on survival could occur over a single decade (Dowse et al. 1991) is wholly incompatible with T2DM. Although it is plausible that putative genes associated with T2DM may have effects earlier in the lifespan, it is not at all clear that such effects would result in the selective pressure required to produce the racialised genetic differentials which are invoked in the name of the TG hypothesis.

Finally, one of the proposed explanations for why those of predominantly European ancestry seem to have a 'resistance' to T2DM is that this group has the most genetic diversity and hence its members are less likely to "harbor two recessive diabetogenes and therefore develop diabetes" (Lev-Ran, 1999:17-8). However, numerous studies have made it clear that sub-Saharan African populations (being the present-day groups most closely related to the common human ancestral population) have the highest genetic diversity in the world, sometimes almost twice that of non-African populations (Cavalli-Sforza et al., 1996:154; Jorde et al., 2000; Kittles & Weiss, 2003:44; Przeworski et al., 2000). Furthermore, the insinuation that T2DM could be monogenic in origin is highly questionable. Despite all of these difficulties, however, the TG hypothesis remains one of the most extensively investigated theories in all of genetic epidemiology. Below, the evidence that has accrued for and against this hypothesis is reviewed.

2.6 The Thrifty Genotype Hypothesis: Genetic and/or Epidemiological Evidence

Before reviewing the evidence that has accrued for the TG hypothesis, it is useful to first distinguish between two approaches to research on this topic: (1) research that seeks to understand the genetics of complex disease and (2) genetic research that seeks to understand variations in complex disease incidence/prevalence rates across populations. The former type of research is referred to here as *genetic* epidemiology while the latter is labelled genetic *epidemiology* (Montoya, 2003). *Genetic* epidemiological research is the process of utilising of population DNA to understand the genetics of disease. There are two main reasons why ethnoracially identified study

populations are utilised in *genetic* epidemiological research: 1) to identify genetic contributions (variants or haplotypes) to disease susceptibility in a given population, and 2) to guard against confounding due to population structure (i.e. mistaking a chance difference in allele frequency across cases and controls as being related to disease aetiology). Ethnoracial labels (Indigenous, White, African-American etc.) are used describe the study populations involved in this form of research, rather than to define them *a priori*.

In genetic *epidemiology*, researchers investigate genetic contributions to disease differentials among populations. This type of research asks: How do the specific susceptibility variants segregating in defined populations explain observed differences in disease incidence/prevalence? When, as in this form of research, ethnoracial labels are used as independent variables in models of disease causality, there is a tendency to conflate biological/genetic meaning with socially derived ethnoracial definitions. This results in a form of geneticisation which overemphasies the genetic contribution to ethnoracial inequalities in health while ignoring the overwhelming social determinants of these inequalities. Often, these two distinct forms of research occur simultaneously, which confuses rather than clarifies evidential claims in this area of research.

2.6.1 *Genetic* Epidemiological Research

Neel once described diabetes as a geneticist's nightmare, of which the main ingredient has always been T2DM (Neel, 1976). There are at least sixty distinct genetic disorders associated with glucose intolerance and clinical diabetes (Vadheim & Rotter, 1992:37). Multiple loci are almost always involved in a complex disease such as T2DM, often with a plethora of associated alleles as well as gene-gene interactions and interactions with gene products (proteins) and environmental signals (Permutt et al., 2005; Stroham, 1993:114; Weiss & Terwilliger, 2000). *Genetic* epidemiological research undertakes the challenging task of identifying the genetic basis of complex human diseases, such as T2DM, through research involving various ethnoracial study populations.

A number of studies have attempted to find genes or genetic variants associated with T2DM in ethnoracial populations as diverse as Pima Indians (Baier et al., 2000; Baier & Hanson, 2004; Majer et al., 1996; Muller et al., 2005; Thompson et al., 1995), Native

Canadians (Hegele et al., 1999; Hegele et al., 2000a; Hegele, 2001), Indigenous Australians (Busfield et al., 2002), people of Indian ethnic origin in Guadelupe (Boullu-Sanchis et al., 1999), Japanese (Horikawa et al., 2003), Caucasians (Evans et al., 2001; Fingerlin et al., 2002; Rasmussen et al., 2002;), African-Americans (Elbein et al., 2000) and Mexican-Americans (Horikawa et al., 2000), with many of these studies failing to find either candidate loci or associated susceptibility variants (Baier et al., 2000; Baier & Hanson, 2004; Boullu-Sanchis et al., 1999; Elbein et al., 2000; Evans et al., 2001; Fingerlin et al., 2002; Horikawa et al., 2003; Matsutani et al., 1990b; Rasmussen et al., 2002; Thompson et al., 1995). A brief review of the most compelling evidence available for the influence of specific genes or genetic regions on T2DM susceptibility is presented below, highlighting the use of socially-defined ethnoracial groupings for genetic discovery.

Only a relatively small number of genomic regions have shown consistent evidence of linkage to T2DM: chromosomes 1q, 3q, 8p, 10q, 12q, and 20q (Permutt et al., 2005). A single study of Indigenous Australians also identified a region of significant positive linkage with T2DM on chromosome 2q (Busfield et al., 2002). The protocol of this study included an interview that recorded age at onset of diabetes, history of treatment and complications, measurements of height, weight, body-mass index (BMI), blood pressure, and waist and hip circumference. However, in common with many genetic investigations, none of these measured variables were adjusted for in the analysis to determine whether they confounded the genetic association identified (Busfield et al., 2002). Linkage to chromosome 20q has been reported for a number of populations including Finns (Ghosh et al., 1999), Japanese (Mori et al., 2004), Chinese (Luo et al., 2001), Caucasians (Ji et al., 1997; Klupa et al., 2000) and West Africans (Rotimi et al., 2004).

Even fewer investigations have been able to trace susceptibility to specific candidate genes and variants. A landmark study, published in 2000, found a complex genotype (defined by a pair of multi-variant haplotypes) of the *CAPN10* gene on chromosome 2 was associated with a 2.80-fold increased risk of T2DM (95% CI: 1.23 - 6.34) in Mexican Americans. The 112/121-haplotype combination was also associated with similarly increased risk in a second, independent group of Mexican Americans (OR = 3.58, 95% CI: 1.43 - 8.92), as well as in Finnish and German cohorts (Horikawa et al., 2000). It has been estimated that the haplotype combination accounts for 14% of the

population-attributable risk for T2DM in Mexican Americans and 4% in Europeans (Horikawa et al., 2000). However, while several studies have been able to replicate this finding (Weedon et al., 2003), a number of others have failed to do so (Baier et al., 2000; Evans et al., 2001; Fingerlin et al., 2002; Horikawa et al., 2003; Rasmussen et al., 2002). A meta-analysis of 10 published case/control studies consisting of 3,303 subjects concluded that a *CAPN10* haplotype defined by the SNP-44 polymorphism was associated with increased risk of T2DM (OR 1.17 [1.02–1.34]) (Weedon et al., 2003). Independent population genetic evidence suggests that nucleotide polymorphism in the vicinity of the *CAPN10* gene has been subject to the effects of natural selection (Fullerton et al., 2002; Vander Molen et al., 2005), making it a plausible candidate for an evolutionary hypothesis such as the TG.

A series of studies by Hegele et al. found a polymorphism (S319) for hepatocyte nuclear transcription factor (HNF-1α) that appeared to be unique to Canadian Oji-Cree Indians and strongly related to T2DM, although the majority of diabetics did not have this variant (Hegele et al., 1999; Hegele et al., 2000a; Hegele, 2001). The mechanism by which this variant may relate to T2DM is not clear, although one study has suggested it may relate to increased plasma concentrations of high density lipoprotein (Hegele et al., 2000b) and accelerated disease onset in the population of Indigenous Canadians in question (Triggs-Raine et al., 2002). However, a study of African Americans found no association between variations in the HNF-1α gene and T2DM (Elbein et al., 2000). Variations in a related gene, HNF-4α, have also been linked to T2DM susceptibility in Pima Indians (Muller et al., 2005) as well as in Ashkenazi Jewish (Love-Gregory et al., 2004) and other European populations (Silander et al., 2004; Weedon et al., 2004). Both genes were first identified as candidate loci by virtue of their involvement in MODY. Variations linked to T2DM susceptibility in Caucasian populations have also been identified for the KCJN11 gene (Hani et al., 1998), PPARγ (Altshuler et al., 2000), and the VNTR region of the insulin locus (Huxtable et al., 2000).

In summary, current *genetic* epidemiological evidence suggests a diffuse and highly heterogenous picture of genetic susceptibility to T2DM, with a restricted number of variants at a small number of loci showing any appreciable degree of contribution to disease risk. Many of the best described gene-disease associations have been observed only in white/Caucasian samples, and replication across different ethnoracial groups has in certain cases proven problematic.

2.6.2 Genetic *Epidemiological* Research

Genetic *epidemiological* research seeks to explain differentials in disease incidence/prevalence between various ethnoracial populations via the identification of genetic differences between these populations. In comparison to *genetic* epidemiological resarch, there has been relatively less genetic *epidemiological* research conducted. This is particularly so in contempoarary research which is attracted by the allure of more sophisticated genomic technologies.

Of the existing research literature, admixture studies are the most common approach to genetic *epidemiological* research on the TG hypothesis. These studies seek to determine whether and how much the incidence of T2DM (and/or associated factors) varies with an individual's degree of Indigeneity after controlling for (or, more typically, assuming the absence of) differences in social/environmental risk factors between members of the study population. An association found between reduced admixture (i.e. more Indigenous ancestry) and an increased prevalence of T2DM is taken to indicate that one or more genes inherent to the indigenous population in question causes a predisposition to T2DM.

There are a variety of approaches to measuring admixture in studies of the TG hypothesis. An assessment of self-reported ancestry (King et al., 1984; Serjeanston et al., 1983; Stein et al., 1965) is the most common approach with skin colour (Gardner et al., 1984) and HLA antigen status (Williams D.R.R. et al., 1987) also utilised. Taken together, admixture studies provide some evidence that the prevalence of T2DM increases with an increasing degree of Indigeneity (i.e. reduced admixture) among Native Americans (Brosseau et al, 1979; Stein et al., 1965; Williams R.C. et al., 2000), Nauruans (Serjeanston et al., 1983) and Mexican Americans (Gardner et al., 1984). There is also some research indicating similar associations between blood glucose/insulin levels and Indigeneity among populations in the Pacific (King et al., 1984) and Indigenous Australians (Williams D.R.R. et al., 1987).

Unfortunately, many of these studies fail to adjust for potential confounders (Brosseau et al, 1979; Stein et al., 1965; Williams D.R.R. et al., 1987) or adjust only for factors

such as age, BMI and tricep skinfold thickness (King et al., 1984), BMI, age and sex (Serjeanston et al., 1983; Williams R.C. et al., 2000) or obesity (Gardner et al., 1984); rather than for possible social/environmental influences. In fact, data on income (Gardner et al., 1984) as well as employment status, diet, smoking (Williams D.R.R. et al., 1987) were collected in two of these studies but were not utilised in analyses. Similarly, BMI was measured in one study and found to vary by admixture but was still not utilised in analysis (Williams R.C. et al., 2000).

Of particular concern is the offhand manner in which a number of these studies dismiss the relevance of sociocultural variables by failing to measure but nonetheless rejecting lifestyle factors (e.g. diet and physical activity) as well as environmental and sociocultural characteristics as possible influences on study findings (Brosseau et al, 1979; King et al., 1984). Such disregard for non-genetic factors is especially surprising given that there have also been several studies that provide genetic *epidemiological* evidence against the TG hypothesis. These studies found that degree of Indigeneity was unrelated to T2DM in Native Americans (Marshall et al., 1993), Pacific Islanders (Zimmet et al., 1982), Mexicans (Stern et al., 1992) and Aboriginal Australians (Guest & O'Dea, 1992).

As a whole, the genetic *epidemiological* research detailed above suffers from a failure to measure, adjust for and consider in interpretation, the social and environmental risk factors of known relevance to T2DM etiology. Furthermore, weak, inconsistent, contrary and even non-existent evidence for a TG is frequently misconstrued as supporting the TG hypothesis and a number of researchers continue to perpetuate the misconception that there are *a priori* genetic differences between people of various ethnoracial groups, ancestry and skin color (Stern, 1999:S67), as well as homogeneity within these groups (Osei, 1999; Schulz & Weidensee, 1993; Williams R.C. et al., 2000; Zimmet et al., 1982).

Overall there are clearly considerable limitations to the evidence from both forms of genetic epidemiological research on the TG. There is only very preliminary and ambiguous evidence for specific thrifty genes and both these gene-specific studies and admixture studies fail to sufficiently account for known and possible social/environmental causes of T2DM. The fact that over 250 genes have been studied as possible causes of T2DM, but together these genes explain less than 1% of diabetes

prevalence worldwide (Lev-Ran, 1999:12), should give researchers – and others – pause.

2.7 Ethnorace, Thrifty Genes and Social Inequality

Given the theoretical limitations inherent to the TG hypothesis, together with a lack of consistent empirical evidence with which to eliminate other, potentially more compelling, causative mechanisms as well as continuing critiques of the hypothesis (e.g. Fee, 2006; McDermott, 1998; Speakman, 2006), it is unclear why investigators continue to hypothesise a 'pre-existing genetic susceptibility which is 'amplified' or 'unmasked' by rapid environmental change (Bennett, 1999; O'Dea, 1997). With the recognised difficulties in determining the independent effects of genes and environments, and the overwhelming influence of environmental factors on recent disease trends, Occam's razor would suggest that, in the absence of reliable evidence for such a susceptibility, the TG hypothesis is superfluous. Unfortunately, a feature of adaptive scenarios such as the TG hypothesis is that they cannot be explicitly tested (McGarvey, 1994), and hence are, in essence, non-falsifiable. As long as researchers continue to be influenced by folk knowledge that conflates race and genetics, the TG hypothesis is likely to continue to thrive despite the strong conceptual and empirical evidence refuting it.

This chapter has demonstrated that the etiology of T2DM is not explained by the genetics of population differences alone – genetic *epidemiology*. It has been shown that T2DM is not wholly explained as a function of specific genetic predispositions of individuals, from whatever group – *genetic* epidemiology. Rather, the continued use of ethnoracial groups in genetic epidemiology is an instance of the ascription of biological difference to socially defined groups. This practice is problematic in light of the pernicious use of scientific rhetoric to explain educational, professional and socioeconomic attainment based upon presumed biological differences (Hernstein and Murray, 1994; Hutchinson, 1997; Rushton, 1994). Ethnographic research demonstrates that scientists are often aware that ethnoracial labels are both inaccurate and politically loaded (Montoya, 2003) and are concerned by the social consequences of their research (Montoya, in press).

Scientific claims have long been recognised as deeply reflective of momentary social concerns (Latour, 1991; Ziman, 2000), especially claims about ethnoracial groups (Harrison 1994, 1995, 1998). It is only because the populations used in T2DM research have an *a priori* ethnoracial identity that their genetic material can be imbued with their racialising potential. In general, ethnoracial groups are specifically selected for genetic epidemiological research because of the high prevalence of a disease of interest. It should be evident that such increased disease prevalence is due to specific environmental, historical, social and political pressures interacting with individual genetic predisposition in complex and varying ways.

Thus, ethnoracial population labels, if at all informative for genetic epidemiology, reference the biological effects of a specific population's history rather than their inherent genetic constitution. Therefore, to derive genetic explanations for such prevalence differences without controlling for environmental confounding tautologically reiterates "the social conditions from which the ethnic groups are derived rather than [providing] any etiological information pertinent to an individual's or groups' biology" (Montoya, 2003:39). At the same time such research, almost paradoxically, renders invisible the social etiology of diabetes.[8] As a consequence, social inequality is embodied as a form of individual pathology while the "hegemony of biological solutions for a chronic sociological dis-ease" is asserted (Montoya, 2003:40). Moreover, this use of ethnoracial labels distances researchers from the important work of locating the physiological and environmental triggers for complex diseases (Braun, 2002).

It is important to recognise that racialisation is a sociocultural phenomenon and not a characteristic of individual scientific projects much less individual scientists. In this light, the use of ethnoracial categories in TG research is an example of racialised genetics more generally and is premised upon a trained incapacity (Wellman et al., 2003) to appreciate the nature of ethnorace as a social rather than a genetic

[8] Interestingly, advocates of the TG hypothesis regularly recommend environmental (lifestyle and socio-political) change as a remedy for disparate rates of T2DM even as they situate the disease as being primarily genetic in origin (see Osei, 1999; Ravussin et. al., 1994; Zimmet, 2000).

phenomenon. There are considerable epistemological barriers to imagining disease etiology as both socially and biologically mediated – barriers that reinforce professional pressures to conduct disembodied genetic epidemiology.

However, it is possible for the field of public health to incorporate genetic knowledge as a way of attending to the production of 'local biologies' (Lock, 1994) as they relate to health and illness. To do so will require rigorous transdiciplinary research between geneticists and social scientists in order to develop new approaches to understanding the intersections between ethnorace, genetics, biology and society (Fee, 2006; Hammonds, 2005).

2.8 Rehabilitating Genetic Epidemiological Research

The use of ethnorace as a proxy for genetic differences limits understanding of the complex interactions among socio-political-economic processes, lived experiences, and human biologies. As such, it is imperative that both *genetic* epidemiological and genetic *epidemiological* research is conducted in a way that does not imply that ethnoracial categories are biologically distinct. In keeping with this, perhaps it is appropriate for genetic research to always include a caveat that genetic variation exists between any human groups – however defined – and thus it is erroneous to assume that such variation reflects 'racial categories' (Duster, 2005). At the same time research must examine existing health inequalities as well as genuine genetic variation across human populations (Lee et al., 2001:69). It is vital that such studies measure, stratify by and control for social/environmental factors (Weiss & Terwilliger, 2000), including sociocultural and sociopolitical variables such as racism, stress and class (Cooper et al., 2003; Kittles & Weiss, 2003:54), as well as gene-environment interactions and exposure to environmental toxins (Ellison & Jones, 2002).

Transdisciplinary research has emerged to link population health parameters, including environmental exposures and social conditions, with both pathophysiology and genetic risk estimates. The challenge for such research is to develop interlinked biological, social and ecological constructs, constructs which can only be derived via careful attention to the life course of individuals, families and particular groups of people. Methods include *in situ* participant-observation, informal interviews, time use and

behavioral inventories, life histories and other data collection strategies. Such proximally captured data will lend untold power to biomatrices, biobank inventories, quantitative surveys, and other biophysiological data including genetic data. An absence of ecologically oriented biocultural data (Goodman & Leatherman, 1998; Krieger, 2005b; Trostle, 2005) will perpetuate the reductionistic disconnect between genotype and phenotype, ecology and well being. In short the dynamic between human life conditions and human biology will remain a mystery.

The problem of racialisation in contemporary genetic epidemiology has been identified previously, with calls for alternatives to the unreflexive recapitulation of biological distinctions among ethnoracial categories falling into three classes of proposed rehabilitation. First, many have been interested in by-passing ethnorace altogether via an exclusive emphasis on the identification of disease-linked genes shared among family members. Family-based studies, which traditionally have been the preferred methodology of human molecular genetics, represent the best way to enrich one's sample for defined genetic determinants and provide some reduction in genetic heterogeneity (Khoury & Yang, 1998) relative to population-scale association studies. Moreover, family-based studies largely bypass the issues arising from population stratification. These advantages, however, must be weighed against the difficulties inherent to family-based recruitment, the greater degree of genotyping required to achieve similar statistical power (Hirshchhorn et al., 2002), and the fact that the variants identified may be rare and family-specific (hence contribute little to population attributable risk). Also, because families are embedded in wider sociocultural contexts, ethnorace is often still designated by investigators in these studies (Ghosh et al., 1999; Ji et al., 1997; Klupa et al., 2000; Luo et al., 2001; Mori et al., 2004; Rotimi et al., 2004).

One, increasingly popular, alternative to family-based genetic identification is to employ so-called 'race neutral' approaches to the problem of identifying allele frequency differences associated with population stratification that have the potential to confound tests of genetic association with disease. These methods, which fall broadly under the banner of 'genomic control,' involve either the genotyping of several dozen to several hundred random genomic markers to determine the degree of sample stratification (Devlin et al., 2001, Rosenberg et al., 2003) or the genotyping of a smaller number of 'Ancestry Informative Markers', or AIMs, preferentially chosen on the basis

of their ability to distinguish the presumed ancestral populations contributing to varying degrees of admixture among cases and controls (McKeigue, 2005). While such methods may appear, at first blush, an appropriate work-around, allowing genetic epidemiologists to control for confounding without any recourse to ethnoracial ascription, they misleadingly imply that genetic confounding is the only confounding of interest, and that environmental exposures and other life experiences are irrelevant to the assessment of disease susceptibility. Research that attempts to explain disease prevalence between human groups using such techniques alone will almost always conflate the descriptors of the population with the attributes of the population. It is precisely this racialisation that the above discussion seeks to thwart.

Analytical approaches to epidemiological research - genetic and social - that begin with the premise that population differences in health reflect the embodied interaction of biological and social conditions (Goodman & Leatherman, 1998; Krieger, 2005b; Trostle, 2005) are clearly to be preferred. Some investigators (e.g. Kaufman & Cooper, 2001; Shields et al., 2005) have recommended that ethnorace, seen largely as a proxy of social disadvantage, be replaced by concerted investigation of robust markers of more direct etiological relevance to disease (or health outcome) wherever possible. Ironically, however, such approaches may inadvertently serve to reinforce the misapprehension that biological and sociopolitical determinants of disease manifestation are, in fact, etiologically separable. From this standpoint, there may be compelling reasons to include ethnoracial identity alongside other indicator variables, not as an aggregate proxy for yet-to-be-identified causal determinants, but as a higher-order manifestation of the inseparable joint effects of population history and biological predisposition. Thus, to establish causality will require detailed information on genetic variation throughout the genome *and* detailed information on social and environmental exposures (Ellison & Jones, 2002).

Research combining the study of genetic and social etiology without reinforcing dangerous notions of racialised biology will allow us to understand why some gene-disease associations have varying strengths across human groups. To ignore such ethnoracial differences in disease outcomes would be just as wrong as essentialising such differences as simply biological in origin. However, when prioritising research, it is important to consider that, at present, environmental factors are (at least technically) far more amenable to modification than genetic ones and are likely to have a greater

impact on health (Kittles & Weiss, 2003:58; Merikangas & Risch, 2003;Weiss & Terwilliger, 2000).

Genetic and epidemiological researchers need to move beyond the mere recognition of ethnorace as socially constructed towards the practice of studying ethnorace as politically constructed. It is hoped that this will lead to a decline in geneticised notions of ethnoracial health disparities in the public imagination. Just as serfs—also treated as a hereditary group—vanished as such with the elimination of the feudal legal structure, it is possible that abandoning the interpellation of race as biological/genetic by the scientific community may also result in a vanquishing of biological connotations of ethnorace, such that perhaps, one day, being Black will be regarded as no more genetic than being from New York (Stevens, 2003:1077-8). Researchers interested in the causes of complex disease like T2DM will profit by examining ethnorace as a complex sociocultural phenomenon and by directing their attention to the biological consequences that ensue from such phenomena.

Chapter 3. ***Defining, conceptualising and characterising racism in health research***[9]

3.1 Introduction

Having introduced racialisation in Chapter 2, this chapter further explores this concept in relation to the sociocultural phenomena of racism, the examination of which, as a determinant of health, is an emerging area of research. Of the 138 published studies on this topic (reviewed in Chapter 4), 65% were published between 2000 and 2004. A range of definitions, conceptualisations and characterisations of racism as a determinant of health have been formulated and utilised within this nascent area of study. However, the approaches used have varied in coherence and clarity. There are three levels at which coherence and conceptual clarity can be sought in this area of study: in defining and theorising racism itself, in clarifying how racism may relate to health and in characterising racism as an exposure which can be operationalised and measured. Drawing on the existing literature, this chapter examines and expands upon the various approaches to the study of racism and health in relation to these three levels, with a view to guiding future research in this area.

3.2 Defining and theorising privilege/oppression and racism

Most studies of racism and health to date have not defined or theorized racism before going on to operationalise and measure this phenomenon. For instance, only 34 of the 138 studies reviewed in Chapter 4 offer a definition of racism, and where definitions are included they suffer from a range of limitations. However, before discussing these limitations, it will be useful to first present a definition and theorisation of racism which overcomes them.

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[9] This chapter is taken from Paradies Y. (2006). Defining, conceptualizing and characterizing racism in health research. *Critical Public Health* 16(2): 143-157.

Racism can be thought of as one of many types of oppression which, along with its dialectical opposite privilege, can be based on a range of social characteristics including gender (sexism), sexuality (heterosexism), physical and mental able-ness (ableism), age (ageism), class (classism), nationality, body size/shape, criminality, religion, and language/accent among others. The intertwined concept of privilege/oppression can be defined as:

A societal system in which actors are divided along socially constructed dimensions with power unevenly distributed (or produced) based on these dimensions.

The social system of privilege/oppression is based on ideologies (worldviews) about differences between groups which are embodied through attitudes, beliefs, behaviours, laws, norms and practices. Despite numerous existing forms of privilege/oppression, the vast majority of identifiable differences between, for instance, people are not sites for privilege/oppression (i.e. power is not distributed or produced based on these dimensions). For instance, although they may be relevant in limited social circumstances, one's blood group or the last three digits of one's phone number are not characteristics along which power is distributed. Clearly, which attributes are salient in terms of power relations (or even perceptible as differences at all) differ dramatically across time and space.

It is also important to note that individuals can be differently placed in relation to various types of oppression. For example, a White woman may be positioned as privileged in terms of 'racism' but positioned as oppressed in terms of 'sexism'. Hence types of privilege/oppression are cross-cutting and potentially in tension.

Racism, as a particular form of privilege/oppression, exists in a dialectical relationship with anti-racism, both of which depend on the concept of racialisation as described in Chapter 2. It is important to note that, regardless of ongoing scientific debate on the biological reality of ethnorace (see Chapter 2), popular and lay ideas of ethnorace often centre on essentialised concepts of culture as markers of innate difference between social groups. As such, ethnorace is about much more than just skin colour or phenotype and is instead an intrinsically protean concept which is not necessarily either immutable or inheritable (see Black & Solomos, 2000).

As with privilege/oppression more generally, racism is embodied through attitudes, beliefs, behaviours, laws, norms and practices and can be similarly defined as:

A societal system in which actors are divided into ethnoraces, with power unevenly distributed (or produced) based on these ethnoracial classifications.

3.3 Theorising privilege/oppression and racism

There are a number of issues fundamental to the definitions of privilege/oppression in general and racism in particular (as presented above) which require further explication. These issues include the concept of power and its relation to privilege/oppression and racism as well as anti-privilege/oppression and anti-racism; the perpetration of privilege/oppression; the interplay between intention and effect; and objective vs. subjective racism.

3.3.1 Power

There are three broad approaches to conceptualising power in the social sciences – power 'over' (conflictual power), power 'to' (consensual power) and power as constitutive of reality. Power 'over' entails the ability of an actor to prevail over another to do something they are resistant to, would not otherwise have done, and/or isn't in their interests, while power 'to' is the generalised capacity of an actor to make something happen (Haugaard, 2002). Conflictual approaches to power are focused on social relations alone whereas consensual approaches focus more on material and/or social action. Also, some conflictual approaches adopt a 'zero-sum' conception of power wherein power is finite (i.e. cannot be produced) and in which power gained by an actor requires power to be lost by another (see Haugaard, 2002). The postmodern notion of power as 'constitutive of reality' contends that power is fundamental in determining who actors are, what they want to 'make happen' and/or their interests and who they want to influence (Haugaard, 2002). It is important to note that the definitions and concepts developed in this chapter are consonant with any of the above approaches to power, rather than being based on any one in particular.

There are two dimensions across which both privilege/oppression and racism can vary in terms of power. First, power can decrease or increase in an *absolute* sense for one or more social groups as a result of privilege/oppression. Second, these absolute changes in power can have the *relative* effect of either attenuating or enhancing power asymmetries. For example, a dominant (i.e. privileged) ethnoracial group such as Whites has relatively more power than a subordinate (i.e. oppressed) ethnoracial group such as indigenous peoples. If a dominant ethnoracial group experienced an absolute increase in power this would have the relative effect of enhancing power asymmetries and would be an instance of racism in particular and privilege in general. On the other hand, if a subordinate ethnoracial group experienced an absolute increase in power this would attenuate existing power asymmetries and would be an instance of anti-racism in particular and anti-oppression in general. Similarly, if Whites experienced an absolute decrease in power this would have also the relative effect of attenuating existing power asymmetries and would be an instance of anti-racism in particular and anti-privilege in general, whereas if an Indigenous group experienced an absolute decrease in power this would enhance existing power asymmetries and would be an instance of racism in particular and oppression in general. So, in summary, attenuation of existing power asymmetries (i.e. anti-privilege/oppression) occurs when dominant group members experience an absolute decrease in power (i.e. anti-privilege) and/or subordinate group members experience an absolute increase in power (i.e. anti-oppression) and existing power asymmetries are enhanced when dominant group members experience an absolute increase in power (i.e. privilege) and/or subordinate group members experience an absolute decrease in power (i.e. oppression).

When using power analyses to unpack the concept of (anti-)privilege/oppression or (anti-) racism it is important to consider the scale or level of the analysis in question. Power differentials between ethnoracial groups may exist in one direction at a micro-level of analysis and have a different pattern at meso or macro levels. For example, a social interaction between a Black and White colleague within an organisation managed and staffed primarily by Black people that was itself situated in a nation with a dominant White culture may have various and cross-cutting power differentials at each level of analysis.

Under the above definition, racism can be either about privilege or oppression and given this, it is now possible (using data from Chapter 4) to understand one of the most

common weaknesses in existing definitions of racism: an asymmetry whereby only the *oppressive* effects of racism for subordinate ethnoracial groups are considered (as in Broman, 1996; Clark 2000; Finch et al., 2000; Fischer & Shaw, 1999; Jackson et al., 1996; Kennedy et al., 1997; Landrine & Klonoff, 1996; Loo et al., 2001; Morris-Prather et al., 1996; Peters, 2004; Ren et al., 1999; Sanders-Thompson 1996; Schneider et al., 2000; Tull et al., 1999; Valentine et al., 1999) rather than also considering the *privileging* effects of racism for dominant ethnoracial groups (as in Gee, 2002; Jackson & Inglehart 1995; Murrell, 1996; Noh et al., 1999; Nyborg & Curry 2003). Similarly, only one definition of racism in the 138 articles reviewed in Chapter 4 recognised ideologies of inferiority and superiority as inseparable aspects of the same phenomenon (Murrell et al., 1996), while other definitions attributed racism to an ideology of inferiority (Nyborg & Curry 2003; Ren et al., 1999; Wiking et al., 2004) or superiority (Din-Dzietham et al., 2004; Peters, 2004; Utsey et al., 2002b; Williams D.R. et al., 1997) in isolation. Several of these definitions of racism also refer to differential treatment by ethnorace without specifying this as *negative* differential treatment (Jackson et al., 1996; Roberts et al., 2004; Stuber et al., 2003; Williams D.R. et al., 1997), thus implicitly classifying anti-racism as indistinguishable from racism.

3.3.2 Perpetrating privilege/oppression and intention vs. effect

The term ‘actors’, as used in the definitions of privilege/oppression and racism above, requires further explanation. This term is derived from actor-network theory as developed by Latour (1986) and Callon (1988) in the field of science studies. In this theory, actors or social actors (also known as ‘actants’) are entities which exist within social networks where they are both acted upon and can act upon other entities. This theory adopts a symmetrical approach to analysis in which the form actors can take (i.e. (non-)human or (non-)living) is not decided *a priori*. Actor-network theory contends that social networks consist of humans, machines, animals, and matter in general and, for the purposes of this chapter, highlights the fact that any actors in these networks can be implicated in privilege/oppression (e.g. non-human actors such as laws and institutions can perpetrate systemic racism).

It is important to note that, under the above definition, privilege/oppression can be perpetrated by members of any social group. For example, racism against Indigenous

peoples can be perpetrated by either non-Indigenous peoples (i.e. inter-racial racism) or by Indigenous people themselves (i.e. intra-racial racism). Intra-racial racism occurs when the actions of one or more members of an ethnoracial group have the effect of enhancing power asymmetries for either themselves or other members of their ethnoracial group. Despite clear descriptions of both the existence and deleterious consequences of intra-racial racism in contemporary health research (Brondolo et al., 2005a; Clark et al., 1999:806; Clark, 2004; Major et al., 2002a), several existing definitions of racism in health research implicitly define subordinate ethnoracial group members as being incapable of perpetrating racism (Broman, 1996; Finch et al., 2000), with only a single definition in the research reviewed in Chapter 4 explicitly recognising the possibility of intra-racial racism (Peters, 2004).

Related to the definition of actors is the question of intentionality in perpetrating privilege/oppression and racism. The above definition of privilege/oppression (and hence racism) does not depend on the intentions of actors, but instead is concerned with the social effects of their actions. Although 'intention' is certainly an important factor in assigning moral culpability and/or reparations for privileging/oppressive acts, it is not a sufficiently clear phenomenon by which to define the notion of privilege/oppression. Attempting to define privilege/oppression in relation to only intentional acts (as in Noh et al., 1999) fails to recognise that oppression is systemic in society and is unwittingly and unconsciously (re-)produced by many actors who have no racist intentions whatsoever.

3.3.3 Subjective vs. objective racism

One of the reasons why the phenomenon of racism is difficult to operationalise and measure objectively is because it is based on *differential* effects (in relation to power) by ethnorace. Any particular experience cannot be objectively determined as racist without comparison to another experience that is, at least for practical purposes, identical except for the ethnorace of actors involved in the situation or event. For example, when seeking service at a department store the behaviour of a service provider towards a White client may be starkly and observably different to his/her behaviour towards an Asian client who is served after the White client, hence establishing a differential by ethnorace. Alternatively a real estate agent may fail to show a Black

client housing that (s)he showed to a White client. Without setting up an experiment to test for this differential (i.e. by having both Black and White clients who are otherwise identical seek housing from the same real estate agent), a particular individual may be unable to determine if they are being subject to objective racism. Thus, in determining whether a situation or event is racist, it is first necessary to establish that racialisation is occurring (i.e. that ethnorace is a salient feature of the situation/event) and then it must be determined that a differential effect in terms of power is involved.

Given the 'objective' occurrence of racism as an increase in power differentials based on ethnorace, there are a number of 'subjective' interpretations of such an occurrence. Some forms of racism such as systemic or internalised racism (see below for definitions of these terms) are frequently not perceived subjectively (and may not be perceivable) by individuals who experience these phenomena. It is also possible that racism can be perceived as occurring subjectively when it has not occurred objectively and, of course, subjective and objective occurrences of racism can coincide. Following on from the example above, a particular actor may subjectively experience racism during his/her interaction with a real estate agent when an experiment would determine that objective racism did not occur, or, alternatively could have the same subjective experience when an experiment would show that objective racism had occurred. Further discussion of how objective and subjective racism have been studied in health research and how these phenomena may relate to health outcomes can be found in the section on characterising racism below.

3.4 ***Conceptualising Racism in Health Research***

Although there have been a range of theoretical approaches to the study of racism in sociology, psychology and other social science disciplines, these theories are primarily concerned with the causes of racism (i.e. the why of racism) including how personality, identity, group dynamics, and ideology are implicated in creating and sustaining racism. However, the study of racism in health research is concerned, at least at present, with how racism may influence health rather than why racism occurs.

In the field of public health itself a number of scholars have formulated theories of racism including ecosocial theory (Krieger, 2001), biopsychosocial models (Clark et al.,

1999), and the weathering (Geronimus, 1992) and double jeopardy hypotheses (Ferraro & Farmer, 1996). The 'weathering' hypothesis contends that 'accelerated aging' due to the cumulative impact of racism is the cause of ill-health (Geronimus, 1992). Similarly, the 'double jeopardy' hypothesis contends that older African Americans are particularly at risk of poor health due to the interaction between racism and aging (Ferraro & Farmer, 1996). Krieger's ecosocial theory is a multi-level epidemiological framework that seeks to integrate social and biological reasoning through a dynamic, historical and ecological perspective, while biopsychosocial models conceptualise racism as a form of stress that results in psychological/physiological damage which is moderated/mediated by a range of characteristics and coping responses (Clark et al., 1999).

Theories and hypotheses about racism are useful in health research in so far as they provide either *predictive power* to explain how racism contributes to patterns of population health (i.e. the weathering/double jeopardy hypotheses have this potential) or provide a *paradigm* through which to understand racism as a determinant of health (i.e. ecosocial theory and biopsychosocial models have this potential). However, the patterns of associations between racism and health are only now becoming apparent in any detail (see Chapter 4 below), and hence paradigmatic conceptualisations of racism may be more useful at present in order to understand how racism, in a general sense, may influence health. Figure 1, which is influenced by a model of the social determinants of health (Turrell et al., 1999) and incorporates aspects of both ecosocial theory and biopsychosocial models, conceptualises racism as one type of oppression in relation to human actors.

In the structural realm shown in Figure 1 fundamental ontological, epistemological, and axiological features of society are encapsulated in notions of culture, ideology and worldview. These factors interact with major institutions of society and together are influenced by and produce systemic oppression. Giddens' theory of structuration (1984) can explain the process by which the structural realm is reproduced and transformed through the action of actors, with these actors simultaneously constituting themselves through this structured action. The structural realm exists through co-constructed meaning which is used to produce certain ordered states such as social institutions. As noted by Haugaard (2002), Giddens' theory conceptualizes the structural realm as both constraining (in that its ordered nature restricts the universe of all possible actions) and enabling (in that actors have agency and realise their specific goals through the existing

structure even as they recreate it). Systemic oppression (defined below in relation to racism) acts, through structuration, to differentially construct the nature of the inter-personal realm.

It is within the inter-personal realm in Figure 1 that social identity is constituted in interaction with the structural realm. Given the focus here on social effects rather than intentions, identity can be seen as existing in *the space between actors* where it is constantly (re-)produced through the meaning conferred on it by these actors. Social identity (which can be considered here as synonymous with social location) can be understood through Bourdieu's notion of 'habitus' in that identities/locations are the set of dispositions (tendencies, propensities or inclinations) which structure an actor's social, spatial and temporal orientation to the world and which are conceived dialectically at the point where the actor and society intersect (Smaje, 1997). Social identities/locations are fluid, contextual and contested and are both inextricably bound to inter-personal and internalised oppression (defined below in relation to racism) and perennially (re-)created through the lens of systemic oppression.

Finally, at the level of the individual a biopsychosocial approach can be used to show how inter-personal and internalised oppression influences and is influenced by psycho-social and behavioural factors as well as bio-physical (including genetic) processes. These pathways lead to various (and differential) health outcomes for human actors who occupy differing social identities/locations and are variously exposed to systemic, inter-personal and internalised racism.

An important caveat to this conceptual diagram is that power as used in defining privilege/oppression is not directly synonymous with the notion of health. For instance, it is not necessarily the case that power gained (or produced) through (anti-)racism will lead directly to improved health. Conversely, it is also not necessarily true that improved health will be associated with increased power as such improvement may come about due to paternalistic or fascist social action which is by definition oppressive. Hence, although it is not possible to show this graphically, it must be remembered that the relationship between power and health is complex and even contradictory in nature.

The idealised processes shown in Figure 1 occur across the dimensions of both time (history) and space (geography) as well as across the whole life course. This conceptualisation of racism in particular, and oppression more generally, as a determinant of health is designed to aid thinking in this emerging area of research in relation to the organisation and synthesis of knowledge, generation of specific research questions, definition of concepts and variables, and in determining particular analytical approaches (Paradies & Stevens, 2005). However, in conducting studies on racism and health, a closer examination of racism as a health risk condition is also required in order to determine how best to characterise, operationalise and measure exposure to racism.

3.5 ***Characterising Racism as a Determinant of Health***

As discussed above there are two main approaches to characterising racism as a determinant of health: subjectively as a direct perception or experience (known as perceived racism) and objectively by indirect inference (either experimentally or observationally), with both of these approaches suffering from different limitations. Direct measurement approaches involve self-reports of racism as an exposure in association with health-related outcomes. It is important to emphasise that, along with similar concepts such as stress, self-reported perceptions/experiences of subjective racism can affect health regardless of the objectivity of such reporting. The term 'self-reported racism' is taken from Krieger et al. (2005) and is not meant to imply that the racism experienced/perceived and then reported by respondents is in any way imagined, fabricated or illusionary. Nonetheless, such self-reports are influenced by a range of psychological and socio-demographic factors that are, at present, poorly understood (Major et al., 2002b). Indirect measurement approaches infer that exposure to racism is associated with health-related measures by eliminating (in either design or analysis) other possible explanations. A study utilising an indirect measurement approach may, for example, conclude that health differentials between Black and White sub-groups of a study population that remain after adjustment for age, gender, sexuality and socioeconomic position are due to experiences of racism. An indirect approach to studying racism is the only available option when racism is not perceivable or expressible by individuals (Krieger, 1999) or when racism cannot be readily or comprehensively measured – as is the case for some forms of systemic and internalised racism. However, especially for observational studies, there are clear methodological

limitations to indirect approaches due to the possibility of incomplete coverage and poor measurement of confounding factors as well as an inability to investigate possible interactions between racism and other variables (Bonilla-Silva, 1997).

Because of difficulties in measuring racism objectively, much of the study of racism in health research has focused on perceived racism. Figure 2 details the range of dimensions across which perceived (subjective) racism (without regard to the existence of objective racism) can be characterised in health research, including dimensions of exposure to racism as well as possible reactions and responses to this exposure. As discussed above, racism can be due to either oppression or privilege. However, to date virtually no research has been conducted in relation to racism as privilege and its association with health. As a result the characterisation of racism, as shown in Figure 2 and discussed below, relates only to the oppressive aspects of perceived racism.

3.5.1 Exposure to Racism

Exposure to racism, as with any exposure, can occur at different stages of the life course, with various intensities in relation to mental (cognitive load) or physical (allostatic load) stress and at different frequencies in time and in relation to regularity, acuteness, chronicity (Krieger, 1999). The duration of exposure to racism can also vary from fleeting to constant and can occur cumulatively across generations, across processes within a particular setting (see below) over time, or across settings over time (Blank et al., 2004).

3.5.2 Characteristics of Racism

The characteristics of racism include its mode, form, level, expression and setting as well as the perpetrators/targets of racism. As discussed above, racism can occur either between actors of different ethnoraces (inter-racial) or actors of the same ethnorace (intra-racial) and can take a variety of forms including legal or illegal, direct or indirect, overt or covert (Krieger, 1999) as well as blatant or subtle, and vicarious via other targets of racism (Harrell, 2000). As discussed above in defining racism, this phenomenon can also be unintentional as well as intentional and may occur through both action and inaction.

Jones (2000) has described racism as occurring at three distinct levels – internalised, inter-personal and systemic (Jones refers to the latter two levels as personally mediated and institutionalised, respectively). Using the framework presented by Jones, internalised racism is defined in this chapter as the incorporation of racist attitudes, beliefs or ideologies within an actor's worldview. There are two forms – *internalised dominance* (i.e. privilege) and *internalised oppression.* Internalised dominance is the incorporation of attitudes, beliefs or ideologies about the inferiority of other ethnoracial groups and/or the superiority of one's own ethnoracial group. On the other hand, internalised oppression is the incorporation of attitudes, beliefs or ideologies about the superiority of other ethnoracial groups and/or the inferiority of one's own ethnoracial group.

Inter-personal racism is here defined as racist interactions between actors whilst systemic racism is defined as the racist production, control and access to material, informational and symbolic resources within a society. Systemic racism includes what others have referred to as institutional(ised), structural (Jones, 2000), cultural (Jones J.M., 1997), societal, and civilisational racism (Scheurick & Young, 1997). As shown in Figure 1 in relation to oppression, these levels of racism are intertwined with an individual's worldview being created through socialisation while societies themselves are constantly (re-)created by the behavior of individuals carrying out established roles in interaction with one another.

Racism can be expressed through stereotypes, prejudice or discrimination (i.e. racist beliefs (cognition), emotions (affect) and behaviours, respectively). As detailed above, racism must have some social effect in order to be considered as such and in this sense some action is required to *express* racist beliefs and emotions (requiring some form of behaviour), whereas behaviours need not involve beliefs or emotions. As shown in Figure 2, racism can also be expressed in a range of settings (Krieger, 1999) which correspond to the institutions detailed in the structural realm in Figure 1. There are also a range of actors who can be either perpetrators or targets of racism. Both the settings and perpetrators/targets shown in Figure 2 are intended to be indicative rather than exhaustive of the range of possibilities.

3.5.3 Reactions/Responses to Racism

As with the expression of racism itself, a human actor's reactions/responses to racism may be cognitive, affective or behavioural (McNeilly et al., 1996a) and can come in active or passive (Harrell, 2000) as well as adaptive or maladaptive forms as detailed in Figure 2. Self-blame is a cognitive passive maladaptive response which occurs when a racist experience is given an internal attribution by an individual (i.e. when (s)he blame him/herself for racism occurring because of his/her action). On the other hand the cognitive active adaptive response of system-blame occurs when a racist experience is given an external attribution (i.e. the racist experience is attributed to other actors) (Neighbors et al., 1996). 'Imagining responses to racism' occurs when targets of racism think about what responses they would have made to a perpetrator if the situation occurred over again. This is a cognitive active adaptive response that can be self-assuring and comforting for targets of racism (Mellor, 2004).

Cognitive maladaptive active responses include hypervigilance in which an individual devotes an extreme amount of cognitive effort to determining whether or not racism occurred, thus creating additional stress for themselves above and beyond the effects of racism itself. Similarly, 'attributional anxiety' is the process of questioning whether an experience was racist through replaying situations in one's mind or entertaining alternative explanations and this can also be stressful above and beyond the original racist experience (Harrell, 2000). 'Denial of racism' and 'self-blame' (active responses) 'resigned acceptance' (passive response) are cognitive responses which attempt to avoid the need to deal with racism at all.

Affective or emotional responses to racism can be classified as either inner-directed or outer-directed in relation to an individual's experience of racism (Harrell, 2000). Inner-directed disempowered affective responses can be either active (e.g. shame, self-hatred, humiliation, anxiety, fear) or passive (e.g. powerlessness, hopelessness, confusion, depression) while outer-directed emotional responses which are, by definition, active can be either in the empowered or disempowered mode. Empowered responses occur when a human actor who experiences racism projects inferiority onto the perpetrator (Mellor, 2004) and feels contempt, amusement and/or sorrow/sympathy for them (McNeilly et al., 1996a), whilst disempowered responses include emotions such as anger, annoyance or frustration.

Behavioural responses to racism can either be adaptive or maladaptive and within these categories can be problem- or emotion-focused (Harrell, 2000). As with affective response, adaptive behavioural responses can be inner- or outer-directed and problem- or emotion-focused. Inner-directed problem-focused adaptive responses include avoiding situations where racism is likely to occur as well as strategic decisions not to respond to racist experiences in order to "protect one's time, energy, sanity [and] bodily integrity" (Harrell, 2000:46) and enable one to "fight those battles that are worth fighting" (Durodoye, 1999:47). Inner-directed emotion-focused adaptive responses include contemplative/relaxing techniques such as praying and meditation while outer-directed adaptive behavioural responses include the problem-focused approach of confronting perpetrator(s) as well as the emotion-focused approach of establishing and/or utilising social support (Krieger, 1999; McNeilly et al., 1996a) and writing, drawing, singing or painting about, or otherwise expressing, racism experiences.

Over and under achievement/striving are maladaptive problem-focused behavioural responses to racism. An example of over-striving is the active coping style known as 'John Henryism' which is the tendency to work extremely hard and with great pressure to disprove stereotypes of laziness and inability (James et al., 1984). On the other hand, an example of under-achievement is the phenomenon of 'stereotype threat' which occurs when a negative stereotype about one's ethnorace causes self-stigmatisation and reduced performance in an activity, experience or situation (Steele, 1997). Emotion-focused maladaptive behavioural responses include alienation from other ethnoracial group members and risk-taking/self-harming activities.

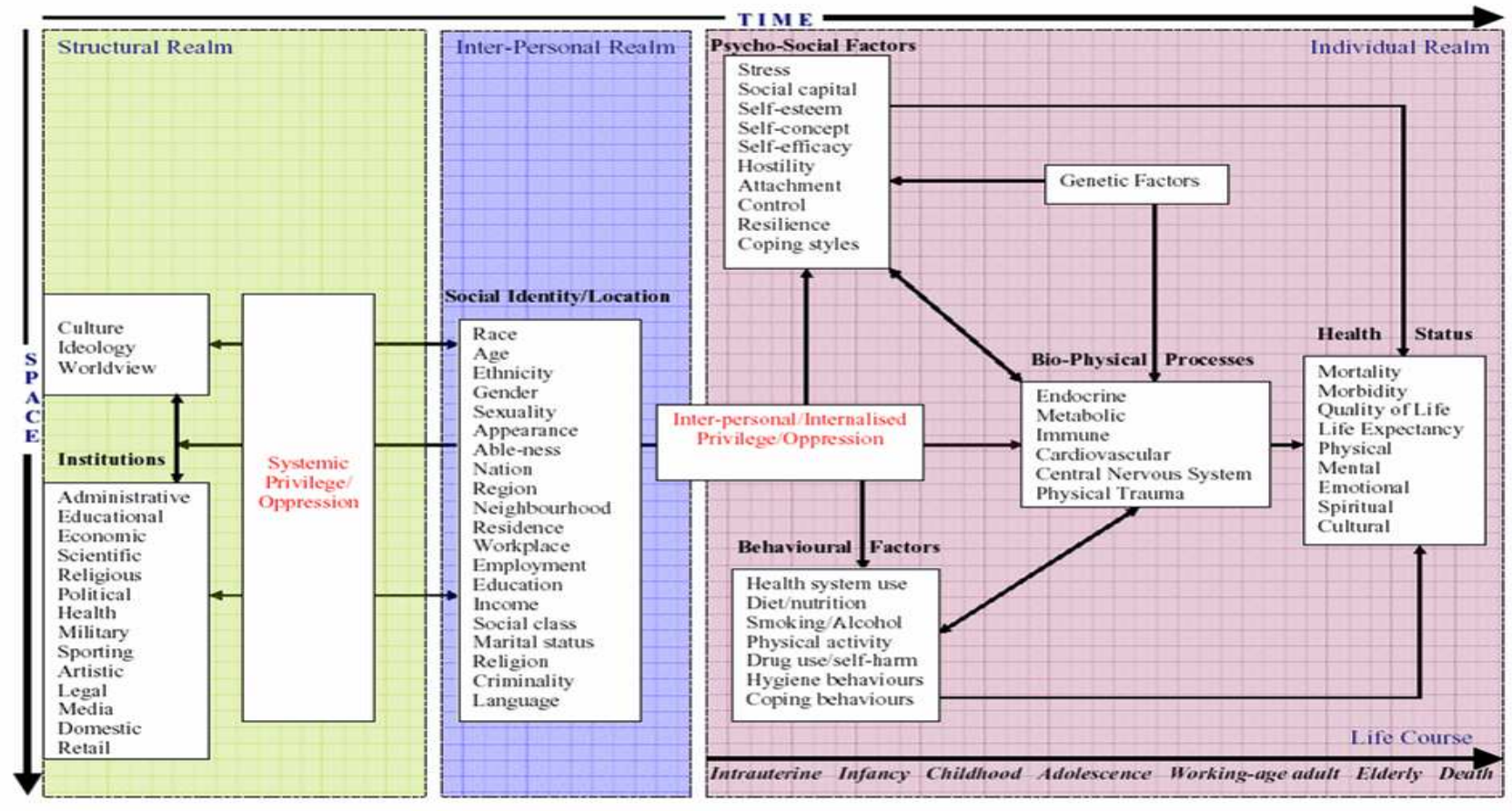
Finally, 'passing' is a problem-focused response (Mellor, 2004), available to some people in some contexts, in which one's ethnoracial identity is either not deployed, or is denied in order to avoid racism. Passing can be either adaptive or maladaptive with short-term benefits in avoiding racist situations but implications in the long-term in relation to strength of ethnoracial identity and social isolation from other ethnoracial group members.

3.6 Conclusion

The study of racism and health is a newly emerging area of study in health research. Clearly, this research requires conceptual clarity in order to unravel, through rigorous theoretically-based empirical research, the complex ways in which racism may act as a determinant of health (Williams D.R. et al., 1997). In striving towards conceptual clarity this chapter has presented a comprehensive definition of racism which, unlike previous definitions, does not exclude possible manifestations of racism (such as privilege) before these manifestations have been studied empirically.

Although Chapter 4 below goes on to review in detail the findings from empirical research in the field of racism and health, this chapter has highlighted two key theoretical lacunae that are evident in this nascent field of research. First, there is a clear need for future research to investigate the privileges of racism (Krieger et al., 1999) by utilising the, as yet, untapped insights from the field of Whiteness studies (Frankenberg, 1993; Hartigan, 2005; Helms, 1995; Hurtado & Steward, 1997; Rasmussen et al., 2001). Second, exploration of power analyses focused on social effects, the role of various (non-)human perpetrators/targets, and the interplay between objective and subjective racism is clearly warranted. The frameworks for conceptualising and characterising racism presented here, which combine and expand upon previous approaches, are designed to assist in determining the focus, scope and methodology of future empirical research in this area. It is hoped that through improved conceptual and theoretical tools future research will be better able to explain how racism relates to the distribution and determinants of population health.

Figure 3.1 Conceptualising Privilege/Oppression as a Determinant of Health



This figure draws on conceptual and theoretical work by Clark et al. (1999), Giddens (1984), Haugaard (2002), Krieger (2001), Smaje (1997) and Turrell et al. (1999).

Figure 3.2 Characterising Oppressive Perceived Racism as a Determinant of Health

Exposure to Racism

Timing: Intrauterine, infancy, childhood, adolescence, early/middle/late adulthood, elderly

Intensity: Degree of induced mental or physical stress (i.e. cognitive or allostatic load)

Frequency: Acute/chronic, sporadic/regular, weekly, monthly, yearly

Duration: Fleeting, prolonged, constant, cumulative

Characteristics of Racism

Mode: Inter- vs. intra-racial

Form: (Il)legal, blatant/subtle, (in)direct (vicarious), (c)overt, (un)intentional, (in)action

Level: Internalised (dominance and oppression), inter-personal, systemic

Expression: Stereotype, prejudice, discrimination

Setting: Domestic/familial, academic/educational, employment, media, police/security, legal/justice, administrative/political, housing/infrastructure, financial, health, goods/services, recreational, public

Perpetrators/Targets: self, family, friends, ethnoracial groups, neighbours, acquaintances, employers/ees, peers, pets, property, strangers, officials, practices, policies, laws

Reactions/Responses to Racism

Cognitive

Active: adaptive – rejecting dominant ideology, strengthened ethnoracial identity, system blame, imagining responses to racism; *maladaptive* – hypervigilance, attribution anxiety, denial of racism, self-blame

Passive: maladaptive – adopting dominant ideology, weakened ethnoracial identity, resigned acceptance

Affective

Inner-directed disempowered: Active – shame, self-hatred, humiliation, anxiety, fear; *Passive* – powerlessness, hopelessness, confusion, depression

Outer-directed: Empowered – contempt, amusement, sorrow/sympathy; *disempowered* – anger, annoyance, frustration

Behavioural

Inner-directed adaptive: problem-focused – passing, avoidance, strategic responses; *emotion-focused* – praying, meditation

Outer-directed adaptive: *problem-focused* – verbal, physical, or legal confrontation; *emotion-focused* – establishing and utilising social networks/safe spaces, write, draw, sing, or paint about racism

Maladaptive: problem-focused – passing, over/under achievement/striving (e.g. John Henryism, stereotype threat); *emotion-focused* – alienation from other ethnoracial group members, risk-taking/self-harming activities

This figure draws on work by Blank et al. (2004), Durodoye (1999), (Harrell, 2000) Jones (2000), Krieger (1999), McNeilly et al. (1996a) and Mellor (2004).

Chapter 4. ***A systematic review of empirical research on self-reported racism and health***[10]

4.1 ***Introduction***

In recent years, there has been an emerging interest in the epidemiological study of racism and health. This review examines empirical research on self-reported perceptions/experiences of racism (hereafter referred to as 'self-reported racism') and various health outcomes. Due to its pervasive nature in contemporary societies racism is frequently not perceptible to individuals or, if perceived, may not always be reported (Krieger et al., 2005). Hence, it should be clear that in focusing on self-reported racism this chapter considers only a very specific subset of this multifarious phenomenon and does not attempt a comprehensive assessment of the impact of racism on population health.

A handful of previous reviews have attempted to provide an overview of certain aspects of this emerging area of epidemiological research. The first article to consider the health impact of racism (along with sexism and social class) was published by Krieger et al. (1993). This was followed in 1999 by a review, also by Krieger, of 15 studies examining ethnoracial discrimination and health and by a review of 13 studies examining racism and mental health for African Americans by Williams & Williams-Morris (2000). There were also three reviews published in 2003, with Wyatt et al. summarising 19 studies relating racism to cardiovascular disease for African Americans, Brondolo et al. discussing studies relating racism to blood pressure (6 studies) and cardiovascular reactivity (11 studies), and Williams et al. reviewing 53 population-based empirical studies of ethnoracial discrimination which were published from 1998 onwards and related to various health outcomes.

[10] This chapter is taken from Paradies Y. (2006). A systematic review of empirical research on self-reported racism and health. *International Journal of Epidemiology* 35(4): 888-901.

Drawing in particular on the most recent review by Williams et al. (2003), this chapter updates and expands upon these earlier reviews by examining the key characteristics of epidemiological studies of self-reported racism and health – where and when studies have been conducted, the ethnorace, age and gender of study populations, study designs, sample sizes and data sources used. This review also aims to describe how self-reported racism is defined in the existing literature, how this exposure is measured in terms of method of administration, content and time frames of exposure, targets and perpetrators of racism and settings in which racism is experienced as well as the variation in reporting of racism by various socio-demographic factors. The nature of associations found between self-reported racism and health is detailed for a range of health outcomes across various study and exposure characteristics along with identified effect modifiers and mediators of these associations. Possible future directions for research in this area are also presented.

4.2 ***Methods***

A search of the PubMed, PsychINFO and Sociological Abstracts databases was conducted using the terms: racism, racial discrimination, race AND discrimination, and perceived discrimination from the earliest records in these databases until the end of 2004. The inclusion criteria for this review required studies to: (1) be empirical; (2) be quantitative; (3) include a direct measure of self-reported or experienced racism as an exposure; (4) include a direct measure of a health-related factor as an outcome; and (5) not be theses/treatises or conference proceedings (due to the difficulty of obtaining these documents). A sequential process of examining the title, abstract and main text of each article or book (chapter) was undertaken, with exclusion of documents occurring at each stage. The bibliographies of all articles which met the inclusion criteria, as well as those of previous review articles, were also searched for further relevant studies which were in turn acquired and checked against the inclusion criteria above. The study of racism in medical/health care is a fast-growing body of research in its own right (Smedley et al., 2002) and there is also existing research on the intersection of racism with other forms of oppressions such as (hetero)sexism, classism etc. (Schnittker & McLeod, 2005). However, reviewing these topics along with self-reported racism is beyond the scope of this chapter and, as such, these issues are not addressed below.

Data pertaining to studies which met the inclusion criteria were entered into a spreadsheet and analysed using basic descriptive statistics in Stata 8.0 for Windows. A formal meta-analysis was not conducted due to both the heterogeneity of studies in term of design, study populations, exposure and outcome measures and because only a small number of studies provided odds ratios (ORs) or similar effect sizes (see below) which could be weighted by study sample size into a combined estimate. Furthermore, publication bias was not assessed in this review.

4.3 ***Results***

A total of 138 separate studies, with several studies published in the same paper, met the inclusion criteria for this review. Table 4.1 details the key characteristics of these studies.[11] Most of the 138 studies were published in the last five years (65%), used a cross-sectional study design (76%) with convenience (i.e. non-representative) samples (59%) of between 100 and 1000 adults (59%) and were conducted in the United States (US) (86%) with African Americans (69%).

Several large surveys were utilised in a number of studies in this review, with studies utilising the same dataset focusing on different exposures, outcomes and/or sub-samples. The surveys utilised were the National Survey of Black Americans (Brown et al., 2000; Brown et al., 2002; Forman et al., 2003; Jackson et al., 1996; Jackson & Inglehart, 1995; La Veist et al., 2001; Mays et al., 1996), Detroit Area Study (Boardman, 2004; Forman et al., 2003; Schulz et al., 2000a; Williams et al., 1997), Fourth National Survey of Ethnic Minorities (Karlsen & Nazroo, 2002a; Karlsen & Nazroo, 2002b), General Social Survey (Jackson & Inglehart, 1995; Kennedy et al., 1997; Roberts et al., 2004), Mexican American

[11] Data on socioeconomic position (SEP) was not included in Tables 4.1-4.2 due to inconsistent stratification by and conceptualisation/measurement of SEP in these 138 studies. See Krieger et al. (1997) as well as Oakes and Rossi (2003) for further discussion of the conceptualisation of SEP in health research.

Prevalences and Services Survey in California (Finch et al., 2000; Finch et al., 2001;), San Francisco Muni Health and Safety (Yen et al., 1999a; Yen et al., 1999b), MIDUS (Kessler et al., 1999; Ryff et al., 2003) and CARDIA surveys (Krieger & Sidney, 1996; Mustillo et al., 2004).

4.3.1 Defining Racism

Only a quarter of the studies (34 of the 138) in this review offered a definition of racism (Broman, 1996; Broman, 1997; Brown et al., 2002; Clark, 2000; Contrada et al., 2001; Din-Dzietham et al., 2004; Finch et al., 2000; Finch et al., 2001; Fischer & Shaw, 1999; Forman et al., 2003; Franzini & Fernandez-Esquer, 2004; Gary, 1995; Gee, 2002; Jackson & Inglehart, 1995; Jackson et al., 1996; Karlsen & Nazroo, 2002; Kennedy et al., 1997; Landrine & Klonoff, 1996; Loo et al., 2001; Morris-Prather et al., 1996; Murrell, 1996; Noh et al., 1999; Nyborg & Curry, 2003; Peters, 2004; Ren et al., 1999; Roberts et al., 2004; Sanders-Thompson, 1996; Schneider et al., 2000; Stuber et al., 2003; Tull et al., 1999; Utsey et al., 2002b; Valentine et al., 1999; Williams et al., 1997). Many of these definitions focused on the detrimental effects of racism (i.e. for non-White ethnoracial groups) (Broman, 1996; Broman, 1997; Clark, 2000; Finch et al., 2000; Finch et al., 2001; Fischer & Shaw, 1999; Jackson et al., 1996; Kennedy et al., 1997; Landrine & Klonoff, 1996; Loo et al., 2001; Morris-Prather et al., 1996; Peters, 2004; Ren et al., 1999; Sanders-Thompson, 1996; Schneider et al., 2000; Tull et al., 1999; Valentine et al., 1999), with only a few also highlighting the privileges accrued through racism (Gee, 2002; Jackson & Inglehart, 1995; Murrell, 1996; Noh et al., 1999; Nyborg & Curry, 2003). Similarly, racism was generally attributed to an ideology of inferiority (Nyborg & Curry, 2003; Ren et al., 1999; Wiking et al., 2004) or superiority (Din-Dzietham et al., 2004; Peters, 2004; Utsey et al., 2002b; Williams et al., 1997), rather than both (Jackson & Inglehart, 1995). Notably, only half of these definitions recognised systemic racism (i.e. racism occurring through societal organisations, institutions, laws, policies, practices etc.) as well as inter-personal racism (i.e. racist interactions between people) (Clark, 2000; Din-Dzietham et al., 2004; Finch et al., 2000; Finch et al., 2001; Forman et al., 2003; Gee, 2002; Jackson & Inglehart, 1995; Jackson et al., 1996; Karlsen & Nazroo, 2002; Kennedy et al., 1997; Loo et al., 2001; Morris-Prather et al., 1996; Nyborg & Curry, 2003; Peters, 2004; Ren et al., 1999; Tull et

al., 1999; Utsey et al., 2002b). Furthermore, several studies defined racism as differential treatment by race (Jackson et al., 1996; Roberts et al., 2004; Stuber et al., 2003; Williams et al., 1997), without specifying for which ethnoracial groups this treatment was positive or negative; hence, failing to differentiate between racism and anti-racism.

4.3.2 Exposure Measurement

A total of 154 different instruments/scales were used in the 133 studies in this review which included a measure of self-reported racism (five experimental studies (Armstead et al., 1989; Fang & Myers, 2001; Jones et al., 1996; McNeilly et al., 1995; Morris-Prather et al., 1996) utilised racist stimuli instead of assessing self-reported racism). There was considerable variation in exposure measurement across these instruments, in terms of both content and length. Of these 152 measures, 106 were between 1 and 9 items in length (with 28 of these consisting of only a single item) and 48 were between 10 and 128 items in length. Self-reported racism was measured across a range of conceptual dimensions including experiences of discrimination (i.e. racist behaviours/actions) (Karlsen & Nazroo, 2002; Murrell, 1996; Steffen et al., 2003) such as racist attacks (Caputo, 2003; Karlsen & Nazroo, 2002; Mak & Nesdale, 2001), harassment (Schneider et al., 2000), exclusion (Cassidy et al., 2004; Iyer & Haslam, 2003; Noh et al., 2003; Schneider et al., 2000; Whitbeck et al., 2002), life events (e.g. being fired) (Blankenau et al., 2000; Boardman, 2004; Kessler et al., 1999; Krieger et al., 1993; Landrine & Klonoff, 1996; Schulz et al., 2000a; Williams et al., 1997), and everyday discrimination/daily hassles (e.g. being refused service) (Barnes et al., 2004; Bird & Bogart, 2001; Boardman, 2004; Guthrie et al., 2002; Guyll et al., 2001; Kessler et al., 1999; Ryff et al., 2003; Schulz et al., 2000a; Schulz et al., 2000b; Sellers et al., 2003; Taylor & Turner, 2002; Troxel et al., 2003; Williams et al., 1997), as well racist affect (emotions) (Murrell, 1996) and stereotypes/beliefs (Essed, 1991; Iyer & Haslam, 2003; Jackson & Inglehart, 1995; Kennedy et al., 1997; Murrell, 1996; Neblett et al., 2004). With the exception of two instruments (Din-Dzietham et al., 2004; McNeilly et al., 1996a) used in seven studies (Bennett et al., 2004; Clark, 2000; Din-Dzietham et al., 2004; Nyborg & Curry, 2003; Peters, 2004; Poston et al., 2001; Steffen et al., 2003), self-reported racism in these studies was assessed without collecting information on the ethnorace of the perpetrator.

Racism was also conceptualised as a form of stress by over a fifth of studies in this review (Blankenau et al., 2000; Broman, 1996; Cassidy et al., 2004; Clark, 2000; Dion et al., 1992; Dole et al., 2003; Fang & Myers, 2001; Guthrie et al., 2002; Kessler et al., 1999; Kwate et al., 2003; MacCarthy & Craissati, 1989; Manuel, 2004; Morgan et al., 2000; Murrell, 1996; Mustillo et al., 2004; Noh et al., 1999; Nyborg & Curry, 2003; Resnicow et al., 1999; Sanders-Thompson, 1996; Schulz et al., 2000b; Simons et al., 2002; Taylor & Jackson, 1991; Troxel et al., 2003; Utsey & Ponterotto, 1996; Utsey et al., 2000; Utsey et al., 2002a; Whitbeck et al., 2001; Wiking et al., 2004). Self-reported stress associated with self-reported racism was assessed in 14 studies included in this review. Of the 10 studies which measured self-reported racism and racism-related stress as two separate constructs, three failed to differentiate between these measures in their analysis (Iyer & Haslam, 2003; Moradi & Hasan, 2004; Salgado de Synder, 1987). The remaining seven studies (Bowen-Reid & Harrell, 2002; Klonoff et al., 1999; Kwate et al., 2003; Landrine & Klonoff, 1996; Landrine & Klonoff, 2000; Mustillo et al., 2004) allow a comparison between self-reported racism and racism-related stress (i.e. the experience of racism as a form of stress) in terms of associations with health outcomes, as detailed below.

Generally, the studies in this review assessed self-reported experiences of inter-personal racism and/or systemic racism, without indicating which of these levels of racism were being examined. Only a small number of studies explicitly assessed systemic racism (Boardman, 2004; Clark et al., 2004; Fisher et al., 2000; Forman et al., 2003; Gee, 2002; Harrell, 1997a; Koomen & Frankel, 1992; La Veist et al., 2000; Martin et al., 2003; McNeilly et al., 1996a; Phinney et al., 1998; Resnicow et al., 1999; Taylor & Jackson, 1991), internalised racism (i.e. the incorporation of racist attitudes, beliefs or ideologies into an individual's worldview) (Caughy et al., 2004; Contrada et al., 2001; Green, 1995; Holder & Vaux, 1998; James et al., 1994; Kennedy et al., 1997; Myers et al., 1989; Taylor & Grundy, 1996; Tomes et al., 1990; Whitbeck et al., 2004), or reactions and responses to racism (Clark, 2000; Krieger, 1990; Krieger & Sidney, 1996; Noh et al., 2003; Stancil et al., 2000; Yen et al., 1999a). This review included 15 studies which specifically assessed self-reported racism for a respondent's entire ethnoracial group (Barry & Grilo, 2003; Brown et al., 2002; Caughy et al., 2004; Dion et al., 1992; Forman et al., 2003; Karlsen & Nazroo, 2002; Kennedy et al., 1997; La Veist et al., 2001; Martin et al., 2003; Morgan et

al., 2000; Nyborg & Curry, 2003; Resnicow et al., 1999; Tomes et al., 1990; Utsey et al., 2002b; Verkuyten, 1998).

A number of exposure instruments examined experiences of discrimination which were not specifically attributed to ethnorace but, rather, measured generic discrimination without attributing to, or differentiating between, discrimination due to ethnorace, gender, sexuality etc. during measurement and/or analysis. Instruments such as the Everyday Discrimination Scale (EDS) were used as such a non-specific measure of discrimination (Barnes et al., 2004; Boardman, 2004; Ryff et al., 2003; Schulz et al., 2000b; Williams et al., 1997; Williams et al., 2003) as was the 3-item measure of major discrimination (Boardman, 2004; Schulz et al., 2000a; Williams et al., 1997) along with several other measures (Janssen et al., 2003; Pavalko et al., 2003; Werkuyten & Nekuee 1999).

Of the 133 studies which included a measure of self-reported racism, 65 studies used an interviewer-administered measure with a further 68 studies using self-administration. There were also six studies which assessed reported racism for family and friends of respondents (Dion et al., 1992; Finch et al., 2001; Jackson et al., 1996; La Veist et al., 2001; Noh et al., 2003; Murry et al., 2001). Exposure to racism was assessed across a range of settings including at work (reported as assessed in 52 studies), from goods/service providers (38 studies), in courts of law/from police (33), when seeking employment (31), in education (30), housing (22), and health care (21), in public (16), from neighbours (12), friends (10), government agencies (7) and at home (4).

About a quarter (34) of the 138 studies used versions of three instruments as exposure measures. These were the EDS (Essed, 1991) used in 12 studies (Barnes et al., 2004; Bird & Bogart, 2001; Boardman, 2004; Guthrie et al., 2002; Guyll et al., 2001; Kessler et al., 1999; Ryff et al., 2003; Schulz et al., 2000a; Schulz et al., 2000b; Taylor & Turner, 2002; Troxel et al., 2003; Williams et al., 1997), a set of items developed by Krieger (1990) (now known, in a slightly varied form as the Experiences of Discrimination (EOD) (Krieger et al., 2005) and referred to below as such) used in 13 studies (Broman et al., 2000; Broman, 1996; Broman, 1997; Collins et al., 2004; Dole et al., 2003; Dole et al., 2004; Krieger & Sidney, 1996; Krieger, 1990; Lespinasse et al., 2004; Mustillo et al., 2004; Peters, 2004; Ren et al., 1999; Yen et al., 1999a), and the Schedule of Racist Events (SRE) (Landrine &

Klonoff, 1996) used in 9 studies (Bowen-Reid & Harrell, 2002; Fischer & Shaw, 1999; Gibbons et al., 2004; Klonoff et al., 1999; Kwate et al., 2003; Landrine & Klonoff, 2000; Moradi & Hasan, 2004; Simons et al., 2002). These instruments have shown good internal reliability within a single study for both the EOD (Krieger & Sidney, 1996) and the SRE (Simons et al., 2002) and in five studies for the EDS (Guthrie et al., 2002; Guyll et al., 2001; Schulz et al., 2000b; Taylor & Turner, 2002; Troxel et al., 2003). Furthermore, the EDS has shown good construct validity in a sample of African American adolescents (Harrell, 1997b), the EOD has been psychometrically validated in a sample of African American, Latino and White participants, showing high scale reliability and validity including test-retest reliability (Krieger et al., 2005) and the SRE has been found to have good construct and concurrent validity (Landrine & Klonoff, 1996) as well as good psychometric properties when adapted for use across ethnoracial groups (Landrine et al., 2006). Other exposure measures that have been psychometrically validated include the Racism and Life Experiences Scale (Harrell, 1997a; Harrell, 1997b), the Perceived Racism Scale (McNeilly et al., 1996b), and the Perceived Ethnic Discrimination Questionnaire (Brondolo et al., 2005; Contrada et al., 2001).

By their nature, 20 of the studies in this review could not include a time frame associated with exposure measurement as these studies assessed stereotypes/beliefs or used experimental interventions (Armstead et al., 1989; Barry & Grilo, 2003; Brown et al., 2002; Butler et al., 2002; Chambers et al., 2004; Fang & Myers, 2001; Jackson & Inglehart, 1995; Jones et al., 1996; Krieger et al., 1993; McNeilly et al., 1995; Morgan et al., 2000; Morris-Prather et al., 1996; Myers et al., 1989; Resnicow et al., 1999; Taylor & Jackson, 1990a; Taylor & Jackson, 1990b; Taylor & Jackson, 1991; Tomes et al., 1990; Tull & Chambers, 2001; Tull et al., 1999). However, of the remaining 118 studies in which it was possible to specify a time frame, this occurred in only 42 studies. Of these 42 studies, 10 measured both past year and lifetime (Bowen-Reid & Harrell, 2002; Caputo, 2003; Caughy et al., 2004; Collins et al., 2004; Fischer & Shaw, 1999; Klonoff et al., 1999; Kwate et al., 2003; Landrine & Klonoff, 1996; Landrine & Klonoff, 2000), or past 6 month (Casey & O'Connell, 2000), self-reported racism. A further nine studies measured self-reported racism over the past year (Gary, 1995; Guthrie et al., 2002; Janssen et al., 2003; Karlsen & Nazroo, 2002; Karlsen & Nazroo, 2002; Moradi & Hasan, 2004; Sellers et al., 2003;

Szalacha et al., 2003) and 20 studies measured self-reported racism over a lifetime (Amaro et al., 1987; Bird & Bogart, 2001; Boardman, 2004; Gee, 2002; Kessler et al., 1999; Krieger & Sidney, 1996; Krieger, 1990; Manuel, 2004; Martin et al., 2003; McCord & Ensminger, 1997; Mustillo et al., 2004; Noh et al., 2003; Poston et al., 2001; Stuber et al., 2003; Taylor & Turner, 2002; Utsey et al., 2000; Utsey et al., 2002a; Utsey et al., 2002b; Williams et al., 1997; Yen et al., 1999a; Yen et al., 1999b). Self-reported racism was assessed over the past three years (Broman, 1996; Broman, 1997; Broman et al., 2000), two years (Schneider et al., 2000) and five years (Pavalko et al., 2003) as well as over the previous six months (Sanders-Thompson, 1996), three months (Contrada et al., 2001; Salgado de Synder, 1987), and a month (Brown et al., 2000; Jackson et al., 1996; Jackson & Inglehart, 1995; La Veist et al., 2001; Mossakowski, 2003), as well as during childhood (Diaz et al., 2001; Dion et al., 1992), adulthood (Diaz et al., 2001; Dion et al., 1992), since arriving in a particular country (Liebkind & Jasinskaja-Lahti, 2000; Noh et al., 1999) or during a specific pregnancy (Collins et al., 2000; Collins et al., 2004; Dole et al., 2004).

4.3.3 Socio-demographic variations in self-reported racism

The studies included in this review do not provide representative information on the prevalence of racism and, as such, data on this topic are not presented here. However, at present little is known about how self-reported racism varies by socio-demographic characteristics. Hence, this section presents available information from this review on such variation. The most consistent finding from the studies included in this review is that the prevalence of self-reported racism varies strongly and consistently by ethnorace, with non-White respondents experiencing more racism than White respondents (Barnes et al., 2004; Barnes et al., 2004; Boardman, 2004; Broman et al., 2000; Contrada et al., 2001; Dole et al., 2004; Guyll et al., 2001; Kessler et al., 1999; Krieger & Sidney, 1996; Krieger, 1990; La Veist et al., 2000; MacCarthy & Craissati, 1989; Mustillo et al., 2004; Pavalko et al., 2003; Ren et al., 1999; Roberts et al., 2004; Sanders-Thompson, 1996; Schulz et al., 2000a; Schulz et al., 2000b; Striegel-Moore et al., 2002; Taylor & Turner, 2002; Troxel et al., 2003; Valentine et al., 1999; Williams et al., 1997). Increased reporting of racism was generally associated with higher SEP (Dion et al., 1992; Gee, 2002; Kennedy et al., 1997;

Ren et al., 1999; Schulz et al., 2000a; Williams et al., 1997; Yen et al., 1999a; Yen et al., 1999b) but also, in a few studies, with lower SEP (Gary, 1995; Janssen et al., 2003; Schulz et al., 2000a), with a number of studies finding no variation in self-reported racism by income (Barnes et al., 2004; Broman et al., 2000; Gary, 1995; Kwate et al., 2003; Sanders-Thompson, 1996) or education (Barnes et al., 2004; Broman et al., 2000; Kwate et al., 2003; Sanders-Thompson, 1996). Many studies have found that self-reported racism varies by gender with males reporting more racism than females (Barnes et al., 2004; Cassidy et al., 2004; Clark, 2004; Fischer & Shaw, 1999; Liebkind & Jasinskaja-Lahti, 2000; Mak & Nesdale, 2001; McCord & Ensminger, 1997; Sellers & Shelton, 2003; Utsey et al., 2002a). In some studies this higher reporting by males occurred only in specific settings such as when being threatened or harassed (Clark, 2004; Contrada et al., 2001), at work (Krieger & Sidney, 1996), in seeking employment (Broman et al., 2000), in educational settings (Kessler et al., 1999), from the police (Broman et al., 2000; Clark, 2003a; Kessler et al., 1999), legal system (Clark, 2003a; Clark, 2004), in money and finances (Clark, 2004; Kessler et al., 1999) and in seeking medical care (Clark, 2004). However, a few studies have found that females report more racist experiences than males (Contrada et al., 2001; Gibbons et al., 2004; Janssen et al., 2003; Wiking et al., 2004), with this occurring in the service provision setting in two studies (Kessler et al., 1999; Whitbeck et al., 2001). A few studies have found no variation in self-reported racism by sex/gender (Bird et al., 2004; Sanders-Thompson, 1996; Yen et al., 1999b).

The prevalence of self-reported racism was found to vary inconsistently by age, with some studies in this review finding more self-reported racism for older people (Peters, 2004; Stancil et al., 2000; Steffen et al., 2003), other studies finding the opposite (Broman et al., 2000; Gary, 1995; Kennedy et al., 1997; Krieger, 1990), and a few studies finding no variation in self-reported racism by age (Clark, 2004; Kwate et al., 2003; Yen et al., 1999a). One study found that reports of discrimination (not specifically attributed to ethnorace) increased slightly with age for African Americans but decreased with age for European Americans (Barnes et al., 2004).

A high level of ethnoracial centrality (i.e. the extent to which a person defines him/herself with regard to race) has also been associated with increased reporting of racism, but as with the socio-demographic variations in self-reported racism presented above, this association

is complex and, at present, poorly understood. In one study, higher levels of acculturation were associated with increased reporting of racism for Latino/a respondents born or educated in Mexico, but acculturation was also associated with decreased reporting of racism for Latino/a respondents born or educated in the US (Finch et al., 2000). Self-reported racism was also found to increase with years spent in the US and decreased fluency in English among recent East Asian migrants (Barry & Grilo, 2003) and, in one study, reported racism tended to increase for African Americans along with an increasing belief that other groups hold negative attitudes towards them (Sellers et al., 2003).

4.3.4 Associations between self-reported racism and health-related outcomes

Table 4.2 shows the associations found between self-reported racism and health-related measures in the 138 studies included in this review. These outcomes, grouped into broad categories, are shown alongside information on the extent to which associations between these outcomes and self-reported racism were statistically significant and in which direction. Most of these studies adjusted for a range of confounders which are not shown in Table 4.2. Confounders adjusted for in three or more studies were socioeconomic and demographic factors such as age (adjusted for in 64 studies), education (55 studies), sex/gender (50), income/poverty (48), marital/partner status (25), ethnoracial group (23) and employment/occupation (21). Health risk factors adjusted for in three or more studies were stress (17), body mass index (12), smoking (10), social support (10), self-esteem (9), nativity (7) and acculturation (6).

Overall, 54% of health-related measures examined were significantly associated with self-reported racism (either positively or negatively). With 314 of the 613 health outcomes examined and 62 of the 138 studies in this review relating to mental health, these outcomes were the most commonly studied. The most consistent association between self-reported racism and health was found for negative mental health outcomes, for which 72% of examined outcomes were significantly associated with self-reported racism, all in the expected direction (i.e. more self-reported racism associated with worse mental health outcomes). Although examined in far fewer studies, 62% of health-related behaviours were

also significantly associated with racism (almost all in the expected direction). Only 48% and 36% of examined positive mental health outcomes and negative physical health outcomes, respectively, were significantly associated with self-reported racism with 8% and 1% of these associations, respectively, being in the opposite direction to the expected (i.e. more self-reported racism associated with better health outcomes).

There were 10 studies which reported the percentage of variance explained by self-reported racism (Clark, 2000; Holder & Vaux, 1998; Littleford & Wright 1998; Murrell, 1996; Sellers et al., 2003; Steffen et al., 2003; Troxel et al., 2003; Utsey et al., 2000; Utsey et al., 2002a). The variance explained ranged from 4% (Littleford & Wright 1998; Steffen et al., 2003) to 42% (Holder & Vaux, 1998) with a median and mode of 18%. A total of 43 statistically significant ORs were also reported across 21 studies,[12] of which 18 fell between 1.00 and 1.9, 20 between 2.0 and 2.8, and 5 between 3.0 and 3.5. A handful of studies reported various non-linear associations between self-reported racism and ill-health (Din-Dzietham et al., 2004; Krieger, 1990; Krieger & Sidney, 1996; Yen et al., 1999a), while twice as many studies found a linear dose-response association (Collins et al., 2004; Din-Dzietham et al., 2004; Guthrie et al., 2002; Janssen et al., 2003; Kennedy et al., 1997; Lespinasse et al., 2004; Mustillo et al., 2004; Whitbeck et al., 2002).

This review included 26 studies in which health outcomes including blood pressure (Armstead et al., 1989; Bowen-Reid & Harrell, 2002; Clark & Adams, 2004; Clark, 2000; Clark, 2003a; Guyll et al., 2001; James et al., 1984; James et al., 1994; Jones et al., 1996; Krieger & Sidney, 1996; Morris-Prather et al., 1996; Peters, 2004; Poston et al., 2001; Stancil et al., 2000; Steffen et al., 2003), birth weight (Collins et al., 2000; Collins et al., 2004; Dole et al., 2003; Dole et al., 2004; Lespinasse et al., 2004; Mustillo et al., 2004; Shiono et al., 1997), BMI/obesity (Butler et al., 2002; Chambers et al., 2004; Tull et al., 1999), and mortality (La Veist et al., 2001) were objectively measured by physicians/researchers rather than self-reported by participants. Of these objectively-

[12] There were also five relative risks reported from three studies (Dole et al., 2003; Krieger, 1990; Stuber et al., 2003) and two ORs of less than 1.0 reported for positive health outcomes (Blankenau et al., 2000; La Veist et al., 2001) which are not included here.

measured negative physical health outcomes, 44% were significantly associated with self-reported racism compared to 36% of all negative physical health outcomes included in this review (which included these objectively measured outcomes along with self-reported outcomes). Such a discrepancy may be due to confounding between exposure and outcome measures which are both self-reported; an effect which has been noted in the broader stress literature (Lynch, 2002).

As discussed above, there were seven studies which examined health outcomes in relation to both racism and racism-related stress. Both racism and racism-related stress had similar associations with health-related measures in three of these studies (Landrine & Klonoff, 1996; Klonoff et al., 1999; Landrine & Klonoff, 2000). In the remaining four studies, racism-related stress, but not racism itself, was associated with increased systolic blood pressure (Din-Dzietham et al., 2004); psychological distress (Kwate et al., 2003); somatisation, depression, anxiety, psychoticism (Bowen-Reid & Harrell, 2002); and smoking (Landrine & Klonoff, 2000).

The finding that racism-related stress is associated with systolic, but not diastolic, blood pressure (Din-Dzietham et al., 2004) is notable given support for such an association in relation to stress more generally as detailed in Chapter 6. Overall, there were 10 studies of racism in this review which differentiated between systolic and diastolic blood pressure in analysis (Clark, 2003a; Clark & Adams, 2004; Fang & Myers, 2001; Guyll et al., 2001; James et al., 1984; McNeilly et al., 1995; Morris-Prather et al., 1996; Peters, 2004; Stancil et al., 2000; Steffen et al., 2003). Across these 10 studies, 4 of 18 (22%) associations between racism and systolic blood pressure were significant and 8 of 17 (47%) associations between racism and diastolic blood pressure were significant (with one study reporting only that a similar pattern of association was found for systolic and diastolic) (Krieger & Sidney, 1996). This suggests that racism may have a different pattern of association with blood pressure than stress; being more strongly association with diastolic, rather than systolic, blood pressure. This has implications for the mechanisms via which racism and stress affect health, as discussed below.

4.3.5 Associations between study/exposure characteristics and health- related outcomes

The statistical significance (at the $p < 0.05$ level) of associations between self-reported racism and health-related measures also differed by a number of study and exposure characteristics as shown in Table 4.3. The highest proportion of significant associations occurred in studies which were published before the year 2000, measured self-reported racism over the past year/using no specified timeframe, used an exposure measure of 10 items or more in length or the SRE. Cross-tabulations of year of publication by other variables in Table 4.3 (analysis not shown) did not reveal any consistent patterns that would explain this finding. Publication bias, which was not assessed in this review, may have contributed to this result.

As shown in Table 4.3, studies with exposure instruments of 10 or more items in length had somewhat more significant associations (72%) than studies with exposure instruments of less than 10 items in length (50%), despite a range of health outcomes (both mental and physical) measured by instruments of these differing lengths. Less than half of the health outcomes examined using the EDS (45%) and the EOD (30%) showed significant associations whereas 86% of health outcomes examined using the SRE were significant. This strong finding for the SRE occurred despite a range of study populations and health outcomes examined in the studies utilising this instrument (results not shown).

Of those studies measuring racism over an explicit timeframe of 12 months or less, 57% of examined associations were significant whereas the corresponding figure was only 49% for studies measuring self-reported racism over a lifetime. This difference occurred despite the fact that these two categories were not mutually exclusive with 10 studies measuring self-reported racism over the past 12 months as well as over a lifetime (Bowen-Reid & Harrell, 2002; Caughy et al., 2004; Collins et al., 2004; Fischer & Shaw, 1999; Klonoff et al., 1999; Kwate et al., 2003; Landrine & Klonoff, 1996; Landrine & Klonoff, 2000; Sanders-Thompson, 1996; Simons et al., 2002). Of these 10 studies, five studies measured, analysed

and reported on these two different exposure timeframes for the same study sample (Bowen-Reid & Harrell, 2002; Collins et al., 2004; Kwate et al., 2003; Landrine & Klonoff, 1996; Landrine & Klonoff, 2000). These five studies found that both exposure timeframes were associated with markers of psychological distress (Bowen-Reid & Harrell, 2002; Landrine & Klonoff, 1996), past year but not lifetime self-reported racism was associated with very low birth weight of offspring (Collins et al., 2004) and with other markers of psychological distress (Bowen-Reid & Harrell, 2002; Kwate et al., 2003), and lifetime but not past year self-reported racism was associated with smoking (Landrine & Klonoff, 2000).

A number of studies specifically compared associations between self-reported racism and health for various ethnoracial groups. Several of these studies found that self-reported racism was related to ill-health for African Americans and Latino/as but not Whites (Dole et al., 2004; Kennedy et al., 1997; Krieger & Sidney, 1996; MacCarthy & Craissati, 1989; Roberts et al., 2004; Schulz et al., 2000b; Troxel et al., 2003) while, in other studies, similar associations were found between self-reported racism and health outcomes for Whites and non-Whites (Dole et al., 2004; Fang & Myers, 2001; Roberts et al., 2004; Ryff et al., 2003; Striegel-Moore et al., 2002), with one study finding an association between self-reported racism and psychological distress for Whites but not African Americans (Pavalko et al., 2003). Results from all the studies reviewed here, as shown in Table 4.3, suggest that there was little difference in the proportion of significant associations between self-reported racism and health-related measures for White respondents compared to other ethnoracial groups.

4.3.6 Effect modification of the association between self-reported racism and health-related outcomes

The associations between self-reported racism and health-related measures examined in this review were modified by a number of psychosocial factors which either intensified or attenuated the association between self-reported racism and health. Having a strong sense of ethnoracial identity or concept (Mossakowski, 2003; Sellers et al., 2003; Sellers & Shelton, 2003), participation in traditional activities (Whitbeck et al., 2002), spirituality

(Bowen-Reid & Harrell, 2002), religious support seeking/instrumental social support (Finch & Vega, 2003), and having personality traits such as hardiness (Dion et al., 1992) were found to attenuate the adverse effects of self-reported racism on depressive symptoms (Mossakowski, 2003; Sellers et al., 2003; Whitbeck et al., 2002), psychological distress (Bowen-Reid & Harrell, 2002; Dion et al., 1992; Sellers & Shelton, 2003) and self-assessed health status (Finch & Vega, 2003). Other research suggests that racial socialisation (i.e. explicit discussion of race/racism) imparted by parents or caregivers, but not personal beliefs about racial socialisation, may attenuate the detrimental effects of self-reported racism on mental health (Fischer & Shaw, 1999). Self-esteem (Fischer & Shaw, 1999), stressful events (Morgan et al., 2000) and substance misuse (Utsey et al., 2000) have all been noted as effect modifiers which intensify the detrimental effect of self-reported racism on mental health (Fischer & Shaw, 1999), life satisfaction (Morgan et al., 2000) and anxiety/depression (Utsey et al., 2000).

A range of socio-demographic factors have also been found to act as effect modifiers of the association between self-reported racism and health. One study found that the ill-effects of self-reported racism on psychological health were intensified for women compared to men and attenuated for Mexican Americans compared to African Americans or Whites (Ryff et al., 2003). In another study, self-reported systemic racism in the workplace increased psychological distress to a greater extent among African Americans of higher SEP compared to those of lower SEP (Forman et al., 2003). Another study found that the deleterious effect of mother-reported racism on infant birth weight was exacerbated for mothers aged 20-29, with over 12 years of education, or income in the second lowest bracket (Collins et al., 2004) and other research has noted that self-reported racism has an intensified association with depression for respondents who were native to the US, had higher levels of acculturation or were female (Finch et al., 2000).

Coping responses have also received some attention as effect modifiers of the association between self-reported racism and health in the studies reviewed here. Active/expressive responses to racism (including seeking social support), but not passive/internalising responses, have been found to attenuate the association of self-reported racism with depression (Noh et al., 2003). There is also evidence more generally of the beneficial effect of active rather than passive coping on health outcomes (Caughy et al., 2004; Din-

Dzietham et al., 2004; Dole et al., 2004; Krieger, 1990; Krieger & Sidney, 1996; Stancil et al., 2000) where these factors have been assessed as independent variables in their own right rather than as effect modifiers. However, it has been found that seeking social support may not be protective among those who lack sufficient social resources (Noh et al., 1999; Noh et al., 2003) and the effectiveness of coping strategies more generally are likely to be highly context dependent (Scott & House, 2005). Another potential effect modifier is the phenomenon of John Henryism (the tendency to work extremely hard to disprove stereotypes of laziness and inability) (James et al., 1984) which has been found to reduce blood pressure reactivity in African American women exposed to low levels of acute racism (Clark & Adams, 2004). Emotion-focused (as opposed to problem-focused) coping has also been found to reduce blood pressure reactivity during an acute speaking task about racist experiences (Clark, 2003b). More general research which does not examine effect modification has suggested that the passive response of self-blame and the active response of John Henryism may both be detrimental in the context of chronic racism (Clark, 2000), while emphasising the positive (a passive response) and trying to change the chronic experience of racism itself (an active response) may be protective (Clark, 2000). Respondents in the studies reviewed here appear to be 2-3 times more likely to use active coping responses such as talking with others and doing something about racism rather than passive coping responses such as keeping quiet, accepting or ignoring racism (Clark, 2000; Krieger, 1990; Krieger & Sidney, 1996; Noh et al., 2003; Stancil et al., 2000; Yen et al., 1999a).

4.3.7 Mediation of the association between self-reported racism and health-related outcomes

A number of mediators (i.e. intervening variables on the causal pathway) between self-reported racism and health have also been identified. Several studies found that the association between self-reported racism and health is completely or partially mediated by stress (Finch et al., 2001;Guthrie et al., 2002; Sellers et al., 2003; Taylor & Turner, 2002; Williams et al., 1997). Self-esteem was also found to mediate between self-reported racism and: blood pressure (James et al., 1994), psychological distress (Diaz et al., 2001), and depression/ anxiety for male but not female adolescents (Cassidy et al., 2004). The

associations between self-reported racism and substance use, self-assessed health, alcohol abuse and impaired fasting glucose have also been found to be mediated by psychological distress (Gibbons et al., 2004), depression (Finch et al., 2001), historical loss (Whitbeck et al., 2004), and waist circumference (Butler et al., 2002), respectively. Self-reported racism was itself found to be a mediator of the association between healthy food beliefs and healthy dietary behaviour (Manuel, 2004), between financial stress and well-being (Murrell, 1996) and between religious orientation and alcohol consumption (Taylor & Jackson, 1990a; Taylor & Jackson, 1990b).

4.4 ***Discussion***

This review suggests that there is an association between self-reported racism and ill-health after adjustment for a range of commonly measured confounders. The most consistent findings in this body of research to date have been for negative mental health outcomes and health-related behaviours and evidence from longitudinal studies also suggests that self-reported racism precedes ill-health rather than vice versa. Overall, it is clear that further investigation of self-reported racism as a widespread and apparently damaging health risk condition is warranted and rigorous methodological approaches and conceptual clarity will be needed to reveal how self-reported racism functions as a determinant of population health.

In such a vein, this review has highlighted continuing debate in this emerging field of research centred on the aetiology of self-reported racism and health in terms of both relevant time periods and pathways/mechanisms of effect, especially in relation to the relatively stronger association between self-reported racism and mental health outcomes as compared to physical health outcomes, and the relationship between stress and self-reported racism. These issues are discussed below along with current limitations of this nascent research field and possible directions for future research. It should be noted that considerable research has been conducted into factors influencing the perception, attribution and reporting of racism (Blank et al., 2004; Branscombe et al., 1999; Brondolo et al., 2003; Brown, 2001; Gomez & Trierweiler, 2001; Krieger, 1999; Major et al., 2002a; Major et al., 2002b; Operario & Fiske, 2001; Schnittker & McLeod, 2005; Sechrist et al.,

2004; Williams & Williams-Morris, 2000) and, although vital to the study of self-reported racism and health, an overview of this research is beyond the scope of this chapter.

Although it is clear that the aetiologically-relevant time period for self-reported racism varies markedly by the health outcome in question, there is nonetheless debate on the appropriateness of specifying timeframes in general for measuring self-reported racism. Some have argued that respondents do not assess or report racism in accordance with specified time frames and that, given the long lasting nature of racist experiences, an unspecified timeframe is the most appropriate (Utsey & Ponterotto, 1996). However, it is unclear whether respondents report recent and/or highly salient/traumatic experiences of racism when responding to questions without a specified timeframe. Also, the inclusion of an explicit time frame on surveys in general (Armstrong et al., 1992), as well as those assessing racism in particular (Blank et al., 2004) is necessary to estimate the rate of exposure and to avoid confounding time-series analyses (Blank et al., 2004). The results from this review suggest that studies which either leave exposure timeframes unspecified or utilise a timeframe equal to a year or less are associated with the highest proportion of statistically significant associations and may tap into the same set of recent experiences. It may be that experiences of racism in the past year are more aetiologically-relevant to the health outcomes assessed in this review or to health outcomes in general. It is also possible that a shorter time frame of exposure measurement ameliorates the effects of recall bias and/or maximises the variability of self-reported racism given that lifetime prevalence of this exposure is very high (60-84%) (Brondolo et al., 2003). By measuring, and comparing with various health outcomes, self-reported racism over different timeframes within the same study, future research will be able to further understanding of the aetiologically-relevant time period of exposure for this health risk condition.

This emerging field of research has also been hampered to date by relatively little reporting of effect sizes, a reliance on non-representative sampling, cross-sectional studies involving adults and a lack of psychometric validation of commonly used exposure instruments. Although one cross-sectional study has measured retrospective exposure to racism in childhood (Diaz et al., 2001) and another found an association between parental denial of racism and child mental health status (Caughy et al., 2004), only 12% and 15% of studies have used longitudinal designs or involved children, respectively. More consistent reporting

of effect sizes across different levels of exposure will allow meta-analyses of the association between self-reported racism and health. As detailed above, only a handful of studies have undertaken psychometric validation of the commonly used exposure instruments and further validation and comparison of these instruments is warranted. Furthermore, only 19% of the studies reviewed here have used data from existing regular omnibus surveys and only 19% of studies have utilised objectively reported, rather than self-reported, health outcomes. Inclusion of self-reported racism measures in existing survey vehicles which collect data on objectively measured health outcomes should be sought by researchers.

One topic which has received little attention in the literature to date is intra-racial racism. This form of racism occurs when an individual is discriminated against because of their ethnorace by a member of their own ethnoracial group, as opposed to inter-racial racism where discrimination is also based on the notion of ethnorace but where the perpetrator and target of racism are from different ethnoracial groups. There is evidence to suggest that members of oppressed ethnoracial groups are more likely to consider negative behaviours from members of their own ethnoracial group to be discriminatory compared to similar behaviours from members of other ethnoracial groups (Major et al., 2002b). One recent study found that 28% of African Americans and 15% of Latinos reported intra-racial racism as the most prevalent form of racism they experienced (Brondolo et al., 2005) and in another study 15% of 'problematic life experiences' were attributed to intra-racial racism (Clark, 2004). However, only a single study included in this review specifically examined experiences of intra-racial racism (Din-Dzietham et al., 2004). This study found that intra-racial racism-related stress was associated with greater increases in blood pressure in comparison to inter-racial racism-related stress (Din-Dzietham et al., 2004). Clearly, further research on intra-racial racism and its associations with health, is required. Similarly, a better understanding of vicarious racism (racism experienced by family or friends), systemic racism and setting-specific racism (e.g. racism in the workplace, home etc.) is needed to advance the study of self-reported racism and health. Furthermore, although many studies in this review examined White as well as non-White study populations, most of these studies statistically adjusted for ethnorace rather than undertaking stratified analyses. Such stratification will be required to uncover any differing associations between self-reported racism and health across ethnoracial groups.

There are a number of possible explanations for the finding that self-reported racism is more strongly association with mental rather than physical health outcomes. As noted by others (Meyer, 2003), it is possible that the association between racism and mental health is exaggerated or spurious due to factors in the self-reporting of both exposure and outcome. Such confounding has been found in relation to stress (Macleod et al., 2002) and as detailed above in relation to objectively measured vs. self-reported health outcomes, may be at play in the studies reviewed here. If not caused by measurement artifice, it is possible that the relatively weaker association between racism and physical health occurs because racism has a lagged effect on physical health (Pavalko et al., 2003) mediated by negative mental health outcomes. However, none of the studies in this review reported on interactions between mental and physical health outcomes in relation to self-reported racism and future research on such putative interactions and possible lagged effects is required.

The relatively stronger association between self-reported racism and mental health outcomes also raises questions about the mechanisms by which racism affects health. It may be that either direct pathopsychological effects on the brain or indirect neurophysiological changes wrought through other body systems (or a combination of both) mediate the association between self-reported racism and poor mental health outcomes. Studies that employ pharmacological blocks as well as brain imaging techniques should shed light on the psychophysiology of racism (Harrell et al., 2003). Researchers in the field of stress are now examining biomarkers specific to body systems (i.e. cardiovascular, neuroendocrine, immune) (Theorell, 2003) with evidence emerging that specific stressors differentially affect various physiological systems (Lucas, 2003). Similar research on racism would add to currently scarce knowledge of the biological processes through which this exposure affects health (Williams et al., 2003).

In addition to duplicating physiological research on the effects of stress, there is a need to disentangle the relationship between racism and stress to advance the study of self-reported racism and health. Although most studies reviewed here have conceptualised racism as a construct separate from stress, there are many who do consider racism to be a form of stress. As shown in Table 4.2, of the 19 associations between self-reported racism and stress as a health outcome examined in this review, 13 were found to be significant positive

associations. As detailed above, there is also evidence that the association between self-reported racism and health can be both mediated and moderated by stress and that the association between stress and health can be mediated by self-reported racism. At the same time, several studies have found that stress and self-reported racism are both independently related to health. It has been suggested that it is not appropriate to weight the stressfulness of racist experiences, as this approach confounds exposure measurement with reactions to exposure (Williams et al., 2003) and may not add to the predictive power of exposure instruments (Cohen et al., 1997). However, findings from studies which examined racism-related stress, as presented above, indicate that the constructs of racism and racism-related stress may be distinct. Unfortunately, there is also considerable debate in the stress literature more broadly about both how to characterise stress as a determinant of health (Cohen et al., 1997) and the complex processes by which coping factors modify the association between stress and health. In the hope of shedding light on these unresolved issues, the next chapter attempts to bring some clarity to the diverse field of stress research by reviewing the variety of concepts used in the study of psychosocial stress and health.

Table 4.1: Characteristics of 138 empirical quantitative studies of self-reported racism and health

	No. of studies	% of total studies
Year of Publication		
1980-1984	1	1
1985-1989	5	4
1990-1994	7	5
1995-1999	36	26
2000-2004	89	65
Study Design		
Cross-sectional	105	76
Cohort	16	12
Experimental	12	9
Case-control	5	4
Sampling Procedure		
Representative/Probability	57	41
Convenience	81	59
Sample Size		
n < 100	20	14
100 <= n < 200	34	25
200 <= n < 1000	47	34
n >= 1000	37	27
Region of Study		
US	118	86
Europe	12	9
Canada	3	2
Australia/New Zealand	2	1
Caribbean	3	2
Study Population		

	No. of studies	% of total studies
Ethnoraical group[a]		
African American	95	69
White	36	26
Asian	27	20
Latino/a	26	19
Immigrants/refugees	15	11
Indigenous	5	4
Age[a]		
Adults	126	91
Children/Adolescents	21	15
College students	15	11
Elderly	2	1
Gender (in studies of adults)		
Men and women	97	70
Men only	8	6
Women only	33	24
Exposure Instruments Utilised[b,c]		
Experiences of Discrimination (Krieger, 1990; Krieger & Sidney, 1996)	13	9
Everyday Discrimination Scale (Essed, 1991; Williams et al., 1997)	12	9
Schedule of Racist Events (Landrine & Klonoff, 1996)	9	7
Nadanolitization Scale (Taylor & Grundy, 1996)	7	5
Racism and Life Experience Scales (Harrell, 1997a; Harrell, 1997c)	7	5
Perceived Racism Scale (McNeilly et al., 1996a)	6	4
Major discrimination scale (Williams et al., 1997)	4	3
Perceived Discrimination Scale (Finch et al., 2001)	3	2

	No. of studies	% of total studies
Index of Race-Related Stress (Utsey & Ponterotto, 1996; Utsey, 1999)	3	2
Exposure Administration[b]		
Self-administered	68	49
Interviewer-administered	65	47
Exposure Timeframe[a,b]		
None	76	55
Past year or less	30	22
> one year <= five years	5	4
Ever/lifetime	32	23
Length of Exposure Instruments Utilised[a]		
<= 9 items	111	80
>= 10 items	48	33

a Categories are not mutually exclusive in relation to the unit of analysis (i.e. studies).

b Categories in this section are not complete.

c Includes only those instruments used in two or more studies.

Note: Percentages may not add to 100% due to rounding. Studies included in this table are as follows: Amaro et al., 1987; Armstead et al., 1989; Auslander et al., 1997; Barnes et al., 2004; Barry & Grilo, 2003; Bennett et al., 2004; Bird & Bogart, 2001; Bird et al., 2004; Blankenau et al., 2000; Boardman, 2004; Bowen-Reid & Harrell, 2002; Broman, 1996; Broman, 1997; Broman et al., 2000; Brown et al., 2000; Brown et al., 2002; Butler et al., 2002; Caputo, 2003; Casey & O'Connell, 2000; Cassidy et al., 2004; Caughy et al., 2004; Chambers et al., 2004; Clark & Adams, 2004; Clark, 2000; Clark, 2003; Collins et al., 2000; Collins et al., 2004; Contrada et al., 2001; Diaz et al., 2001; Din-Dzietham et al., 2004; Dion et al., 1992; Dole et al., 2003; Dole et al., 2004; Fang & Myers, 2001; Finch et al., 2000; Finch et al., 2001; Finch & Vega, 2003; Fischer & Shaw, 1999; Fisher et al., 2000; Forman et al., 2003; Franzini et al., 2004; Gary, 1995; Gee, 2002; Gibbons et al., 2004; Guthrie et al., 2002; Guyll et al., 2001; Holder & Vaux, 1998; Iyer & Haslam, 2003; Jackson & Inglehart, 1995; Jackson et al., 1996; James et al., 1984; James et al., 1994; Janssen et al., 2003; Jones et al., 1996; Karlsen & Nazroo, 2002a; Karlsen & Nazroo, 2002b; Kennedy et al., 1997; Kessler et al., 1999; Klonoff et al., 1999; Koomen & Frankel, 1992; Krieger, 1990; Krieger & Sidney, 1996; Kwate et al., 2003; La Veist et al., 2000; La Veist et al., 2001; Landrine & Klonoff, 1996; Landrine & Klonoff, 2000; Lespinasse et al., 2004; Liebkind & Jasinskaja-Lahti, 2000; Littleford & Wright 1998; Loo et al., 2001; MacCarthy & Craissati, 1989; Mak & Nesdale, 2001; Manuel, 2004; Martin et al., 2003; Mays et al., 1996; McCord & Ensminger, 1997; McNeilly et al., 1995; Moradi & Hasan, 2004; Morgan et al., 2000; Morris-Prather et al.,

1996; Mossakowski, 2003; Murrell, 1996; Murry et al., 2001; Mustillo et al., 2004; Myers et al., 1989; Noh et al., 1999; Noh et al., 2003; Nyborg & Curry, 2003; Pavalko et al., 2003; Pernice & Brook, 1996; Peters, 2004; Phinney et al., 1998; Poston et al., 2001; Ren et al., 1999; Resnicow et al., 1999; Roberts et al., 2004; Rosenberg et al., 2002; Rumbaut, 1994; Ryff et al., 2003; Salgado de Synder, 1987; Sanders-Thompson, 1996; Schneider et al., 2000; Schulz et al., 2000a; Schulz et al., 2000b; Sellers & Shelton, 2003; Sellers et al., 2003; Shiono et al., 1997; Simons et al., 2002; Stancil et al., 2000; Steffen et al., 2003; Striegel-Moore et al., 2002; Stuber et al., 2003; Szalacha et al., 2003; Taylor & Jackson, 1990a; Taylor & Jackson, 1990b; Taylor & Jackson, 1991; Taylor & Turner, 2002; Tomes et al., 1990; Troxel et al., 2003; Tull & Chambers, 2001; Tull et al., 1999; Utsey et al., 2000; Utsey et al., 2002a; Utsey et al., 2002b; Valentine et al., 1999; Verkuyten, 1998; Werkuyten & Nekuee 1999; Whitbeck et al., 2001; Whitbeck et al., 2002; Whitbeck et al., 2004; Wiking et al., 2004; Williams et al., 1997; Yen et al., 1999a; Yen et al., 1999b; Yoshikawa et al., 2004.

Table 4.2: Findings of 138 empirical quantitative studies of self-reported racism and health (p < 0.05 unless otherwise indicated)

	Positive Association[a]	Negative Association[b]	No Association[c]	Total
Negative mental health outcomes	**148(8)[d] (72%)**	**1 (0%)**	**57 (28%)**	**206(8)[d]**
Psychological/psychiatric /emotional distress	40(3)	1	21	62(3)
Depression/depressive symptoms	39(2)	0	13	52(2)
Obsessive-compulsive symptoms	5	0	0	5
Somatisation	5	0	0	5
Anxiety	15	0	7	22
Stress	13	0	6	19
Negative affect	9	0	5	14
Miscellaneous outcomes (examined in < 3 studies)	22(3)	0	5	27(3)
Positive mental health outcomes	**9 (8%)**	**52(7)[d] (48)%**	**47 (44%)**	**108(7)[d]**
Life/personal/patient/ work satisfaction/quality	1	27(6)	16	44(6)
Self-esteem	4	9	13	26
General mental health	4	10(1)	11	25(1)
Miscellaneous outcomes (examined in < 3 studies)	0	6	7	13
Negative physical health outcomes	**61(2)[d] (36%)**	**2 (1%)**	**108 (63%)**	**171(2)[d]**
Increased blood pressure/hypertension	19(1)	1	59	79(1)
Infant low birth	15(1)	0	12	27(1)

	Positive Association[a]	Negative Association[b]	No Association[c]	Total
weight/decreased gestational age				
Heart disease	0	0	12	12
Increased heart rate	5	1	4	10
Diabetes	1	0	6	7
Increased Body Mass Index	1	0	3	4
Miscellaneous outcomes (examined in < 3 studies)	20	0	12	32
SF-12/36 positive physical health	**1**	**0**	**6**	**7**
Health-related behaviours	**21(1) (62%)**	**0 (0%)**	**13 (38%)**	**34(1)**
Cigarette smoking	4	0	0	4
Alcohol (mis)use	8	0	6	14
Substance (mis)use	5	0	1	6
Miscellaneous outcomes (examined in < 3 studies)	4(1)	0	6	10(1)
Other Outcomes	**15(3) (17%)**	**20(2) (23%)**	**52 (60%)**	**87(5)**
Positive health assessed via the SF-12/36	1	2(1)	2	5(1)
Positive self-assessed health status	3	17	20	40
Miscellaneous outcomes (examined in < 3 studies)	11(3)	1(1)	30	42(4)
All Outcomes	**255(14) (42%)**	**75(9) (12%)**	**283 (46%)**	**613(23)**

Note: Many articles examined multiple outcomes and hence the number of associations (613) is greater than the number of articles (138). Percentages may not add to 100% due to rounding.

a Increased exposure associated with higher levels of the health-related outcome.

b Increased exposure associated with lower levels of the health-related outcome.

c The exposure is unrelated to the health-related outcome.

d The figure in brackets indicates the subset of total associations in this cell which are significant at the $0.05 < p < 0.10$ level rather than at the $p < 0.05$ level. So, for example, the first cell of this table, 8 of the 148 associations in this cell were marginally significant ($0.05 < p < 0.10$) and the remaining 140 were significant at $p < 0.05$.

Table 4.3: Significance of associations examined in 138 empirical quantitative studies of self-reported racism and health (p < 0.05)

	No. of significant associations	Total no. of associations examined	% of significant associations
Year of Publication			
Before 2000	114	197	58
2000-2004	216	416	52
Study Design			
Cross-sectional	242	456	53
Cohort	41	77	53
Experimental	39	68	57
Case-control	8	12	67
Sampling Procedure			
Representative/Probability	155	283	55
Convenience	175	330	53
Sample Size			
n < 100	50	100	50
100 <= n < 200	76	132	58
200 <= n < 1000	103	203	51
n >= 1000	101	178	57
Region of Study			
US	274	507	54
Outside the US	56	106	53
Study Population			
Ethnoracial group[a,b]			
African American	218	419	52
White	71	132	54
Asian	76	137	55
Latino/a	62	111	56
Immigrants/refugees	25	40	63

	No. of significant associations	Total no. of associations examined	% of significant associations
Age[a,b]			
Adults	303	570	53
Children/Adolescents	69	99	70
College students	53	79	67
Gender (in studies of adults)			
Men and women	243	463	52
Men only	15	28	54
Women only	72	122	59
Exposure Instruments Utilised[b]			
Everyday Discrimination Scale (Essed, 1991; Williams et al., 1997)	33	74	45
Experiences of Discrimination (Krieger, 1990; Krieger & Sidney, 1996)	17	56	30
Schedule of Racist Events (Landrine & Klonoff, 1996)	49	56	88
Exposure Administration[b]			
Self-administered	154	293	53
Interviewer-administered	154	294	52
Exposure Timeframe[a,b]			
None	154	258	60
Past year or less	113	198	57
> one year <= five years	7	21	33
Ever/lifetime	100	205	49
Length of Exposure Instruments Utilised[a]			
<=9 items	237	470	50
>=10 items	139	192	72

Note: Percentages may not add to 100% due to rounding.

a Categories in this section are not mutually exclusive. **b** Categories in this section are not complete.

Chapter 5. ***Psychosocial stress and health: An overview of conceptual approaches***

5.1 ***Introduction***

The notion of stress first arose during the 17th century and was synonymous with 'hardship, straits, adversity or affliction', while in the 18th and 19th centuries the term came to be associated with 'force, pressure, strain or strong effort' (Pollock, 1988). It wasn't until the 1920s when Walter Cannon conducted research into the physiological responses to emotional arousal, that stress came to be associated with health and wellbeing. Cannon introduced the concept of *fight-or-flight* as an adaptive function of humans and speculated that the physiological changes associated with this reaction were influenced by emotional states (Pollock, 1988). The first contemporary notion of stress, based on the work of Cannon and others, was proposed by Hans Selye in the 1950s (Doublet, 2000). Selye's *General Adaptation Syndrome* (GAS) model held that although change is a normal and inexorable feature of social life, all change is potentially harmful because all change requires (re)adjustment (Selye, 1956).

In the GAS model, stress is characterised by: (i) an alarm reaction where natural resistance is lowered and bodily defences are mobilised; (ii) increased resistance/adaptation; and (iii) exhaustion of energy for adaptation followed by collapse (Mulhall, 1996). The GAS model contends that stress is non-specific in that the same physiological changes result from every experience of stress and hence, it is the intensity of required (re)adjustment that is relevant rather than whether stress is pleasant (eustress) or unpleasant (distress) (Selye, 1956). In modern stress research, the strong physical and biological elements of the GAS model have been superceded by heavily psychologised notions of stress. Rather than change per se, (Bartley et al., 1998) only changes which are perceived as undesirable, unscheduled, non-normative, uncontrolled, or unpredictable are now thought to be stressful (McQueen & Siegrist, 1982; Pearlin, 1989).

Psychosocial stress has now become one of the most ubiquitous concepts in contemporary lay and scientific health discourses. Since the 1960s a plethora of disciplines including psychology, psychiatry, nursing, medicine, sociology, anthropology and pharmacology have studied psychosocial stress (Mulhall, 1996). In the 21st century alone there have already been over 100,000 articles indexed in PubMed with the keyword *stress*. This veritable mountain of research has been accompanied by a profusion of conceptual approaches which, as a whole, suffer from difficulties in characterising stress (Hinkle, 1987; Cohen et al., 1997; Quick & Nelson, 2001), and have lead to the criticism that 'stress in addition to being itself, and the result of itself, is also the cause of itself' (Ellis & Thompson, 1983).

This chapter aims is to bring some clarity to this diverse field by reviewing the variety of concepts used in the study of psychosocial stress and health. A brief history of stress research is followed by an overview of its contemporary conceptualisation and definition as well as a discussion of approaches to studying stressors, the stress process, stress sequelae and the distribution of stressors in society. The qualifier 'psychosocial' stress has been used thus far (hereafter referred to simply as 'stress') in order to exclude the study of physical stress in fields such as sports science, which is beyond the scope of this chapter. Furthermore, it is important to note that this chapter does not review empirical findings on the relationship between psychosocial stress and health or consider measurement issues in such empirical research. Given the enormous amount of empirical research in this area such an empirical review is not feasible. However, due to dearth of information on the relationship between stress and health for indigenous people (a particular focus of this thesis), a review of empirical findings in the specific area of stress and chronic disease in relation to these population groups is attempted in Chapter 6 below.

5.2 ***Conceptualising Stress***

There are two broad approaches to conceptualising stress in contemporary research: as a stimulus or as a response. The *stimulus-based* or *objective stress* approach has much in common with the GAS model in viewing stress as synonymous with a stressor (i.e. a

disturbing stimuli produced by an external/internal environment). On the other hand, the *response-based* or *subjective stress* model defines stress as an individual's response to stressors (Mulhall, 1996), which are defined more broadly as external/internal stimuli that are *potential* causes of stress (Aneshensel, 1992). Subjective stress involves a process of *appraisal* whereby an external/internal stimulus is perceived as a stressor if it is considered to be undesirable and/or if it results in a loss of power/resources. *Primary appraisal* involves the assessment of a stimulus as a stressor while *secondary appraisal* determines if an individual can alleviate/eliminate the effects of the stressor (Lazarus & Folkman, 1984) through *coping* or maintaining *homeostasis* (see below).

Much contemporary research attempts to combine stimulus and response based models into an *interactional* or *transactional* model (Lazarus & Folkman, 1984) which emphasises, to varying degrees, environmental stimuli as stressors; perceptual appraisal; and social, psychological and biological factors that affect both the occurrence of, and responses to, potential stressors (Hobfoll, 1989). Hence, as discussed in the remainder of this chapter, there are three key foci in contemporary stress research: the distribution, nature and meaning of (i) stressors; (ii) contextual factors that influence the stress process; and (iii) the biological and behavioural effects of, and responses to, stressors (i.e. stress sequelae).

The stress concept is based on a complex relationship between an individual and his/her environment and, as such, is a fundamentally relational phenomenon in which neither the interplay between objective and subjective approaches, nor a tautological emphasis on process can, or should be, entirely avoided (Hobfoll, 1989). Nonetheless, in conceptualising stress it is important to clearly delineate between stressors, the stress process and stress sequelae. Even very recent attempts to theorise stress have tended to fall short of this required clarity. For example, Aneshensel and Sucoff (1996) and Wheaton (1999) have explored the concept of an *ecological stressor* defined as 'stressors that occur at any level of the social unit above the individual' (Wheaton, 1999). They have suggested that the effect of ecological stress occurs through either a 'change in the probability of exposure to that stressor at the individual level' or by directly affecting 'individual functioning, regardless of the actual occurrence or nonoccurrence of that stressor at the individual level' (Wheaton, 1999). Yet Wheaton (1996) himself notes that 'stress is not the contingent or probabilistic processes that arise from it'. To contend that a stressor operates by affecting

the probability of exposure to itself or directly whether or not it actually occurs, only further obfuscates a term which has already experienced more than enough confusion.

5.3 ***Stressors***

Most stress research classifies stressors into three categories – *life events*, *chronic stressors*, and *daily hassles/uplifts*. Life events are discrete, acute, observable events which require major readjustment within a relatively short period of time (e.g. birth of a child, divorce) and are essentially self-limiting in nature (Wheaton, 1999). Chronic stressors are relatively enduring, persistent or recurrent demands, conflicts, threats, or problems which require readjustments over much longer periods of time (e.g. disabling injury, poverty) (Wheaton, 1999). Daily hassles and uplifts are mini-events, which require small behavioural (re)adjustments during the course of a day (e.g. traffic jams and a good meal, respectively) (Thoits, 1995). To date, most research attention has focused on life events and chronic stressors, with a more limited focus on daily hassles/uplifts.

Life events are among the oldest approaches utilised in stress research, generally taking the form of a checklist of events sampled from various domains across different hierarchies and weighted either by standardised importance of each event or subjectively by respondents. In general, these checklists were designed to be a representative sample of the major events that occur in people's lives (Cohen et al., 1997). However, it has been suggested that the universe of possible life events have not been sampled uniformly by such checklists, with events occurring to young adults being over-represented, while those occurring to women, minorities, and the poor being under- represented (Wheaton, 1994).

Traumatic events are a type of life event characterised by their suddenness and extreme magnitude of impact. Their effects on health tend to be persistent and responses to traumas may become fixed personality traits (Pearlin, 1999) such as *rumination* (repetitive passive thoughts about negative emotions and their consequences) (Folkman & Moskowitz, 2004) and post-traumatic stress disorder. Although the term *trauma* is almost always associated with an event, it has been suggested that living in a violent neighbourhood, for example,

should also be considered a form of trauma that could be labeled as a *chronic traumatic stressor* (Wheaton, 1999).

Chronic stressors, more generally, have been classified into a number of different types: role overload (e.g. caring for seriously impaired relatives), inter-personal conflicts within role sets (e.g. conflict between spouses), inter-role conflict (e.g. demands of work vs. family), role captivity (e.g. inability to leave an undesirable job), role restructuring (e.g. the changing relationship between adolescents and their parents) and ambient stressors (e.g. poverty, crime, and violence) (Pearlin, 1989), frustration of role expectations (Wheaton, 1983), also known as status inconsistency (Dressler, 1988) (e.g. less than expected income for a given occupation), goal-striving stress (i.e. discrepancy between aspiration/efforts and rewards/achievements (Wheaton, 1983; Dressler, 1988) and lifestyle incongruity (i.e. consumption patterns/behaviours inconsistent with social class (Dressler, 1988). A distinction has also been made between *chronic intermittent stressors* (e.g. examinations) and *chronic stressors* such as prolonged marital discord (Hobfoll, 1989).

Daily hassles are the irritating, frustrating, distressing demands that to some degree characterise everyday life, while uplifts are the daily events that are satisfying, pleasing and/or relaxing (Kanner et al., 1981). Daily hassles occupy an intermediate position between chronic stressors and life events, being in a sense ‘recurrent microevents’ (Wheaton, 1999) which may even mediate the relationship between these other two types of stressors (Weinberger et al., 1987; Hewitt & Flett, 1993).

Although life events and chronic stressors are usually considered to be distinct, life events tend to re-occur over time due to ongoing social, economic, and psychological determinants (Aneshensel, 1992) and chronic stressors can be thought of as a series of events in their own right. Hence, stressors labeled as acute and chronic are often interrelated in that they can lead to one another and also provide meaning and context for each other (Pearlin, 1989). In fact, some researchers have argued for the concept of *operant stress* which refers to a combination of recent/distal life events with chronic stressors that affect an individual at any one point in time (Turner et al., 1995). A similar concept is that of *cumulative stress*: an amalgamation of current stressors along with previous significant traumas that continue to act as stressors (Turner et al., 1995).

Another approach to the interplay between stressors is the notion of *stress sequences* in which the importance of the order and combination of stressors is recognised. For example, a chronic stressor in a particular role (such as work) followed by role exit/loss (such as being fired) may actually decrease rather than increase stress sequelae (Pearlin, 1989; Thoits, 1995) and other research has found increased reporting of daily uplifts after exposure to a chronic stressor (Folkman & Moskowitz, 2000). Related to stress sequences is the concept of *stress proliferation* in which a primary stressor leads to one or more secondary stressors, such as when the death of a loved one leads to social isolation (Pearlin, 1999). The *carry over* effect of stressors (and stress coping) is another approach taken in stress research. Carry over occurs when stress is translated between people (e.g. from wife to husband), across role domains (e.g. from work to home), across stages of life (e.g. from childhood to adulthood) (Thoits, 1995) or when coping with stress for a particular individual becomes a stressor for others in his/her social sphere (Pearlin, 1999).

Some researchers have also considered the notion that under-demand (e.g. boredom) is a type of stressor because it is not stimulating enough (Wheaton, 1996) and that *non-events* such as the failure of an expected or desired outcome may also be a type of stressor (Pearlin, 1999). For example, an anticipated event may cause stress, whether or not it actually occurs (Pollock, 1988). However, as others have noted (Wheaton, 1994), if a non-event is simply considered an event in itself this conceptual distinction becomes superfluous.

5.4 ***The Stress Process***

Psychological research on stress and health has explored the role that cognitive and behavioural traits, resources and responses play in the stress process (Taylor & Aspinwall, 1996; Bartley et al., 1998) with several different conceptual models being utilised in order to understand how contextual factors/resources influence the stress process. In the *stress-suppression/enhancement* model (Wheaton, 1985), a stressor mobilises resource(s) which

then alleviates or exacerbates this stressor by affecting its appraisal, responses to the stressor, further stressor proliferation and/or the relation between stressors and ill-health (Taylor & Aspinwall, 1996). A variation on this model entails stressors depleting the resource which alleviates or exacerbates it. In the *stress-deterrent/stimulant* model (Wheaton, 1985), these resources, which are causally antecedent to the stressor in question, reduce or increase the exposure to stressors, rather than influence the stress process itself (Pearlin, 1999). A third model conceptualises stressors and resources as having separate and opposite effects while remaining independent of one another. Resources offset or buttress the stressor, but are not involved directly involved in the stress process because they operate along a separate causal pathway, even in the absence of stressors (Wheaton, 1985).

The role that psychological resources play in the stress process (under any of the above models) is commonly studied under the rubric of stress *coping* – defined as cognitive and/or behavioural resources used to master, tolerate, reduce, offset, prevent, or avoid stressors, contain stressor proliferation, alter the meaning of stressors, and/or manage states of arousal caused by stressors (Pearlin, 1989; Aneshensel, 1992). Coping can be either a set of habitual preferences activated in situations of (potential) undesirability or loss, or more specific responses invoked selectively for specific situations (Pearlin, 1989; Aneshensel, 1992). *Coping strategies* can be categorised into either *problem-focused coping* (i.e. instrumental thoughts and behaviours) (Folkman & Moskowitz, 2000) or *emotion-focused coping* (i.e. efforts to ameliorate stress-mediated negative emotions) (Folkman & Moskowitz, 2004). Problem-focused and emotion-focused coping are also known as *approach* and *avoidance* coping strategies, respectively (Moos & Schaefer, 1993). Although these two general strategies have commonly been opposed, others have noted that they are in fact complementary (Lazarus, 2000), with the former being more effective in situations of high situational control and the latter in uncontrollable situations (Folkman & Moskowitz, 2004).

Emotion-focused coping such as *positive reappraisal* (in which stimuli are (re-)appraised as desirable or a gain) and *role de-valuation/distancing* (in which stimuli are deemed less or completely irrelevant and hence are neither (un)desirable nor a gain/loss) can also play a part in primary and secondary appraisal, and may even do so at an unconscious rather than

conscious level (Lazarus, 2000). Coping such as this, which involves reinterpreting the meaning of stimuli, is also known as *meaning-focused coping* (Folkman & Moskowitz, 2004). The nature of meaning-focused coping, in particular, highlights the conceptual difficulties inherent in differentiating between coping and secondary appraisal and also raises questions about how effective such coping is in reducing or eliminating stress sequelae (discussed below).

5.5 ***Stress Sequelae***

There are two proposed pathways leading from stress to ill-health: a direct effect on somatic health and disease development, and an indirect effect, where stress is expressed through health-damaging behaviour (Bartley et al., 1998). The field of biomedicine, with its emphasis on the body and disease, has lead research relating to the former type of stress sequelae (Hinkle, 1987), while there has been relatively little research on either the psychological/physiological processes by which stressors lead to the adoption, maintenance, and cessation of health-related behaviours (Yen & Syme, 1999; Steptoe, 2000) or the direct pathopsychological pathways through which stress affects personality traits such as self-esteem.

Two important concepts in the biomedical approach to stress research are *homeostasis* and *allostasis.* Homeostasis is the process of maintaining a constant internal equilibrium within an organism in opposition to stressors which may disrupt this equilibrium (Brunner & Marmot, 1999). In allostasis the body does not conserve the same internal state but, rather, establishes a new equilibrium in response to stressors. The cost of accommodating stressors through this process is known as allostatic load (O'Dea & Daniel, 2001).

A premise of studying stress sequelae is that the central nervous system (CNS), as the source of perception and responses to the external world, mediates the effects of stressors on health (O'Dea & Daniel, 2001). Given this premise, the biomedical study of stressors has, in the main, been pursued through two new sub-disciplines of physiology – psychoneuroendocrinology and pscyhoneuroimmunology – focusing on the effects of stressors on the endocrine and immune systems, respectively (Bartley et al., 1998). Other

biomedical research has also investigated the effects of stressors on cellular aging (Epel et al., 2004) as well as differing responses to stressors for women (Kudielka et al., 2000; Taylor et al., 2000; Ennis et al., 2001; Klein & Corwin, 2002) and African Americans (Myers et al., 1998).

The concept of fight-or-flight as originally proposed by Cannon is utilised in psychoneuroendocrinology to explain the responses to stressors in terms of two main pathways of the endocrine system – the Sympathetic-Adrenal Medullary (SAM) system and the Hypothalamic-Pituitary-Adrenocortical (HPA) axis (Brunner & Marmot, 1999). The fight reaction, associated with the SAM system, is activated very rapidly through the sympathetic branch of the autonomic nervous system. Stressor-mediated activation of the SAM system results in increased output of adrenaline (epinephrine) secreted into the bloodstream and noradrenaline (norepinephrine) released at the nerve endings. These hormones may produce suppression of cellular immune function, abnormal heart rhythms and neurochemical imbalances (Cohen et al., 1997; Cacioppo et al., 1998). Activation of this system also leads to the release of testosterone, heightened blood pressure, heart and metabolic rate, sweating, and constriction of peripheral blood vessels (Cohen et al., 1997; Brunner & Marmot, 1999; O'Dea & Daniel, 2001).

The flight reaction is associated with the HPA axis which is activated in response to chronic stress. When this occurs the anterior pituitary gland secretes andrenocorticotrophic hormone (ACTH), leading to activation of the adrenal cortex and secretion of corticosteroids such as cortisol which is an antagonist of insulin and leads to raised blood glucose, fatty acid release and increased output of cholesterol-carrying particles from the liver into the blood (Brunner & Marmot, 1999). The HPA axis also interacts with and influences the hypothalamus-pituitary-thyroid (HPT) axis (critical to normal metabolism) and the hypothalamus- pituitary-gonadal (HPG) axis (responsible for the regulation of reproduction) (Williams & Kurina, 2002). Psychoneuroendocrinological studies have also examined the interaction of the HPA axis with circadian rhythms (van Eekelen et al., 2003) and stress sequelae such as blood clotting, decrease in plasma volume, the build up of fibrofatty plaques, arteriosclerosis and impaired memory (Kaplan et al., 1993; McEwen & Sapolsky, 1995; Patterson et al., 1995).

Stress research in the field of psychoneuroimmunology has focused on cytokines as mediators between the CNS and the immune system (Muller & Ackenheil, 1998). The upregulation of factors such as interleukin-1 during the stress process leads to immunosuppression (Muller & Ackenheil, 1998) and chronic heightened cortisol levels have also been shown to depress immunologic function (McEwen, 1988). Prolactin secreted by the pituitary gland, as well as natural opiates such as beta endorphin and enkephalin excreted during HPA axis activation are also known to interact with the immune system in complex ways (Cohen et al., 1997; Brunner & Marmot, 1999). Pscyhoneuroimmunological research is also revealing how stressors contribute physiologically to damaging mental health sequelae such as schizophrenia, depression and anxiety. These sequelae may be mediated through the effects of cytokines on neurotransmitters such as dopamine and serotonin (Muller & Ackenheil, 1998; van Praag, 2004).

The association between stressors and biomarkers specific to physiological systems (i.e. cardiovascular, neuroendocrine, immune etc.) is an emerging area of interest in biomedical research (Theorell, 2003), with evidence that specific stressors may differentially affect various physiological systems (Lucas, 2003:344-5). This contradicts the non-specific stress response proposed in the GAS model and highlights the need to further understand the distribution of various kinds of stressors (as discussed below) in order to explain concomitant stress sequelae.

5.6 ***The Distribution of Stressors***

The study of the distribution of stressors has mainly occurred in the fields of sociology and anthropology where the social factors that determine who is exposed to stressors as well as when, where, how and why have been of interest. Although representing a very small body of research, there is also evidence from studies of monozygotic and dizygotic twins that genetics play a role in how many stressors an individual is exposed to, particularly in relation to life events such as inter-personal difficulties which may be influenced by individual behaviour as well as social context (Blackwood, 2000). It is also possible that genetic factors influence the nature and degree of stress sequelae (Yehuda & McEwen,

2004) as well as aspects of the stress process but there has been very little research on these topics to date.

Sociological approaches to stress research have tended to focus on analysing differences in group vulnerability across class, ethnorace, gender and age. Anthropological studies have studied stressors and disease in the complex interplay of social organisation, historical change and cultural context (Mulhall, 1996; Bartley et al., 1998). It has been suggested that the stress concept can provide a bridge linking large-scale organisation and social life with individual experience, action and functioning (Pearlin, 1989; Thoits, 1995).

Sociological research seeks to understand why health inequalities mirror social inequalities (Bartley et al., 1998). Such research focuses on stressors in the organisation and structure of life experience (Pearlin, 1989). From this perspective, the distribution of stress can be summarised as follows: (i) an individual's location in the social structure reflects inequality in resources, status, and power that differentially expose him/her to stressors (Aneshensel, 1992); and (ii) factors in the stress process (e.g. coping styles) are socially patterned in ways which, at least partially, leave members of disadvantaged groups more vulnerable to stress sequelae (Pearlin, 1989; Thoits, 1995; Turner et al., 1995).

To the extent that class, ethnorace, gender and age are systems that embody the unequal distribution of resources and opportunities, a low status within them may itself be a stressor (Pearlin, 1989; Brunner & Marmot, 1999), with stressors therefore being an inevitable outcome of systemic discrimination and inequity (Thoits, 1995). A related hypothesis is that societies prescribe a variety of forms of behaviour, conditions and relationships that are proper for its members – variable by social positionality. These prescribed forms are internalised by individuals as value systems, with sanctions for stepping outside these prescriptions manifesting as stressors (Hinkle, 1987).

A key question in sociological research on the distribution of stressors is whether social disadvantage is associated with exposure to stressors. A recent review indicates that people of lower SEP encounter more frequent negative life events and chronic stressors, and experience more emotional distress after equivalent exposure to stressors compared to people in higher SEP (Gallo & Matthews, 2003), while other research has found that low

SEP is associated with fewer (but more severe) daily hassles (Gryzwacz et al., 2004). Alternatively, negative experiences may lose their subjective impact when they are common (i.e. people may become accustomed to disadvantage). A number of anthropological studies provide evidence for this latter alternative, wherein social contexts of extreme poverty or endemic violence are considered normal and routine by those living within them (Nguyen & Peschard, 2003).

5.7 ***Conclusion***

It is vital that researchers in this field conceptualise stress clearly and unambiguously in terms of the models adopted, the types of stressors considered, the contextual factors relevant to the stress process, and the stress sequelae of aetiological importance. Such clarity in the study of stress will be a necessary prerequisite to elucidating the complex relationship between stressors, the stress process and racism as they relate to health sequelae and outcomes.

This review has also highlighted a number of emerging issues that will challenge the study of stress, including the need to better understand the role of stressors in pathopsychological processes and in modifying health behaviours as well as the interplay between genotypical and phenotypical influences on both stress sequelae and exposure/responses to stressors. The relationship between types of stressors and between emotion-/meaning-focused coping and secondary appraisal as well as the effect of such coping on individuals across social contexts and locations will also require further elucidation as will the possibility that stressors may have differential effects on various physiological systems and may vary in their importance by social location (e.g. ethnorace and gender), both psychologically and behaviourally as well in the physiological pathways through which they operate.

Given the possibility that the association between stressors and health may vary by social location, the next chapter considers both the empirical evidence for an association between stress and chronic disease, and the effectiveness of concomitant interventions, in relation to the population group of particular interest in this thesis – indigenous peoples.

Chapter 6. ***A review of psychosocial stress and chronic disease for fourth world Indigenous peoples and African Americans***[13]

6.1 ***Introduction***

1. There is now clear evidence that psychosocial stress is a risk factor for the development of a range of chronic diseases (Brunner, 1997; Brunner & Marmot, 1999; Cassidy, 1999; Krantz et al., 2000; Majer & Saper, 2000). It is also well-known that fourth world indigenous peoples are exposed to higher levels of stress and suffer a particularly high burden of chronic disease morbidity and mortality compared to dominant members of their respective societies (Australian Bureau of Statistics & Australian Institute of Health and Welfare, 2005; Bramley et al., 2005; Dressler, 2000; Kunitz, 1994). It is therefore surprising that there is very little empirical research on the association between stress and chronic disease for fourth world indigenous peoples. Furthermore, few evaluations of interventions to reduce stress or its ill-effects for indigenous peoples could be found in the published literature. Because a review of this very small body of existing research would be of dubious utility, this chapter broadens out the examination of these two topics to include African Americans as an oppressed ethnoracial group who live in a similar social context within a first world nation about which considerable research has been conducted in relation to stress and chronic disease. This chapter, therefore, aims to address two research questions: 1) what evidence is there linking psychosocial stress to the development of chronic disease or the complication of its management for fourth world indigenous peoples (hereafter refered to as indigenous peoples) and African Americans; 2) in these populations,

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13 This chapter is taken from Paradies Y. (2006). A review of psychosocial stress and chronic disease for 4th world Indigenous peoples and African Americans. *Ethnicity and Disease* 16(1): 295-308.

what interventions have been tested and proven successful in reducing psychosocial stress and/or its detrimental health effects?

6.2 ***Methods***

A review was conducted by searching available electronic databases using PubMed, Ovid, and the World Wide Web. The key words and Boolean operators used in searching these databases were based on a recent review of stress and coronary heart disease (Bunker et al., 2003) and Medline MeSH terms (see Box 6.1 below). Stress was defined broadly as psychological or social phenomena that tax an organism in such a way that physical, psychological, or social change results. The key exclusion resulting from this definition was of studies relating only to physical or physiological stress such as those in the field of sports science. Chronic disease was defined as non-acute illnesses and conditions, including mental illness, drug and alcohol misuse, and chronic infectious diseases. Studies that examined stress as an outcome of chronic disease were not included in this review.

A sequential process of examining article titles followed by abstracts and then full text was conducted whereby articles were excluded if: they were not available in English; the study population was not indigenous or African-American; or if the study did not include stress as an exposure and health as an outcome. This review includes published and unpublished items produced until the end of July, 2004. Communication with relevant experts in the field and manual searching of bibliographies were also undertaken as part of this review. In total, approximately 5000 journal articles, monographs, and conference abstracts were located and examined. Within this identified literature, 50 studies were found which met the inclusion criteria.

6.3 ***Results***

6.3.1 Associational Studies

A number of the 50 included studies were associational studies which examined the effects of various forms of stress on the development of chronic disease among indigenous peoples and African Americans. Of these, 13 were conducted with indigenous populations and the remaining 37 with African Americans. Twelve studies were qualitative in nature with the others being quantitative cross-sectional studies (n=31) and cohort studies (n=7).

Fifteen studies involved women only (10 cross-sectional, three cohort and two qualitative), five studies (four cross-sectional and one qualitative) involved men only and four studies (two cross-sectional and two cohort) related to adolescents only. Ten studies specifically considered the effect of stress on the management of chronic conditions such as HIV/AIDS (Byrnes et al., 1998; Catz et al., 2002; Delahanty et al.,2004; Kimerling et al.,1999; Simoni & Cooperman, 2000; Owens, 2003), hypertension (Boutain, 2001; Thompson et al., 2002), sickle cell disease (Thompson et al., 1992), mental illness (Ford, 2002), and myocardial infarction (Ong & Weeramanthri, 2002). Summary details of associational studies included in this review are shown in Table 6.1.

The most commonly used measures in these 50 studies were stressful life events/traumas (Delahanty et al., 2004; Dinges & Duong-Tran, 1992; Dole et al., 2003; Dressler, 1987; Ford, 2002; George & Lynch, 2003; Kimerling et al., 1999; Lincoln et al., 2003; Murry et al., 2001; Shiono et al., 1997; Stancil et al., 2000; Taylor et al., 1997; Turner & Lloyd, 2004; Whitbeck et al., 2002), financial/economic stress (Boutain, 2001; Dressler, 1987; Ellison et al., 2001; Harburg et al.,1973; Lincoln et al., 2003; Murry et al., 2001; Thompson, 1997; Troxel et al., 2003; Whitbeck et al., 2002), and perceived stress (Din-Dzietham et al., 2004; Sellers et al., 2003; Strogatz et al., 1997; Stancil et al., 2000; Thompson et al., 2002; Young et al., 2004). Half of these studies also adjusted for one or more confounders in their analysis. The most common confounding factors adjusted for in these studies were age (Din-Dzietham et al., 2004; Dressler, 1987; Ford, 2002; George & Lynch, 2003; Guthrie et al., 2002; Harburg et al., 1973; James et al., 1984; Knox et al., 2002; Shiono et al., 1997; Taylor et al., 1997; Tull et al., 2003; Utsey & Payne, 2000; Utsey et al., 2002; Webb & Beckstead, 2002; Whitbeck et al., 2002; Young et al., 2004),

education (Catz et al., 2002; Ford, 2002; George & Lynch, 2003; Shiono et al., 1997; Taylor et al., 1997; Tull et al., 2003; Utsey et al., 2002; Utsey & Payne, 2000; Whitbeck et al., 2002; Young et al., 2004), sex (George & Lynch, 2003; Gore & Aseltine, 2003; Thompson et al., 2002; Turner & Lloyd, 2004; Utsey et al., 2002), Body Mass Index (BMI) (Din-Dzietham et al., 2004; Knox et al., 2002; Thompson et al., 2002; Tull et al., 2003; Young et al., 2004), gender (Din-Dzietham et al., 2004; Guthrie et al., 2002; Tull et al., 2003; Whitbeck et al., 2002), and income (Taylor et al., 1997; Utsey et al., 2002; Whitbeck et al., 2002). Adjusting for these confounders was common in both cross-sectional and cohort studies but did not occur in qualitative studies. A number of cross-sectional studies used convenience (Din-Dzietham et al., 2004; Simoni & Cooperman, 2000; Stancil et al., 2000; Taylor et al., 1997; Utsey et al., 2002; Utsey & Payne, 2000; Webb & Beckstead, 2002; Williams & Lawler, 2001; Young et al., 2004) or non-random samples (Byrnes et al., 1998; Catz et al., 2002; Delahanty et al., 2004; Dinges & Duong-Tran, 1992; Ford, 2002; Guthrie et al., 2002; Thompson et al., 1992) and several cohort studies also used non-random sampling techniques (Dole et al., 2003; Kimerling et al., 1999; Shiono et al., 1997), with all qualitative studies using convenience samples.

Across this range of research a number of poor health outcomes were found to be positively associated with increased stress exposure as variously measured. Mental health outcomes associated with stress were psychological distress (Ellison et al., 2001; Lincoln et al., 2003; Taylor et al., 1997), depression (Catz et al., 2002; George & Lynch, 2003; Gore & Aseltine, 2003; Simoni & Cooperman, 2000; Thompson et al., 1992; Turner & Lloyd, 2004; Whitbeck et al., 2002), reduced psychological well-being (Ellison et al., 2001; Young et al., 2004) and psychological functioning (Murry et al., 2001), poor mental health (Swan & Fagan, 1991; Utsey et al., 2002; Young et al., 2004), increased anxiety (Catz et al., 2002; Dole et al., 2003; Thompson et al., 1992; Utsey & Payne, 2000), suicidal ideation (Dinges & Duong-Tran, 1992; Radford et al., 1990) and suicidal outcomes (Dinges & Duong-Tran, 1992; O'Neil, 1986b).

Physical health outcomes associated with stress were elevated blood pressure (Boutain, 2001; Din-Dzietham et al., 2004; Knox et al., 2002; Stancil et al., 2000; Thompson et al., 2002), hypertension (Din-Dzietham et al., 2004), self-reported illness (Williams & Lawler, 2001), impaired immune function (Kimerling et al., 1999), diabetes (Garro, 1995; London

& Guthridge, 1998; Skinner & Silverman-Peach, 1989; Sunday et al., 2001; Thompson, 1997), hyperglycemia (Skinner & Silverman-Peach, 1989), heart disease (Ong & Weeramanthri, 2002; Thompson, 1997), pre-term birth (Dole et al., 2003), reduced physical well-being (Young et al., 2004), increased heart rate (Knox et al., 2002) and increased carotid artery intima-media thickness (Troxel et al., 2003). In addition, health damaging behaviors such as drug misuse (Dinges & Duong-Tran, 1992) and cigarette smoking (Guthrie et al., 2002) were also found to be associated with stress exposure.

On the other hand, there were also a number of studies which did not find a statistically significant association with stress exposure. Psychological distress (Lincoln et al., 2003), blood pressure (Alexander et al., 1996; Din-Dzietham et al., 2004; Dressler et al., 1996 ; Harburg et al.,1973; James et al., 1984; Stancil et al., 2000; Strogatz et al., 1997; Thompson et al., 2002; Webb et al., 2000), hypertension (Din-Dzietham et al., 2004; Webb & Beckstead, 2002), insulin resistance (Tull et al., 2003), blood glucose (Dressler et al., 1996), life satisfaction (Utsey et al., 2002), physical well-being (Utsey et al., 2002), general well-being (Ford, 2002), depression (Utsey & Payne, 2000; Whitbeck et al., 2002), anxiety (Utsey & Payne, 2000), self-reported illness (Williams & Lawler, 2001), atherosclerotic plaque (Troxel et al., 2003), natural killer cell cytotoxicity (Byrnes et al., 1998), and birth weight (Shiono et al., 1997) were found to have a null association with stress exposure in the studies cited above. Only one study found an association between stress and improved health with posttraumatic stress disorder symptoms related to higher CD4 count in HIV positive African Americans (Delahanty et al., 2004).

In the 50 studies overall, there were 25 positive associations and five null associations between a stress exposure and a mental health outcome while for physical health outcomes and stress exposure there were 18 positive associations and 18 null associations. Notably, there was a marked difference in associations between stress and increased systolic vs. diastolic blood pressure with 11 out of 17 associations (65%) being positive, rather than null, for the former and only five out of 14 (34%) being positive, rather than null, for the latter.

A number of factors were found to moderate the effects of stressors. These factors were social support (Dressler, 1987; Lincoln et al., 2003), personal control (Lincoln et al., 2003),

hardiness (Williams & Lawler, 2001), racism (Murry et al., 2001), religiosity (Ellison et al., 2001), substance misuse (Utsey & Payne, 2000) and several coping strategies (Dressler, 1987; Littrell & Beck, 2001; Stancil et al., 2000). Active problem-focused responses to stress (Dressler, 1987; Littrell & Beck, 2001; Stancil et al., 2000), social support (Dressler, 1987; Strogatz et al., 1997), hardiness (Williams & Lawler, 2001) and religiosity (Ellison et al., 2001) were found to buffer the ill-effects of stress on health while factors found to exacerbate these ill-effects were neuroticism (Lincoln et al., 2003), racism (Murry et al., 2001) and substance misuse (Utsey & Payne, 2000).

The associational studies in this review were also considered by study type and study population. The seven cohort studies found that blood pressure (Knox et al., 2002), heart rate (Knox et al., 2002), depression (Gore & Aseltine, 2003; George & Lynch, 2003), psychological distress (Sellers et al., 2003), immune function (Kimerling et al., 1999) and pre-term birth (Dole et al., 2003) were associated with stress. The health outcomes associated with stress in the twelve qualitative studies were general health status (Sibthorpe, 1988), poor mental health (Swan & Fagan, 1991), suicidal ideation (Radford et al., 1990), suicidal outcomes (Dinges & Duong-Tran, 1992; O'Neil, 1986b), elevated blood pressure (Boutain, 2001; Stancil et al., 2000), diabetes (Garro, 1995; London & Guthridge, 1998; Skinner & Silverman-Peach, 1989; Sunday et al., 2001; Thompson, 1997), hyperglycemia (Skinner & Silverman-Peach, 1989) and heart disease (Ong & Weeramanthri, 2002; Thompson, 1997).

The 13 studies of indigenous groups in Australia, Canada, and the United States found that health outcomes associated with stress were general health status (Sibthorpe, 1988), poor mental health (Swan & Fagan, 1991), depression (Whitbeck et al., 2002), suicidal ideation (Dinges & Duong-Tran, 1992), suicidal outcomes (Dinges & Duong-Tran, 1992; O'Neil, 1986b; Radford et al., 1990), diabetes (Garro, 1995; London & Guthridge, 1998; Skinner & Silverman-Peach, 1989; Sunday et al., 2001; Thompson, 1997), hyperglycemia (Skinner & Silverman-Peach, 1989), heart disease (Ong & Weeramanthri, 2002) and substance misuse (Dinges & Duong-Tran, 1992). Findings from the four studies of adolescents indicate that depression (Gore & Aseltine, 2003), cigarette smoking (Guthrie et al., 2002), suicidal ideation (Dinges & Duong-Tran, 1992) and suicidal outcomes (Dinges & Duong-Tran, 1992; Radford et al., 1990) are associated with stress and the ten studies which examined the effects of stress on the management of chronic diseases suggest that elevated blood pressure

(Boutain, 2001; Din-Dzietham et al., 2004; Stancil et al., 2000; Thompson et al., 2002), impaired immune function (Kimerling et al., 1999), depression (Catz et al., 2002; Simoni & Cooperman, 2000; Thompson et al., 1992) and anxiety (Thompson et al., 1992) are associated with stress.

The 15 studies involving only women found that health outcomes associated with stress were depression (Catz et al., 2002; Simoni & Cooperman, 2000), psychological distress (Utsey & Payne, 2000), anxiety (Radford et al., 1990; Troxel et al., 2003), reduced self-esteem (Utsey & Payne, 2000), reduced well-being (Alexander et al., 1996), poor mental health (Ellison et al., 2001; Taylor et al., 1997; Young et al., 2004), elevated blood pressure (Boutain, 2001; Din-Dzietham et al., 2004; Stancil et al., 2000), self-reported illness (Williams & Lawler, 2001), immune function (Kimerling et al., 1999), pre-term birth (Dole et al., 2003) and increased carotid artery intima-media thickness (Troxel et al., 2003). In one of these studies, hardiness was also found to buffer the ill-effects of stress on health (Williams & Lawler, 2001). Finally, the five studies involving only men found that stress was associated with increased depression (Littrell & Beck, 2001), anxiety (Utsey & Payne, 2000), suicidal outcomes (O'Neil, 1986b), mortality (James et al., 1976) and elevated blood pressure (James et al., 1984), with substance misuse acting as a stress exacerbator in one of these studies (Utsey & Payne, 2000).

6.3.2 Intervention Studies

This review identified a total of 15 studies which examined the treatment and/or management of stress in Indigenous or African-American populations. Of these, six were randomised controlled trials (Alexander et al., 1996; Barnes et al., 2001; Castillo-Richmond et al., 2000; Gaylord et al., 1989; Kondwani & Lollis, 2001; Schneider et al., 1995; Webb et al., 2000), one was a retrospective cohort study (Schneider et al., 2001) and the remaining eight were demonstration studies of programs or activities, without control groups, that ranged over periods from a day to almost a year (Brave Heart, 1999; Haber, 1986; Harrison & Rao, 1979; Lee et al., 1999; Mays, 1995; McGrady & Roberts, 1992; Moshinsky et al., 2000; Tsey & Every, 2000). Of these interventions, 11 involved African Americans, four Indigenous

Australians and one Native Americans. Summary details of intervention studies included in this review are shown in Table 6.2.

The types of interventions trialed were transcendental meditation (TM) (Alexander et al., 1996; Barnes et al., 2001; Castillo-Richmond et al., 2000; Gaylord et al., 1989; Kondwani & Lollis, 2001; Schneider et al., 1995; Schneider et al., 2001), progressive muscle relaxation (PMR) (Alexander et al., 1996; Gaylord et al., 1989; Schneider et al., 1995; Schneider et al., 2001; Webb et al., 2000), yoga and aerobics (Haber, 1986), biofeedback relaxation (Harrison & Rao, 1979), focused support groups (Mays, 1995), empowerment programs (McGrady & Roberts, 1992; Tsey & Every, 2000), psychoeducational group experiences (Brave Heart, 1999) and stress reduction/management workshops (Lee et al., 1999; Moshinsky et al., 2000).

Six randomised controlled trials involving African Americans used TM as the intervention, with PMR also used as a secondary intervention arm in several trials. These studies were conducted with both hypertensive and normotensive African Americans. Compared with the control group, those who undertook the TM intervention had decreased carotid intima-media thickness (Castillo-Richmond et al., 2000), systolic blood pressure (Alexander et al., 1996; Barnes et al., 2001; Schneider et al., 1995) and diastolic blood pressure (Alexander et al., 1996; Schneider et al., 1995), heart rate and cardiac output (Barnes et al., 2001), anxiety (Kondwani & Lollis, 2001; Schneider et al., 1995), depression (Kondwani & Lollis, 2001), neuroticism (Gaylord et al., 1989), and sleep dysfunction (Kondwani & Lollis, 2001) as well as increased energy (Kondwani & Lollis, 2001), general mental health (Gaylord et al., 1989) and health locus of control (Kondwani & Lollis, 2001). However, a few TM and PMR interventions failed to produce changes in health outcomes with one study finding no significant change in heart rate for the TM group (Schneider et al., 1995) and another finding no change in vocational strain (Webb et al., 2000) for the PMR group. In general, TM was more effective than PMR in an eight year follow-up of 530 African-American participants which found a 63% reduction in all-cause mortality and a 82% reduction in heart disease mortality in the intervention group compared to the control groups (Schneider et al., 2001).

The single study that used a yoga and aerobics intervention did not produce any change in blood pressure (Haber, 1986) while the two studies which utilised biofeedback relaxation had mixed results showing improvement in diastolic blood pressure and forehead muscle tension

but not in systolic blood pressure or finger temperature (Harrison & Rao, 1979; McGrady & Roberts, 1992).

The focused support group (FSG) model aims to: 1) build and enhance group bonding and group cohesion in a short period of time; 2) present participants with a model of facilitation that they can easily execute; and 3) change patterns of coping through increased awareness of behaviors (Mays, 1995). The FSG model also seeks to buffer the effects of stress through the provision of positive support networks and by increasing knowledge of gender and ethnic discrimination as a significant source of stress (Mays, 1995). The study considered in this review of the FSG model found that, according to participant self-reports, the intervention reduced stress and increased personal and professional support networks for the 65 African-American women participants (Mays, 1995).

An empowerment program conducted with Indigenous Australians was, according to the post-intervention evaluation, effective in enhancing participants' awareness, resilience, and problem solving ability (Tsey & Every, 2000). This program was based on the principles of psychosynthesis, analytical skills, mastery training, transformation, and meditation and has since been delivered in a number of Indigenous communities in Australia (Tsey & Every, 2000). Another study, underway in north Queensland since 2001, supports earlier findings that an empowerment program is able to equip Indigenous Australians with greater analytical and problem-solving skills, provide space for self-reflection and growth, encourage confidence, improve communication skills, promote empathy and an ability to help others, and create an improved understanding of root causes of problems (Tsey et al., 2003). A similar program conducted in the United States used a psycho-educational group experience based on notions of group sharing and support which incorporated Native American cultural traditions. Self-report data after this intervention suggests that it was effective in promoting positive and reducing negative emotions (Brave Heart, 1999).

Two stress reduction/management workshops have also been conducted in Australia with Indigenous people using techniques such as identifying signs of stress and strategies to promote well-being, stress management techniques, balancing stress, problem-solving skills, simple relaxation techniques, and dealing with anger (Lee et al., 1999; Moshinsky et al., 2000). The only one of these studies which was quantitatively evaluated showed a reduction

in both the incidence and complications of diabetes (Lee et al., 1999).

6.4 ***Discussion***

Within the 50 associational studies examined in this review there was a diversity of stress measures utilised. Of these, stressful life events (which was used in eleven studies) was the most commonly utilised measure. The literature has noted that stress measures have tended to under-sample the experiences of women, minorities, and the poor especially in the case of life events measures (Wheaton, 1994). In addition, many of the exposure measures used were not psychometrically validated. The Perceived Stress Scale was the most commonly used psychometrically validated scale in these studies (Sellers et al., 2003; Stancil et al., 2000; Strogatz et al., 1997; Thompson et al., 2002; Young et al., 2004). In general, however, more work is needed to standardise stress exposures in the study of chronic disease development to allow greater comparability across studies.

Another limitation of this body of work is that the majority of associational studies (31 in total), including several cohort studies (Dole et al., 2003; Kimerling et al., 1999; Shiono et al., 1997) and all qualitative studies, used either convenience or non-random sampling approaches. However, an examination of studies using random/representative sampling against those using convenience/non-random sampling suggests that these two sub-sets are comparable in terms of stress exposure measurement, confounder adjustment, and associated health outcomes.

Although there are only seven cohort studies included in this review, it is worth noting that the findings from this sub-set are comparable to that of the review as a whole in terms of stress exposure measurement, sampling methods, and adjustment for confounders. Both physical (four studies) and mental (three studies) health outcomes were examined in these seven studies and all three null associations related to physical health outcomes. There were also similar findings for African Americans and indigenous groups, with a range of both

physical and mental health outcomes associated with stress exposure for both these groups and more null associations for physical rather than mental health outcomes.

The studies in this review, as a whole, adjusted for a range of well-known demographic and anthropometric confounders. However, many studies did not take account of possible confounders at all or did so insufficiently; although in some cases, this was because stress was not considered as the primary exposure of interest and was itself adjusted for in other examined relationships. More importantly, only one study adjusted for factors that were identified in this body of literature as moderators of the relationship between stress and chronic disease (Whitbeck et al., 2002).

The ways in which these factors can affect the relationship between stress and health for members of oppressed ethnoracial groups are complex. For example, the active coping style of 'John Henryism' is one of the few stress-related constructs hypothesised to apply particularly to members of oppressed ethnoracial groups. This coping style is named after a legendary African-American railroad steel-driver in the late 19th century who, after winning a celebrated race against a mechanical steel drill, dropped dead from exhaustion. As such, it is used to describe the tendency to work extremely hard to disprove the stereotype of laziness and inability (James et al., 1984). A number of studies have shown that African Americans with high levels of 'John Henryism' are at increased risk from the detrimental effects of stress on their health, especially high blood pressure (Dressler, 1987; Fernander et al., 2004; James et al., 1984; James et al., 1992; Merritt et al., 2004; Tsey et al., 2003; Wheaton, 1994). This relationship appears to be strongest for African Americans of low SEP (Fernander et al., 2004). However, in one study the relationship only held for low SEP African-American women and high SEP men but not vice versa (Wheaton, 1994). Other studies failed to find an association with blood pressure (Jackson & Adams-Campbell, 1994) in one case and in another, found an association between John Henryism and reduced stress for African-American women (Williams & Lawler, 2001). Clearly, further research on stress moderators is needed as is the measurement and analysis of those moderating factors that have already been identified.

Turning to the second research question considered in this review, there is evidence that two intervention techniques are effective in ameliorating stress and its effects – Transcendental

Meditation and group-oriented stress management. Unfortunately this evidence is limited by the fact that only two of the RCTs of TM in this review used blinding (Alexander et al., 1996; Castillo-Richmond et al., 2000; Schneider et al., 1995) with other studies being either non-blinded (Webb et al., 2000) or not stating their blinding status. Also the interventions involving group-oriented stress management were demonstration studies with only limited follow-up and long-term evaluation. However, TM has been investigated in several quantitative meta-analyses of first-world populations, with evidence of its effectiveness in reducing physiological arousal, anxiety, smoking, alcohol and drug misuse, and improving psychological health (Schneider et al., 1995). In a meta-analysis of 146 studies, anxiety measures decreased more in the groups that practiced TM compared to those using other forms of meditation or relaxation and individuals who practiced TM also showed decreases in illicit drug use, depression as well as increased self-actualisation and positive health habits (Kondwani & Lollis, 2001). It should be noted that, although there is a clear place for individual-level interventions to reduce stress and/or its health effects, the increased burden of stress for oppressed ethnoracial groups will only be eliminated through attention to the societal-level causes (as addressed in relation to racism in Chapter 10).

This review has a number of limitations which should be noted. By focusing on published articles, it is likely that this review has overstated the strength of the evidence due to publication bias. Also, given the difficulties in weighting evidence across such studies with such heterogeneity in exposure and outcome measures as well as study populations, it was not possible to perform a meta-analysis with the studies identified in this review. For similar reasons, it was not thought that an in-depth examination of the methodological strengths and weaknesses of the studies in this review would be appropriate.

Bearing in mind these limitations, this review indicates that stress is a determinant of chronic mental, more so than physical, ill-health for indigenous groups and African Americans, and highlights two promising interventions that warrant further investigation. This differential strength of association between stress and mental vs. physical health corresponds to the findings for racism discussed in Chapter 4 and raises similar questions about the mechanisms by which stress affects health, suggesting the need for further research on the possible pathopsychological and/or neurophysiological aspects of the stress process that lead to ill-health for oppressed ethnoracial groups. Such research is especially warranted for indigenous

populations about whom less is known in comparison to African Americans. Given that chronic disease susceptibility starts early in life (Kuh & Ben-Shlomo, 2004), more research should also be undertaken with children and adolescents and longitudinal studies will be required to establish the direction of causation for stress processes.

Having established the nature and validity of racism as a determinant of health as well as elucidating the concept of stress and demonstrating its association with chronic disease for indigenous peoples, this thesis now details the development and validation of an instrument designed to assess racism in relation to Indigenous Australians.

Box 6.1: Search terms

Chronic disease OR coronary heart disease OR coronary artery disease OR acute myocardial infarction OR sudden death OR ventricular fibrillation OR ventricular tachycardia OR mortality OR atherosclerosis OR atherogenesis OR ischaemic heart disease OR acute coronary syndrome OR diabetes OR renal disease OR mental health OR arthritis OR hypertension OR lipids OR HIV OR AIDS OR COPD OR Chronic Obstructive Pulmonary Disease OR emphysema OR Parkinson disease OR Alzheimer's disease OR epilepsy OR inflammatory bowl disease OR cirrhosis OR hepatitis OR leukaemia OR leukemia OR anaemia OR anemia or lupus.

AND

stress OR social stress OR psychosocial OR psychosocial stress OR psycho-social stress OR psychological stress OR psychological distress OR burnout OR depression OR anxiety OR hostility OR anger OR hopelessness OR helplessness OR vital exhaustion OR work stress OR job control OR Type A behaviour OR Type A behavior OR life events OR stressful events OR PTSD (adapted from Bunker et al., 2003).

AND

Aborigines OR aboriginal OR Aborigine OR indigenous OR Indigenous Australians OR australoid OR australoid race OR Indians, north American OR north American Indians OR Eskimos OR maori OR Indians, central American OR central American Indians OR Negroid OR Negroid race OR African Americans OR inuit OR cree OR mulatto OR mestizo OR miao OR Indians, south American, OR south American Indians (adapted from Medline MeSH terms)

Table 6.1: Associational Studies of Psychosocial Stress and Chronic Disease in Indigenous and African American Populations

Study Population	Exposure Measure	Outcome Measure	Findings
	Cross-sectional studies (men and women)		
(Delahanty et al., 2004) Nonrandom clinical sample of 110 HIV adults from a Midwestern city (49 African American), NRRG	PTSD measured by the 15-item Impact of Event Scale	Self-reported physician determined CD4 count	PA*
(Din-Dzietham et al., 2004) Convenience sample of 356 African Americans aged 21 years and over in Atlanta, Georgia (196 men and 160 women)	Perceived stress from race-based discrimination at work (RBDW) from non-African Americans (inter-RBDW) and from other African Americans (intra-RBDW)		Perceived stress by: Intra- Inter-RBDW
		Self-reported physician diagnosed hypertension[a,bmi,c,g,]	No A PA*
		SBP [a,bmi,c,g,]	PA* PA*
		DBP [a,bmi,c,g,]	No A No A
Nonrandom sample of 124 American Indian and Alaskan Native adolescents with a depressive disorder, aged 14 to 18 years in Alaskan boarding schools (60 male and 64 female), NRRG (Dinges & Duong-Tran, 1992)	Life events: family/ parental conflicts, marital/ pregnancy fears, inter-personal conflicts, loss of culture supports/ academic	Suicide ideation	PA*
		Suicide attempt	PA*
		Drug misuse	PA*

Study Population	Exposure Measure	Outcome Measure	Findings
	demands, social rejections & close death		
SRS of 285 African-Americans aged 17 and over in a southern US city (101 male and 184 female), NRRG (Dressler, 1987)	8-item measure of economic stress	Depression sub-scale of the Hopkins Symptom Checklista,m,ses	PA* (men, women & total sample)
SRS of 93 Mississippi Choctaw aged 21 years or older (43 men and 50 women), 67% RR (Dressler et al., 1996)	10-item psychological stress scale combining mood, acute/chronic stress	Blood glucosedi,hy,me SBPdi,hy,me DBPdi,hy,me	No A No A No A
Multistage area probability sample of 1,139 African American adults as part of the Detroit Area Study, 70% RR (Ellison et al., 2001)	1-item work problems 2-item financial problems 4-item family problems	6-item PD index 2-item psychological well-being	PA* (all three measures) PA* (financial, family stress)
Nonrandom network sample of 105 African American girls aged between 11 and 19 years in the US who were not pregnant, NRRG (Guthrie et al., 2002)	26-item Daily Hassles for Adolescents Inventory including extent of hassle for each item	Ever smoked	PA*

Study Population	Exposure Measure	Outcome Measure	Findings
All census tracks in Detroit using a random sample (stratified by sex, race, stress and area) of 487 married African Americans aged 25 to 60 years who had relatives in the area, 94% RR (Harburg et al.,1973)	Stress as measured by economic deprivation, residential/ family instability, crime & density	SBP[a, h,o, p, rr, se, ti] DBP [a, h,o, p, rr, se, ti]	PA* (men), No A (women) PA* (men), No A (women)
Nationally representative sample of 549 African Americans aged 18-54 in the US National Comorbidity Survey, 82.4% RR (Lincoln et al., 2003)	10-item Traumatic events	4-item PD[a,e]	PA*
	2-item Financial strain	4-item PD[a,e]	No A
Area probability sample of 897 caregivers (93% women) of African American children (10-11 years old) in Iowa and Georgia, 61-68% RR (Murry et al., 2001)	29-item negative life events, chronic financial stressors and job stress	Psychological functioning measured by the 5-item distress-depression and 3-item distress-anxiety subscales of the Mini-Mood and Anxiety Symptom Questionnaire	NA*
Stratified random sample of 1,784 African Americans aged 25-50 years in Pitt County, North Carolina, 80% RR (Strogatz et al., 1997)	8-item adaptation of PSS	SBP DBP	PA* (women), No A (men) PA* (women), No A (men)

Study Population	Exposure Measure	Outcome Measure	Findings
Nonrandom clinical roster sample of 109 African American adults with sickle cell disease in the US, 94.7% RR (Thompson et al., 1992)	Daily stress measured by the 17-item Hassles Scale	Depression (SC-90)[a,com,g,pa,scd,ses]	PA*
		Anxiety (SC-90)[a,com,g,pa,scd,ses]	PA*
		Global Severity Index (SC-90)[a,com,g,pa,scd,ses]	PA*
Random sample of 126 non-obese normotensive or mildly hypertensive African Americans (97 male and 29 female) in Pennsylvania, NRRG (Thompson et al., 2002)	14-item PSS	Resting SBP[a,al,bmi,s]	PA*
		Resting DBP [a,al,bmi,s]	PA†
		Daytime ambulatory SBP [a,al,bmi,s]	PA*
		Daytime ambulatory DBP [a,al,bmi,s]	No A
Random sample of 296 African Americans aged 20 years older who were both born and currently living in the US Virgin Islands (97 men and 199 women), 77.2% RR (Tull et al., 2003)	Psychosocial stress as measured by sub-scales of the Roger's Life Attitude Inventory	Insulin resistance as measured by fasting serum insulin using the homeostasis model assessment method[a,bmi,ed,f,g,w]	No A
Representation sample of 1,803 young adults (<23 years) in South Florida (25% African Americans), 70.1% RR (Turner & Lloyd, 2004)	33-item lifetime exposure to adversity including major/traumatic life events	Depressive disorder (DSM-IV)[et,s,t,ses]	PA* (African Americans)
		Anxiety disorder (DSM-IV)[et,s,t,ses]	PA* (African Americans)

Study Population	Exposure Measure	Outcome Measure	Findings
Convenience sample of 127 African Americans aged 55 to 93 years (87 women and 26 men, 14 missing sex) in the US (Utsey et al., 2002)	46-item IRRS with four subscales: Individual, Institutional, Collective and Cultural	5-item Satisfaction with Life Scale [a,ed,i,s]	No A
		MCS of the Short Form-36 [a,ed,i,s]	NA* (institutional subscale)
		PCS of the Short Form-36 [a,ed,i,s]	No A
Random sample of 287 Native American adults (96 men and 191 women) in the upper Midwest, 85% RR (Whitbeck et al., 2002)	6-item financial strain scale (alpha =0.86)	Depression (CES-D) [a,al,ch,d,ed,g,ho,pi,pt,ss]	No A
	12-item negative life events scale	Depression (CES-D) [a,al,ch,d,ed,g,ho,pi,pt,ss]	PA*
Cross-sectional studies (women only)			
Nonrandom clinical sample of 36 HIV+ African American women in Miami, NRRG (Byrnes et al., 1998)	10-item version of the Life Experiences Survey measuring stress over past 12 months	Natural killer cell cytotoxicity [ar,cs,hp,pe,sm]	No A
Nonrandom clinical sample of 100 women with HIV/AIDS receiving medical care in Louisiana (84% African American), 80% RR (Catz et al., 2002)	43-item SRRS	Depression (20-item CES-D) [ds,e,thiv]	PA*
		Anxiety measured by the State-Trait Anxiety Inventory [ds,e,thiv]	PA*

Study Population	Exposure Measure	Outcome Measure	Findings
Nonrandom clinical sample of 55 African American women with severe and persistent mental illness aged 18 to 68 years in Pennsylvania who were mental health service outpatients, 78.6% RR (Ford, 2002)	Violence and trauma measured by the 49-item self-report Posttraumatic Diagnostic Scale	Well-being (SF-36)[a,e,m] No. of psychiatric disorders [a,e,m]	No A PA†
Convenience sample of 373 women with HIV/AIDS in New York city (44% African American) (Simoni & Cooperman, 2000)	Stress measured by frequency of physical and sexual abuse	Depression (20-item CES-D)	PA*
Convenience sample of 94 pregnant African American women from the Pittsburg area without hypertension, diabetes or previous adverse pregnancy outcomes (Stancil et al., 2000)	Life Events Scale PSS Life Events Inventory	SBP SBP SBP	No A PA* No A
Convenience sample of 79 African American mothers of adolescents attending high school (Taylor et al., 1997)	Stressful life events measured by the 35-item Psychiatric Epidemiology Research Interview Life Events Scale	Psychological distress (20-item CES-D)[a,e,i,fs] Self-esteem (Rosenberg Self-Esteem Scale)[a,e,i,fs]	PA* NA*

Study Population	Exposure Measure	Outcome Measure	Findings
Representative sample of 334 pre-menopausal women (225 non-Hispanic Caucasians and 109 African Americans) aged 42-52 years from the Pittsburgh area, RR: 88.6% (African Americans), 84.9% (Caucasians) (Troxel et al., 2003)	Life events Ongoing stressors Economic hardship	Carotid artery intima-media thickness measured by ultrasound [HDL-C,SBP,whr] Artherosclerotic Plaque[HDL-C,SBP,whr] Hypertensive status[a,py,whr]	PA* (composite stress score) PA† (life events), PA† (ongoing stressors) PA† (economic hardships) No A No A
Convenience sample of 90 African American women employed in a health care facility in the US (Webb & Beckstead, 2002)	The Personal Strain Inventory (four 10-item scales: vocational, inter-personal, psychological & physical strain		
Convenience sample of 50 low-income African American women in the US (Williams & Lawler, 2001)	126-item SRRS in last 12 months for: Self, ignificant other, Community stress (social problems, violence, & fear in their community)	Illness measured using self-report checklist of disease[ba,dd,di,ddi,du,th]	PA* (self), PA* (significant other), No A (community stress)

Study Population	Exposure Measure	Outcome Measure	Findings
Convenience sample of 128 urban African American women enrolled in a church-based physical activity randomised trial from Baltimore, Maryland (Young et al., 2004)	PSS	Physical health status (SF-36)[a,e,bmi] Mental health status (SF-36)[a,e,bmi] Wellbeing measured by Cantril's Ladder of Life[a,e,bmi]	No A NA* NA*
	Cross-sectional studies (men only)		
Ecological study of all 45-54 year-old men residing in 86 counties in North Carolina (James et al., 1976)	Stress measured by a composite of children not living at home with both parents, divorce, delinquency, correction school admissions, incarceration and murder rates	Hypertension, heart disease and stroke related mortality rates	PA* (non-whites in the difference between mortality rates for high and low stress groups)

Study Population	Exposure Measure	Outcome Measure	Findings
Area probability sample of 112 African American male workers aged 17-60 years in rural North Carolina, 91% RR (James et al., 1984)	1-item job security	SBP[a,bmi,s,ti]	NA*
		DBP[a,bmi,s,ti]	No A
	1-item race as a hindrance to job success	SBP[a,bmi,s,ti]	PA†
		DBP[a,bmi,s,ti]	PA†
	1-item unfair wages	SBP[a,bmi,s,ti]	No A
		DBP[a,bmi,s,ti]	No A
Random sample of 90 African-American homeless men from a church soup-kitchen in the US, NRRG (Littrell & Beck, 2001)	20 item hassles/stressors in the week before the survey	CES-D (week prior to the survey)	PA*
Convenience sample of 126 African American men, 56 from a residential substance abuse treatment program (clinical sub-sample) and 70 undergraduate college students (non-clinical sub-sample) (Utsey & Payne, 2000)	22-item Index of Race-Related Stress using the overall Global Racism scale	21-item Beck Depression Inventory II[a,e,i]	No A (both sub-sample)
		21-item Beck Anxiety Inventory[a,e,i]	PA* (clinical sub-sample)
			No A (non-clinical sample)
Cohort Studies			

Study Population	Exposure Measure	Outcome Measure	Findings
4-year study using a non-random clinical sample of 1,962 pregnant women without multiple gestation (36% African American) in North Carolina aged over 16 years who spoke English, 83% follow-up (Dole et al., 2003)	39-item Life Experiences Survey weighted by perception of life impact Pregnancy-related anxiety	Preterm birth (<37 weeks)[al,arh,mp,sm]	No A (all life events), PA* (life events with negative impact) PA*
6-year study using an area probability sample of 1,060 African Americans aged 65 years and over in 5 counties in North Carolina (321 men, 739 women), 47.4% follow-up (George & Lynch, 2003)	4-item life events: death of spouse/child/friend or other family member and serious illness/injury of close friend/family member	Depressive symptoms (CES-D)[a,ed,fs,s]	PA*
2-year study of an area probability sample of 279 African American high school seniors in the Boston area (132 male and 147 female), 72% follow-up rate (Gore & Aseltine, 2003)	1-item conflict with parents	Depressed mood[dr,fa,ls,ped,s] (12-item version of the CES-D)	PA*
	1-item conflict with peers	Depressed mood[dr,fa,ls,ped,s]	PA*

Study Population	Exposure Measure	Outcome Measure	Findings
1-year study of a nonrandom clinical sample of 67 HIV+ African American women aged 18-45 years in New Orleans, no follow-up rate given (Kimerling et al., 1999)	Traumatic life events measured by the 22-item Life Stressor Checklist	Lower CD4 to CD8 rati[obt]	PA*
	PTSD (DSM-IV‡) (22-item Impact of Events Scale (Revised))	Lower CD4 to CD8 ratio	PA*
3-year study involving a random sample of 204 African Americans adults (88 men and 116 women) aged 20-33 years in the Coronary Artery Risk Development in Young Adults Study, 79% follow-up (Knox et al., 2002)	Stress reactivity using a video game and mirror star-tracing task	SBP[a,al,bmi,fh,ph,SBP,sm]	PA* (men and women)
		DBP[a,al,bmi,fh,ph,SBP,sm]	PA* (women, No A (men)
		Ambulatory heart rate[a,al,bmi,fh,ph,SBP,sm]	PA*
5-year study involving a census of 555 African American students in ninth-grade (at baseline) in a school district in Michigan (301 females and 254 males), 81% follow-up rate (Sellers et al., 2003)	12-item version of the PSS	PD measured by 6-item depression and 6-item anxiety subscale of the Brief Symptom Inventory	PA*

Study Population	Exposure Measure	Outcome Measure	Findings
Nonrandom clinical sample of 1,150 pregnant women over 17 years of age from six ethnic groups in New York and Chicago (30% African American) followed from 16-28 weeks gestation till birth, no follow-up rate given (Shiono et al., 1997)	Material hardships in food, housing, medical care and from lack of money	Mean birth weight	No A
	Negative stressful life events (loss of a loved one, legal problems etc.)	Mean birth weight[a,ed,et,m,po,tm]	No A
Qualitative Studies			
Convenience sample of 314 African American women with diagnosed hypertension in south Louisiana (Boutain, 2001)	Worry and stress	Self-reported high blood pressure	Participants noted that just 'making ends meet' financially caused their elevated blood pressure
Convenience sample of 34 Anishnaabe (Ojibway), 26 women and 8 men with a mean age of 49 years, from a community near Manitoba (Garro, 1995)	Open-ended interviews	Explanations for the cause of diabetes	Worry and stress were mentioned by several individuals and there was an overall consensus that worrying too much could raise one's sugar level
Convenience sample of 14 Aboriginal adults (11 with Type 2 diabetes) aged 31-63 in a remote	Group discussions, semi-structured interviews and	Explanations for the cause of diabetes	Many participants, particularly middle-aged

Study Population	Exposure Measure	Outcome Measure	Findings
Aboriginal community in Australia (3 men and 11 women) (London & Guthridge, 1998)	informal conversations		women, considered worry about family & social problems to be the primary cause of their diabetes
Convenience sample of 12 Inuit men aged 15 to 30 years in the Arctic (O'Neil, 1986b)	Various coping styles and forms of stress including the stress of definition, isolation, transition, timing and consolidation	General Health status	The experience of stress was found to be a risk factor for increased contact with the health care system and suicidal outcomes
Convenience sample of 14 Indigenous adults from an urban and three rural areas in Australia who had suffered a myocardial infarction (10 men and 4 women) (Ong & Weeramanthri, 2002)	Open-ended unstructured interviews	Personal beliefs about the causes of heart attacks and heart disease in general	Many participants said that a stressful lifestyle was the main cause of their heart attacks

Study Population	Exposure Measure	Outcome Measure	Findings
Convenience sample of 18 African American women aged 31-49 years with HIV/AIDS in New Jersey (Owens, 2003)	Open-ended interviews	Major concerns or problems with experience of living with HIV/AIDS	Family was identified as a source of stress in relation to communication, family availability, endurance, resources, and over-estimation of their ability to cope by family members
Convenience sample of 88 low SES Aboriginal heads of households in Adelaide (72 women and 16 men) (Radford et al., 1990)	Stress	Suicide ideation Suicide attempt	PA* PA*
Convenience sample of an urban Indigenous community in Australia (Sibthorpe, 1988)	Participant observation with a focus on dietary factors	General health status	High levels of stress were evident in the community and were thought to be a more important factor than diet in the health status of the community

Study Population	Exposure Measure	Outcome Measure	Findings
Convenience sample of four case histories of Native Americans (Skinner & Silverman-Peach, 1989)	Stress caused by socio-cultural change	Self-reported diabetes Self-reported hyperglycemia	Positive associations between abrupt lifestyle changes/extreme stress and the two outcome factors
Convenience sample of 28 Anishnaabe (Ojibway) in Canada (Sunday et al., 2001)	Open-ended interviews	Explanations for the cause of diabetes	Stress was noted as a cause of diabetes by 14/28 people
Nonrandom clinical sample of patients presenting at an Aboriginal Medical Service, NRRG (Swan & Fagan, 1991)	Current stressful life events	Mental health	NA*
Convenience sample of Aboriginal Community Elders and Victorian Aboriginal Health Service in Melbourne (Thompson, 1997)	29 months of participant observation, 38 in-depth interviews, 2 focus groups, 2 small group discussions.	Financial/material and emotional worries associated with diabetes	No. of worries 0 1-4 5+ Cases 28% 33% 39% Cont. 15% 38% 46%
	Pilot questionnaire with 31 Aboriginal respondents (18 with diabetes and 13 age and sex matched controls without diabetes)	Worries included physical distance, separation, conflict, illness and death in families, young people and elders, role fulfillment and social support	Diabetes in the family Yes 29% 29% 43% No 10% 60% 30%

*p < 0.05 (two-tailed), † p < 0.10 (two-tailed). **CES-D**: Center for Epidemiological Studies-Depression, **DBP**: Diastolic blood pressure, **DSM-IV**: Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition, **HDL-C**: High density lipoprotein-C, **IRRS**: Index of Race-Related Stress, **MCS**: Mental component scale, **NRRG**: No Response Rate Given, **No A**: No association, **PA**: Positive association, **PCS**: Physical component scale, **PD**: Psychological distress, **PSS**: Perceived Stress Scale, **PTSD**: Posttraumatic stress disorder, **RR**: Response rate, **SBP**: Systolic blood pressure, **SC-90**: Symptom Checklist-90 (revised), **SF-36**: Short Form-36, **SRS**: Simple random sample, **SRRS**: Social Readjustment Rating Scale. Lettered superscripts indicate statistical adjustment for variables with abbreviations as detailed above or below: **a**: age, **al**: alcohol consumption, **ar**: antiretroviral use, **arh**: adverse reproductive history, **ba**: basis of prognosis, **bmi**: Body Mass Index, **bt**: baseline t-cell populations, **c**: coping ability, **ch**: childhood events and behaviours, **com**: complications, **cs**: No. of current sex partners, **d**: discrimination, **dd**: degree of disability, **ddi**: degree of discomfort, **di**: diabetic status, **du**: duration, **dr**: dropout status, **ds**: disease severity, **e**: extroversion, **ed**: education, **et**: ethnicity, **f**: family history of diabetes, **fa**: family's standard of living, **fh**: family history of hypertension, **fs**: family structure, **fu**: functional status, **g**: gender, **h**: hours since last meal, **ho**: household income per capita, **hp**: presence of human papillomarvirus, **hy**: hyptertensive status, **i**: income, **ls**: living situation, **m**: marital status, **me**: medicative status, **mp**: medical problems, **n**: neuroticism, **o**: overweight, **p**: ponderal index, **pa**: pain frequency, **pe**: pessimistic attitude, **ped**: parents education, **ph**: physical activity, **pi**: parenting influences, **po**: poverty level, **pt**: participation in traditional activities, **py**: pack-years of smoking, **rr**: rated tension at reading, **s**: sex, **se**: season, **scd**: sickle cell disease type, **ses**: socio-economic status, **sm**: smoking, **ss**: social support, **t**: time of event, **ti**: time of interview, **th**: threat to life, **thiv**: time since HIV diagnosis, **tm**: type of medical insurance, **w**: waist circumference, **wa**: warmth and support, **whr**: waist/hip ratio.

Table 6.2: Studies of Psychosocial Stress Interventions in Indigenous and African American Populations

Study	Trialed Intervention	Outcome Measure	Findings	
3 month single-blind RCT of 127 hypertensive African Americans aged 55 to 85 years in a primary care health centre (half of the participants were taking antihypertensive medications) (Alexander et al., 1996; Schneider et al., 1995)	T P C (lifestyle modification (nutrition and physical activity)	SBP DBP Heart rate Adjusted for baseline BP and age.	NA* (T) NA† (P) NA* (T & P) No A T was about twice as effective as P overall. Associations hold for those with high & low stress levels	
2 month RCT (blinding status unspecified) of 34 African Americans and 1 Caucasian American aged 15-18 years with resting SBP between the 85 and 95th percentile for their age and gender (Barnes et al., 2001)	T, n= 17 (8 female, 9 male) C (Health education), n = 18 (8 female, 10 male) All reported associations in comparison to control group	Resting SBP in response to (i) simulated car driving stressor; (ii) inter-personal social stressor: SBP Heart rate Cardiac output	NA* (i) NA* NA* NA*	 (ii) NA* No A No A

A trial of a historical trauma intervention with 54 Lakota Native Americans (32 female and 12 male) in the US (Brave Heart, 1999)	Brief 1 day intensive psycho-educational group experience integrated with Lakota culture and ceremonies		Before	After
		Pride	51.5%	81.8%
		Joy	45.5%	75.8%
		Sadness	66.7%	18.2%
		Grief	54.5%	27.3%
		Anger	69.7%	18.2%
		Hopelessness	45.5%	0.0%
		Helplessness	54.5%	0.0%
		Shame	60.6%	6.1%
		Guilt	60.6%	6.1%
6-9 month single-blind randomised controlled trial (RCT) of 60 hypertensive African Americans (19 men and 41 women) aged over 20 years (Castillo-Richmond et al., 2000)	T, n =31 (9 men, 22 women) HE, n = 29 (10 men, 19 women) Both groups instructed by African American practitioners	Blood pressure	NA* (T & HE)	
		Carotid intima-media thickness	NA* (T only) Adjusted for antihypertensive medication and smoking status	
1 year RCT (blinding status unspecified) of 83 African American college students in the US (39 male and 44 female) (Gaylord et al., 1989)	T P C (Cognitive-behavioural strategies)	General mental health	PA* (T & P)	
		Anxiety	NA* (T & P)	
		Neuroticism	NA* (T only)	
88 low-income elderly African-Americans (Haber, 1986)	10-week health promotion program including yoga and aerobics	Blood pressure	No A	

22 African-American hypertensive patients (Harrison & Rao, 1979)	Biofeedback relaxation	Blood pressure	No A
1 year RCT (blinding status unspecified) of African Americans with hypertension (Kondwani & Lollis, 2001)	T HE	Left ventricular mass Anxiety Depression Sleep dysfunction Energy Health locus of control	NA* (T & HE) NA* (T only) NA* (T only) NA* (T only) PA* (T only) PA* (T only)
Three to four day diabetes camps, available to both Aboriginal people with diabetes and family members in New South Wales, Australia (Lee et al., 1999)	Stress management techniques	Diabetes incidence Hospital admissions for diabetics	Decrease in incidence among non-diabetic family members 15% reduction in the number of Aboriginal diabetics being admitted to hospital
5-week trial with 65 African American working women (Mays, 1995)	Focused Support Group Model with the goals of building and enhancing group bonding cohesion, changing patterns of coping and maladaptive response strategies in the resulting situations through increased awareness of behaviours	Feelings of stress Personal support networks Professional support networks	Reduction for 40% of participants Increase for 82% of participants Increase for 60% of participants

Unspecified number of African American hypertensives (McGrady & Roberts, 1992)	Biofeedback relaxation	DBP SBP Forehead muscle tension Finger temperature	NA No A NA No A
Aboriginal women aged 40-60 years in Victoria, Australia participating in a workshop called 'Finding a balance: the next step' held in May 2000 (Moshinsky et al., 2000)	Included identifying signs of stress and strategies to promote wellbeing and discussions in small groups. A feminist approach and community development framework was utilised	Evaluation of the program	Women were extremely enthusiastic and appreciative of being able to explore these issues in a secure place
Eight year follow-up of a total of 530 African Americans (Schneider et al., 2001)	T P Lifestyle modification program	All-cause mortality Heart disease mortality	63% reduction 82% reduction T group compared to P+L groups
Empowerment program structured into four stages running for 10 weeks each with 12-31 participants and Aboriginal facilitators (more than 80% of participants were Aboriginal Australians) (Tsey & Every, 2000)	Based on the principles of psychosynthesis, analytical skills, mastery training, transformation and meditation. Participants gained a formal qualification in counselling upon completion.	Participant observation and narrative interviews	Participants reported enhanced awareness, resilience and problem solving ability

8-week non-blinded RCT of 43 African American women employed in a health service in the southeastern US (Webb et al., 2000)	P, n=22 C (30 minutes of relaxation, n=21)	Physical strain Inter-personal strain Psychological strain Vocational strain Blood pressure	NA* (P only) NA* (P only) NA* (P & R) No A (P & R) No A (P & R)

*p < 0.05 (two-tailed), † p < 0.10 (two-tailed), **C**: Control group, **DBP**: Diastolic blood pressure, **HE**: Health education, **NA**: Negative Association, **No A**: No Association, **P**: Progressive muscle relaxation, **PA**: Positive Association, **RCT**: Randomised Controlled Trial, **SBP**: Systolic blood pressure, **T**: Transcendental Meditation.

Chapter 7. **Development of and Validation of the Measure of Indigenous Racism Experiences (MIRE)**

7.1 Introduction

As detailed in Chapter 4, there has been an emerging interest in the relationship between self-reported racism and health for various ethnoracial groups. This nascent research includes numerous instruments which utilise a range of approaches to measuring racism experiences. However, as also described in Chapter 4, the conceptual bases and psychometric validity of such instruments are rarely discussed in the published literature. Furthermore, there is evidence that both single item global questions about racism (Blank et al., 2004:168), and short instruments (less than 10 items) appear to be of limited utility (see Chapter 4).

The Measure of Indigenous Racism Experiences (MIRE) questionnaire was developed in response to an identified need for an instrument which assesses the multiple facets of racism experiences, including reactions and responses to racism (Blank et al., 2004:172; Krieger, 1999:323; Krieger et al., 1993:109) and that was applicable in the Australian Indigenous context. The MIRE is a 31-item questionnaire designed to assess self-reported racism across a range of dimensions for Indigenous Australians. It appears that the MIRE is the first such instrument to be developed for an indigenous population that assesses racism together with reactions/responses to such racism.

This chapter describes the process of developing and validating the MIRE. Focus group research is first described in 7.2, including exploratory research conducted before the MIRE was drafted and confirmatory research undertaken to determine the applicability of the draft MIRE for Indigenous people. A description of the final MIRE instrument is detailed in 7.3 and a discussion of the pilot testing and validation of the MIRE in 7.4.

7.2 ***Focus group research***

7.2.1 Introduction

Following a number of other racism researchers (Green, 1995; Loo et al., 2001; Utsey & Ponterotto, 1996; Vines et al., 2001:252-3), three focus groups were conducted as an adjunct to the development of the MIRE. By gaining a more detailed understanding of the topic under consideration, focus groups enhance the face validity of questionnaire research (Wilkinson, 1998) and also act as an aid to questionnaire design by: contributing to the identification of relevant theoretical concepts; assisting in the formulation of appropriate hypotheses; and suggesting how to communicate effectively with the target population (Fuller et al., 1993).

Focus groups were chosen over in-depth interviews as they are able to create a sense of involvement for communities (especially those previously alienated from research) and are also more efficient at establishing the local credibility of a researcher (O'Brien, 1993). Focus groups have also been found to be particularly effective for participants who have historically had limited power and influence (Kidd & Parshall, 2000; Morgan & Krueger, 1993) and in relation to phenomena such as racism, for which participants may value the opportunity to share difficult experiences (Farquhar & Das, 1999).

7.2.2 Methods

Pre-existing groups of people who were known to each other were recruited in this focus group research. Although it has been suggested that such pre-existing groups should not be used as they tend to discuss a narrow set of concerns (Hughes et al., 1993), such pre-existing groups have better rapport, comfort, and trust and can be more effective due to shared experiences with similar problems or issues (Wilkinson, 1998). Only a small number of focus groups were conducted as this research was an adjunct to questionnaire research rather than as a primary method of data collection. Furthermore, it has been

suggested that saturation can occur over only three focus group interviews (Asbury, 1995; Hughes & DuMont, 1993), and, indeed, much of the results presented below were common to all three of the focus groups conducted.

A focus group guide containing a scripted introduction to the research and five topics, with sub-questions and possible probes, was utilised in this research (see Box 7.1). These topics were experiences, exposure and reactions/responses to racism, ethnoracial identity and, for the two confirmatory focus groups, comments on the draft questionnaire. Topics were sequenced from the general to the specific (Krueger, 1993), starting with an initial question which was designed to be easy to respond to for all participants (Asbury, 1995). A project information sheet and a consent form were also provided to participants (see Box 7.2 and 7.3).

There was no attempt to define 'racism' in these focus groups, as the intention was to tap into participants' concepts rather than constrain discussion to a pre-determined framework, especially as there is no "taken-for-granted criteria for determining racism" (McLeod & Yates, 2003:34). The author was the sole moderator for all three interviews and an observer who had arranged the interview was also present for focus group B. Recruitment for all three focus group interviews occurred through the author's personal and professional networks. Light refreshments were offered during focus group B as this interview was conducted over lunch and such provision has been shown to act as an incentive for participation (Asbury, 1995; Morgan, 1995).

QSR NVivo 1.3 was used to analyse all focus group interviews via a thematic approach which entailed an iterative process of analysis informed by the characterisation of racism developed in Chapter 3. In the section below, reference to individual participants is made using an alpha-numeric code consisting of the focus group they attended following by their unique single-digit identifier. Where a reference is not provided this participant was from focus group B and their identity was not available from the field notes taken (see below for details). When used as an adjunct to questionnaire development, focus group interviews do not normally require extensive analysis (Carey & Smith, 1994) and, as such, only broad findings from this research are presented below.

7.2.3 Exploratory focus group research

The development of the MIRE commenced with exploratory focus group research conducted in Sept 2001 (focus group A). This initial research included the construction of draft versions of the project summary sheet, consent form and focus group guide which would be finalised for later use in confirmatory focus group research. This initial focus group explored experiences of discrimination (including racism) for Indigenous people living in Darwin (the area in which the MIRE would later be piloted, see 7.4) and was designed to assess both the importance of racism as a form of discrimination and the salient dimensions of racism experiences for Indigenous people.

Focus group A was conducted, on site, with four Indigenous staff at a research institution in Darwin and went for 2 hours and 15 minutes, of which all but the last 15 minutes was audio recorded, with field notes taken for the last portion. One male and two females aged 25-30 years and one female around 50 years of age participated in this focus group. The majority of experiences of discrimination discussed in this pilot focus group were concerned with racism, confirming the important of this phenomenon for Indigenous people (with experiences of sexism being the next most common experience of discrimination, occupying almost all the remaining discussion). As very similar findings in relation to dimensions of racism were gleaned from both this exploratory focus group and the two confirmatory focus groups that followed, results from all three focus groups are discussed together in 7.2.5 below.

Following this exploratory focus group, existing instruments designed to measure racism in relation to health (see Chapter 4) were used, along with conceptual work in Chapter 3, as a basis for designing a draft of the MIRE. This draft instrument then underwent several cycles of review and modification involving consultation with (in chronological order) two social psychologists specialising in racism in Australia, fellow PhD students, the author's primary supervisor and other senior researchers, a workshop session at the 2002 Indigenous Researchers' Forum (Paradies, 2002), the Darwin Region Urban Indigenous Diabetes (DRUID) survey working group, the DRUID study Indigenous steering group and five

Indigenous researchers working at the Menzies School of Health Research. The instrument was then subject to confirmatory focus group research as described below.

7.2.4 Confirmatory focus group research

Two focus groups (B and C) were conducted in March 2003. Focus groups B and C were conducted with eight bureaucrats at a Commonwealth government department in Darwin and with five university students at the Northern Territory University (now Charles Darwin University) respectively. All participants were Indigenous and the focus groups, which were conducted on site at the department and university, went for 1:12 minutes and 35 minutes respectively and were audio recorded.[14] A mixture of males and females attended focus groups B and C, with one male and seven females in focus group B and two males and three females in focus group C. Focus group B participants were mostly middle-aged and focus group C participants were mostly in their 20s and 30s. Both focus groups B and C started with the moderator sharing the general flavour of his experiences of racism as an Indigenous person.

7.2.5 Focus group findings

7.2.5.1 Focus Group Process

The moderation and conduct of the focus groups went smoothly overall. There was little disagreement among participants in the interviews. Although people evinced contradictory ideas about, for instance, how to deal with racism, it appeared that these ideas were seen as personalised approaches that didn't necessarily contradict the approach of other participants or require discussion leading to a consensus on the 'correct' approach. No censoring,

[14] However, due to technical difficulties the audio recording of focus group B was not recoverable and field notes were instead taken immediately following the conclusion of the interview.

coercion or conflict avoidance was apparent in the interviews. In focus group A, the discussion moved off the primary focus of discrimination on several occasions but such a loss of focus was an acceptable trade-off for the gains made in enhancing disclosure and creating a relaxed atmosphere for all involved. Additionally, such free-ranging discussion allowed the moderator to fade into the background and allow the focus group to drive the discussion themselves. In focus group B the discussion was somewhat more directed by the moderator with most of the discussion still occurring between participants. In focus group C participants talked primarily one-to-one with the moderator, with numerous but short interjections from other participants after the first third of the interview had elapsed.

Overall it was clear the status of the moderator as an Indigenous person, which was well-known to participants in all focus groups, was very important in establishing legitimacy, rapport and trust and in creating the rich, open and honest discussion that took place in these interviews. The participants appeared to have a lot of fun in the interviews with (including the moderator) 27 occurrences of laughter in focus group A throughout the two hours of transcribed session and 29 occurrences of laughter in focus group C in only 35 minutes.[15] Also, several days after the session, participant A1 commented to me that he had found the session very therapeutic and had really appreciated being able to discuss these issues with others in the same position as him and similarly C1 said that "it has been…helpful to talk about [racism] in the focus group and to find out who [else] is Indigenous as well".

7.2.5.2 Experiences of Racism

Participants reported experiencing racism, from childhood to adulthood, across a range of different settings such as work, university or school (including their children's schools), at shops, pubs, services stations, banks (including difficulty getting loans), in taxis, when purchasing or being provided other goods and services, during sports, and on the street. Racism occurred from a range of perpetrators including police, service-providers, peers,

[15] Laughter was also common in focus group B, although it was not possible to numerically estimate its occurrence as no transcript for this interview was available.

colleagues, friends, family members and relations by marriage (such as parents of children's spouses) and was targeted at participants, their children, other family members, teachers and pets. Racism was in most cases expressed verbally or through body language and included name such as "Coons", "Abos" and "Boongs", with these terms used both as intentionally offensive as well as in ignorance of their derogatory nature. Participants also discussed expressions of being avoided due to a perceived threat, as a form of racism.

Participants reported experiencing racism at all three levels (internalised, inter-personal and systemic). Examples of inter-personal racism included being thought of and treated as stupid or incapable, being treated with less respect and courtesy than others, being treated like a child, being ignored, experiencing delayed service, being refused a refund, being treated suspiciously or considered untrustworthy, people refusing to make eye contact, being subject to stereotypes of Indigenous people as "dirty", "messy" or "drunk", being given poor grades at school, being talked to slowly and others assuming s/he spoke English poorly, being accused of "getting a free ride" and "getting help because [they are] Aboriginal", not having their skills or abilities recognised, being treated as a "token Aborigine" or as a "troublemaker" and feeling isolated among non-Indigenous people.

B1 provided an example of internalised racism in describing how she told her children not to hang around other Aboriginal children at school, just as she was told to do by her parents. When she was told by her children's teachers that these other Aboriginal children were model students, she felt guilty, but maintained that these children were "gangsters" that should be avoided. Although the term itself was not used, there were a number of other examples of internalised racism raised by participants. B2 talked about how her child wanted to be called "brown" instead of "black" while other participants described a similar instance of a child insisting that s/he was "black" but not "Aboriginal" and a different participant discussed how the father of one of her children didn't like being called "black" by his child. B7 suggested that Indigenous people pass on racist beliefs to their children.

Participants also discussed systemic racism at their workplace, expressed through low numbers of employed Indigenous people and their generally low status. "[N]ot a single program that I saw…would empower and train Indigenous people" (A4). Institutional racism against Indigenous clients of the organisation was also discussed with A4 saying

that people “don’t need to be friendly and smile or know what [Indigenous] culture is about” and A1 noting that work at the institution “doesn’t involve the well-being of [Indigenous] people”. Some participants also related historical incidents of systemic racism. B4 said she remembered having to get a pass from police in order to leave the mission where she lived and B5 said that her mum was sent to a different mission as punishment for giving birth out of wedlock.

There was a considerable range in the frequency of racist experiences with C3 saying that he experiences a lot of racism with others saying they didn’t experience racism much (B1, C4) or that it is uncommon in certain settings such as work (A2) and school (A3) but more common in others such as academia (A2, A3). C3 suggested that “it was harder when I got…older and out of school and you start to see it in, you know, real life” because there “was a lot more stereotyping,” while C2 said he “got a lot of it” at school. Also, some participants seemed to experience attributional anxiety in determining which particular types of discrimination were being experienced (i.e. was it racism, classism, ageism etc.) and C2 said that he “didn’t realise it [that he was experiencing racism] most of the time” because he didn’t learn “about it at school.”

Intra-racial racism (i.e. racism from other Indigenous people, see Chapter 3) was raised as an issue in all the interviews. A3 described experiencing racism from other Indigenous people in her workplace who thought that she “dress[ed] wrong, talk[ed] wrong, associated with the wrong people there, [and] was sucking up to the wrong people.” She described how “for a number of years I got the most dreadful discrimination from other Aboriginal people” because she didn’t “belong to any of [the local] families” and “because I came in at a higher [employment] level…”

B5, who was relatively well-educated and successful, was accused of being a “snob” by family members because they perceived her as thinking she was better than they were, while B2 (who is fair-skinned) talked about how her father didn’t want her to have a relationship with a dark-skinned Aboriginal man. There was also some discussion of Indigenous people being racist against Asians even though many Indigenous people were

"part-Asian" themselves. C3 said that he was called a "yella fella"[16] by people in the community that he grew up in because he wasn't black enough and because he was educated in a private school and C5 talked about experiencing racism at school from "full-blood"[17] Aboriginal students who swore at her and told her she was White.

In focus group B, participants discussed their own racist attitudes, stereotypes and behaviours freely. In some cases they justified their beliefs with statements such as "but those Asians win more at the casino anyway, so it's okay." In other cases, where people admitted being prejudiced against other Aborigines this was explained, although not excused or condoned, by the fact that the were taught to be like this by their parents and Euro-Australians. Participants made remarks that many would label as racist without much concern even when others responded with comments like "that sounds pretty racist." Generally participants laughed at such dialogue and certainly there was far less sensitivity to being called, or having your comments labelled, racist in comparison to middle-class liberal White Australians (see Aveling, 2004; McLeod & Yates, 2003:39).

Participants also described several natural experiments which, although not recognised as such, were conducted to test for the presence of racism. One participant told of when one of their children was in primary school and she was having trouble with her English subject. Despite hiring a tutor, her child kept getting bad marks. The tutor (who held a Master of English) become suspicious and wrote an assignment himself, submitting it with the child's name and still got a bad mark. Another participant told of a "full-blood" friend of hers who sought a refund for a car part which, although unopened and with a receipt, was refused. When the participant went in there herself with the same materials she was given a refund because, she suggested, "she has brown rather than black skin." Participants also related instances of racism being resolved and of anti-racism with B7 noting that "talking to each other can sometimes dispel racism that is otherwise a self-fulfilling prophecy". After

[16] A derogatory term used to refer to Indigenous people of mixed heritage.

[17] A term used to describe Indigenous people who have no (or very little) non-Indigenous ancestry.

witnessing White people intervening to assist Indigenous people, B6 said that her "faith in White people had been restored".

7.2.5.3 Reactions/Responses to Racism

Reactions to racism ranged from verbal confrontation, anger and violence (i.e. slapping people) to embarrassment and shock (being "stunned and…[not] know[ing] what to say" (C5)). Responses to racism included actions such as making a complaint to staff in situations where White people were served out of turn or "avoiding" the situation or people where racism occurred: "I used to miss some days at work…" (A1). Others' responses included making an "extra effort" to prove that one wasn't getting a "free ride" ("I also used to work a lot longer to try and prove that I was doing the work, the same work as them." (A1)), letting things go rather than allowing them to bother you or make you angry and seeing racism as amusing, funny or a game ("after a while you just let it slide off and you just think it's a bit of a game watching people's reaction" (A4)).

B6 thought that "talking about racism was helpful" and C3 agreed that "it's always good to talk about [racism] with someone". B5 said she always confronts racism whenever she experiences it while others, such as B6, thought it best to just ignore racism. C2 thought it was better to respond "verbally" to racism rather than "fighting and swearing" which "just makes it worse".

The consequences of racism discussed by participants included reduced self-esteem and increased self-doubt: "you actually question yourself. Am I getting free ride...Am I getting something that I shouldn't get" (A1) and feeling a necessity to conform: "Basically…I was the only Indigenous person…So you go rocking the boat you'll just end up washing dishes" (A1).

7.2.5.4 Racism and Identity

The intersection between identity as an Indigenous person and experiences of racism was evident in all the focus groups. Participants frequently reported that assumptions were made about them based on their look, manner and bearing and that Indigenous people were stereotyped as a group:

> "[W]hen I used to go out all the time" people would ask "where do you work and you say I work at the hospital" and they would ask "what do you do and I say …what do you think I do, and they said I was a cleaner" or that "I worked in the kitchen". "It's the assumption that you do nothing" (A1).

> "[W]hen you look at me you just go 'ohh she must be from somewhere overseas' and … I just find it hilarious cause it happens everywhere" (A4).

C1 said that "people don't really think that I'm part Aboriginal" and similarly C5 reported that people say "ohh she's not Black" to her friends and "people I talk to, that I just meet [say] 'ohh you don't look Aboriginal'" "because I don't have any colour." C5 also told of how she was singled out once her friends knew she was Indigenous through appending "no offence" to any comments about Aboriginal people that she may have construed as offensive.

C5 said that she tries to explain that it's about your "background and you don't have to have colour, black colour in your skin." People respond to this by suggesting that she has to "prove" that she is Aboriginal. Other university students have said to her that "Aboriginal people get [benefits]" and she feels "uncomfortable saying" that she has "Aboriginal in me" because they respond with question like "how can you say that and not prove it", "[y]ou can get all these benefits and don't have to prove it."

Participants in focus group B also made jokes about their Aboriginality, saying look at my skin I must be at least 1/8th, maybe 1/16th Aboriginal and laughing. B6 discussed how she was out with her children (who are lighter-skinned than her) and somebody thought that she was their nanny. B2 also related an experience of being denied certain services provided to Indigenous people because she didn't look Aboriginal enough.

The tendency for Indigenous people to be stereotyped as a group is illustrated in an incident in which B3 was standing in the aisle of a full bus when a middle-aged White women boarded and stared at B3. When B3 finally said "what", the woman looked at a 12 year old Aboriginal boy who was sitting nearby and said indignantly 'Well aren't you going to put him on your lap?' B3 was absolutely shocked because she didn't know the child at all but the White woman had assumed that because they were both Aboriginal they must be related. B3 didn't know what to say in response and, after looking over to the mother of the child who was farther back in the bus, took the child into her lap – leaving the White women ignorant of her erroneous assumption and the anguish it had caused B3. Similarly, one participant discussed how White people look at her as if it was her fault when other Indigenous people are causing trouble or a scene.

7.2.5.5 Feedback on the MIRE

Only one query was raised about the meaning or appropriateness of MIRE items in confirmatory focus groups B and C. In focus group C, the following exchange occurred:

> C1: "What do you mean…where do you say 'How often do you think about being Indigenous' [MIRE Q4]?"
>
> Moderator: Yeah, good question. Ahh, it's a funny question. They've been using the question over in the, in the US and, ummm, well it's just about how relevant, how relevant it is to you. Like I could just as easily say how often do you think about the fact that you're a student or the fact that you're a man or a young person, you know what I mean. Like how relevant is that part of…
>
> C1: yeah, yeah…

> Moderator: to who you are. Cause you know it can be for any reason. Like, sometimes if you just like, maybe living on remote community or something like everyone is Aboriginal…
>
> C4: You never think about it…

Although at first this question was confusing it made sense to C1 after the moderator's explanation which was completed by C4 who also clearly understood the substance of the question.

7.2.5.6 Summary of focus group research

The findings presented above demonstrate that focus group research conducted by an Indigenous researcher is effective in tapping into the experiences of racism for Indigenous people. The Indigenous undergraduate students and professionals in this study appear to experience (and be familiar with) many different forms of racism, including internalised, systemic and intra-racial racism. These participants also appear to utilise a range of responses and reactions to these experiences. This research also suggests a complex relationship between racism and identity (explored further in Chapter 8).

The participants in this research are not representative of all Indigenous people living in the Darwin area and, for instance, may not encapsulate well the experiences of Indigenous people of lower SEP. However, the considerable breadth of findings, as well as commonalities across focus groups, suggest that this research has achieved reasonable coverage of the phenomenon in question. Furthermore, the validity of using the MIRE in this population is supported by the various experiences of racism reported, the range of reactions/responses to these experiences, and by direct endorsement of the MIRE by focus group participants.

7.3 The final MIRE instrument

The final MIRE consists of six multi-part questions which assess exposure to inter-personal racism (Q1), responses (Q2a-f) and reactions to racism (Q2g-k), internalised racism (Q3a-d), systemic racism (Q3af-g) and aspects of ethnoracial identity (Q4-6). Table 7.1 details the items and scales of the final MIRE instrument along with response categories, coding, conceptual underpinnings and sources for these items/scales.

The MIRE does not include an explicit time frame for measuring exposure to racism. It is acknowledged that explicit time frames on questionnaires are necessary in order to estimate the rate of exposure to racism and to avoid confounding time-series analysis (Armstrong et al., 1992:149; Blank et al., 2004:172-3). It has also suggested that lifetime or 'ever' questions are cognitively difficult and lead to underreporting (Blank et al., 2004:172-3). However, no time period was specified in the MIRE because measuring "experiences of racism is best served by not restricting the period of time in which it occurred" given that the effects of traumatic experiences are potentially long lasting (Utsey & Ponterotto, 1996:500).

A number of other instruments that measure racism do not specify an explicit time period, including the perceptions of racism scale (Green, 1995), the telephone-administered perceived racism scale (Vines et al., 2001) and Essed's nine item measure of everyday discrimination (Essed, 1991). As in the MIRE, these instruments instead seek to measure 'current' exposure as determined by respondents. This approach also avoids the problem of 'telescoping' in which respondents report experiences that occur outside the exposure period specified in a questionnaire (Armstrong et al., 1992:24).

It has also been suggested that frequency scales which use subjective (i.e. sometimes) rather than objective (i.e. once a week) indicators may not be directly comparable with each other as individual respondents can interpret 'sometimes' in different ways (Postmes et al., 1999:336). However, it is preferable to allow respondents to estimate how much racism they experienced in relation to the amount of time they spent in a particular setting in order

to avoid differences in setting occupancy driving variations in the self-reported prevalence of racism across settings (Williams et al., 2003). For example, a once a week experience of racism at work may be rated as occurring 'very often' if the respondent only works a day a week or 'sometimes' if the respondent works full-time Furthermore, allowing respondents' to determine the extent and impact of their own personal experiences of racism is of particular importance for phenomena such as perceived racism which are fundamentally subjective experiences.

7.4 Pilot study of the MIRE

The MIRE was piloted in the DRUID study which collected data on diabetes and related conditions from people aged 15 years and over who identified as Aboriginal and/or Torres Strait Islander and who had lived (for at least six months) in a private dwelling within a specific region in and around Darwin in the Northern Territory (NT) of Australia (see Cunningham et al., 2006 and below for details). The study included a health examination that involved the collection of blood and urine samples, clinical and anthropometric measurements and the administration of questionnaires which assessed health status as well as socio-demographic, psychosocial and behavioural characteristics. Between 12 September 2003 and 31 March 2005 the DRUID study recruited a non-random sample of 1004 eligible participants who gave consent and provided at least one measurement (Cunningham et al., 2006).

However, part way through the study period, DRUID questionnaires were substantially shortened by the study chief investigators resulting in the majority of items from the MIRE being removed from these questionnaires. As such, only the first 363 eligible participants were recruited while the full MIRE instrument was included in the DRUID questionnaires. Of these 363 eligible participants, 312 completed at least some of the MIRE. This sample of 312 is the study population considered here. It should also be noted that the layout of the DRUID questionnaire resulted in MIRE Q4-6 being administered prior to and separate from the rest of the MIRE items.

7.4.1 Validation of the MIRE

7.4.1.1 Face validity

In addition to the consonance of MIRE items with the findings of focus group research described above in 7.2, MIRE items also appear to have face validity in relation to Australian research published after its development. The settings covered in Q1 of the MIRE are very similar to those reported by Koori[18] people in interview research (Mellor, 2003). Responses to racism as detailed in this research (Mellor, 2004) also appear to be very similar to those utilised in the MIRE. A scale developed to measure modern prejudice using qualitative data from surveys on Indigenous issues conducted in Perth included the following statements: (1) Aborigines should try harder to fit in with western society; (2) Aboriginal people get given more government money than they should; (3) the media is often biased against Aborigines (Pedersen et al., 2004). The first of these statements is very similar to Q3c of the MIRE whereas the other two statements are consonant with MIRE Q3e and Q3g.

Q4 was the only question queried during the pre-pilot focus group research, with one participant expressing confusion as to its meaning. Feedback from DRUID interviewers and participants suggest that this question continued to cause confusion during the pilot study. Given that Q4 had plausible patterns of association with other elements of the MIRE (see below) and that only ten participants had missing data for this item (a proportion of missing data much lower than the average for DRUID variables), it does not appear that this confusion had a marked affect on the validity of this item. However, until the reasons underlying such interpretational difficulty are uncovered this item should be interpreted with caution.

[18] Koori is a term commonly used in the southeast of Australia, particularly in the states of New South Wales and Victoria to indicate an Aboriginal person.

7.4.1.2 Psychometric validity

7.4.1.2.1 Methods

In order to determine the match between empirically derived factors and the a priori theoretical constructs detailed above, the psychometric properties of the MIRE were assessed using data from the pilot study. All coding and analyses were conducted using Stata 8.0 for Windows. For each question or scale that consisted of more than one item, intra-scale/question validity was assessed via measures of internal reliability (Cronbach's α) and, where appropriate, exploratory principle components factor analyses. The use of Cronbach's α as a measure of scale reliability is only appropriate when the items being analysed are essentially τ –equivalent, that is when each item evaluates the same underlying latent dimension in the same units (but not necessarily with the same precision) (Raykov, 2001). Although more general tests of scale reliability are available for items with differing units of measurement and precision (known as congeneric tests) (Zubrick et al., 2006), such tests are not utilised here as the items included in each test of reliability below have both a common latent dimension and the same measurement scale. Also, it has been shown that positively correlated measurement error can result in Cronbach's α overestimating the true reliability of a scale (Raykov, 2001). However, there is some debate as to whether such correlated error is a part of reliability assessment (Komaroff, 1997; Rae, 2006), and approaches to adjusting for any such correlated error (Komaroff, 1997; Zubrick et al., 2006) are beyond the scope of the analyses conducted here.

Because ordinal categories are best analysed using a weighted least squares rather than maximum likelihood procedures (Zubrick et al., 2006:14), principle components factor analysis was used instead of maximum likelihood factor analysis to determine empirical fit with underlying theoretical constructs. The use of exploratory factor analysis when more explicit model fitting could be undertaken using confirmatory factor analysis has been criticised by some (Russell, 2002). However, given that this is the first use of the MIRE and that exploratory principle components factor analysis is commonly used to assess racism instruments (Brondolo et al., 2005b; Green, 1995; Clark et al., 2004; Utsey, 1998; Utsey,

1999; Utsey & Ponterotto, 1996), this approach was considered preferable to confirmatory factor analysis which has been used in this field to assess much more well-established and widely used racism instruments (Krieger et al., 2005; Landrine et al., 2006). There is also debate as to the merit of using principle components vs. principle axis approach to factor analysis (Russell, 2002). These two approaches tend to produce very similar results with the former approach selected in this case as it assumes that all item variance is potentially explicable by the factors derived during the analysis (Russell, 2002), and so is considered more appropriate given the strong a priori theoretical basis of the MIRE in which items are designed to be fully explicable by underlying constructs.

In general, it has been noted that factor loadings and factor correlations are subject to under-estimation where ordinal variables are used (especially variables with two to three categories and non-normal distributions that vary across items) (Zubrick et al., 2006:14). Although the variables considered here are not normally distributed, they have at least five response categories and so more closely simulate continuous data. The use of polychoric or polysemic correlations to adjust for this under-estimation (Zubrick et al., 2006) is beyond the scope of this analysis and so the results presented below are conservative in the sense that the loadings and correlations reported may be under-estimated. It is also recommended that factor analyses be conducted only when the ratio of participants to items is at least five (Grimm & Yarnold, 2001). The analyses presented below meet this requirement in that no more than nine items are examined in any of the factor analyses and the sample size in these analyses is never less than 200. The Kaiser criterion was used to determine the number of factors retained (i.e. factors with eigenvalues equal to or greater than one were retained) as it is the method recommended when there are fewer than 30 items and at least 250 observations (Grimm & Yarnold, 2001) in a factor analysis. Single item constructs not considered in intra-scale analyses were Q2k and Q4-6.

For factor analysis, the full five categories of Q1-3 were retained with ‘not applicable’ responses for Q1 recoded as missing. Table 7.2 shows detailed results of factor analyses conducted on the MIRE subscales. Following factor analyses, inter-item and scale correlations were used to assess convergent and discriminant validity via cross-tabulations and chi-squared tests of association. Only tests of associations which were significant at a $p < 0.05$ level (or marginally significant ($0.05 <= p < 0.10$ as indicated) are presented below.

Significant results were reported if a consistent linear trend across the categories in questions was evident (curvilinear trends were not considered) and if no cells had zero values and no more than 20% of cells had values less than five.

7.4.1.2.2 Coding

For inter-item and scale analyses, items in Q1 and Q2 were recoded to a three-point scale (i.e. never/hardly ever: 0, sometimes: 1, often/very often: 2) and items in Q3 were re-coded to a three-point scale (i.e. strongly agree/agree: 1, neither agree nor disagree: 2, disagree/ strongly disagree: 3). An average score across the nine settings in Q1 was coded as follows: no inter-personal racism (all responses 'never': 0); low inter-personal racism (average response of 'hardly ever': 1); high inter-personal racism (average response of 'sometimes', 'often' or 'very often': 2). A composite variable consisting of the number of settings in which racism was experienced (0-9) was also coded and this variable was then partitioned into quartiles.

After reverse coding Q3c, an average internalised racism score was coded from Q3a-d as follows: no internalised racism (average response of 'strongly agree': 0); low internalised racism (average response of 'agree': 1); high internalised racism (average response of 'neither agree nor disagree' or 'disagree': 2). No participants had very high level of internalised racism (i.e. average response of 'strongly disagree').

An average systemic racism score was also coded from Q3e-g as follows: low systemic racism (average of 'strongly disagree'/'disagree': 1); moderate systemic racism (average of 'neither agree nor disagree': 2); high systemic racism (average of 'agree': 3); very high systemic racism (average of 'strongly agree': 4). Q4 was re-coded to a five-point scale (i.e. never/once a year: 0, 1/month: 1, 1/week: 2, 1/day: 3, 1/hour or constantly: 4) while Q5 and Q6 were re-coded to a four-point scale (i.e. no, hardly anybody/not many people: 0, some people: 1, yes, most people: 2, yes, everyone: 3). 'Unsure' responses for Q5 and Q6 were recoded as missing.

7.4.1.2.3 Intra-scale validity

The items in Q1 are designed to be mutually exclusive and comprehensive of the settings in which inter-personal racism may occur and, as such, are not based on one or more underlying theoretical constructs. The experience of inter-personal racism in any one of these settings does not necessarily increase the likelihood of experiencing inter-personal racism in any of the other settings. Therefore, although inter-dependence among these items is quite possible, internal reliability is not required for such a checklist and removal of items to decrease redundancy is not appropriate (Williams et al., 2003). For study participants with at least one non-missing item in Q1a-i (n=301), these items showed a good level of internal reliability ($\alpha = 0.83$) suggesting that participants who experienced inter-personal racism in one setting tend to also experience inter-personal racism in other settings. Furthermore, the addition of Q1j did not change the internal reliability ($\alpha = 0.83$), suggesting that self-reported inter-personal racism from other Indigenous people is also associated with self-reported inter-personal racism in other settings.

The internal reliability of the items assessing responses to inter-personal racism (Q2a-f) ($\alpha = 0.48$) for the 212 participants who reported experiencing some inter-personal racism and had non-missing data, suggests a number of underlying theoretical constructs for these items. As shown in Table 7.2, factor analysis of these items produced two factors with items that loaded on both of these factors suggesting that an oblique rather than an orthogonal rotation was appropriate. Factor one, which explained 30% of item variance, loaded positively on Q2a and Q2f, negatively on Q2d, Q2e and Q2c, and insignificantly on Q2b.

Both Q2a and Q2f assess passive maladaptive cognitive responses and hence would be expected to co-vary. Furthermore, the fact that Q2a and Q2f are non-adjacent items on the MIRE reduces the possibility that these findings are due to an order effect bias in which adjoining items are answered in a similar manner by respondents due to their proximity. Items Q2d-e assess outer-directed adaptive problem- and emotion-focused behavioural responses. These responses are as theoretically different from Q2a and Q2f as possible, in that they are: (1) affective/behavioural instead of cognitive; (2) outer-directed (active)

instead of passive; and (3) adaptive instead of maladaptive. As such it is appropriate that factor one loads positively on Q2a and Q2f and negatively on Q2d-e. Item Q2c assesses an inner-directed adaptive problem-focused behavioural response which for similar conceptual reasons is much more similar to Q2d-e than to Q2a or Q2f.

Factor two, which explained 56% of item variance, loaded positively on Q2b and Q2c as well as on Q2a, Q2f and Q2d but loaded insignificantly on Q2e (Table 7.2). Items Q2b-c measure inner-directed adaptive problem-focused behavioural responses and hence their correlation supports this single underlying conceptual dimension. The relatively weaker loadings on Q2a and Q2f may result from these items measuring an underlying construct with is also by definition inner-directed (i.e. cognitive) and hence somewhat related to items Q2b-c. The weak loading of Q2d on factor two may result from the fact that this item assesses an outer-directed response while items Q2b-c assess inner-directed responses. Item Q2e is even more conceptually distinct from Q2b-c in that it assesses a response to inter-personal racism which is outer- instead of inner-directed and emotion- instead of problem-focused.

For these same 212 participants, the reactions to inter-personal racism items Q2g-j were characterised by a moderate internal reliability ($\alpha = 0.55$). A factor analysis of these items produced one factor which explained 45% of item variance and loaded highly on Q2g, Q2h and Q2j but poorly on Q2i. This factor explained less than 10% of the variance of Q2i, hence supporting the conceptual divergence of this item which assesses an outer-directed empowered reaction to inter-personal racism whilst other items measured disempowered reactions to racism.

The data from the 287 participants with non-missing items in Q3a-d showed a very low degree of internal reliability ($\alpha = 0.23$). A factor analysis of these items produced two factors which explained 35% and 25% of the item variance, respectively. As these factors appeared to be orthogonal (i.e. no items appeared to load significantly on both), a varimax rotation of the factors was conducted with results indicating that factor one loaded primarily on Q3a and Q3d, both of which measure affective aspects of Indigenous identity. As shown in Table 7.2, this factor also loaded to a lesser degree on Q3b which assessed agreement with a statement of fact about the disadvantaged situation of Indigenous

Australians. While factor one did not load to any significant degree on Q3c, this item was highly loaded on factor two while other items had weak loadings with this factor. Although there was no a priori reason to expect this, it appears that the factual item (Q3b) converges with the two affective items (Q3a, d) while the normative statement is divergent and assesses a distinct construct to the other items in this subscale. The data from the 288 participants with non-missing items in Q3e-g also evinced a moderate degree of internal reliability ($\alpha = 0.47$) with a factor analysis of these items producing a single factor which explained 50% of item variance and loaded adequately on all three items in this scale (Q3e (0.57), Q3f (0.84) and Q3f (0.69)), hence supporting its unidimensional structure.

7.4.1.2.4 Inter-item and inter-scale correlations

Participants with a high inter-personal racism score were more likely to use adaptive behavioural responses (Q2c and Q2d significant at p=0.08 and p=0.03, respectively) than those who had low inter-personal racism scores. Similarly, the more settings that racism was experienced in, the less likely respondents were to try and avoid racism (Q2b) (p=0.06) and the more likely they were to talk about/express experiences of racism (Q2g) (p=0.08) and use adaptive cognitive and behavioural responses. However, the lack of variation in maladaptive cognitive responses by inter-personal racism suggests that these responses were not used any less by participants exposed to high levels of inter-personal racism. Such a finding is not contradictory because active and passive coping responses are not mutually exclusive (Vines et al., 2006), but rather complement each other in differing contexts (Lazarus, 2000). No consistent linear trends were found between Q2 items and Q1j (i.e. intra-racial racism).

Those who reported experiencing high inter-personal racism also reported experiencing all four affective (and one somatic) reactions to inter-personal racism more often than participants reporting low inter-personal racism. This finding suggests that increased exposure to inter-personal racism may lead to higher reactivity or susceptibility to the effects of inter-personal racism. Such a result is supported by other research which has found that exposure to racism is associated with heightened feelings of threat and harm from future racism experiences (Brondolo et al., 2005a).

There were no significant associations between Q3 items/composite variables and inter-personal racism score. However, the more settings that racism was experienced in, the less likely respondents were to agree that Indigenous people should try to think and act more like other Australians (Q3c) (p=0.06). Although there were no associations between Q1j and Q3b, Q3c or Q3e-f, expected associations between more frequent experiences of racism from other Indigenous people and feeling less accepted by other Indigenous people (Q3a) as well as not feeling good about being an Indigenous person (Q3d), were found. While 88% of respondents who had never/hardly ever experienced racism from other Indigenous people strongly agreed/agreed that they felt accepted by other Indigenous people, only 68% of those who often/very often experienced this form of racism strongly agreed/agreed with this statement (p=0.04). Similarly, while 95% of respondents who had never/hardly ever experienced racism from other Indigenous people strongly agreed/agreed that they felt good about being Indigenous, only 79% of those who often/very often experienced this form of racism strongly agreed/agreed with this statement (p=0.01).

As the level of inter-personal racism and settings of racism experience increased, the frequency with which participants thought about being Indigenous (Q4) also increased, with 77% of those reporting a high level inter-personal racism also reporting that they think constantly about being Indigenous while only 49% of those who experienced no inter-personal racism thought constantly about being Indigenous (p=0.01). Although the direction of causation of this association is far from clear, there is considerable research to support the finding that stronger ethnoracial identity (as variously operationalised) is related to increased reporting of racism (Beiser & Hou, 2006; Branscombe et al., 1999:137; Major et al., 2002a; Operario & Fiske, 2001:554-5; Sellers & Shelton, 2003:1087; Swim et al., 2003; Watts & Carter, 1991; Werkuyten & Nekuee, 1999). Agreeing that ‘Indigenous people have less opportunities than other Australians’ (Q3b) was also associated with increased race-consciousness (Q4) (p=0.01), while there were no consistent linear associations between level of inter-personal racism/settings of racism experienced and Q5 or Q6, or between racism from other Indigenous people (Q1j) and Q4, Q5 or Q6.

A tendency to ignore, forget about or accept racism as a fact of life (Q2a) was associated with higher internalised racism (p=0.07), and agreeing with the statement that ‘Indigenous

people should try to think and act more like other Australians' (Q3c) was associated with more frequent attempts to change the way one is or the things that one does to prevent racism (Q2c) ($p < 0.001$). Talking about or expressing racism experiences (Q2e) was associated with lower internalised racism ($p=0.04$) and higher systemic racism ($p=0.03$) as well as with agreeing that 'there is hardly ever anything good about Indigenous people in the media' (Q3g) ($p=0.07$). Similarly, those who reported that they were more likely to keep racism to themselves (Q2f) were also more likely to have higher levels of internalised racism and to agree that 'Indigenous people should try to think and act more like other Australians' (Q3c). Overall, these finding suggests that, as expected, incorporating dominant racist norms into one's worldview (i.e. internalised racism) was associated with more frequent use of maladaptive and inner-directed responses, while perceiving racism to be pervasive in society (i.e. systemic racism) was related to being more likely to talk about/express racist experiences.

More frequent angry, annoyed or frustrated reactions (Q2h) to inter-personal racism were associated with lower internalised racism in general ($p=0.06$) as well as higher agreement that 'Indigenous people have less opportunities than other Australians' (Q3a) ($p=0.07$) and that 'other Australians think they are better than Indigenous people' (Q3f) ($p=0.02$). As with responses to racism above, these findings suggest that an outer-directed disempowered reaction to inter-personal racism is more common amongst those who felt that Indigenous Australians are genuinely disadvantaged and experience pervasive racism from other Australians.

Q5 provides information on how well-known a participant's Indigeneity was within their social group, while Q6 attempts to capture how well a participant's appearance, manner and attire match with popular notions of what Indigenous people are like. Participants who 'look' Indigenous to strangers should also 'look' Indigenous to those in their social group. A cross-tabulation of Q5 and Q6 shows that, as expected, respondents who reported that most/all of the people they meet for the first time knew they were Indigenous also reported that most/all of those in their social group know they are Indigenous. Also supporting the construct validity of these items was the fact that among those participants who reported that hardly anybody/some people they meet for the first time knew they were Indigenous, those who felt more accepted by other Indigenous people (Q3a) and who thought more

about being Indigenous (Q4) were more likely to disclose their Indigeneity to their social group (Q5) (p=0.08).

7.4.1.3 Convergent validity

An assessment of the MIRE's convergent validity was possible using a question within a stress checklist administered earlier in the DRUID questionnaire. This question asked if, in the last 12 months, the participant had experienced discrimination (not specifically attributed to ethnorace), or if their family/friends had experienced discrimination that affected them personally. This question had a four-point response scale: (1) No; (2) Yes, had minimal or no effect; (3) Yes, affected me somewhat; and (4) Yes, affected me a great deal. As shown in Table 7.3, there was a high degree of concordance between any 'yes' response to this discrimination question and the summary score of inter-personal racism for those respondents with non-missing data for both the discrimination question and the inter-personal racism summary score. Only 6% of participants answered 'yes' to discrimination and reported no inter-personal racism experiences. However, there was much less concordance between 'no' responses with 64% of participants who reported no discrimination going on to report some form of inter-personal racism (see Table 7.3). Put another way, only 25% of participants answered 'yes' to the discrimination question while about 70% of participants reported some experience of inter-personal racism across the nine settings included in the MIRE.

There are a number of possible reasons for this large discrepancy in reported prevalence rates between these two apparently similar items. One possibility is that, as noted in other research (Brown, 2001; Gomez & Trierweiler, 2001), the use of ethnoracial terminology in the MIRE introductory text (see Table 7.1) increased the reporting of racism. In one study, participants were asked about both unfair treatment due to race (explicit measure) and unfair treatment in six domains later attributed to one of nine reasons including race (implicit measure) (Brown, 2001). The prevalence of reported racial discrimination was 67% when measured explicitly and 50% when measured implicitly. On the other hand, it is also possible that increased reporting of racism resulted from prompting respondents' recall via explicitly listing a range of possible settings in which racism may have occurred. The

proportion of participants reporting some level of racism in any one setting in Q1 (25-45%) is comparable to the 25% of participants who answered 'yes' to the discrimination question, suggesting that, perhaps, a single item does not cue respondents to report the full extent of their racism experiences. It is also possible that this discordance may be caused by the fact that the discrimination item assesses exposure over the past year while the MIRE includes no explicit timeframe. If this timeframe divergence was responsible for the differing prevalence rates, it would suggest that the majority of racism experiences reported by participants occurred more than a year prior to the administration of the MIRE.

Despite the lack of concordance in responses between discrimination and inter-personal racism, discrimination exposure has a pattern of association with other MIRE items/scales that is similar to those found for inter-personal racism as presented above (given that far fewer 'yes' responses would be expected to reduce the statistical power to detect such associations). Compared to participants reporting no discrimination, participants who reported experiencing discrimination were more likely to report using adaptive behavioural responses (Q2d and Q2e) and to report experiencing all three disempowered affective and one somatic reaction to inter-personal racism. Experiencing discrimination was also associated with more frequent thoughts about being Indigenous (Q4). Furthermore, the discrimination item and MIRE Q1 items had similar patterns of association with the health outcomes examined in Chapter 9. Such findings support the convergent validity of the MIRE, suggesting that discordance between the discrimination and racism items is unlikely to be due to a variation in underlying constructs.

7.4.1.4 Summary of MIRE validity

In addition to good face validity, the empirical data available from this pilot study suggest that the MIRE also has excellent intra and inter-scale construct validity. Such findings support both the comprehensive a priori theoretical and methodological basis of the instrument as well as its extensive process of development. The convergent validity for the MIRE was also supported to some degree, although further testing against other existing psychometrically-validated instruments is warranted.

It is evident that the MIRE has significant potential to be adapted for use with other indigenous and oppressed ethnoracial groups as an instrument that effectively assesses multiple facets of racism together with a diverse set of responses/reactions to this exposure. It is hoped that such a psychometrically validated instrument will assist in redressing the dearth of racism research conducted in relation to indigenous peoples around the globe who are affected by this potentially deleterious health risk.

Box 7.1: Focus Group Guide

Introduction

Thanks to everyone for coming along to talk with me today. As you know my name is Yin Paradies, I'm an Aboriginal man and have lived most of my life in Darwin. My people are from the eastern part of the NT around the Barkley tablelands and I was born in Townsville where I lived till I was 9 years old.

As I described in the information sheet this focus group discussion is part of research I am doing for my PhD which is about the health effects of racism for Indigenous Australians.

The reasoning behind this research is that recent scientific evidence from overseas has shown that experiences of racism have a negative impact on health in terms of things like high blood pressure, stress, self-esteem, anxiety among other things.

These effects may seem pretty obvious to us but from a scientific perspective investigations are only just beginning and in Australia there is very little scientific evidence about these issues.

I am doing this research because I feel that racism is experienced by a lot of Indigenous Australians and it is important that we find out what impact these experiences are having on our health and then get this information out into the public arena.

What I want to find out about are people's experiences of racism. I am trying to understand what experiences of racism, prejudice and unfair treatment are like for Indigenous people who live in the Darwin area.

The second main thing I want to talk to you about a survey I have designed about racism for Indigenous people. I want you to have a look at it and tell me what you think of the survey including any ideas for improving it.

The whole purpose of focus groups is to hear from you about your experiences, thoughts and ideas so I won't be talking the whole time, don't worry. I want you to feel free to say anything that comes to mind. There aren't any right or wrong answers, just different perspectives which I want to learn about.

I expect the discussion to last from an hour to an hour and a half and I would like to tape-record this session so I don't miss anything you say and can go back and make sure I represent it accurately as well as look at what I'm saying and see how well I can run a focus group.

All information from this research will remain strictly confidential and will be accessible only by the researcher conducting the project and nobody will be able to tell that you took part in this study from any of the reports or discussions that are produced from this project.

Topic 1: Experiences of Racism

1.1 Have you ever experienced racism? Tell us about your experiences.
[Probe: Where? From Whom? How?]

1.2 What about witnessing other Indigenous people experiencing racism? Tell us about that.
[Probe: Where? From Whom? How?]

1.3 What do you think that racism is exactly?

How easy is it to know if something that happens is due to racism?

[Probe: Treated unfairly, as if you are inferior, rudely, with disrespect, being ignored, insulted, harassed, stereotyped, discriminated against or having unfair assumptions made about you]

Topic 2: Exposure to Racism

2.1 How much do you encounter racism in your everyday life?

What about your family, friends and others?

How common do you think it is?

Is it an accepted part of life? Is it take for granted?

[Probe: What about racism when you were younger?]

Topic 3: Reactions/Responses to Racism

3.1 How do you feel when you experience racism?

How upset do you get?

What is the affect of racism on your life?

[Probe: Both physical and mental/emotional reactions]

3.2 How do you feel when you see or hear about other Indigenous people experiencing racism?

How upset do you get?

3.3 What do you do about racism?

How do you respond to racism?

What is the best way to deal with racism?

Topic 4: Identity

4.1 Are there times when you feel like it would be easier if you weren't Indigenous?

4.2 How much do people or situations make you feel that you are different because you are Indigenous?

Topic 5: The Draft Survey

5.1 Could you answer these questions for yourself? Do they make sense? Are they confusing in any way?

How would you change them to make them better?

5.2 What would be some of the difficulties for you in answering questions like this?

What would make the questions easier to answer?

Box 7.2: Project Information Sheet

RACISM, STRESS & THE HEALTH OF INDIGENOUS AUSTRALIANS

This research is being done by Mr Yin Paradies, an Aboriginal man who is doing his PhD with the Menzies School of Health Research.

Although it is clear that racism against Indigenous people occurs in Australia there is little scientific evidence of how racism affects our health as Indigenous people.

In this project I am trying to understand what experiences of racism, prejudice and unfair treatment are like for Indigenous people who live in the Darwin area.

This includes your views, perceptions, and attitudes on racism, what different experiences of racism people have and how it affects them and what you feel, think and do when you experience racism.

I will also be asking you to have a look at a survey I have designed about racism for Indigenous people and I would like you to tell me what you think of the survey.

All information collected from you for this research is **strictly confidential** and nobody will be able to tell that you took part in this study from any of the reports or discussions that are produced from this project.

I am happy to answer any questions about the research so please ask me…

My phone number at work is PH 89 227 825.

Talking through experiences of racism can sometimes be difficult and painful. If you would like to talk to a professional counsellor about any issues that are worrying you, a free counselling service is provided by Danila Dilba Medical Service. The number for this service is PH 89 361 717.

Reports of racism can also be made to the Northern Territory Anti-Discrimination Commission on 1800 81 846 or the Racism Hotline run by the Multicultural Council of the Northern Territory on 8981 4729.

If you want to talk to an independent person about this research please contact:

Ms Gabrielle Falls
Secretary
Top End Human Research Ethics Committee
PH: (08) 89 228 624

Box 7.3: Consent Form

CONSENT TO BE INVOLVED IN RESEARCH

This research is being done by Mr Yin Paradies, an Aboriginal man who is doing his studying with the Menzies School of Health Research and the University of Melbourne.

In this project I am trying to understand what the experiences of racism, prejudice and unfair treatment are like for Indigenous people, including what sort of experiences people have and how it affects them.

Today I want to learn about your experiences of racism. This includes your views, perceptions, and attitudes on racism and what you feel, think and do when you experience racism.

You should only be involved in this research if you want to be.

At any time just tell me if you don't want to be involved anymore.

If you agree to be involved our conversation will be taped so that so I don't miss anything you say and so I can go back and listen to it again. This tape and all information about you is **strictly confidential.**

Nobody will be able to tell that you took part in this study from any of the project reports.

I am happy to answer any questions about the research at any time…

My phone number at work is 89 227 825.

If you would like to be involved in this project please write your name below and also write the date and your signature.

NAME: _____

DATE: _____

SIGNATURE: _____

If you want to talk to an independent person about this research please contact:

Ms Gabrielle Falls
Secretary
Top End Human Research Ethics Committee
PH: 89 228 624

Table 7.1: The Measure of Indigenous Racism Experiences (MIRE)

MIRE introductory text

The following questions are about your experiences of being Aboriginal or Torres Strait Islander. In the questions the word 'Indigenous' is used to mean Aboriginal or Torres Strait Islander people including people who are both Aboriginal and Torres Strait Islander. Some questions ask about your experiences of 'being treated unfairly'. People can be treated unfairly for all sorts of reasons. For these questions we only want to find out about times when you were treated unfairly because you are Aboriginal or Torres Strait Islander. Being 'treated unfairly' means being treated as if you were inferior, rudely, with disrespect, being ignored, insulted, harassed, stereotyped, discriminated against or having unfair assumptions made about you.

MIRE item	Response categories	Conceptual basis	Sources
Question 1 (Q1) How often are you treated unfairly because you are Indigenous in each of the following situations? (If the situation doesn't apply to you, please tell me.) (a) At work or on the job?; (b) At home, by neighbours or at somebody else's house?; (c) At school, university or other academic setting?; (d)While doing sporting, recreational or leisure activities?; (e) By the police, security personnel, lawyers or in a court of law?; (f) By doctors, nurses or other staff at hospitals or doctors' surgeries?; (g) By staff of government agencies like Centrelink, ATSIC, etc.?; (h) By staff at restaurants, bars,	0 = Never 1 = Hardly ever 2 = Sometimes 3 = Often 4 = Very often 9 = This doesn't apply to me	Assesses inter-personal racism across nine settings as well as a non-mutually exclusive item on intra-racial racism from other Indigenous people	Krieger (1990:1274), the Telephone-administered Perceived Racism Scale (TPRS) (Vines et al., 2001:254) and the Schedule of Racist Events (SRE) (Landrine & Klonoff, 1996)

MIRE item	Response categories	Conceptual basis	Sources
shops, banks, motels, real estate agents, in taxis or when getting any other services?; (i) By other people on the street, at shopping centres, sporting events, concerts, nightclubs etc?; (j) By other Indigenous people?			
Question 2 (Q2) When you are treated unfairly because you are Indigenous how often do you: (a) Ignore it, forget about it or accept it as a fact of life?; (b) Try to avoid it in the future?; (c) Try to change the way you are or things that you do so that it won't happen again?; (d) Try to do something about the people who did it or the situation in which it happened?; (e) Talk to other people like family or friends about it, or write, draw, sing or paint about it?; (f) Keep it to yourself?; (g) Feel ashamed, humiliated, anxious or fearful?; (h) Feel angry, annoyed or frustrated?; (i) Feel amused, contemptuous or sorry for the person who did it?; (j) Feel powerless, hopeless or depressed?; (k) Get a headache, an upset stomach, tensing of your muscles, or a pounding heart?	0 = Never 1 = Hardly ever 2 = Sometimes 3 = Often 4 = Very often	Q2a and Q2f assess maladaptive passive cognitive responses. Q2b and Q2c assess adaptive inner-directed problem-focused behavioural responses. Q2d assesses an adaptive outer-directed problem-focused behavioural response. Q2e assesses an adaptive outer-directed emotion-focused behavioural response. Q2g assesses an inner-directed	Q2a and Q2d-f are based on Krieger (1990:1274) and Q2b-c on Q3 of the racism-encounter emotions and coping section of The Racism and Life Experiences Scales (RaLES-Revised) (Harrell, 1997a). The section of Q2e where respondents are asked whether they "write, draw, sing or paint" is based on Q92H from the Koori young people's health study (Victorian Aboriginal Health Service, 1999; see

MIRE item	Response categories	Conceptual basis	Sources
		disempowered active reaction. Q2h assesses an outer-directed disempowered reaction. Q2i assesses an outer-directed empowered reaction. Q2j assesses an inner-directed disempowered passive reaction. Q2k assesses possible somatic reactions to racism	also Holmes, Stewart et al., 2002). Q2g-j was informed by items in the TPRS (Vines et al., 2001:254), Q5 of the Reactions to Race Module in the US 2002 Behavioral Risk Factor Surveillance System (BRFSS) and the Perceived Racism Scale (McNeilly et al., 1996a:157). Q2k is taken directly from Q6 of the 2002 BRFSS
Question 3 (Q3) Please indicate how much you agree or disagree with each of the following statements. There are no right or wrong answers. (a) I feel accepted by other Indigenous people; (b) Indigenous people have less opportunities than other Australians; (c) Indigenous people should try to think and act more like other Australians; (d) I feel good about being an Indigenous person; (e) Other Australians think that	1 = Strongly agree 2 = Agree 3 = Neither agree nor disagree 4 = Disagree 5 = Strongly disagree	Q3a-d forms the internalised racism scale. Q3a and Q3d assess a personal affective dimension, Q3b a factual dimension and Q3c a normative dimension of	Q3a is based on Q108 from the Koori young people's health study (Victorian Aboriginal Health Service, 1999) while Q3b is based on Q11 of the Perception of Racism Scale (Green, 1995).

MIRE item	Response categories	Conceptual basis	Sources
Indigenous people are better off because they get special treatment from the government; (f) Other Australians think they are better than Indigenous people; (g) There is hardly ever anything good about Indigenous people in the media (TV, radio, newspapers, etc.)		internalised racism. Q3e-g forms the systemic racism scale which taps into common stereotypes and prejudices about Indigenous Australians	Q3c and Q3e are based on Q16 and 20 of Section 4 in Pedersen & Griffiths, 2002, Q3d is based on Q10 of the Racial Identity Attitude Scale (Helms & Parham, 1996:173) and Q3f is based on Q2 of Section four in Pedersen, Griffiths, 2002. Q3g is adapted from question 15 of the Index of Race-Related Stress (Utsey & Ponterotto, 1996:494)
Question 4 (Q4) How often do you think about being Indigenou	0 = Never 1 = Once a year 2 = Once a month 3 = Once a week 4 = Once a day 5 = Once an hour	Assesses respondents' race-consciousness (Jones, 1999)	Q4 is based on Jones (2001)

MIRE item	Response categories	Conceptual basis	Sources
	6 = Constantly		
Question 5 (Q5) Do the people that you mix with know that you are Indigenous?	0 = No, hardly anybody 1 = No, not many people 2 = Some people 3 = Yes, most people 4 = Yes, everyone 8 = Unsure	Assesses how salient (i.e. well-known) a respondents' ethnoracial identity is within that respondent's social group	Q5 is adapted from Q73 of the Koori young people's health study (Victorian Aboriginal Health Service, 1999)
Question 6 (Q6) Do people you meet for the first time know that you are Indigenous?	0 = No, hardly anybody 1 = No, not many people 2 = Some people 3 = Yes, most people 4 = Yes, everyone 8 = Unsure	Assesses how salient (i.e. well-known) a respondents' ethnoracial identity is among strangers/in public	Q6 is inspired by Q1 of the 2002 BRFSS

Table 7.2: Principle component factor analysis of MIRE Q2-3

	Factor 1	Factor 2	Item variance explained
Responses to inter-personal racism			
Q2a	0.36	0.46	0.37
Q2b	-0.06	0.76	0.57
Q2c	-0.39	0.71	0.59
Q2d	-0.78	0.28	0.63
Q2e	-0.79	-0.05	0.63
Q2f	0.46	0.55	0.57
Reactions to inter-personal racism			
Q2g	0.75	NA	0.56
Q2h	0.68	NA	0.47
Q2i	0.31	NA	0.09
Q2j	0.82	NA	0.67
Internalised racism			
Q3a	0.75	0.13	0.58
Q3b	0.51	-0.25	0.32
Q3c	0.03	0.96	0.93
Q3d	0.76	0.00	0.58
Systemic racism			
Q3e	0.57	NA	0.32
Q3f	0.84	NA	0.71
Q3g	0.69	NA	0.47

NA: Not applicable as only one factor was identified for these items

Table 7.3: Convergent validity of the MIRE

	Inter-personal racism score			Total
Self, family or friends experience of discrimination in the past 12 months[a]	0 (none)	1-9 (hardly ever)	10-36 (sometimes/ often/very often)	
No	72	106	25	203
Any 'Yes' response	5	29	36	70
Total	77	135	61	273

a Details of responses to this item are shown in Table 8.8 (stress item 16).

Chapter 8. **Prevalence and correlates of ethnoracial identity, racism and stress in the DRUID study**

8.1 Introduction

Having described the development and validation of the MIRE in Chapter 7, this chapter utilises data available from the DRUID study on the MIRE as well as items on stress and other aspects of ethnoracial identity in order to understand the prevalence and correlates of ethnoracial identity, racism and stress for Indigenous Australians in this pilot study. The same sample of 312 participants from the DRUID study as detailed in Chapter 7 is utilised in this chapter. After describing the methods of analysis used, this chapter examines the representativeness of the pilot study population in relation to both the area where the survey was conducted (in and around Darwin, NT) and the Indigenous Australian population as a whole. Following this, the prevalence of racism and stress is detailed along with the characteristics (or correlates) across which the reporting of racism and stress varies.

8.2 Methods

Appendix A contains copies of DRUID study questionnaires one and two, which, together with details of laboratory tests in Cunningham et al. (2006), show the question wording and response categories of all variables potentially available for analysis. A selection of these variables is utilised in the analyses undertaken in this section. As discussed above the MIRE was used to assess measures of self-reported racism including responses/reactions and aspects of race-consciousness and identity. Racism variables used in the analysis below were coded as detailed in Chapter 7.

In terms of socio-demographic variables, the common characteristics of age, sex and marital status are utilised together with a dichotomous variable denoted ‘household composition’ which assessed whether a respondent lived in a household consisting of only

Indigenous people, or if some non-Indigenous people lived in their household. It was thought that this variable may relate to self-reporting of ethnoracial identity or experiences of racism among respondents.

A number of markers of socio-economic position (SEP) were selected from those available in the DRUID study. Education-based measures of socio-economic status have the benefit of being stable over the adult lifespan, easy and reliable to measure and not subject to reverse causation (i.e. poor health causing poor SEP) (Dutton et al., 2005:50). Of the available DRUID variables, highest qualification appeared to be the most easily measured and interpreted as well as the most stable. In terms of income-based measured, only gross household income (in categories) was collected in the DRUID and this variable is included in analyses below after conversion to per capita income based on the number of people in each household. In response to the recognised need to include wealth-based as well as income-based measures as indicators of SEP (Dutton et al., 2005:59), housing tenure is also considered below as a marker of SEP. A question on how well respondents manage on their available income was included in the DRUID. However, this item was not used in analyses due to ambiguities of interpretation which exist in the absence of information on cash flow, accumulated assets and purchasing power. Data on employment status, occupational and subjective social status were not included in analyses due to concerns about data quality.

Several measures of ethnoracial identity collected in the DRUID are also considered in this chapter. These included questions on whether respondents: identified with a clan, tribe, or language group; recognised a homeland/traditional country; had any non-Indigenous grandparents; or were part of or a member of the 'Stolen Generations'.[19] Table 8.1 contains details on all socio-demographic, socio-economic and ethnoracial identity variables utilised in this chapter.

[19] This is a self-identified term referring to Indigenous children of mixed parentage who, between 1910 and 1970, were forcibly removed from their families by government authorities and placed in institutions or foster homes, with the intention of weakening their cultural links to Indigenous society and strengthening their affiliation to White society. The wording of this question was determined by the DRUID Indigenous Steering Group.

It has been noted that a number of factors can affect the accuracy and completeness of self-reported racism, including mode of data collection (self- or interviewer-administration), interviewer ethnorace, venue, and time of occurrence, as well as question format (e.g. explicit or implicit, phrasing, order) and context (Blank et al., 2004:169-70). Furthermore, perceived racism that is subtle or indirect may not be readily captured by explicit questions (Blank et al., 2004:169-70). In light of these previous findings, data from the DRUID study on mode of data collection (self- or interviewer-administration) is utilised in analyses below as a potential correlate of self-reported racism and stress.

All socio-demographic variables used in the analyses were coded as shown in Table 8.1. The prevalence of responses to MIRE items are detailed in Tables 8.2-8.7 below. The DRUID study also included 31 items which assessed exposure to stressors over the past 12 months. Each question was coded as shown in Table 8.8 (discussed below in relation to prevalence) and assessed either acute or chronic stressors. As the internal reliabilities of the acute stress items ($\alpha = 0.66$), chronic stress items ($\alpha = 0.83$) and all stress items combined ($\alpha = 0.86$) were adequate to good, average summary scores for acute, chronic and total stress were calculated (see Table 8.8). By collapsing all three 'yes' categories into one category and then summing these across the relevant items, three summary variables were also created for the total number of acute, chronic and total stressors experienced. These summed variables were further categorised into quartiles. Three other dichotomous variables were created to indicate whether or not participants had experienced: any stressors at all, any stressors which affected them somewhat/a great deal, and any stressors that had affected them a great deal.

The distribution of responses to the MIRE by socio-demographic characteristics is examined below via cross-tabulations and chi-squared tests of association. Unless otherwise stated, tests of associations were significant at the $p < 0.05$ level. Results were reported if a consistent linear trend across the categories in question was evident (curvilinear trends were not considered) and if no cells have zero values and no more than 20% of cells had values of less than five. All possible cross-tabulations between the socio-demographic variables in Table 8.1 were examined with significant associations reported below. The same analytical approach was used to assess the effect of method of

administration on racism/stress prevalence. Logistic regressions were also performed to check whether unadjusted associations between method administration and any racism/stress items remained after adjusting for age, highest qualification, identification with a clan, tribal or language group, recognition of a homeland/traditional country and being a member or part of the Stolen Generations (the socio-demographic factors found to relate to method of administration as detailed below).

8.3 Representativeness of the study population

Key socio-demographic characteristics of study participants are shown in Table 8.1 along with comparative information, where available, on residents of the Yilli Rreung ATSIC which includes and surrounds the city of Darwin and defines the study area (known hereafter as the Darwin area) and the Indigenous Australian population as a whole. From this information it is clear that Indigenous people in the Darwin area and Indigenous Australians overall had similar distributions of age, sex and household tenure as well as similar levels of education. However, compared to Darwin and Australia, early DRUID participants were more likely to be older and female. Members of this study population were also more likely to own or be purchasing, rather than renting, their home.

Educational qualifications of the DRUID participants considered here are not directly comparable to data from the Darwin area or Australia. This is because highest non-school qualification was assessed in the DRUID whereas the available Census and 2002-3 National Aboriginal and Torres Strait Islander Social Survey (NATSISS) data pertains to whether or not a qualification is held by the respondent rather than if it is their highest qualification. Bearing this limitation in mind, Table 8.1 suggests that the study population tended to have a higher level of education than Indigenous people either in Darwin or across Australia. As a university degree (batchelor or higher) is the highest category of qualification, data for this category are directly comparable, with 11% of the study population vs. 2% of both the Darwin Australian Indigenous population having a degree qualification.

Following advice from the DRUID Indigenous Steering Group (see Cunningham et al., 2006 for details of this group), a question which asked participants "Do you consider yourself to be part of or a member of the Stolen Generations?" was included. This item was intended to be answered in the affirmative by those who were themselves taken from their natural families as well as by those who were affected by having family members who were taken from their natural family. In order to be comparable, the Australian data on the Stolen Generations shown in Table 8.1 relates to those who had either been removed themselves and/or had relatives who had been removed from their natural family. Given this caveat it is clear that the proportion of study participants who indicated that they were part of or a member of the Stolen Generation (36%) is similar to the corresponding proportion of Indigenous Australians who also reported such in the NATSISS (38%) (Australian Bureau of Statistics, 2004). Similar proportions of study participants (77%) and Indigenous Australians (70%) also recognised a homeland/traditional country (Australian Bureau of Statistics, 2004), while a higher proportion of study participants identified with a clan, tribe, or language group (71%) compared to Indigenous Australians as a whole (54%) (Australian Bureau of Statistics, 2004).

Taken as a whole, the data in Table 8.1 show that the study population are older, more qualified, of higher SEP, more likely to be female and to identify with a clan, tribe, or language group than Indigenous Australian as a whole. As such, the limited external validity of this study should be taken into account when interpreting the data below. However, it should be noted that the external validity concerns discussed above do not impact on associations among study participants themselves, which is a function of the internal validity of a study. As noted in Chapter 4, such analysis is of particular interest given that little is currently known about variation in self-reported racism by socio-demographic characteristics.

8.4 Prevalence of self-reported racism and stress

The prevalence of responses to MIRE items and scales are shown in Tables 8.2-8.7 along with prevalence of reported stressors in Table 8.8. Overall, 70% of study participants reported experiencing inter-personal racism in at least one of the nine settings listed in Q1, between 25-45% reported experiencing inter-personal racism in each of these nine settings

and 25% of participants answered ‘yes’ to some degree in response to the question about discrimination experiences. Surprisingly, the most prevalent form of racism reported in this study population was racism from other Indigenous people (Q1j), followed closely by racism in employment (Q1a), public places (Q1i) and from services providers (Q1h) (Table 8.2). The fewest number of respondents reported racism in health care (Q1f) and domestic settings (Q1b).

There was considerable variation in reporting of reactions and responses to racism among the study population. Feeling angry, annoyed or frustrated (Q2h) was the most commonly reported reaction/response to racism, with feeling amused or sorry for the perpetrator (Q2i) and talking about/expressing racism (Q2e) also being very prevalent (Table 8.3). Trying to change oneself or one’s behaviour (Q2c) along with feeling ashamed, humiliated, anxious or fearful (Q2g) were the least prevalent reactions/responses to racism (Table 8.3).

There was a relatively high level of internalised racism reported in this pilot study with 30% of respondents having a high internalised racism score, 63% a low score and only 8% showing no indication of internalised racism (Table 8.4). The most notable finding in Table 8.4 was that a high proportion of respondents feel both accepted by other Indigenous people and good about being an Indigenous person. Recognition of systemic racism was also quite prevalent in this pilot study, with 68% of respondents reporting high/very high levels of systemic racism, 26% a moderate level and only 6% a low level of systemic racism (Table 8.5).

Results for race consciousness (Q4) are presented in Table 8.6. Further examination of these findings in the context of existing literature is presented in Table 8.10 and the discussion. Table 8.7 presents the results of Q5-Q6. Q5 provides information on how well-known a participant’s Indigeneity was within their social group while Q6 attempts to capture how well a participant’s appearance, manner, attire etc. match with popular notions of what Indigenous people ‘look’ like (i.e. how easily they are identified as Indigenous by strangers). It is likely that those who don’t have a stereotyped Indigenous ‘look’ are, to a greater degree, free to disclose, or control, knowledge about their Indigeneity among their social group. Of those participants who said that *hardly anybody* they meet for the first time knew they were Indigenous, over 75% reported that most/all of their social group knew

they were Indigenous. Of those who said that *some people* they meet for the first time knew they were Indigenous, over 90% reported that most/all of their social group knew they were Indigenous. These findings suggest a high level of Indigenous identity disclosure for study participants. Of course, given concerns about external validity above, it is not clear whether such a level of disclosure is representative of Indigenous Australians more generally. It is also of note that the DRUID study was targeted specifically at Indigenous people and hence to volunteer for this study would have required a certain level of ethnoracial identity disclosure in itself.

The prevalence of stressors in this study population also revealed some stark patterns (Table 8.8). By far the most commonly reported stressor was worry about family members (item 20) which was reported by 77% of respondents (with 36% of respondents being affected a great deal). A higher proportion of respondents experienced this chronic stressor then reported experiencing any acute stressor (76%). Other common chronic stressors included pressure to fulfill family or carer responsibilities (item 18), money (item 24) and health (item 26) problems and trouble with relationships. The death of a child, close family member or close friend (acute stressor) affected a staggering 40% of respondents over the previous 12 months (with 22% being affected a great deal). One in three respondents (34%) reported being affected by a serious illness (17% affected a great deal) and a similar proportion of respondents reported being affected by a marriage/relationship breakup (31%).

8.5 Variation in self-reported racism by method of administration

Given concerns about possible low levels of English literacy among the target population, at the outset of the DRUID study it was intended that all instruments be administered by trained interviewers. However, feedback from some participants that self-administration was preferable lead to this option being made available part-way through the study. As such, very early responders to the DRUID study (the first 58 respondents) had their questionnaires interviewer-adminstered while later participants could choose either self- or interviewer- administration of survey instruments. Of the 312 participants who completed some or all of DRUID questionnaire one, 122 self-administered the questionnaire while 190

respondents were interviewer-administered. Participants who were interviewer-administered were more likely than those who self-administered to be older, to have certificate/degree level qualifications, to identify with a clan, tribal or language, group, recognise a homeland/traditional country and report being a part of the Stolen Generations. For all items in Q1, inter-personal racism summary score and the number of settings in which racism was experienced, participants who self-administered the DRUID questionnaire were less likely to report racism then those whose questionnaires were interviewer-administered. Only 60% of self-administered participants reported experiencing some inter-personal racism compared to 79% of interviewer-administered participants (p=0.003).

Participants whose questionnaires were interviewer-administered were more likely than those who self-administered to report 'often/very often': trying to avoid racism (Q2b) (34% vs. 15%); talking about or expressing experiences of racism (Q2e) (47% vs. 26%); and have somatic reaction to racism (Q2k) (21% vs. 6%). Compared to those who self-administered , participants whose questionnaires were interviewer-administered had lower levels of internalised racism and were more likely to 'strongly agree/agree' that 'there is hardly ever anything good about Indigenous people in the media' (Q3g) (62% vs. 46%) and to 'disagree/strongly disagree' that 'Indigenous people should try to think and act more like other Australians' (Q3c) (69% vs. 52%).

Participants who were interviewer-administered had a higher acute, chronic and total stress score, reported more acute, chronic and total stressors and were more likely to report a stressor, to report one or more stressors that affected the person somewhat/a great deal and to report one or more stressors that affected the person a great deal, than those who self-administered. In logistic regressions, as described above, interviewer-administered participants were still more likely to have a higher inter-personal racism score, to report inter-personal racism in more settings, feel amused at or sorry for the perpetrator of racism (Q2i) and agree that 'there is hardly ever anything good about Indigenous people in the media' (Q3g), even after adjustment for a range of possible confounders as described above.

8.6 Socio-demographic variation in self-reported racism and stress

There were a number of associations among socio-demographic variables themselves that are noted here before going on to consider associations with racism and stress. Identifying with a clan, tribal or language group, recognising a homeland/traditional country, and being a part of the Stolen Generations were all positively associated with each other and with increasing age. Of those who identified with a clan, tribal or language group, 90% recognised a homeland/traditional country compared with 46% of those who did not identify with a clan, tribal or language group. Of those who were part of the Stolen Generations, 89% recognised a homeland/traditional and 83% identified with a clan, tribal or language group. A principle components factor analysis revealed one underlying factor which explained 53% of the variance in these three factors. Recognition of a homeland/traditional country and identification with a clan, tribal or language group loaded particularly highly on this factor which explained 66% of the variance across these two characteristics. Being part of the Stolen Generations loaded somewhat less highly, with only 27% of the variance of this characteristic being explained by this underlying construct.

A higher weekly per capita household income, higher educational qualifications, owning/buying a home and living in a mixed (Indigenous and non-Indigenous) household were strongly related characteristics for participants, with factor analysis indicating that the former two characteristics (income and education) loaded together on one factor while the latter two characteristics (home ownership and household composition) loaded on a distinct factor. Conversely, participants who lived in Indigenous only households were more likely to have no formal qualifications, only 10 years of schooling or a trade/apprenticeship. Respondents who lived in Indigenous-only households were less likely to report non-Indigenous grandparents.

Females were more likely than males to have a degree qualification and less likely to be training for, or qualified in, a trade, with those aged 30-44 having the highest educational qualifications of any age group. Being married/in a de facto relationship was associated with increased age, living in an Indigenous-only household and owning/buying a home.

Married/de facto respondents were also more likely to identify with a clan, tribal or language group, and recognise a homeland/traditional country than never married participants.

Responses to Q1 varied by a number of socio-demographic characteristics ($p < 0.05$ unless otherwise indicated). Racism at work/on the job (Q1a) was experienced more often by participants of older ages and by those who identified with a clan, tribal or language group, recognised a homeland/traditional country, or were part of the Stolen Generations. Racism in domestic (Q1b) and educational settings (Q1c) was also more frequently reported by participants who were part of the Stolen Generations while having trade or degree qualifications was associated with reporting racism in educational settings (Q1c). Higher per capita household income was associated with reporting racism in more settings.

Males experienced racism while engaged in sporting, recreational or leisure activities (Q1d) and from the police, security personnel, lawyers or in a court of law (Q1e) more frequently than females. On the other hand, females ($p=0.08$) and those participants with at least one non-Indigenous grandparent reported experiencing racism more frequently from other Indigenous people. Racism from the police, security personnel, lawyers or in a court of law (Q1e), by government agency staff (Q1f), service providers (Q1h), other Indigenous people (Q1j) as well as the overall level of inter-personal racism and number of settings of experienced racism were all positively related to identifying with a clan, tribal or language group, recognising a homeland/traditional country ($p=0.07$ for association with Q1h), and being part of the Stolen Generations.

Responses/reactions to racism evinced less variation by socio-demographic characteristics. Participants who recognised a homeland/traditional country ($p=0.06$) and were married (correlated factors) were more likely to try to do something about the perpetrator of, or the situation in which racism occurred (Q2d). Males, those from mixed households ($p=0.08$) and those with lower incomes were more likely to keep racism to themselves (Q2f). Participants who were part of the Stolen Generations were more likely to feel ashamed, humiliated, anxious or fearful (Q2g), powerless, hopeless or depressed (Q2j) and angry, annoyed or frustrated (Q2h, $p=0.06$) as a reaction to racism. Similarly, respondents who recognised a homeland/traditional country were more likely to feel powerless, hopeless or

depressed (Q2j) as a reaction to racism, while females were more likely than males to have a physical reaction (Q2k) to racism and to feel powerless, hopeless or depressed (Q2j, p=0.08) as a reaction to racism.

Participants who were younger, never married and not part of the Stolen Generations were less likely to agree (and more likely to disagree) that 'Indigenous people have less opportunities than other Australians' (Q3b). Concomitantly, those who were never married, had less qualifications, and didn't identify with a clan, tribal or language group were more likely to agree (and less likely to disagree) that 'Indigenous people should try to think and act more like other Australians' (Q3c). In a similar vein, not identifying with a clan, tribal or language group, not recognising a homeland/traditional country, being male, of younger age, never married, and earning a lower income were all associated with a higher internalised racism score.

Systemic racism tended to have an opposite pattern of association to internalised racism. Those respondents who were older, better educated (p=0.06) and identified with a clan, tribal or language group (p=0.06) were more likely to agree (and less likely to disagree) that that 'other Australians think they are better than Indigenous people' (Q3f). Similarly, older participants and those who were part of the Stolen Generations were more likely to agree (and less likely to disagree) that 'there is hardly ever anything good about Indigenous people in the media' (Q3g). A higher level of reported systemic racism, in general, was associated with increasing age (p=0.06).

A greater degree of race-consciousness (Q4) was associated with increasing age, identifying with a clan, tribal or language group, recognising a homeland/traditional country and being a part of the Stolen Generations (correlated factors). Knowledge of Indigeneity among a participant's social group (Q5) was higher for those who identified with a clan, tribal or language group, recognised a homeland/traditional country and who lived in all-Indigenous households. Knowledge of a participant's Indigeneity among those they meet for the first time was higher for males, older participants, those with less education, and those who identified with a clan, tribal or language group, recognised a homeland/traditional country and who had only Indigenous grandparents.

Reported stressors were also associated with a number of socio-demographic characteristics. When compared to males, females were more likely to have a higher acute stress score (p=0.07), experience more acute (p=0.07), chronic (p=0.08) and total stressors and report one or more stressors that affected them a great deal as well as somewhat/a great deal with younger age also related to more acute, chronic and total stressors. On the other hand, having only Indigenous grandparents was associated with reporting fewer chronic stressors and with reporting no stressors in general (p=0.08).

Participants who identified with a clan, tribal or language, group were more likely to have a higher chronic stress score, to report at least one stressor (p=0.06) and to report at least one stressor that affected them a great deal. Similarly, those who recognised a homeland/traditional country were more likely to have experienced discrimination stress (item 16), have a higher chronic stress score, to report experiencing any stressor at all as well as any stressor that affected them a great deal as well as somewhat/a great deal. Participants who reported being a part of the Stolen Generations were also more likely to have a higher acute, chronic and total stress score, to experience more acute (p=0.09), chronic, and total stressors, any stressors, and any stressor that affected them a great deal.

8.7 Discussion

This pilot study found that 70% of Indigenous participants reported some experience of inter-personal racism, with between 25-45% reporting inter-personal racism in each of the nine settings examined. Racism from other Indigenous people, in employment, in public and from service providers were the most commonly reported experiences of inter-personal racism. Outer-directed reactions/responses to inter-personal racism were more commonly reported by participants than inner-directed reactions/responses.

Respondents in the National Aboriginal and Torres Strait Islander Social Survey (NATSISS) were asked if they experienced discrimination as a stressor in the last 12 months, with over 18% country-wide answering in the affirmative (Australian Bureau of Statistics, 2004). These findings are similar to the proportion of participants reporting discrimination in the study population considered here (25%). Given that the definition of

racism used in the 2001-2 Western Australian Aboriginal Child Health Survey is most akin to a single setting from Q1 of the MIRE, the fact that 22% of the children in this survey reported experiencing racism (Zubrick et al., 2005) is also consonant with the findings from this pilot study in which 25-45% of respondents reported experiencing inter-personal racism in any one of the nine settings assessed.

Although there has been very little research conducted on intra-racial racism (see Chapter 3 for an explanation of this term), there is some support for the finding that the most prevalent form of racism reported in this study was racism from other Indigenous people. A recent US study found that 28% of African Americans and 15% of Latinos reported intra-racial racism as the most prevalent form of racism they experienced (Brondolo et al., 2005a), while 15% of 'problematic life experiences' were attributed to intra-racial racism in another study (Clark, 2004). Such findings support the importance of these experiences for Indigenous and other oppressed racial groups around the world.

The small body of published research on the prevalence of reactions and coping responses to racism is summarised in Table 8.9. These studies involve very different populations including 39 African American women college students (Clark, 2000), 42 African Americans aged 50 and older with type 2 diabetes mellitus (Moody-Ayers et al., 2005), 476 African American women from a large health plan (Vines et al., 2006) and 147 Aboriginal people in Australia (Gilles et al., 2004). Perhaps due to these varied populations as well as differences in the exact questions asked, there are no clear patterns that emerge from these studies. Although a number of studies have found that participants are more likely to use active coping responses rather than passive coping responses (Clark, 2000; Krieger, 1990; Krieger & Sidney, 1996; Noh & Kaspar, 2003; Stancil et al., 2000; Vines et al., 2006; Yen et al., 1999a;), the findings from this study population, as well as from one other study (Moody-Ayers et al., 2005), suggest that a similar proportion of participants use both types of coping responses. As noted above in Chapter 7, this latter finding is not contradictory, in that active and passive responses are complementary in differing contexts (Lazarus, 2000) rather than mutually exclusive (Vines et al, 2006).

There was a relatively high level of internalised racism reported in this pilot study, with the small amount of comparable research on this topic suggesting a relatively lower level of

internalised racism among African Americans (Caughy et al., 2004) and moderate levels among Caribbeans (Butler et al., 2002; Chambers et al., 2004; Tull et al., 1999; Tull & Chambers, 2001). Systemic racism was also quite prevalent in this pilot study but the plethora of approaches to measuring this construct in previous research precludes useful comparison with other study populations. It is notable that a high proportion of respondents feel both accepted by other Indigenous people and good about being an Indigenous person and that there is a high level of Indigenous identity disclosure for study participants.

Several patterns emerged in relation to socio-demographic variation in self-reported racism. Identifying with a clan, tribal or language group, recognising a homeland/traditional country and being part of the Stolen Generations (all co-related characteristics), older age and higher SEP were associated with increased inter-personal and systemic racism, reduced internalised racism and a greater degree of race-consciousness. The first three related characteristics were also associated with reporting more frequent reactions to racism as well as increased knowledge of a participant's Indigeneity. Identifying with a clan, tribal or language group, recognising a homeland/traditional country, being part of the Stolen Generations as well as being female and of younger age were also all associated with higher reported stress. Given that these three characteristics appear to capture aspects of ethnoracial identity salience, their strong association with racism and stress is consonant with the finding, as noted above, that stronger ethnoracial identity is related to increased reporting of racism (Beiser & Hou, 2006; Branscombe et al., 1999:137; Major et al., 2002a; Operario & Fiske, 2001:554-5; Sellers & Shelton, 2003:1087; Swim et al., 2003; Watts & Carter, 1991; Werkuyten & Nekuee, 1999).

As found in several other studies (see Chapter 4), there was evidence of some variation by gender in self-reported inter-personal racism and responses/reactions to racism. Method of administration was significantly associated with racism and stress prevalence even after adjustment for a range of socio-demographic as detailed above. Although it is not clear why this is so, given these findings, method of administration will be retained as a potential confounder in analyses conducted in Chapter 9.

Of respondents in this pilot study, 56% reported constantly thinking about being Indigenous. The item on which Q4 (race-consciousness) was based was worded: 'how often

do you think of your race?,' with the response categories as in the DRUID (Jones, 1999). Prevalence of race-consciousness using this original question is available for Indigenous people in Western Australia (Gilles et al., 2004), African American women (Jones, 1999; Vines et al., 2006), White, Asian, and Hispanic women (Jones, 1999) and also for New Zealand Pakeha and Maori (those who identify exclusively as Maori as well as those who also identify with other social identities – i.e. mixed-Maori) (Jones, 1999). Indigenous Western Australians, mixed-Maori, exclusive Maori and members of the study population considered here all reported thinking about their race to a similar degree (see Table 8.10). Of those populations not included in Table 8.10, no more than 23% reported thinking about their race constantly (Jones, 1999; Vines et al., 2006). As shown in Table 8.10, all DRUID participants combined had a pattern of race-consciousness that was most similar to mixed-Maori. Those with an exclusive-Maori identity had a higher degree of race-consciousness. However, Table 8.10 shows that those DRUID participants who recognised a homeland/traditional country or identified with clan, tribal or language group had a higher degree of race-consciousness than other members of the study population. Those who reported being part of the Stolen Generations had a pattern of race-consciousness that approached that reported by exclusive-Maoris (Table 8.10), suggesting that membership in this group is consonant with a strong ethnoracial identity.

There were a number of differences between this pilot study and the study in Western Australia that could explain their somewhat different findings. The Western Australian study was a representative survey conducted in a rural area that involved a mix of Indigenous and non-Indigenous respondents while this pilot study was a non-representative survey undertaken in an urban setting which involved only Indigenous respondents. Furthermore, the original wording of this question was utilised in the Western Australian study (how often do you think of your race?) while an adapted version was used in the DRUID study.

This chapter has explored the prevalence and correlates racism and stress among a subset of participants in the DRUID study. Given the relatively high prevalence of racism and stress found in this study population, it is important to also consider the impact that exposure to these phenomena has on the health and well-being of Indigenous people. An examination of this topic, using data from the DRUID study, is undertaken in the following chapter.

Table 8.1: Comparative demographic characteristics of the study population

	N	Study population (%)	Yilli Rreung ATSIC region (%)	Total Australian Indigenous population (%)
Sex				
Male	99	32	49[b]	50[a]
Female	213	68	51[b]	50[a]
Age (excluding those < 15 years)				
15-24	52	17	30[b]	30[a]
25-34	59	19	27[b]	26[a]
35-44	70	22	21[b]	20[a]
45-54	76	24	13[b]	13[a]
55-64	32	10	6[b]	6[a]
65+	23	7	4[b]	5[a]
Marital status				
Never Married	76	25	-	-
Married/de facto	161	53	-	-
Separated/divorced/ widowed	69	23	-	-
Gross weekly per capita household income ($)				
0-99	53	21	-	-
100-199	67	26	-	-
200-299	41	16	-	-
300-399	52	20	-	-
400+	44	17	-	-
Household tenure				
Rented	155	52	64[b]	63[a]
Owned/being purchased	142	48	36[b]	32[a]
Household composition				

	N	Study population (%)	Yilli Rreung ATSIC region (%)	Total Australian Indigenous population (%)
All household members Indigenous	197	63	-	-
Household members a mix of Indigenous and non-Indigenous	115	37	-	-
Qualification (for those >= 15 years)		**Highest qualification (%)**	**% who held qualification**	**% who held qualification**
No formal qualification	91	31	45[b]	45[a]
Year 10	40	13	25[b]	29[a]
Year 12	27	9	20[b]	18[a]
Trade/apprenticeship	16	5	-	-
Certificate/diploma	91	31	8[b]	12[a]
Degree	32	11	2[b]	3[a]
Part of or a member of the Stolen Generations				
Yes	106	36	26	38[c]
No	191	64	74	62[c]
Identify with a clan, tribe, or language group				
Yes	218	71	-	54[c]
No	90	29	-	46[c]
Recognises homeland/traditional country				
Yes	238	77	-	70[c]
No	71	23	-	30[c]
Any non-Indigenous				

	N	Study population (%)	Yilli Rreung ATSIC region (%)	Total Australian Indigenous population (%)
grandparents				
Yes	204	69	-	-
No	91	31	-	-

Note: N may not add to 312 due to missing values; percentages may not add to 100% due to rounding.

a Data from Australian Bureau of Statistics, 2003.

b 2001 Census data available from www.ausstats.abs.gov.au

c Data from Australian Bureau of Statistics, 2004.

Table 8.2: Prevalence of self-reported racism across nine settings and intra-racially (n=301)

How often are you treated unfairly because you are Indigenous in each of the following situations?	Never/hardly ever %(n)	Sometimes %(n)	Often/very often %(n)
a. At work or on the job?	74 (212)	20 (58)	6 (16)
b. At home, by neighbours or at somebody else's house?	88 (260)	9 (28)	3 (9)
c. At school, university or other academic setting?	84 (238)	11 (32)	5 (13)
d. While doing sporting, recreational or leisure activities?	83 (241)	15 (44)	1 (4)
e. By the police, security personnel, lawyers or in a court of law?	83 (241)	14 (40)	4 (11)
f. By doctors, nurses or other staff at hospitals or doctor's surgeries?	89 (263)	10 (28)	1 (3)
g. By staff of government agencies like Centrelink, ATSIC, etc.?	82 (242)	14 (41)	4 (11)
h. By staff at restaurants, bars, shops, banks, motels, real estate agents, in taxis or when getting any other services?	70 (209)	25 (73)	5 (15)
i. By other people on the street, at shopping centres, sporting events, concerts, nightclubs etc?	72 (213)	23 (69)	4 (13)
j. By other Indigenous people?	67 (200)	23 (70)	9 (28)
Total inter-personal racism score	*None %(n)*	*Low %(n)*	*High %(n)*
	28 (77)	50 (136)	22 (61)
# of settings in which racism was experienced[a]	*1-2 settings* 29%	*3-5 settings* 23%	*6-9 settings* 18%

Note: There were 301 participants with at least one non-missing item in Q1. The number of non-missing responses for items a-j varying from n=283 to n=298. Percentages may not add to 100% due to rounding.

a 30% of respondents did not report experiencing racism in any of the nine settings examined.

Table 8.3: Responses/reactions for participants who experienced self-reported racism (n= 221)

When you are treated unfairly because you are Indigenous how often do you:	Never/ hardly ever %(n)	Sometimes %(n)	Often/very often %(n)
a. Ignore it, forget about it or accept it as a fact of life?	32 (70)	41 (88)	27 (58)
b. Try to avoid it in the future?	38 (82)	34 (73)	28 (61)
c. Try to change the way you are or things that you do so that it won't happen again?	65 (139)	18 (39)	17 (37)
d. Try to do something about the people who did it or the situation in which it happened?	34 (73)	40 (85)	27 (57)
e. Talk to other people like family or friends about it, or write, draw, sing or paint about it?	28 (61)	31 (67)	40 (87)
f. Keep it to yourself?	54 (116)	24 (52)	22 (46)
g. Feel ashamed, humiliated, anxious or fearful?	65 (139)	26 (56)	8 (18)
h. Feel angry, annoyed or frustrated?	19 (41)	42 (88)	39 (83)
i. Feel amused at or sorry for the person who did it?	27 (58)	43 (91)	30 (65)
j. Feel powerless, hopeless or depressed?	59 (127)	29 (62)	12 (25)
k. Get a headache, an upset stomach, tensing of your muscles, or a pounding heart?	55 (118)	28 (60)	17 (36)

Note: There were 221 participants who reported experiencing racism (hardly ever, sometimes, often or very often) in at least one item in Q1. The number of non-missing responses for each item a-k varies from n=212 to n=216. Percentages may not add to 100% due to rounding.

Table 8.4: Prevalence of self-reported internalised racism (n= 296)

Please indicate how much you agree or disagree with each of the following statements:	Strongly agree/ Agree %(n)	Neither agree nor disagree %(n)	Disagree/ Strongly disagree %(n)
a. I feel accepted by other Indigenous people.	85 (249)	12 (34)	3 (10)
b. Indigenous people have less opportunities than other Australians.	71 (210)	14 (40)	15 (44)
c. Indigenous people should try to think and act more like other Australians.	16 (47)	21 (61)	63 (183)
d. I feel good about being an Indigenous person.	93 (271)	2 (7)	4 (13)
Total internalised racism score	*None*	*Low*	*High*
	8 (23)	63 (180)	30 (85)

Note: There were 296 participants with at least one non-missing item in Q3. The number of non-missing responses for items a-d varying from n=291 to n=294. Percentages may not add to 100% due to rounding.

Table 8.5: Prevalence of self-reported systemic racism (n= 296)

Please indicate how much you agree or disagree with each of the following statements:	Strongly agree/Agree %(n)	Neither agree nor disagree %(n)	Disagree/ Strongly disagree %(n)	%(n)
e. Other Australians think that Indigenous people are better off because they get special treatment from the government.	50 (144)	14 (40)	37 (106)	-
f. Other Australians think they are better than Indigenous people.	67 (194)	21 (61)	12 (35)	-
g. There is hardly ever anything good about Indigenous people in the media (TV, radio, newspapers, etc.).	56 (164)	22 (63)	22 (65)	-
Total systemic racism score	Low	Moderate	High	Very High
	6 (18)	26 (74)	48 (138)	20 (58)

Note: There were 296 participants with at least one non-missing item in Q3. The number of non-missing responses for items e-g varying from n=290 to n=292. Percentages may not add to 100% due to rounding.

Table 8.6: Race-consciousness (n= 302)

	Never %(n)	Once a year %(n)	Once a month %(n)	Once a week %(n)	Once a day %(n)	Once an hour %(n)	Constantly %(n)
How often do you think about being Indigenous?	13 (41)	4 (13)	6 (19)	8 (25)	9 (28)	2 (6)	56 (170)

Note: There were 302 participants with a non-missing value for Q4. Percentages may not add to 100% due to rounding.

Table 8.7: Social group/stranger knowledge of Indigenous identity (n= 310)

	Yes, everyone %(n)	Most people %(n)	Some people %(n)	No, not many people/ hardly anybody %(n)
Do the people that you mix with know that you are Indigenous?	64% (197)	30% (94)	4% (11)	3% (8)
Do people you meet for the first time know that you are Indigenous?	31% (87)	31% (89)	23% (65)	15% (43)

Note: There were 311 participants with a non-missing value for Q5 and 294 participants with a non-missing value for Q6. One participant who answered 'unsure' to Q5 and ten participants who answered 'unsure' to Q6 are not included in this table. Percentages may not add to 100% due to rounding.

Table 8.8: Prevalence of stressors over the past 12 months

Events occurring to respondent or their family/friends that affected them personally	No (%)	Yes, had minimal or no effect (%)	Yes, affected me somewhat (%)	Yes, affected me a great deal (%)
Acute stressors (explained as one-off, sudden or unexpected events)				
1. Marriage or relationship breakup	69	8	13	11
2. Death of husband / wife or partner	92	1	3	4
3. Death of a child, close family member or close friend	60	7	11	22
4. Laid off or fired at work	95	2	1	2
5. Witness to violence	76	7	9	8
6. Victim of abuse or crime	82	4	8	6
7. Member of family sent to jail	85	4	5	5
8. Serious illness	66	5	12	17
9. Serious accident or injury	86	1	5	8
10. Other stressful events	86	2	3	8
Total average acute stressor score	23%	65%	11%	0%
# of acute stressors experienced	*0 stressors*	*1 stressors*	*2 stressors*	*3-10 stressors*
	24%	26%	18%	*3-11* 32%
Chronic stressors (explained as regular or continuing events)				

Events occurring to respondent or their family/friends that affected them personally	No (%)	Yes, had minimal or no effect (%)	Yes, affected me somewhat (%)	Yes, affected me a great deal (%)
11. Trouble at work or with a boss or co-worker	68	10	15	7
12. Trouble at school or with a teacher	84	7	5	4
13. Trouble with relationships	60	14	16	10
14. Alcohol or drug-related problems	75	8	7	10
15. Overcrowding at home	86	6	4	4
16. Discrimination	75	10	8	7
17. Family members in prison	87	4	3	6
18. Pressure to fulfill cultural responsibilities	75	8	11	6
19. Pressure to fulfill family or carer's responsibilities	56	12	18	14
20. Worry about family members	23	15	26	36
21. Having to rely on other people for assistance with everyday needs	79	10	7	3
22. Problems with access to land/country	85	6	4	5
23. Gambling problems	84	7	5	5
24. Money problems	55	18	17	10
25. Mental or emotional problems	66	14	12	8
26. Health problems	54	13	18	15
27. Problems finding a job	85	4	6	5
28. Problems settling into a new job	93	2	3	2

Events occurring to respondent or their family/friends that affected them personally	No (%)	Yes, had minimal or no effect (%)	Yes, affected me somewhat (%)	Yes, affected me a great deal (%)
29. Abuse or violence	86	4	5	6
30. Problems with the police or the law	88	4	4	4
31. Other troubles	92	1	3	4
Total average chronic stressor score	6%	82%	11%	0%
# of chronic stressors experienced	*0-1 stressors*	*2-4 stressors*	*5-7 stressors*	*8-20 stressors*
	18%	29%	28%	25%
Total average stressor score	5%	85%	10%	0%
# of total stressors experienced	*0-2 stressors*	*3-6 stressors*	*7-10 stressors*	*11-31 stressors*
	19%	31%	25%	26%

Note: Only participants with non-missing value reported. Percentages may not add to 100% due to rounding.

Table 8.9: Studies of responses/reactions to racism

Any reported use of this response/reaction (i.e. hardly ever/sometimes/often/very often for DRUID respondents)	DRUID study population (%)	Clark, (2000) (%)	Moody-Ayers et al. (2005) (%)	Vines et al. (2006) (%)	Gilles et al. (2004) (%)
a. Ignore it, forget about it or accept it as a fact of life?	82	-	-	-	-
Ignore it	-	55	-	-	-
Forget about it	-	40	-	-	-
Accept it as a fact of life	-	40	-	-	-
b. Try to avoid it in the future?	68	23	62	-	-
c. Try to change the way you are or things that you do so that it won't happen again?	44	-	-	-	-
Try to 'change things'	-	68	48	-	-
d. Try to do something about the people who did it or the situation in which it happened?	75	-	-	-	-
e. Talk to other people like family or friends about it, or write, draw, sing or paint about it?	82	88	69	-	-
f. Keep it to yourself?	64	-	52	-	-
g. Feel ashamed, humiliated, anxious or fearful?	48	-	-	-	-
Feel ashamed	-	-	33	-	-
h. Feel angry, annoyed or frustrated?	87	-	-	-	
Feeling angry	-	-	71	-	-

Feeling frustrated	-	-	60	-	-
i. Feel amused at or sorry for the person who did it?	80	-	-	-	-
j. Feel powerless, hopeless or depressed?	60		-	-	-
Feel powerless	-	-	52	-	-
Feel hopeless/powerless	-	-	-	79	-
Feel sad	-	-	57	-	-
k. Get a headache, an upset stomach, tensing of your muscles, or a pounding heart?	49	-	-	-	-
Feel physical stress	-	-	-	-	35

Table 8.10: Studies of race-consciousness

	Never %	Once/ year %	Once/ month %	Once/ week %	Once/ day %	Once/ hour %	Constantly %
Aboriginal respondents in Western Australia (Gilles et al., 2004)	31	6	3	8	4	1	42
Mixed Maori (Jones, 1999)	4	0	4	9	22	7	53
All DRUID participants (n=302)	13	4	6	8	9	2	56
DRUID participants who recognised a homeland/traditional country (n=231)	12	4	5	6	9	3	61
DRUID participants who identified with clan/ tribal/language group (n=210)	11	3	5	7	7	2	65
DRUID participants who were part of the Stolen Generations (n=102)	8	4	2	10	4	1	72
Exclusive Maori (Jones, 1999)	2	2	1	5	9	5	77

Note: Only DRUID participants with non-missing value reported. Percentages may not add to 100% due to rounding.

Chapter 9. ***Associations between ethnoracial identity, racism, stress and health-related measures in the DRUID study***

9.1 Introduction

This chapter examines associations between racism, ethnoracial identity, stress and a range of health outcomes using data from the DRUID pilot study. The primary purpose of this section is to determine whether the same patterns of association between racism and health found in the broader literature (see Chapter 4) are evident for Indigenous Australians who responded to the MIRE in this pilot study, including an initial assessment of the mediation/confounding of such relationships. The pilot study is also used to elucidate the relationship between racism and stress by determining if racism has differing associations with acute vs. chronic stress or with summary stress scores (weighted by impact) vs. number of stressors experienced. In addition, this section builds on work in Chapter 6 in investigating the association between stress and chronic conditions (or their markers) for Indigenous Australians.

9.2 Methods

9.2.1 Data variables and coding

A range of health-related measures and potential covariates, which together cover the main topics on which data was collected in the DRUID pilot study, are analysed in this section (see Appendix A for details on question wording and response categories). Health-related behaviours (smoking status, alcohol consumption, physical activity and junk food consumption) as well as self-report data on depressive symptoms as assessed by the Centre for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) are considered below. Also examined are self-assessed health status and general mental and physical

health from the Short Form 12 (SF-12) (Ware et al., 1995) as well as a number of psychosocial variables (optimism, lack of control, positive social capital (also known as emotional social support), negative social capital, positive instrumental social support and level of social isolation).

A range of physical health outcomes, for which full details on measurement can be found in Cunningham et al. (2006), are also utilised in analyses below. These consist of anthropometric characteristics (waist circumference, waist-to-hip ratio, systolic and diastolic blood pressure); physiological measurements (glycosylated haemoglobin (HbA1c) as a measure of glucose homeostasis (Sargeant, 2000), hypertension, dyslipidaemia, high sensitivity C-reactive protein, fibrinogen and homocysteine as long-term markers of CVD risk (Cummings et al., 2006; Kannel, 2005)); and a measure of dental health. As detailed below, two composite variables were also included. These were (1) chronic conditions suffered by respondents and (2) a measure of allostatic load. All coding and analyses were conducted using Stata 8.0 for Windows.

Racism and stress variables were coded as detailed in Chapter 7 with reduced three-point scaled versions of racism variables being utilised. Health behavior variables were dichotomised as smoker/non-smoker,[20] no/low risk vs. moderate/high risk alcohol consumption, sufficient vs. insufficient (which includes sedentary) physical activity, sufficient vs. insufficient fruit and vegetable consumption and high/low consumption of junk food. Levels of alcohol consumption risk were based on Australian National Health and Medical Research Council (NHMRC) guidelines (2001:6-7), while physical activity was coded as detailed in Active Australia Survey (Australian Institute of Health and Welfare, 2003). Based on the NHMRC (2003:3) dietary guidelines, at least five serves of vegetables and two serves of fruit per day were considered a sufficient intake of fruit and

[20] Preliminary analyses revealed smoking status coded as never vs. ever smoker had much weaker associations with predictor variables than smoking status coded as current smoker vs. non-current smoker and hence the latter coding scheme was adopted when considering smoking as an outcome variable. When considered as a potential confounder, smoking was coded and analyses separately using both of the above coding schemes.

vegetables, while a high intake of junk food was defined as the daily consumption of more than two serves of any combination of take away/snack food, flavoured milk or soft drinks (the relevant data items available in the pilot study).

The CES-D is a 20-item self-report depression scale with each item having a response scale scored from 0-3, giving an overall scale score range of 0-60. This variable was analysed as a dichotomous variable with a score of 16 or above indicating the presence of depressive symptoms (referred to as depression below) (McDowell & Newell, 1996:255). Self-assessed health status was also dichotomised into poor/fair health vs. good/very good/excellent self-reported health. The full SF-12 component scores were analysed as continuous variables using linear regression. The distribution of SF-12 mental component scores (MCS) and physical component scores (PCS) suggested a reasonably normal distribution and sensitivity analysis comparing results for raw scores vs. a squared transformation (the transformed distribution closest to normality) produced very similar results and hence analysis was performed using untransformed scores.

All analyses of mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) excluded the 54 respondents who reported taking anti-hypertensive medication. Both SBP and DBP were marginally normally distributed and were treated as non-normal continuous variables in unadjusted analysis below. However, as for SF-12 scores, sensitivity analyses indicated no benefit in undertaking a reciprocal transformation (the transform closest to normality) and adjusted analysis was performed using untransformed scores. Hypertension was defined as SBP >= 140 mmHg or DBP >=90 mmHg. Dyslipidaemia was considered to be present if any of the following conditions were met: triglycerides >= 2 mmol/L, total cholesterol >= 5.5 mmol/L, low density lipoprotein >= 3.5 mmol/L, high density lipoprotein <=1 mmol/L. These variables were defined solely using clinical criteria in recognition of possible confounding between self-reported exposure and outcomes variables as discussed in Chapter 4.

Waist circumference and waist-to-hip ratio (WHR) were used instead of BMI as these measures appear to be better proxies of cardiovascular disease (CVD) risk for Indigenous populations (Wang & Hoy, 2004). As WHR was only marginally normally distributed it was treated as a non-normal continuous variable in unadjusted analysis below, while WHR

was retained in its raw form for adjusted analyses after sensitivity analyses found no advantage in transforming this variable.

Homocysteine, high sensitivity C-reactive protein (CRP) and fibrinogen were all non-normally distributed and no transformation of these variables produced a normal distribution. As there are no recognised cut point values for either homocysteine or fibrinogen as long-term markers of CVD risk, all analyses were conducted with homocysteine and fibrinogen dichotomised along their medians into low and high levels (< 9.7 umol/L and >= 9.7 umol/L for homocysteine and < 3.8 g/L and >= 3.8 g/L for fibrinogen). Using an established cut point value (Bonora, 2006), CRP was dichotomised as < 3 mg/l and >= 3 mg/l. In this pilot study, as with other indigenous populations (Best et al., 2005), there was a particularly high median of CRP (3.4 mg/l) among participants which produced a fairly equal split along this cut point value of 3 mg/l.

HbA1c was selected as a measure of glucose homeostasis (Sargeant, 2000) which has been found to be related to stress among indigenous populations (Daniel et al., 1999). The analysis of HbA1c was restricted to those respondents who had laboratory confirmed normal blood glucose (fasting glucose < 6.1 mmol/L and 2-hour glucose tolerance <7.8 mmol/L) and who reported that they were not on medication for diabetes. For this group of 177 participants, HbA1c was normally distributed and was analysed as a normal continuous variable.

The item used as a measure of dental health assessed the level of gum/teeth pain or discomfort in the last 12 months. This variable was dichotomised into no pain/discomfort vs. some pain/discomfort for analysis.

An indicator of allostatic load was calculated as the number of the following ten characteristics for which each participant fell into the highest-risk quartile (see Seeman et al., 1997): systolic blood pressure, diastolic blood pressure, WHR, total and HDL cholesterol, triglycerides, HbA1c, homocysteine, fibrinogen and CRP. As neither a Gaussian nor a Poisson distribution matched the distribution of allostatic load, all analyses were conducted with allostatic load dichotomised along the median into low (<= 2 chronic

conditions in the highest quartile) and high levels (> 2 chronic conditions in the highest quartile).

The number of chronic conditions that affected a respondent was calculated for ten conditions via a combination of self-report and clinical/laboratory measurement. History of heart attack, angina, other heart disease, stroke, kidney disease, emphysema/chronic bronchitis, asthma and cancer were self-reported by respondents. Hypertension was considered to be present if self-reported, if medication use for this condition was reported and/or if SBP >= 140 mmHg or DBP >=90 mmHg for a respondent. Dyslipidaemia was considered to be present if self-reported, if medication use for this condition was reported and/or if triglycerides >= 2 mmol/L, total cholesterol >= 5.5 mmol/L, low density lipoprotein >= 3.5 mmol/L or high density lipoprotein <=1 mmol/L for a respondent.[21] As neither a normal nor a Poisson distribution fit this composite variable, all analyses were conducted with number of chronic conditions dichotomised along the median into low (<= 2 chronic conditions) and high levels (> 2 chronic conditions).

As with allostatic load and number of chronic conditions, neither a Poisson nor a normal distribution fit the six stress outcome variables considered here. As such, acute and chronic stress scores (i.e. weighted by impact) were converted into dichotomous variables by splitting respondents along the median of each respective stress score (<= 3 and > 3 for acute stress and <= 9 and > 9 for chronic stress). Similarly, the number of reported acute and chronic stressors were split along the median into high and low levels (< 2 and >= 2 for acute stressors and <= 5 and > 5 for chronic stressors). Converting stress scores into binary variables which indicated whether or not acute or chronic stressors had been experienced at all was also investigated as a possible coding scheme. However, as only 6% of participants reported not experiencing a chronic stressor, a lack of variability in this outcome and hence lack of comparability between acute and chronic stress precluded this coding approach. The

[21] In calculating allostatic load, hyptension and dyslipidaemia were defined using self-report and clinical measures. This is in contrast to the definition of hypertension and dyslipidaemia as separate variables above in which only clinical measures were utilised in defining these conditions.

two dichotomous stress outcome variables (experience of a stressor that affected respondent somewhat/a great deal or a great deal, respectively) were retained in their dichotomous form.

Optimism was assessed using the Life Orientation Test-Revised (see DRUID questionnaire 2, section 8) and was coded as detailed in Scheier et al. (1994). Lack of control was coded and analysed as two separate variables. The first categorised average summary score of the six variables in this scale. The second, categorised average summary score of the four variables in this scale that applied to all respondents (by excluding items on 'job security' and 'work life'). Positive and negative social capital were coded as a categorised average summary score of items 2-7 and items 8-12 of DRUID Questionnaire 2, section 7, respectively (see Appendix A). Positive instrumental social support was coded as a categorised average summary score of items 14-18 of this same section of Questionnaire 2 and level of social isolation dichotomised as very/somewhat isolated vs. not very isolated at all.

Variables with missing values were recoded where possible to 'not stated' (55 participants for gross weekly per capita household income, 15 respondents for highest qualification); 'other' (15 participants for house tenure); and 'not calculable' (31 participants for optimism, 58 for lack of control, 24 for positive social capital, 18 for negative social capital and 24 for positive instrument social support) responses. With the exception of composite variables (which consist of data pooled from a number of individual variables), no more than 10% of respondents had missing values for any variables after recoding (see Table B.1 in Appendix B). For ordinal/dichotomous composite variables it was possible to unambiguously recode some missing values to a valid category. This was undertaken in cases where, regardless of the 'correct' value of missing data, it would not have changed how respondents were categorised for the composite variable in question. This recoding was undertaken for all racism and stress composite scores as well as for depression (CES-D). Table 9.1 details the prevalence of selected health-related characteristics and potential confounders within the study population.

9.2.2 Analytical approach

Initial unadjusted bivariate correlations/cross tabulations between each predictor variable and each outcome variable were performed along with tests of association (chi-squared, Spearman's Rho or Pearson's Rho for categorical, non-normal continuous and normal continuous data, respectively) to identify statistically significant ($p < 0.05$) and marginally statistically significant ($0.05 < p < 0.10$) associations. Results of these unadjusted analyses are not shown here. Each predictor and outcome variable combination were then included in a maximum-likelihood logistic or linear ordinary least squares regression with age and sex in the model.

Following this, confounders/mediators of the association between each predictor and outcome variable combination were considered. Mediation and confounding are statistically identical phenomena, which can only be distinguished through causal reasoning (Paradies, 2005). As such, except in a few cases as discussed below, no distinction was made between these two phenomena. For mediation/confounding to occur four conditions must be met: (1) the predictor must be associated with the outcome; (2) the mediator/confounder must be associated with the predictor; (3) the mediator/confounder must be associated with the outcome; and (4) the association between the predictor and outcome is no longer significant when adjusting for the mediator/confounder (Baron & Kenny, 1986). Due to the possibility of multicollinearity between the mediator and predictor, an additional requirement of step four is that the strength of association between the predictor and outcome is reduced (Baron & Kenny, 1986). Variables that meet the above conditions are referred to here as *substantive* mediators/confounders. In the analyses below a less stringent approach to mediation/confounding was adopted whereby a variable was considered to be a *potential* mediator/confounder if it met condition (1) above (i.e. the variable was statistically significantly associated with the particular outcome in question).

All socio-demographic and psychosocial variables were examined as potential mediators/confounders. In addition, smoking status, alcohol consumption, physical activity, junk food consumption, fruit and vegetable consumption, depression, self-assessed health status and SF-12 mental and physical component scores were examined as potential

mediators/confounders for all physiological outcomes. Mean waist circumference, diabetes status, total cholesterol and triglycerides were also considered as potential mediators/confounders of blood pressure, hypertension, homocysteine, C-reactive protein, fibrinogen and HbA1c.[22]

To determine whether a particular variable acted as a potential mediator/confounder, the variable in question was added to the regression models detailed above which consisted of a predictor and outcome variable combination together with age and sex. If this variable was associated with the outcome in question (at the $p < 0.05$ level) it was considered to be a potential mediator/confounder. All such potential mediator/confounder identified were then included together (along with age and sex) in a further series of regression models that again examined each predictor and outcome variable combination in turn. To increase the stability of models and reduce the width of confidence intervals, the most prevalent category of each predictor variable was selected as the base reference category in regression models.

A common set of potential mediators/confounders were included for all stress variables examined as outcomes. This common set of potential mediators/confounders consisted of all variables found to be potential mediators/confounders for any of these stress outcomes. This resulted in all potential psychosocial and socio-demographic confounders, except household composition and positive instrumental social support, being included in these models.

The analytical approach outlined above involves a large number of statistical tests without adjustment of the significant level to account for such multiple comparisons. Such a situation is taken into account in the results below by noting, and excluding from further discussion, those outcomes which had less than the number of significant associations with predictor variables than would be expected by chance. For those outcomes which had more significant association with predictors than would be expected by chance, the magnitude of selected relationships of interest is also discussed.

[22] HbA1c was stratified by diabetes status as detailed above.

Selected outcomes which were strongly associated with racism predictor variables in the analyses outlined above were analysed further in order to determine whether any of the potential confounders adjusted for in regression analyses were acting as substantive confounders/mediators of the associations in question (as detailed in 9.6 below). All socio-demographic, psychosocial variables and health-related measures considered as potential confounders above were subjected to substantive mediation/confounding analysis for the selected outcomes of interest, with the four conditions outlined above necessary to establish substantive mediation/confounding. Variables were only delineated as mediators vs. confounders when their location along causal pathways was clear. This resulted in variables which preceded exposure to racism such as gender being considered as possible substantive confounders rather than as possible substantive mediators. Multinomial and logistic regression models (adjusted for age and sex) were used for ordinal and dichotomous outcome variables, respectively in substantive mediation/confounding analyses.

The variance explained by predictor items was determined by the change in adjusted R^2 values upon removal of the predictor from the regression model in question. For MIRE predictor variables, all items in each question were included simultaneously in a regression model such that co-variance between items was adjusted for in these models. The variance explained by only those predictor items which were individually statistically significantly related to the outcome in question was also calculated. Due to the influence of missing values on these calculations, the variance explained by this subset of statistically significant items may be greater than that explained by all items in a particular MIRE question. Similarly, due to the influence of missing values, detailed comparisons of the variance explained by question items vs. composite variables or the full set of question items vs. a subset of significant items were not undertaken. Also, because of instability in some regression models which included all 31 MIRE items, the total variance explained, as shown in Table 9.2, was calculated by summing the variance explained across the predictor variables listed in this table. The same approach was used in Table 9.3, in relation to stress predictor variables, in order to ensure comparability. Although not accounting for co-variance among all the MIRE items/stress predictors combined, results from stable regression models suggest that this approach did not overestimate the variance explained.

Full results of the analyses detailed above are contained in Appendix B. In this appendix, Tables B.2-B.29 contain information on the strength of each statistically significant association examined (odds ratios or coefficients as appropriate) as well as the variance explained by both the full regression model and the predictor variable in question (statistically significant findings are shown in bold). Tables B.30 – B.43 summarise these associations in terms of their significance and the direction of association (positive (+), negative (-) or blank cells for no significant association), while Tables B.44 - B.57 summarise the variance explained by predictor variables.

9.3 Associations between self-reported racism and stress

As noted above in Chapter 4, there is considerable debate about the relationship between self-reported racism and health. Although a range of studies have conceptualised racism as a form of stress the causal relationship between these two phenomena is still far from clear. Although unable to establish the causal direction of this relationship, this section considers the empirical association between self-reported racism and stress as measured in the DRUID study.

Of the associations between MIRE items/composite variables and various stress variables examined in analyses (either adjusted for age/sex or for all potential confounders), 22% (200 out of 912 associations) were statistically significant (see Tables B.2-B.7). Of the examined associations between stress variables and MIRE Q1 items/composite variables 40% (120 out of 300 associations) were statistically significant and all were in the expected direction (i.e. increased inter-personal racism associated with increased stress). Combined, 57% of the associations between inter-personal racism score and stress outcomes and between the number of settings of reported inter-personal racism and stress outcomes were significant (34 out of 60 associations). These two composite variables showed a high degree of concordance with both being significantly associated with the same stress variables in all but one instance.

Of the associations between stress outcomes and MIRE Q2 items/composite variables, 19% were significant (51 out of 264 associations), with all but five being in the expected

direction. These unexpected associations included an association between ‘often/very often’ trying to avoid racism (Q2b) and not having experienced a stressor that affected the respondent a great deal as well as associations between ‘often/very often’ keeping racism to oneself (Q2f) and having a low acute stress score, a lower number of experienced acute stressors and not having experienced a stressor that affected the respondent a great deal.

Less than 1% of the examined associations between stress outcomes and MIRE Q3 (10 of out of 228 associations) were statistically significant and many were in a direction other than that which was theoretically expected. Specifically, increasing internalised racism was associated with a reduced acute stress score and fewer experienced acute stressors while disagreeing that ‘other Australians think they are better than Indigenous people’ (Q3f) (i.e. reduced systemic racism) was associated with a higher chronic stress score and a greater number of chronic stressors. There were, however, some relationships in which internalised and systemic racism were associated with increased stress. Disagreeing that ‘other Australians think that Indigenous people are advantaged because they get special treatment from the government’ (Q3e) was associated with fewer experienced chronic stressors and a higher systemic racism score in general was associated with having experienced a stressor that affected the respondent somewhat/a great deal.

Less frequent thought about Indigeneity (Q4) was associated with reduced stress for all six stress measures. Reduced knowledge of Indigeneity among a respondent’s social group was associated with fewer experienced acute stressors while reduced knowledge of Indigeneity among those one meets for the first time (Q6) was associated with a higher chronic stress score and a higher number of chronic stressors. In accordance with the pattern of significant associations discussed above, much more variance in stress outcomes was accounted for by Q1 and Q2 of the MIRE than by Q3-6 (see Table 9.2). Table 9.4 shows the magnitude of associations between stress outcomes and inter-personal racism composite variables. These results indicate that exposure to increased inter-personal racism is associated with a relative odds of reporting a stress outcome of between 2.8-12.9.

As introduced above, analyses were conducted to determine if racism had differing associations with acute vs. chronic stress or with summary stress scores (weighted by impact) vs. number of stressors experienced. It was found that MIRE items/composite

variables were more strongly associated with chronic stress (score and number of stressors) than with acute stress (score and number of stressors). Across regression models (either adjusted for age and sex or for all potential confounders), 33% of the associations examined between racism and chronic stress were significant (99 out of 304 associations), compared to only 17% of the associations between racism and acute stress which were examined (53 out of 304 associations). These findings were supported by the fact that 43-54% of variance in the two chronic stress outcomes was explained by significant MIRE variables while only 40-44% of variance in the two acute stress outcomes was explained by MIRE items (see Table 9.2) and by the higher relative odds of reporting chronic vs. acute stress following experiences of racism (see Table 9.4).

The patterns of association between racism and the two acute stress composite variables (score and number of stressors) were fairly similar with only 16 discordant associations out the 53 significant associations (30%) between the MIRE and these two composite measures (eight more net associations for number of acute stressors than for acute stress score) and an almost identical amount of variance explained by the MIRE for each of these measures (see Table 9.2). Association patterns between the two chronic stress measures (score and number of stressors) and the MIRE were also somewhat consonant, with only 21 discordant associations out the 99 significant associations between the MIRE and these two composite measures (21%), with only one more net association for number of chronic stressors than for chronic stress score. The variance in the number of reported chronic stressors which was explained by the MIRE (43%) was somewhat lower than the variance in chronic stress score explained by the MIRE (54%).

Although there were relatively few associations with the final two stress measures, it appears that racism was more strongly associated with having experienced a stressor that affected the respondent a great deal (23 significant associations) than with having experienced a stressor that affected the respondent somewhat or a great deal (13 significant associations). However, a similar amount of variance in experiencing a stressor that affected a respondent a great deal vs. somewhat/a great deal was explained by MIRE items (41% and 39%, respectively).

9.4 Associations between self-reported racism and health outcomes

Patterns of association between self-reported racism (as measured by the MIRE) and the various health outcomes considered here changed considerably from unadjusted analyses to models adjusted for age and sex and once again when other potential confounders were added to regression models. Given that racism is variously related to age and sex (see Chapter 8) it was expected that the significance of associations in analyses adjusted for age and sex would differ from results of simple cross-tabulations. Changes in the patterns of significant association when adjusting for a range of other potential confounders suggested that substantive mediation/confounding of the primary associations of interest was occurring, as considered further in analyses below. Both socio-demographic and psychosocial variables were commonly identified as potential confounders of the various health outcomes considered here. These variables (and the number of times they were so identified) were lack of control (20), negative social capital (15), income (13), highest qualification (12), level of social isolation (12), positive social capital (11) and method of administration (11). These variables are considered further in analysis of substantive mediation/confounding below.

A number of the health-related measures examined here did not appear to be associated with self-reported racism. Smoking, physical activity, junk food consumption, fruit/vegetable consumption, SF-12 PCS, waist circumference, WHR, hypertension, dyslipidaemia, CRP, fibrinogen, dental health, allostatic load and number of chronic conditions had no more associations with racism items/composite variables than would be expected by chance (i.e. no more than about 1 in 20) in either regression models adjusted for age and sex alone or in regression models adjusted for all identified potential confounders. As such, these outcomes are not considered further in results below which will instead focus on the association between racism and eight health outcomes (alcohol consumption, depression, self-assessed health status, general mental health, SBP, DBP, homocysteine and HbA1c).

Of the examined associations between a Q1 item/composite variable and these eight health outcomes (in analyses either adjusted for age/sex or for all potential confounders), 28% were statistically significant (56 out of 200 associations). All but two these associations were in the expected direction (i.e. increased inter-personal racism associated with poorer health outcomes). Q1 items were associated with alcohol consumption, depression, self-assessed health status, general mental health, SBP (in a direction opposite to the expected), homocysteine and HbA1c (in a direction opposite to the expected) in analyses adjusted for age and sex (see Table B.34 and B.38). As shown in Table 9.5, the relative odds of having a poor health outcome given exposure to inter-personal racism (i.e. composite variables) were in the range of 1.9 to 3.6.

Q1 composite summary variables and their constituent items generally had concordant association with the health outcomes considered here. Both constituent and composite items were associated with alcohol consumption, depression, poor/fair self-assessed health status, general mental health, homocysteine and HbA1c while neither of the two composite items was associated with SBP (see Table B.34 and B.38). Across all regression models, there were some differences in the pattern of association between health outcomes and inter-personal racism score vs. the number of settings of reported inter-personal racism. Although these composite variables had a similar number of significant associations with health outcomes (8 associations for inter-personal racism score and 9 for the number of settings of reported racism), only nine of the 17 significant associations across these two composite variables were concordant. These two composite variables had concordant associations with homocysteine and HbA1c but not with SBP, alcohol consumption, depression, self-assessed health status or general mental health (see Table B.34 and B.38).

Across the eight health outcomes considered here, 17% of the associations with Q2 items were significant in regression models either adjusted for age/sex or all potential confounders (30 out of 176 associations). Q2 items were associated with alcohol consumption, depression, poor/fair self-assessed health status, general mental health, SBP and DBP (in directions opposite to the expected), homocysteine and HbA1c (in a direction opposite to the expected) in analyses adjusted for age and sex (see Table B.34 and B.38). Generally, these associations were in the expected direction whereby maladaptive responses and disempowered reactions to racism were associated with worse health

outcomes. In particular, (in analyses adjusted for age and sex) such associations were as expected for items Q2a and Q2d-k, with the exception that feeling ashamed, humiliated, anxious or fearful as a reaction to racism (Q2g) was associated with decreased HbA1c, and feeling angry, annoyed or frustrated (Q2h) was associated with increased homocysteine (see Table B.34 and B.38). However, Q2b and Q2c had unexpected associations with health outcomes. 'Sometimes' trying to avoid racism (Q2b) and 'sometimes' trying to change oneself or one's behaviour to avoid racism (Q2c) were each associated with depression and this same level of exposure frequency for Q2c was also associated with increased DBP and HbA1c, with similar results found when adjusting for all potential confounders (see Table B.35 and B.39). As summarised in Table 9.6, key reactions/response to racism had a strong relationship with the health outcomes considered here. In general, reported reactions/responses to racism were associated with relative odds of having a poor health outcome that ranged from 2.5 to 4.8.

Of the associations between Q3 items and health outcomes, 15% were significant (23 out of 152 associations) in regression models either adjusted for age/sex or all potential confounders. Q3 items were associated with alcohol consumption, depression, general mental health, systolic and diastolic blood pressure and homocysteine but not self-assessed health status or HbA1c. Both constituent and composite items of Q3 were associated with alcohol consumption and systolic blood pressure but composite items were not associated with depression, general mental health, diastolic blood pressure or homocysteine (see Table B.36-B.37 and B.40-B.41). Across the relatively few associations between Q3 items and the health outcomes examined here, the magnitude of effect appeared similar to those found for Q1-2 above (see Table B.36-B.37 and B.40-B.41).

In analyses either adjusted for age/sex or for all potential confounders, only 8% of examined associations between MIRE Q4-6 and health outcomes (13 out of 160 associations) were significant. Associations between health outcomes and frequency of thought about Indigeneity (Q4) were mixed. In analyses adjusted for either age and sex or all potential confounders, less frequent thought about Indigeneity was associated with reduced alcohol consumption and homocysteine but increased SBP, while less knowledge of Indigeneity among those one meets for the first time (Q6) was also associated with reduced SBP as well as reduced DBP. Reduced knowledge of Indigeneity among a

respondent's social group (Q5) was associated with increased HbA1c, but reduced knowledge of Indigeneity among those one meets for the first time (Q6) was associated with decreased HbA1c. The few associations between Q4-6 items and selected health outcomes were characterised by effect sizes comparable to those found for Q1-2 above (see Table B.36-B.37 and B.40-B.41).

Taken together, the above findings suggest that, in this study population, racism as measured in the MIRE is most strongly and consistently associated with depression (CES-D), poor/fair self-assessed health status and poor general mental health (both of which were assessed via the SF-12), increased homocysteine and decreased HbA1c. Of the 152 associations between MIRE items/composite variables and each of these health outcomes, there were 27 statistically significant associations between racism and general mental health (18% of examined associations), 14 such associations between racism and poor/fair self-assess health status (9% of examined associations) and 18 statistically significant associations between racism and depression (12% of examined associations), across regression analyses adjusted for either age and sex or all identified potential confounders (all in the expected direction). Furthermore, homocysteine had 17 statistically significant associations with MIRE items (11% of examined associations), with all but five in the expected direction. Although HbA1c had 16 statistically significant associations with racism predictors (11% of examined associations), only three of these associations were in the expected direction, with the other 13 associated with decreased, rather than increased, levels of HbA1c.

The strength of association between racism as measured by the MIRE and a range of health outcomes was considerable with the odds ratios ranging from 1.9 to 4.8 in magnitude (see Table 9.5-9.7). Furthermore, the MIRE explained about a third of the variance in depression (33%), self-assessed health status (34%) and homocysteine (32%) and a fifth of the variance in general mental health (22%) (see Table 9.3). As was the case with stress, Q1 and Q2 accounted for the bulk of explained variance in health outcomes with Q3-6 having a relatively weaker association with these four health outcomes. Also of note was the relatively large amount of variance explained by intra-racial racism (Q1j). This item alone accounted for 2.8% of the variance in depression and 5.7% of the variance in general mental health (see Table 9.3).

9.5 Associations between stress and health outcomes

A number of health outcomes had no more associations with stress than would be expected by chance, with many of these being the same outcomes which were identified as being unassociated with racism above. Smoking, alcohol consumption, physical activity, self-assessed health status, general physical health, waist circumference, WHR, DBP, hypertension, dyslipidaemia, CRP, fibrinogen, allostatic load and number of chronic conditions had no more associations than would be expected by chance (i.e. no more than about 1 in 20) in analyses either adjusted for age and sex alone or for all identified potential confounders and these outcomes are not discussed further below.

Stress predictor variables were strongly and consistently related to depression and general mental health (21 significant associations each), moderately related to SBP, HbA1c and dental health (9 significant associations each) and weakly related to junk food consumption and homocysteine (see Tables B.42-B.43). As shown in Table 9.7, the magnitude of significant associations between stress predictors and a selection of these health outcomes was considerable with relative odds ranging from 3.1 to 4.2.

Of the examined associations between stress measures and these eight health outcomes in analyses adjusted for age and sex or for all potential confounders, 29% (94 out of 320 associations) were statistically significant, with only nine being in an unexpected direction in which increased stress was related to better health. These nine associations consisted predominately of a relationship between decreased SBP and each of total stress score, number of total stressors experienced and having experienced a stressor that affected the respondent somewhat/a great deal or a great deal. In addition, reporting a higher number of acute stressors was associated with decreased HbA1c.

Across the eight health outcomes which stress predictors were related to, there was a good degree of concordance in the pattern of associations between scores vs. counts of stressors experienced for acute and total stress. However, number of reported chronic stressors had more significant associations with health outcomes (18) compared to either chronic stress

score (7), number of acute stressors (11) or number of total stressors (12) in analyses adjusted for age and sex or for all potential confounders. As shown in Table 9.8, over half of the variance in general mental health (54%), a third of the variance in depression (39%) and about 13% of the variance in SBP and dental health was explained by stress predictors, with number of reported chronic stressors (together with number of reported total stressors, which consists primarily of chronic stressors) being the biggest contributor to the proportion of variance explained by stress predictors across the eight health outcomes considered here.

9.6 Substantive mediation/confounding of associations between racism and key health outcomes

As detailed in the description of the analytical approach above, further analyses were conducted to determine which, if any, co-variates substantively mediated/confounded the associations between racism and those health outcomes strongly and consistently related to this exposure. Depression (CES-D), general mental health (SF-12 MCS) and homocysteine were selected for this analysis as the health outcomes most strongly related to racism (self-assessed health status was excluded as it is a subset of general mental health in the SF-12). Due to the cross-sectional design of this pilot study, it was rarely possible to determine whether the variables identified in these analyses acted as substantive mediators or confounders of the relationships in question.

Only two substantive mediators/confounders were identified for homocysteine. Number of acute stressors substantively mediated/confounded the association between experiences of inter-personal racism in recreational settings (Q1d) and homocysteine, while the association between feeling accepted by other Indigenous people (Q3a) and homocysteine was substantively confounded by whether respondents had any non-Indigenous grandparents.

Ethnoracial identity variables were also examined as possible substantive mediators/confounders of the relationship between self-reported racism and health. Recognition of homeland/traditional country and membership in the Stolen Generations were identifiable as mediators/confounders of the association between reported inter-

personal racism from government staff (Q1g) and poor mental health. Unfortunately, the complex circular causality between experiences of racism and ethnoracial identity (see Chapter 8) precludes a clear designation of these variables as either confounders or mediators.

Similarly, determining whether findings for psychosocial and stress variables, as shown in Table 9.9, were caused by mediation or confounding was prevented by the complex relationship between self-reported racism and these phenomena. However, racism and stress as measured in this pilot study are both highly associated with each other and with the same health outcomes in a fashion that suggests considerable confounding/mediation between these constructs. In concordance with findings above, the relationship between racism and stress is strongest for chronic stressors (either stress score or number of experienced stressors) with acute stressors found to mediate associations between racism and health about half as often as chronic stressors.

Given that reactions/response clearly follow experiences of inter-personal racism in any causal chain, these can be conceptualised as mediators rather than confounders. Having a physical reaction to inter-personal racism (Q2k) and feeling ashamed, humiliated, anxious or fearful (Q2g) and powerless, hopeless or depressed (Q2j) as a reaction to inter-personal racism were clearly identifiable as mediators of the relationship between reported inter-personal racism (Q1) and depression/poor mental health. In particular, these reactions were found to mediate between the above health outcomes and experiences of inter-personal racism in employment (Q1a), domestic (Q1b) and public settings (Q1i), from government staff (Q1g) and service providers (Q1h).

Other notable mediators/confounders identified in these analyses were lack of control, positive and negative social capital, and level of social isolation (variables which were also commonly identified as potential confounders in analyses above). As expected, higher positive social capital and higher negative social capital were associated with better and worse health outcomes, respectively.

Experiences of inter-personal racism in employment (Q1a) and public settings (Q1i), from service providers (Q1h) and the composite inter-personal racism score were

mediated/confounded by a range of characteristics (see Table 9.9). Also of interest was the fact that while feeling ashamed, humiliated, anxious or fearful (Q2g) and powerless, hopeless or depressed (Q2j) mediated the association between inter-personal racism and poorer health, these reactions were themselves variously mediated/confounded by lack of control, positive and negative social capital, level of social isolation and acute/chronic experiences of stress. As for associations with health outcomes (see 9.3 above), there were discordant patterns of mediation/confounding between inter-personal racism score and the number of settings of reported racism, with no significant mediators/confounders identified for the latter while the former was the variable with the most identified mediators/ confounders.

9.7 Discussion

The findings from this pilot study show strong associations between racism and depression, lower self-assessed health status, poor general mental health and CVD risk markers, with these associations predominately due to inter-personal racism (including intra-racial racism) and reactions/responses to such racism. The strength of association of the MIRE with these health outcomes was higher than comparable effect sizes reported in previous studies (see Chapter 4). Similarly, the amount of variance in stress/health outcomes explained by the MIRE was much higher than the median variance explained (18%) in the previous research reviewed in Chapter 4.

The finding that self-reported racism was more strongly associated with chronic stress than with acute stress adds support to the notion that racism has long lasting and pervasive effects on those who experience it. Such a finding also supports the conceptualisation of racism as a form of stress – an approach adopted by a number of scholars (see Chapter 4). The pattern of associations between racism and acute/chronic stress did appear to differ to some degree by the measure of stress (scores weighted by impact or number of stressors experienced). Moreover, it was found that number of reported chronic stressors was more strongly associated with health outcomes than was chronic stress score (weighted by impact). Hence, the possibility that these two measurement approaches may access distinct underlying constructs, cannot be ruled out.

A strong and consistent relationship was found between inter-personal racism and various stress measures and also between responses/reactions to racism and stressors. The pattern of associations between inter-personal racism and stress composite variables did not appear to vary by measurement approach (weighted score vs. count of settings in which inter-personal racism was reported). However, although these two composite variables were associated with virtually the same number of health outcomes, there were differences in which health outcomes they were related to, as well as dissimilarities in the extent to which they were mediated by other variables. Therefore, as with chronic stress above, the equivalence of these two measures cannot be assumed from the findings of this pilot study.

Trying to avoid racism (Q2b) and 'sometimes' trying to change oneself or one's behaviour to avoid racism (Q2c) are both inner-directed problem-focused behavioural responses – a coping response which is considered to be adaptive in the literature (see Chapter 3). Although an observed association between trying to avoid racism (Q2b) and reduced reporting of stressors supports the existing literature, the associations of Q2b-c with poorer health outcomes detailed above suggest that such inner-directed adaptive problem-focused behavioural responses may be maladaptive in this study population. In contrast, keeping racism to oneself (Q2f) is considered to be a maladaptive coping response (see Chapter 3) and yet it was associated with reduced reporting of stressors in this pilot study. Given that research on coping responses in the context of racism exposure is scarce, further investigation of these finding in relation to indigenous, and other, populations is required.

As with inter-personal racism, it was expected that increased internalised and systemic racism would be associated with increased stress and poorer health outcomes. However, in contrast to inter-personal racism, there were weak and inconsistent relationships between internalised racism and stress as well as between systemic racism and stress. Similarly, neither internalised nor systemic racism were strongly associated with any health outcomes and those associations that were found were not consistent with *a priori* expectations. Such results indicate either that these two levels of racism are not well operationalised in the MIRE for indigenous people or that empirical associations between health outcomes and internalised/systemic racism do not match theoretical constructs. Interpretation of these findings is hampered by a dearth of such empirical research on the association between

these levels of racism and health with which to compare and contrast the findings from this pilot study (see Chapter 4).

A consistent pattern of associations indicated that less frequent thought about Indigeneity (Q4) was associated with reduced stress and perhaps (although the evidence was more equivocal) with better health outcomes. As detailed in 7.3.4.2.4, increased reporting of inter-personal racism related to more frequent thought about Indigeneity and, although no mediators/substantive confounders of Q4 were identified, it is possible that the apparent protective effect of less frequent thought about Indigeneity may be confounded by an association with reduced exposure to inter-personal racism. Very few associations emerged between MIRE Q5-6 and either stress or health outcomes, with those identified showing no consistent patterns.

Mediation/confounding analyses indicated that inner-directed disempowered reactions to racism (Q2g and Q2j) as well as somatic reactions (Q2k) mediate the association between inter-personal racism and poor mental health outcomes. Although the role of responses to racism has been highlighted in a small body of previous research (see Chapter 4), this study is the first to identify affective reactions as an important aspect of the process by which racism affects health.

The above results also suggest that the association between health outcomes and both inter-personal racism and inner-directed disempowered reactions to inter-personal racism may be partially mediated by stress (primarily chronic stress) as well as by psychosocial characteristics such as lack of control, social capital and level of social isolation which have been identified as important predictors of health (Almedom, 2005; Berkman & Kawachi, 2000; Chandola et al., 2004; Marmot et al., 1997; Marmot & Wilkinson, 1999).

The strong and consistent association of racism with depression, lower self-assessed health status, poor general mental health and CVD risk markers is supported by the existing literature in which 75% of associations between racism and depression examined revealed a positive relationship between these two factors. Moderately consistent associations between racism and both poor general mental health and lower self-assessed health status (40% and 43% of examined associations in the expected direction, respectively) (see Chapter 4) has

also been noted in previous research.[23] Furthermore, recent evidence continues to support the association between racism and depression (Beiser & Hou, 2006; Greene et al., 2006; Schulz et al., 2006a; Schulz et al., 2006b; Steffen & Bowden, 2006) as well as racism and lower self-assessed health status (Borrell et al., in press; Harris et al., in press; Schulz et al., 2006b), including research from cohort studies that establishes the temporal relationship of these factors (Greene et al., 2006; Schulz et al., 2006b).

None of the 12 associations between racism and heart disease examined in previous research were significant (see Chapter 4). However the association between homocysteine and the MIRE is supported by recent research which shows a relationship between racism and both coronary artery calcification (Lewis et al., 2006) and self-reported CVD (Harris et al., 2006). Notwithstanding this finding and in accordance with the research literature to date (see Chapters 4 and 6), poor mental health outcomes were more strongly and consistently associated with racism and stress than physical health outcomes in this pilot study.

This pilot study also uncovered some unexpected and anomalous findings. Of the five health-related behaviours considered here, it is somewhat surprising that only alcohol had a moderate association with racism given that associations have been found between, for instance, racism and smoking in a limited number of studies (see Chapter 4; Bennett et al., 2005; Harris et al., in press). Also notable was a consistent but unexpected relationship between increased racism/stress and both lower SBP and DBP. Inter-personal racism, experiences of discrimination, responses/reactions to racism, agreement that 'Indigenous people should try to think and act more like other Australians (Q3c), not feeling good about

[23] The lower proportion of significant associations found in this pilot study (18%, 12% and 9% for general mental health, depression and poor/fair self-assess health status, respectively) may be due to the fact neither internalised/systemic racism nor ethnoracial identity were frequently assessed in previous research and these exposures had relatively weak associations with health outcomes among this study population. Furthermore, publication bias may have resulted in some null findings not being reported in the literature reviewed in Chapter 4.

being an Indigenous person (Q3d), more frequent thought about Indigeneity (Q4) total stress score, number of total stressors experienced and having experienced a stressor that affected the respondent somewhat/a great deal or a great deal were each associated with lower blood pressure.[24] Only one association in the expected direction was found, with lower internalised racism associated with reduced SBP. Although 75% of associations between racism and blood pressure/hypertension examined in the broader literature were non-significant, only 1% of these associations were in unexpected directions (see Chapter 4). Hence it is unclear why this unusual result has occurred in this study population and further replication of this finding should be sought. Similarly, it is uncertain why racism was related to decreased levels of HbA1c, given that the two studies which previously examined the association between racism and diabetes showed a positive association between racism and diabetes-related measures (Piette, 2006; Tull & Chambers, 2001).

It is hoped that further research will be able to clarify the role of internalised/systemic racism and ethnoracial identity in population health, as well as the place of weighted vs. count measures and anomalous findings vis-à-vis blood pressure, in the intricate web of causation hinted at above. Overall, the above findings provide a first glimpse of the complex relationships between health and wellbeing, inter-personal racism, responses and reactions to this racism, chronic stress and a range of psychosocial characteristics for Indigenous Australians.

[24] In general, these associations were with SBP and/or DBP but, as found in the broader literature (see Chapter 6), were only evident with SBP rather than DBP for stress variables.

Table 9.1: Selected health-related characteristics of the study population (N=312)

	N	%
Number of chronic conditions[a]		
0	50	16
1	103	33
2	80	26
3+	79	25
Allostatic load[b]		
0	55	21
1	55	21
2	41	16
3	37	14
4	36	14
5	17	6
6	12	5
7+	11	4
Depressive symptoms		
Yes (CESD >= 16)	90	32
No (CESD < 16)	189	68
Self-assessed health status		
Poor/fair	79	26
Good/very good/excellent	229	74
Waist circumference		
< 0.9 metres	92	32
>= 0.9 metres	197	68
Glycosylated haemoglobin[c]		
HbA1c <= 7%	255	86
HbA1c > 7%	43	14
Clinically-diagnosed hypertension[d]		
Yes	38	12

	N	%
No	270	88
Clinically-diagnosed dyslipidaemia[e]		
Yes	195	66
No	101	34
Gum/teeth pain or discomfort in last 12 months		
Yes	130	42
No	176	58
Physical activity[f]		
Sedentary	28	9
Insufficiently active	83	29
Sufficiently active	185	62
Sufficient fruit and vegetable consumption[g]		
Yes	31	10
No	277	90
High junk food consumption[h]		
Yes	80	26
No	222	74
Alcohol consumption risk[i]		
No risk (non-drinker)	65	22
Low risk	63	21
Moderate risk	108	36
High risk	62	21
Smoking Status		
Current smoker	138	45
Former smoker	67	22
Never smoker	103	33
Level of social isolation		
Very/somewhat isolated	80	27
Not very isolated at all	213	73

Note: Totals may not add to 312 due to missing values; Percentages may not add to 100% due to rounding.

a Out of the following self-reported conditions: history of heart attack, angina, heart disease, stroke, kidney disease, emphysema/chronic bronchitis, asthma, cancer as well as hypertension (defined as self-reported

hypertension or self-reported medication use for hypertension or SBP >= 140 mmHg and DBP >=90 mmHg, dyslipidaemia (self-reported high cholesterol/triglycerides or self-reported medication use for dyslipidaemia or triglycerides >= 2 mmol/L or total cholesterol >= 5.5 mmol/L or low density lipoprotein >= 3.5 mmol/L or high density lipoprotein <=1 mmol/L).

b The number for which the participant fell into the highest-risk quartile of the following ten characteristics: systolic and diastolic blood pressure, waist-to-hip ratio, total and HDL cholesterol, triglycerides, glycosylated haemoglobin, homocysteine, fibrinogen and c-reactive protein (see Seeman et al., 1997).

c The analysis of glycated haemoglobin was restricted to those respondents who had laboratory confirmed normal blood glucose (fasting glucose < 6.1 mmol/L and 2-hour glucose tolerance <7.8 mmol/L) and who reported that they were not on medication for diabetes.

d Hypertension was defined as SBP >= 140 mmHg or DBP >=90 mmHg.

e Dyslipidaemia was considered to be present if any of the following conditions were met: triglycerides >= 2 mmol/L, total cholesterol >= 5.5 mmol/L, low density lipoprotein >= 3.5 mmol/L, high density lipoprotein <=1 mmol/L.

f Based on The Active Australia Survey (Australian Institute of Health and Welfare, 2003)

g At least five serves of vegetables and two serves of fruit per day.

h Daily consumption of more than two serves of take away/snack food, flavoured milk or soft drinks.

i Based on Australian NHMRC (2001:6-7) guidelines.

Table 9.2: Variance of stress outcomes explained by MIRE (%)

	Acute stress score	# of acute stressors	Chronic stress score	# of chronic stressors	Respondent affected somewhat/ a great deal by astressor	Respondent affected a great deal by a stressor
MIRE Q1a-i	13.0 (9.4)	12.1 (9.6)	18.2 (18.2)	15.3 (15.3)	8.3 (2.9)	12.6 (9.6)
MIRE Q1j	5.1	3.0	7.8	6.8	2.2	2.5
inter-personal racism score	*4.4*	*6.0*	*13.7*	*12.0*	*7.3*	*5.5*
# of setting of inter-personal racism	*2.9*	*3.5*	*12.8*	*8.8*	*7.8*	*3.6*
MIRE Q2	17.5 (13.6)	14.3 (9.5)	17.9 (14.8)	11.1 (9.3)	20.9 (8.4)	21.5 (18.5)
MIRE Q3	3.8	4.0	5.5 (2.1)	5.2 (2.4)	3.9	2.0
Internalised racism	*0.0*	*0.6*	*0.0*	*0.3*	*0.0*	*0.5*
Systemic racism	*0.6*	*0.6*	*1.3*	*0.6*	*1.8*	*1.8*
MIRE Q4	3.6	5.0	1.8	1.3	2.2	1.7
MIRE Q5	1.3	1.4	1.3	0.4	0.2	0.4
MIRE Q6	0.1	0.2	1.6	2.4	1.4	0.3
Total	**44.4**	**40.0**	**54.1**	**42.5**	**39.1**	**41.0**

Note: Figures in brackets denote the variance explained by the subset of items which were statistically significantly related to the health outcome in question. Neither the variance explained by composite variables as shown in italics nor figures in brackets are included in the total variance.

Table 9.3: Variance of key health outcomes explained by MIRE (%)

	Depression (CES-D)	Self-assessed health status (SF-12)	General mental health (SF-12)	Homocysteine
MIRE Q1a-i	7.8	7.8	8.7	7.2
	(5.9)	(5.5)	(9.9)	(2.4)
MIRE Q1j	2.8	0.5	5.7	0.4
Inter-personal racism score	*1.6*	*1.4*	*2.3*	*0.9*
# of setting of reported inter-personal racism	*0.8*	*0.3*	*0.4*	*2.1*
MIRE Q2	15.3	14.9	5.9	11.2
	(11.9)	(8.0)	(9.3)	(3.9)
MIRE Q3	6.1	2.9	1.0	6.8
	(3.0)	(0.0)	(2.9)	(2.7)
Internalised racism	*0.9*	*0.5*	*0.0*	*2.2*
Systemic racism	*1.1*	*0.2*	*0.0*	*0.4*
MIRE Q4	0.3	1.0	1.0	2.5
MIRE Q5	0.4	1.7	0.0	2.3
MIRE Q6	0.4	1.0	0.0	1.8
Total (Q1-6)	**33.1**	**34.3**	**22.3**	**32.2**

Note: Figures in brackets denote the variance explained by the subset of items which were statistically significantly related to the health outcome in question. Neither the variance explained by composite variables as shown in italics nor figures in brackets are included in the total variance.

Table 9.4: Magnitude of associations between selected racism predictors and stress outcomes

Odds ratios (95% Confidence Interval)	High inter-personal racism score[a]	# of settings racism experienced in (4th quartile)[b]
High acute stress score	2.82 (1.40-5.67)	2.89 (1.28-6.50)
High # of acute stressors	3.85 (1.79-8.24)	4.18 (1.78-9.81)
High chronic stress score	5.83 (2.58-13.2)	12.9 (5.10-32.8)
High # of chronic stressors	5.68 (2.53-12.7)	9.36 (3.81-23.0)
Reported stressor that affected respondent somewhat/a great deal	NS	2.98 (1.07-8.31)
Reported stressor that affected respondent a great deal	2.98 (1.32-6.75)	3.64 (1.56-8.49)

Note: All reported associations adjusted for age and sex.

NS: No statistically significant association.

a The base category for this variable is low inter-personal racism score.

b The base category for this variable is 1st quartile of # of settings racism experienced in.

Table 9.5: Magnitude of associations between selected MIRE Q1 predictors and health outcomes

	High inter-personal racism score[a]	# of settings racism experienced in (4th quartile)[b]
Moderate/high risk alcohol consumption OR (95% CI)	NS	2.31 (1.02-5.24)[c]
Depression (CES-D) OR (95% CI)	1.91 (1.01, 3.64)[c]	NS
Poor/fair self-assessed health status (SF-12) OR (95% CI)	2.00 (1.02, 3.95)[c]	NS
Poor general mental health (SF-12) (0-100) Coefficient (95% CI)	-4.13 (-7.23, -1.03)[c]	NS
High SBP OR (95% CI)	NS	NS
High DBP OR (95% CI)	NS	NS
High homocysteine OR (95% CI)	NS	3.59 (1.21, 10.6)[d]
High HbA1c OR (95% CI)	NS	NS

CI: Confidence interval; **NS**: no statistically significant association; **OR**: odds ratio.

a The base category for this variable is low inter-personal racism score.

b the base category for this variable is 1st quartile of # of settings racism experienced in.

c Adjusted for age and sex.

d Adjusted for age and sex, household composition, highest qualification, optimism, lack of control, positive and negative social capital, positive instrumental social support, method of administration and SF-12 physical component score.

Table 9.6: Magnitude of associations between selected MIRE Q2 predictors and health outcomes

	'Sometimes' trying to change oneself or one's actions to avoid racism (Q2c)[a]	'Often/very often' feeling ashamed/ humiliated/ anxious/fearful (Q2g)[a]	'Often/very often' feeling powerless/ hopeless/depressed (Q2j)[a]
Moderate/high risk alcohol consumption OR (95% CI)	NS	NS	NS
Depression (CES-D) OR (95% CI)	2.48 (1.16, 5.33)[b]	2.95 (1.05, 8.27)[b]	2.63 (1.36, 5.06)[b]
Poor/fair self-assessed health status (SF-12) OR (95% CI)	NS	3.25 (1.18, 8.98)[b]	4.76 (1.87, 12.1)[b]
Poor general mental health (SF-12) (0-100) Coefficient (95% CI)	NS	-7.15 (-12.3, -1.97)[b]	-7.83 (-12.4, -3.23)[b]
High SBP Coefficient (95% CI)	5.97 (0.11, 11.8)[c]	NS	NS
High DBP Coefficient (95% CI)	3.38 (0.05, 6.71)[b]	NS	NS
High homocysteine OR (95% CI)	NS	NS	NS
High HbA1c Coefficient (95% CI)	0.18 (0.01, 0.36)	-0.50 (-0.77, -0.22)	NS

CI: Confidence interval; **NS**: no statistically significant association; **OR**: odds ratio.

a The base category for this variable is never/hardly ever.

b Adjusted for age and sex.

c Adjusted for age and sex, income, lack of control, positive and negative social capital, level of social isolation, whether respondent had any non-Indigenous grandparents and alcohol consumption level.

Table 9.7: Magnitude of associations between selected stress predictors and health outcomes

Odds ratios (95% CI)	Depression (CES-D) OR (95% CI)	Poor mental health (SF-12) (0-100) Coefficient (95% CI)	High SBP Coefficient (95% CI)	Poor dental health OR (95% CI)
Acute stress score (Yes, affected me somewhat)[a]	NS	-7.33 (-11.66, -3.01)[c]	NS	3.44 (1.43, 8.29)[c]
# of acute stressors (4th quartile)[b]	3.73 (1.73, 8.01)[c]	-6.32 (-9.68, -2.96)[c]	NS	1.99 (1.02, 3.89)[c]
Chronic stress score (Yes, affected me somewhat)[a]	3.11 (1.44, 6.70)[c]	-7.58 (-11.2, -4.02)[c]	NS	NS
Total stress score (Yes, affected me somewhat)[a]	NS	-7.59 (-11.5, -3.64)[c]	-7.28 (-13.9, -0.70)[d]	3.48 (1.47, 8.21)[c]
# of total stressors (4th quartile)[b]	4.20 (1.95, 9.04)[c]	NS	-6.65 (-11.8, -1.53)[c]	2.26 (1.14, 4.49)[c]
Reported stressor that affected respondent somewhat/a great deal	2.59 (1.09, 6.15)[c]	NS	-5.13 (-9.71, -0.54)[c]	NS

Odds ratios (95% CI)	Depression (CES-D) OR (95% CI)	Poor mental health (SF-12) (0-100) Coefficient (95% CI)	High SBP Coefficient (95% CI)	Poor dental health OR (95% CI)
Reported stressor that affected respondent a great deal	3.09 (1.67, 5.71)[c]	-5.38 (-7.91, -2.85)[c]	-4.29 (-8.00, -0.58)[c]	NS

CI: Confidence interval; **NS**: no statistically significant association; **OR**: odds ratio.

a The base category for this variable is no reported stress.

b The base category for this variable is 1st quartile of # of settings racism experienced in.

c Adjusted for age and sex.

d Adjusted for age and sex, income, lack of control, positive and negative social capital, level of social isolation, whether respondent had any non-Indigenous grandparents and alcohol consumption level.

Table 9.8: Variance of key health outcomes explained by stressors (%)

Stress predictors	Depression (CES-D)	General mental health (SF-12)	Systolic blood pressure	Poor dental health
Acute stress score	2.1	5.4	*0.8*	2.1
# of acute stressors	5.0	6.1	*0.6*	1.3
Chronic stress score	3.9	6.4	*0.0*	*1.5*
# of chronic stressors	10.7	10.7	5.8	2.6
Total stress score	0.9	5.6	0.0	2.8
# of total stressors	10.9	8.6	5.3	2.3
Experienced any stressor that affected respondent somewhat/a great deal	1.6	*4.1*	0.0	*0.5*
Experienced any stressor that affected respondent a great deal	4.3	7.2	0.7	*0.0*
Total (all stress composite variables)	**39.4**	**54.1**	**13.2**	**13.1**

Table 9.9: Substantive mediation/confounding of key health outcomes

	Optim	LOC	LOC ~ES	PSC	NSC	ISS	LI	Q2b	Q2c	Q2g	Q2h	Q2j	Q2k	ASS	#AS	CSS	#CS	AS	AGD	GD
Q1a		d	d	d	d					d		d	d			d	d, m			
Q1b												m	m			m	m			
Q1c																m				
Q1g			m							m		m	m			m	m			
Q1h		d	d		d					d		d	d	d	d	d, m	d, m		d	d
Q1i							m			m	m	m	m			m	m			
Q1j			d														d			
IPR			d		d			d	d	d		d, m	d	d	d, m	d, m	d, m			d
#RS																				
Q2b	d		d	d	d		d	-									d			
Q2c				d					-								d			
Q2f				d																
Q2g				d	m	d	d			-					d, m	d, m	d, m			d
Q2j		d	d		d		d					-		d	d	d	d			
Q2k		m											-	m	m		m			

Q: MIRE question with numbers indicating level of variable as coded in Chapter 7.3.4.2.2; **d**: depressive symptoms (CES-D); **m**: poor mental health (SF-12 MCS); **IPR**: inter-personal racism score; **#RS**: number of settings in which inter-personal racism was reported; **Optim**: optimism; **LOC**: lack of control; **LOC~ES**: lack of control (employment settings excluded); **PSC**: positive social capital; **NSC**: negative social capital; **ISS**: positive instrumental social support; **LI**: level of social isolation; **ASS**: high acute stress score; **#AS**: high number of acute stressors experienced; **CSS**: high chronic stress score; **#CS**: high number of acute chronic experienced; **AS**: having experienced any stressor; **AGD**: having experienced a stressor that affected respondent somewhat/a great deal; **GD**: having experienced a stressor that affected respondent a great dea

Chapter 10. ***Anti-racism and Indigenous Australians***[25]

10.1 Introduction

Despite evidence presented in Chapters 1 and 8-9 that racism is a significant social problem for Indigenous Australians, there has been relatively little research conducted on possible anti-racism interventions in relation to Indigenous Australians. This chapter attempts to summarise and synthesise the existing body of research on this topic, beginning with an overview of theoretical issues in the study of anti-racism, drawing from sociology, followed by a review of empirical research from social psychology on effective anti-racism approaches. Institutional and legal policy recommendations are then discussed in relation to Indigenous Australians. In conclusion, strategies for engendering political will to combat racism in the current neo-liberal capitalist climate are explored.

It has been suggested that fundamental reform of racialised systems is necessarily accompanied by struggles that reach the point of overt protest and that, although this need not be in the form of violent racially based revolution, social systems must be shaken if fundamental transformation is to take place (Gilroy, 2002). There is no doubt that it is difficult to create anti-racist structures that combine elements of the anti-racist movement which are both outside the state and dedicated to remaining within it (Gilroy, 2002). At the same time it is also important not to rely exclusively on anti-racist social movements within civil society to the exclusion of efforts undertaken within existing state structures (Gilroy, 2002). This chapter is not a treatise on how to remold society in a single revolutionary event, but instead aims to improve upon state activity in relation to racism (Omi, 2001) through a focus on public policy approaches to anti-racism which could be effective within existing state structures.

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25 This chapter is taken from Paradies Y. (2005). Anti-racism and Indigenous Australians. *Analyses of Social Issues and Public Policy* 5: 1-28.

10.2 Theoretical Considerations

There are a number of theoretical considerations in the study of racism and anti-racism that affect the nature of empirical research and the approach to developing public policy in this area. A fundamental issue is how racism and anti-racism are defined, which has implications for notions of intentionality, morality and the effects of social action. A further issue is the approach to combating racism in society: Is the goal elimination of ethnoraces themselves or only the social effects that membership of different ethnoraces entails? Is anti-racism about highlighting differences, or similarities, between and within ethnoracial groups? This section will consider these issues and questions in the light of their application to the study of anti-racism and the development of anti-racist public policy.

To begin with the definitional and conceptual approach to racism (and hence anti-racism) detailed in Chapter 3 is also adopted here. Under this approach, 'anti-racism' is defined as the actions which reduce power differentials through advantaging subordinate ethnoracial groups and/or disadvantaging dominant ethnoracial groups. As noted in Chapter 3, the distinction between racism and anti-racism does not depend on the intentions of social actors or institutions, but rather the actual effect of these actions. Many forms of racism operate under the guise of anti-racism as a form of legitimation[26] and some actions overtly intended to be racist fail to cause disadvantage (i.e. reduced power) for subordinate ethnoracial groups (Anthias & Lloyd, 2002). Furthermore, anti-racist approaches may produce racist outcomes despite the best of intentions: for instance, attempts to help the

[26] For instance, in 1983 after extensive maneuvering, the Reagan administration in the US completed its reorganisation of the Commission on Civil Rights, a state 'watchdog' organisation which had been established in the 1950's to monitor and report on progress and problems in the achievement of racial (and later, women's) equality. Under its new leadership, the Commission declared that addressing 'reverse discrimination' (i.e. discrimination against Whites resulting from affirmative action and other similar programs) was its highest priority (Omi & Winant, 1994).

targets of racism may be undertaken in ways that are paternalistic and hence disempowering and, by definition, racist. Also of importance is the fact that both racism and anti-racism may be counter-intuitively related to notions of 'morality' – for example, reducing the power of dominant ethnoracial groups may involve an abrogation of their 'universal human rights' and hence be 'immoral'.

In considering approaches to anti-racism in this chapter, the goal sought is a society of harmony and mutual respect (Anthias & Lloyd, 2002) that can only be achieved by combating racism and/or its deleterious effects. It is important to stress that this chapter is not contending that racism be addressed in isolation, neither is there a suggestion that racism is readily seperable from other forms of oppression (Gilroy, 2002). Rather, people are differently placed in relation to racism depending on their particular context and, furthermore, types of oppression are not mutually exclusive and are both fluid and flexible over time and space. For example, a White woman may be positioned as an oppressor in terms of racism but positioned as oppressed in terms of sexism and so forth. Therefore, if such a society as described above is to be created, anti-racism needs to be undertaken alongside efforts to combat (hetero-)sexism, classism, nationalism and other forms of oppression, together with an understanding of the complexity of oppressor/oppressed subject positions. However, the application of the strategies discussed below, as applied to a diverse range of oppression, is beyond the scope of this chapter as is a consideration of how research on intersecting oppressions[27] (i.e. gendered racism) could be utilised in the development of public policy.

Conceptually, there are two main approaches to anti-racist activism: either the elimination of the notion of ethnorace and hence racism from society or the elimination of power differentials across ethnoraces. Some have suggested that racism can only be eliminated by ending racialisation within social systems and hence ending the very notion of ethnoraces altogether. This would result in a situation where it would no longer be possible to have

[27] For examples of this research see Anthias & Yuval-Davis, 1992; Bohmer & Briggs, 1991; Cohen, 1995; Hartung, 1991; Brah & Deem, 1986; Burgess-Macey, 1992; and Gewirtz, 1991.

power differentials because there would be no criteria on which to base them (i.e. no ethnoraces). However, the actual elimination of the idea of ethnorace should not be confused with the frequent absence of any overt reference to ethnorace in new forms of racism which rely on tropes related to culture and identity as well as nation, nationality, and national belonging to create oppression (Dei, 1997; Gilroy, 2002). This confusion aside, such an assimilationist approach begs the question, what would this ethnorace-less society look like? Young (1990) has distinguished between conformist and transformational assimilation. The former simply requires that 'othered' ethnoracial groups in society conform to and become the same as the dominant ethnoracial group whilst the latter approach seeks to transform the dominant ethnoracial group(s) so that the end of racialisation coincides with a newly produced society that is distinct from those which previously existed. Clearly, these two divergent approaches to assimilation have profoundly different consequences for research into social interventions and the development of public policy.

Another approach to anti-racism is to retain notions of difference in terms of ethnorace but as an aspect of equality, whereby ethnoracial individuals/groups are different within equalised power relations. In political theory this approach is known as 'democratic cultural pluralism' or 'radical pluralism' and entails a positive sense of group difference and autonomy and an elimination of hierarchical relationships between ethnoracial groups (Young, 1990). Problematic in this approach is the need to define both the boundaries of group identity (i.e. how fragmentary can ethnoracial groups practically become) and the foundations upon which such a society can be built (i.e. what axioms of social order are not negotiable) in ways which do not lead to exclusions and power differentials.

These two differing theoretical approaches to anti-racism are reflected in practice through the interplay between universalism and particularism. On the one hand, anti-racist activism can emphasise the similarities of all human beings, universal human rights and equality of treatment regardless of ethnorace, and on the other hand, anti-racist discourses can assert the fundamental plurality of ethnoracial groups primarily through notions of 'culture' and 'language'; leading to calls for special, or different, treatment for Indigenous peoples and other subordinate ethnoracial groups. These calls included slogans such as 'equality within difference' and the need for a 'multicultural liberal state'. Similarly, anti-racist approaches

can either seek to suppress or assert differences within subordinate ethnoracial groups (Bonnett, 2000) depending on the implicit answer to the above question on the extent to which groups can practically be fragmented for the purposes of either research or policy.

In practice, and in public policy, it is effective to emphasise both similarities and differences between ethnoracial groups in differing contexts (Pedersen et al., 2005). With this in mind it would nonetheless appear that the elimination of the notion of ethnorace is the more difficult, and, given the rise of colour-blind policies to be discussed further below, the more dangerous task. Thus, this chapter will focus on approaches to creating equality within difference rather than the elimination of ethnorace itself. However, regardless of which approach is adopted, it is important to note that addressing racism is not the sole task of dominant ethnoracial groups (i.e. White people in Australia), but also the responsibility of subordinate ethnoracial groups who need to address issues of racism, including internalised racism. The evidence and recommendations presented here pertain to and can be advanced by any and all members of society.

10.3 Empirical Review

Although there is a vast body of research on racism against various target groups as well as the factors that mediate and moderate its outcomes, there has been relatively little research on how to reduce or eliminate racism itself (Duckitt, 2001). The following sections will consider anti-racism strategies for both the targets and perpetrators of racism drawing primarily from the field of social psychology.

10.3.1 Arm(our)ing Against Racism

Clearly anti-racist education for dominant ethnoracial groups must necessarily be different from anti-racist education for subordinate ethnoracial groups (Bonnett & Carrington, 1996).As such, a number of studies have highlighted approaches to helping targets of racism deal more effectively with this phenomenon. For experiences of chronic racism, it has been found that emphasising the positive and trying to change the situation is most

effective; whilst for experiences of acute racism, emotional distancing is more effective (Clark, 2000). In addition, there are benefits in seeking social support and having a strong sense of ethnoracial identity/concept (see Chapter 4). There have also been recommendations for counselling practices to ameliorate the effects of racism, including ethnorace-specific approaches to psychotherapy (Elligan & Utsey, 1999) and counselling which includes re-establishing an adaptive in-group identity, utilising culturally congruent coping behaviors, encouraging benign inter-group contact experiences and being mindful of the practitioner's ethnorace and its impacts on treatment (Dunbar, 2001).

It has been found that a worldview resonant with one's ethnoracial group, strong ethnoracial identity, an awareness of racism and bicultural adaptation can furnish the racism-resistant armour needed to build positive well-being (Harrell, 2000). Learning about both the ubiquity of racism in society as well as the different forms of internalised racism is important to all ethnoracial group members, as is information on the forms and nature of different ethnoracial identities.[28]

This type of learning is known as 'racial socialisation' and has three main components: (i) learning to identify racism accurately and acknowledge its extent; (ii) role models who can demonstrate appropriate responses to racist situations including conversational skills and strategies utilising polite corrections, witty repartees, jokes and stories rather than just facts and evidence (Guerin, 2003); and (iii) understanding the experience of racism, which may be fraught with feelings of difference, rejection, and confusion (Greene, 1994).

It is also possible to reduce the impact of racism through dismissing the importance of the domain in which racism has occurred (i.e. by asserting that academic achievement isn't important after experiencing racism from a teacher) or by dismissing the significance of the perpetrator or their opinions (Major et al., 2000). Clearly, however, this approach to dealing

[28] There has been considerable recent work in social psychology on racial identity development, and its relation to racial worldviews and internalised racism, which could be used in such education. See for instance Helms, 1995; Kohatsu et al., 2000; LaFleur et al., 2002; and Poston, 1990.

with racism, especially in relation to domain disidentification, has potentially deleterious societal implications.

For some targets of racism it may also be possible and beneficial for them to conceal their ethnorace when faced with potential racist persons or situations. Clearly, this is not an option for many individuals and does not provide protection from (and may even exacerbate) feelings of vicarious racism directed against others from their in-group who won't or can't conceal their ethnorace. It should also be noted that, in the long-term, identification as a member of a subordinate ethnoracial group is important for collective social action (Major et al., 2000).

There are thus a number of strategies that may be employed by targets of racism in an effort to ameliorate its effects. Unfortunately, there is evidence to suggest that, at least amongst African Americans, only half to two thirds of parents actively engage in a process of racial socialisation with their children (Thomas, 1999) and although some Indigenous children are exposed to racial socialisation, including the reclaiming of 'idioms of stigma' to reduce their impact in racist language (Carter, 1994), it is not clear how widespread this practice is. A discussion of effective policy approaches to promoting racial socialisation appears in the final section of this chapter.

10.3.2 Reducing Racism

Duckitt (2001) suggests that efforts to reduce prejudice can be organised around four causal levels of intervention: cognitive, individual, inter-personal and societal. Duckitt's model represents a parsimonious synthesis of a broad range of findings from social psychology on approaches to reducing prejudice and discrimination. Below this framework is applied to a discussion of the empirical evidence for effective anti-racist interventions, with a particular emphasis on evidence in the Indigenous Australian context, where available.

10.3.2.1 **Cognitive Level**

At the cognitive level there is a wealth of experimental evidence to suggest that four kinds of categorisation help to reduce racism: (i) de-categorisation; (ii) re-categorisation; (iii) sub-categorisation; and (iv) cross-cutting categorisation (Duckitt, 2001). It is important for individuals from different ethnoracial groups to experience personalised contact with varied out-group members so that friendship and acquaintanceship can develop (de-categorisation) and this ideally occurs where a common, shared identity through super-ordinate interests and goals is present (re-categorisation). Generalising these experiences from individual contacts to whole subgroups would be assisted when ethnoracial membership was salient (sub-categorisation), and more positive evaluation of others would be likely when at least one category of membership is shared (cross-cutting categorisation) (Duckitt, 2001). Note that cross-cutting categories work only when they are conceptualised *inclusively* rather than exclusively (i.e. a shared sense of group identity between white and black women because they are women rather than exclusive subcategories such as white women and black women) (Brewer, 2000). These perceptual-cognitive conditions are the most primary determinants of racism and changes at the cognitive level flow from interventions at the three causal levels which are discussed below.

10.3.2.2 **Individual Level**

Individual level interventions are of two broad kinds: (1) direct change to individuals' stereotypes, attitudes and behaviors; and (2) change in some attribute related to racism (Duckitt, 2001). A number of studies suggest that the difference between individuals who are low and high in racism is not their ingrained beliefs but, rather, the cognitive schemas that low racist individuals call forth to counteract these ingrained beliefs (Devine, 1989). It is therefore likely that change in racist individuals occurs through internalising new values or standards that motivate the suppression of previous stereotypes or negative attitudes, rather than through direct change (Duckitt, 2001). In general, individual level interventions can be cognitive or affective in focus and may combat both implicit unconscious as well as explicit conscious racism (Dovidio et al., 2000).

In the Australian context it has been found that 'false (ideological) beliefs' about Indigenous Australians are very common and include notions that Indigenous people get

their car loans and funerals paid for them by the government (Pedersen et al., 2005). One study found that the reporting of positive views about Indigenous Australians increased after correct information on such topics had been provided to participants (Pedersen et al., 2005). A three-day program designed to promote awareness of Indigenous history and culture was delivered and evaluated in relation to government employees in South Australia. Immediately after the program, increases in knowledge and decreases in endorsement of negative stereotypes were found for participants exhibiting either old-fashioned or modern forms of racism. These results were attenuated at 3-month follow-up but still remained statistically significant (Hill & Augoustinos, 2001).

Important attributes relating to racism are ideological beliefs, personality and worldview. Two particular attributes which have been found to strongly relate to racism are 'right-wing authoritarianism' and 'social dominance orientation', and their underlying personality traits of 'social conformity' and 'tough-mindedness' respectively. These personality traits can be described in turn as worldviews of a 'dangerous threatening world' and belief in a world akin to a 'competitive jungle' (Duckitt, 2001).

Some studies suggest that, amongst persons with high authoritarianism, legitimate authority figures are important in reducing racism. Similarly, parenting styles that teach perspective-taking, empathy and non-violent conflict resolution have been found to increase the likelihood that children will adopt tolerant and non-racist attitudes and behaviors (Sanson et al., 1998). There is also some evidence that formal education reduces both racism and authoritarianism, and that it is 'liberal' education, which exposes individuals to a diversity of ideas and perspectives, that is most effective at achieving this, rather than 'conservative' education which aims to preserve and incalculate traditional norms and beliefs (Duckitt, 2001). In Australia, it has been found that reduced levels of racism are associated with increased education (Pedersen et al., 2000; Pedersen & Walker, 1997), especially education about Indigenous issues (Pedersen et al., 2004).

Recent research has suggested a promising means of combating racism by combining both kinds of individual-level interventions through value confrontation, an emphasis on democracy and civil liberties, and 'empathy' approaches (Devine et al., 2000; Finlay & Stephan, 2000; Pedersen et al., 2004; Pedersen et al., 2005). These approaches work

through cognitive dissonance and responses to evoked feelings of 'guilt' (Devine et al., 2000; McGarty et al., 2005), and may be particularly effective amongst both highly-racist individuals and people with authoritarian or social dominance personality traits (Duckitt, 2001).

10.3.2.3 **Inter-personal Level**

At the inter-personal level, there are two broad approaches to reducing racism: (i) changing social influence; and (ii) creating favourable inter-group contact (Duckitt, 2001). In terms of changing social influence, effective approaches include: (i) mass persuasion campaigns; (ii) promoting positive and anti-racist media portrayals of target groups; and (iv) anti-racist educational curricula.

It has been found that mass persuasion campaigns that use simple exhortations such as anti-racist sermons and parodies of racist attitudes/beliefs or negative stereotypes are not effective in reducing racism (and may even strengthen racism in highly-racist individuals) (Duckitt, 2001; Murji 2006; Vrij & Smith, 1999). Rather, films and television programs with implicit anti-racist messages can have substantial effects which are inversely related to the baseline level of racism (although long-term evaluations have not been conducted to date) (Duckitt, 2001).[29] As noted above, in relation to authority figures, the use of high-profile individuals as role models of anti-racist behaviors may also be efficacious (Pedersen et al., 2005; Sanson et al., 1998; Vrij & Smith, 1999;) and anti-racist messages from high status subordinate group members may also reduce racism against their own and other subordinate ethnoracial groups (Major et al., 2000). However, it is important to note that these approaches may also produce feeling of resentment amongst some individuals leading to a 'backlash' effect and increased racism (Duckitt, 2001).

[29] Levels of racism in Britain during the 1970's may have been reduced through the efforts of an organisation called 'Rock Against Racism' which sought to communicate anti-racist themes through popular music (Sanson et al., 1998).

In the early 1990s a two-week media campaign designed to challenge negative stereotypes about Aboriginal people in employment was conducted in a country town in Western Australia with a population of 20,000 including about 1,500 Aboriginal people. A comparison of two independent samples of the residents pre- and post-intervention showed considerable improvements in reported stereotypes of Aboriginal people in relation to employment and the most change was found in those people without strong initial beliefs, either positive or negative (Donovan & Leivers, 1993). In general, the mass media is among the most significant sites for both ethnoracial vilification and anti-racism. This importance is particularly strong for those community groups, such as Indigenous Australians, with whom other Australians have little regular personal contact due to their relatively small proportion of the national population (Hollinsworth, 1998).

Multicultural educational curricula which explicitly teach about the history and culture of subordinate ethnoracial groups, and emphasise the value of diversity, are also effective in fostering anti-racist norms and values in society. These programs are most effective when integrated into the overall curricula, introduced as early as possible in a child's education, delivered by teachers with appropriate training, and involve cooperative learning and the development of critical thinking (i.e. liberal educational approaches) (Hollinsworth, 1998; Pedersen et al., 2005; Sanson et al., 1998). This type of education needs to counter the tendency to make dispositional, rather than situational and historical attributions, for social disadvantage, should include explicit teaching on the nature of racism, how to alleviate it (Duckitt, 2001) and respond to it in discourse (Guerin, 2003). The importance of education is highlighted by the fact that the New Right has made it a significant battleground upon which to attack anti-racism through a portrayal of schools sites of authentic national culture (Gilroy, 2002) which are being undermined by anti-racism (Palmer, 1987).

The importance of critical thinking in anti-racist education cannot be overemphasised. This type of education does not seek to merely create a dogmatic and ideological form of anti-racism to equal that of some forms of racism but rather seeks to recognise the complex, contradictory and contentious nature of oppressive and liberatory power relations. Unfortunately, some approaches to 'political correctness' are illustrative of this dogmatic approach to anti-racism. In Australia, for instance, an example of this approach can be found in an academic text which, labouring under the misconception that Charles Darwin

'invented' social Darwinism,[30] condemns the racism that is purported to exist in naming an Australian capital city after this eminent scientist (Patell-Gray, 1998). Such approaches to anti-racism are ineffective as they have 'backlash' effects which both alienate those with a more nuanced and critical approach to anti-racism and provide a easy target for the New Right and others who wish to portray anti-racism activism as 'extreme', out of touch with reality and infringing on freedom of expression and belief.

In addition to the above strategies for changing social influence, a very promising model for improving inter-group relations is the 'contact hypothesis' first articulated by Gordon Allport (1954). The model specifies four essential conditions under which increased contact leads to reduced inter-group racism: (i) equal status; (ii) super-ordinate goals; (iii) no competition; and (iv) institutional/authority sanctioning of the contact (Allport, 1954). It has also been suggested that contact should be of sufficient frequency, duration and closeness to permit the development of meaningful inter-group relationships (Vrij & Smith, 1999) and that the perceived importance of contact to those participating in it may also be of relevance (van Dick et al., 2005). A recent meta-analysis of 203 studies indicates that contact under these conditions is efficacious in reducing prejudice and discrimination for a number of different personal characteristics (sexual orientation, ethnorace, disability, nationality and age), and across a number of different settings (work, school, housing, recreational etc.) (Pettigrew & Tropp, 2000). In Australia, decreased levels of reported racism have been associated with geographic areas that have high cultural diversity (Dunn & Geeraert, 2003) and, in general, the potential to reduce miscommunication across ethnoracial groups through honest discussion of the difficulties caused by racism has been noted (Devine & Vasquez, 1998).

It is also possible to create favourable inter-group contact via the contact hypothesis in schools and workplaces. For instance, evidence for the effectiveness of cooperative learning approaches such as the 'Jigsaw Classroom' is now overwhelming (Duckitt, 2001),

[30] The founding of social Darwinism is attributed to Herbert Spencer who first coined the phrase 'survival of the fittest' 15 years after publication of *The Origin of the Species*, by Charles Darwin, in 1859.

including its efficacy in reducing racism alone, or in combination with curricula on conflict resolution and civic values (Johnson & Johnson, 2000), and its applicability in an Australian context (Walker & Crogan, 1998). However, despite these findings there has, as yet, been little implementation of these types of programs – a point which will be returned to when discussing policy recommendations below.

A recently completed cohort study found that infants as young as 6 months of age show preverbal categorisation of ethnorace and that this measure was associated with racism in these children at 6 years of age (Katz, 2003). This study also found that low levels of racist attitudes and beliefs for children were associated with watching more TV as infants and toddlers (particularly educational programs such as *Sesame Street*), having parents who value diversity and talk explicitly about ethnorace, and having an ethnoracial diversity of friends (Katz, 2003). These findings support the need for appropriate media portrayals, inter-group contact, and racial socialisation. There is also evidence that promoting anti-racist norms is particularly effective amongst adolescences, even for highly racist and authoritarian individuals (Duckitt, 2001).

As part of this discussion of interventions at both the individual and inter-personal level it is important to highlight that engaging in anti-racist activities is not without risk to individuals. This process can arouse feelings of sadness, anxiety, hopelessness, anger, shame and guilt among people of various ethnoracial groups. Intra-personally, the struggle to overcome racism requires that individuals examine aspects of their identity that relate to their socialisation as ethnoracial beings, and that they dare to confront how they are implicated in the practice of racism. Challenging racism can also lead to ostracism and rejection and the breaking of ties with significant people in one's life (Thompson & Neville, 1999) and cannot be achieved, in the end, without appropriate commitment from societies as a whole – as discussed in the section below.

10.3.2.4 **Societal Level**

At the societal level there are a number of interventions which can be influential in reducing racism. Strengthening equality of representation as well as social justice can be achieved through liberal democratic forms of governance. This requires well formulated

and strictly enforced system of anti-discriminatory legislation and affirmative action policies to redress existing social disparities due to socio-historical racism. It should be noted that although affirmative action policies are necessary to achieve social justice they can also generate negative expectancies of subordinate group members such as self-doubt (Dovidio & Gaertner, 1996), and it is clear that these policies are most helpful to members of ethnoracial group who are most advantaged by class and other aspects of social location (Jones, 1999). It is vitally important that affirmative action policies do not result in the compromise of 'merit' approaches (however imperfect these are) wherein people are placed in positions which they do not have the skills, abilities or qualifications to be successful. Backlash to affirmative action policies can be lessened through programs that are both fair and are perceived as such, which avoid simple quota systems and include institutional education that explains the need to remedy historical injustice and the benefits of diversity (Crosby et al., 2006; Dovidio & Gaertner, 1996).

An example of such an affirmative action policy that exists in legislative form is the requirement, by a Presidential Order, for US Federal contractors and subcontractors (not in the construction industry) with 50 or more employees or contracts of more than $50,000 to develop and maintain a written affirmative action program (AAP). As part of its AAP, the contractor must conduct a workforce analysis of each job title, determine workforce availability of women and minorities for each job group, and conduct an utilisation analysis to determine whether women or members of oppressed ethnoracial groups are under-utilised in any job group. Based on these analyses, the contractor establishes goals to overcome under-utilisation, and must make a good faith effort to achieve those goals (Department of Labor, 2004). A policy requiring affirmative action programs for all private sector employers with more than 25 employees as well as all public sector employers has also been used in Northern Ireland in relation to religious discrimination (Lustgarten & Edwards, 1992) and in Australia, the Commonwealth *Equal Opportunity for Women in the Workplace Act 1999* requires that private sector companies, community organisations, non-government schools, unions, group training companies and higher education institutions with 100 or more employees establish a workplace program to remove the barriers to women entering and advancing in their organisation (Commonwealth of Australia, 2004).

It has been suggested that these approaches to promoting positive action rather than banning discriminatory action are more effective in promoting equity and, in fact, have spawned a culture of affirmative action that now extends beyond the domains where legislative provisions apply. Similarly, providing financial incentives for employers to engage in affirmative action is far more effective than the possibility of sanctions for not adopting such approaches (Lustgarten & Edwards, 1992).

Although it is difficult to gauge the impact of anti-discriminatory legislation, there is no doubt that similar legislation can bring about significant changes in expressed attitudes and observable behaviour such as in the case of seat-belt and drink driving legislation. Given the extraordinarily low rates of prosecution of anti-discriminatory legislation in Australia (Hollinsworth, 1998), it is important to recognise that these laws serve primarily symbolic and educational roles as important statements of societal values as well as having secondary deterrent, remedial and punitive roles (Jones M., 1997).

Despite the rhetoric to the contrary (Ben-Tovin et al., 1992; Bonnett, 2000), so-called colour blind policies[31] have been found to be extremely disadvantageous to subordinate ethnoracial groups by reinforcing intolerant attitudes to group differences, maintaining covert racism and discrimination (Duckitt, 2001), and severely curtailing any possible redress through affirmative action. Instead, there is a need for accurate and relevant information on outcomes, by ethnorace, across a range of social domains including data collection on the nature and extent of racism for different ethnoracial groups. As a nation, Australia should strive towards an integrative multiculturalism consisting of strong subgroup identities within an overall super-ordinate identity which exists within a safe, secure, stable environment of social justice and a cooperative and egalitarian ethos (Duckitt, 2001).[32]

[31] Approaches that erase notions of race and ethnicity from public policies preclude measures of differential outcomes across racial groups as well as any form of affirmative action.

[32] Although creating an environment of social justice falls under the purview of anti-racism activism at the societal level, achieving this goal is an enormous challenge in countries like

10.4 Policy Recommendations

Given the review of theory and empirical evidence above, there are a number of policy initiatives that could be fruitfully pursued in the Australian context with a particular focus on Indigenous Australians – including approaches at the national, state and local levels of government. Addressing racism at all three tiers of Australian government is particularly important given the geographically specific nature of racism (Dunn & Geeraert, 2003) which requires racism to be tackled using multiple approaches at all possible levels of intervention. The section below examines existing institutional and legislative policies with a view to possible improvements and expansions to these policies in response to the empirical research findings discussed above.

10.4.1 Institutional Policy

10.4.1.1 **Education and Child-Rearing**

As discussed above, school-based interventions have proved extremely successful in reducing racism. One clear policy recommendation emerging from the above evidence is the need to provide anti-racist education that includes inter-cultural understanding and effective conflict resolution, in age-appropriate format, as an integral part of school curricula (Sanson et al., 1998).[33] The goal of a school environment free of racism is

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Australia, where Indigeneity is powerfully correlated with socio-economic deprivation, and a consideration of policies to address this challenge is beyond the scope of this chapter.

[33] Anti-racist education needs to address false beliefs as well as attitudes and behaviors. Existing websites such as 'Racism No Way' (Conference of Education Systems Chief Executive Officers, 2000) and 'Fair Go Australia' (Fair Go Australia, 2004) could be utilised as well as studies of best practice delivery in diverse format (Hieronymus, 2003).

explicitly set out in the *Adelaide Declaration on National Goals for Schooling in the Twenty-First Century* endorsed by State, Territory and Commonwealth ministers of education in 1999 (HREOC, 2004a). State and Territory education departments are responsible for anti-racism policies and programs, in accordance with this national goal. However, implementation of these policies at a local level is a matter for individual schools and as a result there is a lack of uniformity in how racism is tackled across Australia (HREOC, 2004a). It is recommended that Federal, State and Territory governments work together to promote consistent implementation of anti-racism policies in schools through the Federal Ministerial Council on Education, Employment, Training and Youth Affairs (MCEETYA) (HREOC, 2004a). The MCEETYA taskforce on Teacher Quality and Educational Leadership should also develop standards for anti-racism and diversity training aimed at improving the quality of teaching and learning in schools including the use of cooperative learning models which, as noted above, have been shown to be highly effective.

There is considerable evidence that parenting which involves the use of perspective-taking, empathy and non-violent conflict resolution (Sanson et al., 1998) as well as the valuing of diversity and explicit discussion of ethnorace are effective in combating racism (Katz, 2003). It is thus recommended that parenting skills courses which promote these approaches and discourage the use of punitive, power-assertive strategies be funded by the Federal government and made available through existing organisations and programs such as Relationships Australia (Sanson et al., 1998) and the Positive Parenting Program (Sanders & Markie-Dadds, 1996).[34] As considered above, there is a need to promote racial socialisation that teaches Indigenous children how to identify, respond to and cope with racism and courses for Indigenous parents on effective approaches to racial socialisation could also be provided through these existing organisations.

[34] A recent population-based intervention in Perth indicated that an Australian wide implementation of the Positive Parenting Program would reduce the burden of child behaviour problems across the nation by 36% (Williams A. et al., 1997).

10.4.1.2 **Public Service**

For Commonwealth public servants *The Charter of Public Service in a Culturally Diverse Society* states that government services must prevent discrimination through ongoing cultural diversity training for staff (Department of Immigration and Multicultural Affairs, 1998). This Charter has been endorsed by Commonwealth, State and Territory Governments and by the Australian Local Government Associations and progress towards its implementation is reported on in the *Access and Equity Annual Report* by the Department of Immigration and Multicultural and Indigenous Affairs (2004a). However, it is not clear from this report to what extent this particular recommendation is being meet.

States and Territories also have policies and reporting mechanisms that relate to anti-racism. For example, public sector agencies in New South Wales are required to have an Ethnic Affairs Priorities Statement (EAPS) which shows how they will provide training for staff on cultural diversity issues. Each year the Community Relations Commission for a Multicultural New South Wales assesses and monitors the performance of public sector agencies, and reports on this performance in an annual *Community Relations Report* (HREOC, 2004a). It is recommended that continued efforts are made to monitor and report clearly on the implementation and effectiveness of anti-racism training for public servants and that incentives for private sector organisations to provide anti-racist training are also explored. As detailed above, the most effective anti-racism training will promote an awareness of Indigenous history and culture, dispel false ideological beliefs and use a liberal education approach that focuses on the complexities of racism and anti-racism including the power relations embedded in material and cultural structures (Hollinsworth, 1998). Members of Federal and State parliaments should also receive anti-racism training that emphasises the need to both refrain from and rebut statements that reinforce racism, blame 'victims' and scapegoat on the basis of ethnorace.[35]

[35] Some of the comments made by parliamentarians in the past have not only failed to contribute to anti-racism goals but have actively promoted a racist society. For instance, Senator Ross Lightfoot suggested in 1995 that introducing Indigenous studies into all

10.4.1.3 **Law Enforcement**

Racism in the practice of law enforcement, including ethnoracial labeling and profiling, is particularly detrimental and damaging to Indigenous Australians (Cunneen, 2001; Johnston, 1991) and hence is one area that anti-racist policies are particularly important. Police in all States and Territories receive cultural diversity training as part of basic recruitment. In 1997, the National Police Ethnic Advisory Bureau (now the Australasian Police Multicultural Advisory Bureau (APMAB)) released a national training standard for cultural diversity training of police across States and Territories, replacing stand-alone courses tacked onto previously existing training programs (HREOC, 2004a). This is an important step forward, as is the statement by the Bureau that inappropriate use of ethnoracial labeling is "unhelpful, misleading, offensive and counter-productive to sound community relations in Australia"(HREOC, 2004a:157).

Established reporting procedures recommend the use of other physical descriptors such as height, hair colour, distinguishing marks and clothing before ethnorace and that once a case has progressed beyond identification, information on ethnoracial identity is no longer used in police media reports (HREOC, 2004a). However, a scoping exercise by the APMAB in 2003 found that police were having difficulties in conforming to these procedures and in managing the use of descriptors by the media. As a result the development of a set of national standards in training police to use descriptors in a consistent and culturally informed manner was recommended (HREOC, 2004a), and once completed should be implemented across Australia and evaluated for effectiveness.

Racial profiling in law enforcement (the use of ethnorace to make judgments of criminal suspicion) is both discriminatory and ineffective in preventing crime (Glaser, 2003). The collection of data on ethnoracial profiling is now mandatory in 39 States in the US (West,

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schools was "preposterous" because he found Aboriginal culture "distasteful" (Cunneen, 1997).

2003) and similar data is required in Australia, whereby the ethnorace of people stopped by police in their cars, or in public, is recorded. Data collected should include reason for, length and outcome of the stop, whether a warrant check or search was conducted and police officer demographics (West, 2003).

10.4.1.4 **Media**

The media has a strong influence on societal norms and beliefs and has been shown to be an important area in which to both combat racism and promote anti-racist messages. As discussed above, there is considerable evidence that implicit anti-racist messages in the media, especially those using high-profile individuals as role models, are effective (Duckitt, 2001) and have been shown to reduce negative stereotypes about Indigenous Australians in particular (Donovan & Leivers, 1993).

It is vital that the State media including the Australian Broadcasting Commission, the Special Broadcasting Service and related radio stations adopt policies that encourage the representation of Indigenous Australians in positive and individuating ways rather than ignoring Indigenous people or portraying them in negative or low-status roles (Duckitt, 2001). It is also important for these organisations to create more possibilities for background and investigative reporting, consider the impact of the language of political and official actors and support cooperation and information exchange with Indigenous organisations to promote ethnoracial diversity in the media (ter Wal, 2002). Increased efforts to enforce existing legislation against ethnoracial vilification and incitement to ethnoracial hatred in the media, is also required.

Commercial television stations' current guidelines prevent them from broadcasting "a program, program promotion, station identification or community service announcement which is likely, in all the circumstances, to seriously offend the cultural sensitivities of Aboriginal and Torres Strait Islander people" (Jones M., 1997), and the commercial radio stations' code of practice also precludes the broadcast of programs which are "likely to incite or perpetuate hatred against or vilify any person or group on the basis of race" (Commercial Radio Australia, 2004). The adoption of policies recommended for the State

media should be encouraged, for commercial media, through the administrative bodies of Commercial Television Australia and Commercial Radio Australia. There is also a need to redress the skewed reporting in commercial newspapers in relation to Indigenous people.[36] The Australian Press Council, in their guidelines for reporting of race state that the "press needs to show more sensitivity in reporting issues when oppressed ethnoracial groups are perceived in the community to be more 'different' or when they are the subject of particular public debate" (Australian Press Council, 2001). The Australian Journalists' Association Code of Ethics requires members of the Australian Journalists' Association of the Media Entertainment and Arts Alliance not to "place unnecessary emphasis on personal characteristics, including race…" and the Association accepts complaints from members of the public who believe that a journalist has acted contrary to this Code of Ethics (HREOC, 2004a:152).

It is recommended that the Australian Press Council and Australian Journalists' Association be provided with sufficient resources to actively promote improved practice in relation to reporting of ethnorace in the Australian commercial media. It is also recommended that the Australian Broadcasting Authority and the Australian Press Council undertake information campaigns in a variety of formast to inform Indigenous Australian organisations and community members about their standards and complaint processes (HREOC, 2004a).

A sustainable reduction in racism in Australia requires on-going promotion of anti-racist values and beliefs. It would be appropriate for the Federal government to launch a long-term advertising campaign, including a public statement by the Prime Minister acknowledging that racism is still prevalent in Australia in both overt and covert forms and

[36] For example, an examination of newspaper reporting about youth in Western Australia between 1990-2 found that 87% of articles which identified young people as Aboriginal were primarily about crime and an analysis of the Sydney Morning Herald from January 1987 - April 1990 showed that 40% of all references to the word 'riot' in Australia were related to Aboriginal people (Cunneen, 1997).

that the Government is committed to combating it.[37] In association with this, a Prime Minister's Award for Race Relations could be created to annually recognise the most innovative and successful private or public sector organisation in the area of race relations (Lustgarten & Edwards, 1992).[38]

10.4.1.5 **Monitoring Racism and Promoting Anti-Racism in Civil Society**

The Human Rights and Equal Opportunities Commission is the peak government body that deals with issues of discrimination in Australia. It is recommended that this organisation be sufficiently resourced to: (i) encourage and assist all institutions, professions, and organisations to examine their policies and practices to identify and address institutional racism (Sanson et al., 1998); (ii) examine all new Federal government policy initiatives and legislation to ensure that they promote and maintain the ideals of social justice using social impact assessment methods;[39] (iii) encourage and support programs that provide

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[37] This campaign could be based on documents such as 'As a Matter of Fact' developed to dispel myths about Indigenous Australians (Aboriginal and Torres Strait Islander Commission, 1999). The campaign should utilise music, the arts, and well-known Australians to increase its efficacy. In the 1990's the Irish government announced a 4.5 million pound three-year government-sponsored anti-racism/ interculturalism public awareness program (Lentin & McVeigh, 2002) and it is vital that similar efforts in Australia have sufficient funding to make an impact at the national level.

[38] Another approach that may be effective in fostering anti-racism in civil society is something akin to the 'Museum of Tolerance' in Los Angeles which focuses on the dynamics of racism and prejudice in the US and the history of the Holocaust (The Simon Wiesenthal Center, 2004). A similar institution in Australia on racism and cultural genocide in relation to Indigenous Australians could be explored.

[39] This may best be undertaken by designated senior bureaucrats in each government department, overseen by a cabinet committee chaired by a senior minister (Lustgarten & Edwards, 1992). One approach would be to emulate the social exclusion unit in the UK, a

opportunities for optimal inter-racial contact (Pettigrew & Tropp, 2000);[40] (iv) publicise and promote the services that they provide to the Indigenous community;[41] (v) handle complaints in a timely manner;[42] (vi) provide training to State/Territory and Federal police on referring matters to HREOC and State/Territory anti-discrimination commissions;[43] and (v) in collaboration with State/Territory and Federal Ombudsmans and Industrial Relations Commissions, collect and publish statistics on the number and nature of complaints made in each jurisdiction by ethnorace (Goodstone et al., 2001).

As discussed above, data collection around issues of racism for a variety of ethnoracial groups is vital to ongoing monitoring and policy-making. However, very little information on the prevalence of racism is available from either administrative sources or large-scale surveys in Australia. Although data on ethnorace hate crimes/incidents are collected in Australia, there is varying coverage across States and Territories. Currently, there is limited data collection about ethnorace hate crimes by police in South Australia and Western Australia and none in Tasmania or the Northern Territory where there are no legislative provisions under criminal law for ethnoracial vilification. The other three States have well

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ministerial portfolio of the deputy prime minister, which seeks to formulate cross-government policy relating to social exclusion (Office of the Deputy Prime Minister, 2004).

[40] One approach to consider is that taken by Boston in its 2004 city-wide dialogues on race which sought to have each neighborhood in the city hold its own local dialogue group with 15-20 ethnically and racially diverse participants and two group facilitators over four sessions in the hope of bringing together a 1000 Bostonians city-wide (City-Wide Dialogues on Boston's Ethnic & Racial Diversity, 2004).

[41] Funding should also be provided to the Commonwealth Ombudsman's Office to employ Indigenous complaints and outreach officers (Goodstone et al., 2001).

[42] This should include the option to make oral as well as written complaints (Goodstone et al., 2001).

[43] There is some evidence that the police are not referring matters to these regulatory bodies appropriately (Jones M., 1997), with at least one project in Australia undertaking training to address this matter (Department of Immigration and Multicultural and Indigenous Affairs, 2004b).

developed data collection systems with the most comprehensive system introduced by the New South Wales Police in October 1999. This State-wide system collects data on hate/prejudice motivated crimes under which officers, when recording a crime report, can record a motivation, including ethnorace (HREOC, 2004a). Sharing information about hate crimes across the States and Territories has been identified as a priority by the Australasian Police Minister's Council (APMC) and it is recommended that data sharing is accompanied by an adoption, across Australia, of the 'gold standard' set by the New South Wales Police (HREOC, 2004a).

Finally, whilst some ethnoracial communities in Australia, such as the Jewish community maintain a significant program to monitor racism against their constituency, the Indigenous community does not have any active reporting system. As with Indigenous agencies that deal with specific land, legal and health issues there is a need for the Indigenous community to set up a semi-formal space that provides a forum to report racism against Indigenous Australians (Browning & Jakubowicz, 2004).

10.4.2 Legislative Policy

As considered above, anti-racism legislation is important in that it serves a primarily symbolic and educational purpose in promoting societal values and secondarily deterrent, remedial and punitive roles (Jones M., 1997). As such, this type of legislation has considerable potential to bring about significant changes in expressed attitudes and observable behaviour (Hollinsworth, 1998).

At the Federal level the *Racial Discrimination Act 1975* (RDA) as administered by HREOC (HREOC, 2004c), is the key legal mechanism for addressing racism. The RDA was the first piece of Federal anti-discrimination legislation in Australian and is based to a large extent on the Convention on the Elimination of All Forms of Racial Discrimination (1969) (CERD) (HREOC, 2004b). The *Racial Discrimination Act 1975* s.9 makes it unlawful "to do any act involving a distinction, exclusion, restriction or preference based on race, colour, descent or national or ethnic origin which has the purpose or effect of nullifying or impairing the recognition, enjoyment or exercise, on an equal footing, of any human right

or fundamental freedom in the political, economic, social, cultural or any other field of public life." The Act (s.18) also makes it unlawful to engage in a public act which "is reasonably likely in all the circumstances to offend, insult, humiliate or intimidate a person or group of people, on the basis of race, colour, or national or ethnic origin."[44] The Act specifies that both 'direct' and 'indirect' racial discrimination are illegal. The latter does not require either intent to discriminate or that a particular individual or group is targeted (i.e. it is enough to demonstrate disparities at a ethnoracial group level), whilst the former has also been interpreted in case law as not requiring intention (HREOC, 2004b), although, in this case, a particular individual or group must be directly targeted.

However, the Act is undermined by a number of limitations. First, HREOC is established only as an administrative body and proceedings to enforce a determination by HREOC must be taken to the Federal Court. Second, complaints under the Act may only be lodged by a person aggrieved, which precludes members of the public without connection with the person(s) affected from bringing a complaint to HREOC, hence restricting action which could otherwise be taken in the public interest (Jones M., 1997). Third, a human right or fundamental freedom does not cover all instances of possible racism. For example, in *Hagan v Trustees of the Toowoomba Sports Ground Trust*, a complaint of racial discrimination was brought in relation to the maintenance of a sign saying "The ES 'Nigger Brown' Stand" at an athletic oval. It was found that because only the complainant's personal feelings were affected by the sign, rather than any human rights or fundamental freedoms, there was no racial discrimination involved (HREOC, 2004b). Fourth, case law has made it clear that a high standard of circumstantial evidence is required to prove breaches of the RDA and evidence of statistical differences in outcomes by ethnorace is frequently not sufficient (HREOC, 2004b). Finally the RDA has no provision for criminalising racial hatred although such provisions do exist under the CERD upon which it is based (HREOC, 2004b).

[44] It is not necessary to establish that all people in the relevant group may be offended by the acts complained of. It is sufficient to show that a subset of the broader group may reasonably be affected by the conduct (HREOC, 2004b).

Given these limitations, it is recommended that the *Racial Discrimination Act 1975* be amended to: (i) make determinations by HREOC legally enforceable with more evidential burden of proof on defendants and a less onerous standard of proof on complainants (Hunyor, 2003); (ii) allow third party legal action; (iii) increase the scope of discriminatory acts beyond a human rights framework; and (iv) fully implement CERD by adopting provisions for criminalising acts of racial hatred. In the meantime, the Commonwealth government should legislate to make the Federal Magistrates Service a 'no-cost' jurisdiction for discrimination matters where each party pays their own legal costs regardless of who wins the proceedings and promote its policy on waiver of fees for people on low incomes who lodge discrimination claims (Goodstone et al., 2001).

The various State/Territory anti-discrimination Acts make racial discrimination illegal in employment, education, training, the conferral of qualifications, the provision of goods and services, accommodation, at registered clubs and associations, sport, superannuation, insurance, disposal of land, access to places and vehicles, requests for information, administration of laws and programs, and local government. However, there is considerable variation in the coverage of each Act and no one Act covers all these areas, although all these Acts do make it unlawful to discriminate against a person because they have a relative or associate who is of a particular ethnorace (UTS and UNSW Faculties of Law, 2004).

All States and Territories, except the Northern Territory, also "prohibit any public act which is capable of inciting hatred towards, serious contempt for or severe ridicule of a person or a group of persons because of their race" (racial vilification). Civil remedies are provided in all jurisdictions where these acts are prohibited (Lacey, 2004). However, there are no provisions for criminal offences for racial vilification in Commonwealth law or in Tasmania and, of course, no provisions for such in the Northern Territory. In Western Australia the possession of racial material and the publication of racial material for display to harass a racial group or to incite racial hatred is a criminal offence (Lacey, 2004). The provision used in New South Wales, which serves as a model for those in the Australian Capital Territory, South Australia and Queensland, required the consent of the Attorney-general for prosecution, which has never been given. Victorian legislation appears to be the most comprehensive whereby it is a criminal offence to "intentionally engage in conduct

that the offender knows is likely to incite serious contempt for, or revulsion or severe ridicule" (Lacey, 2004).

Future amendments to these Acts need to standardise the areas where racial discrimination is illegal by including all of the domains listed above, with racial vilification laws amended to follow the Victorian model or alternatively that of a private members Bill entitled 'Racial and Religious Hatred Bill 2003', which seeks to amend the Commonwealth *Crimes Act 1914* to prohibit the intentional incitement of racial hatred (one year imprisonment), threats of physical harm on the ground of race or religion (two years imprisonment) and threats to property on the same grounds (one year imprisonment) (Lacey, 2004).

At the local government level, as town councils exist under State statute law, it is recommended that each State/Territory in Australia legislate to require local councils to institute and report on anti-racism initiatives for their constituency and be provided with training in how to implement anti-racism strategies.[45]

Finally, affirmative action policies need to be strengthened by utilising positive legal mechanisms, as detailed in the examples above, with a view to increasing Indigenous representation in areas such as the media, the legal system (especially law enforcement) and the professions in general. The use of financial and other incentives, such as making these

[45] A 1996-7 survey of 666 local councils in Australia, of which about half responded, found that the majority of community relations policies took the form of information provision about various racial groups in society or events to celebrate cultural diversity rather than addressing institutional racism (Dunn et al., 2001). About 31% of councils stated that they were involved in specific programs to improve local community understanding of Indigenous people but only 24% reported similar programs for council employees. This lack of anti-racism training for council employees is especially problematic given that only 29% of councils reported having an anti-racism policy at all and that some councils seemed to position Indigenous people as outside what they see as their constituency and consider multiculturalism (let alone anti-racism) as marginal to their "core business" (Dunn et al., 2001).

policies a prerequisite for seeking Federal contracts as in the US example above, should also be investigated.

10.5 Engendering Political Will to Combat Racism

This chapter has attempted to present an overview of the theoretical issues in the study of anti-racism of particular relevance to empirical research and public policy. Focusing then on Indigenous Australians as a population that experiences particularly high levels of racism, this chapter has considered the existing research on effective approaches to anti-racism for both targets and perpetrators, followed by recommendations for institutional and legal policies required to implement these approaches in Australia. However, none of these policy recommendations will be implemented without significant political will and there is no doubt that powerful sections of the Australian community currently benefit from racism, and therefore will likely resist attempts to counter it (Pedersen et al., 2005). The adoption of economic rationalist values, the minimisation of state intervention and the fostering of profit has shifted politics away from concern for equality of opportunity (Hollinsworth, 1998) as has the whitewashing of ethnorace and colour-blind politics in Australia and other first-world nations.

'Hard evidence', on an on-going basis, of both the prevalence and detrimental effects of racism in Australia will be necessary to muster the political will to address racism in society. Therefore, as a matter of priority, research that produces national data available down to the local government level on the extent and types of racism in Australia as has been undertaken by geographers at the University of New South Wales (Dunn & Geeraert, 2003) must continue along with the implementation of recommendations for reporting under the Federal and State/Territory anti-discrimination acts as discussed above.

Recommendations from a 1984 Australian National Opinion Poll Market Research survey included the suggestion that the Federal government conduct a campaign to increase public awareness of Aboriginal issues. This call went unanswered. In fact, the political landscape in Australia is littered with the wrecks of reports and inquiries in Indigenous affairs whose recommendations remain partially or completely unimplemented (Jonas, 2000). In contrast

to these ongoing recommendations, the Federal government recently drafted legislation which aims to abolish a number of high-level positions in HREOC and reduce its powers and independence (HREOC, 2004d).

The recent World Conference Against Racism recognised "the negative economic …consequences of racism" (United Nations, 2001) and one scholar has suggested that it is the power of racism to "cause conflict and to 'waste' human resources that most excites the interest of 'state agencies' and 'the ruling class'" (Bonnett, 2000:5). Thus, one possible way forward is to quantify the 'cost of racism' to Australia and to 'sell' anti-racism as a means of ameliorating tensions that threaten the state (Alexis, 1999; Bonnett, 2000). 'Productive Diversity' is a public policy of the Australian Federal government that seeks to utilise Australia's language and cultural diversity for the economic and social benefit of all Australians (Department of Immigration and Multicultural and Indigenous Affairs, 2004c). This policy takes as its basis that cultural and language diversity is one of Australia's greatest resources which can be used to boost business productivity and access local and global niche markets. The co-option of this economic rationalist rhetoric by anti-racist activism in Australia may be an effective approach to implementing anti-racism policies in the current neo-liberal capitalist national and global political climate.

Chapter 11. **Concluding Remarks**

Drawing from the disciplines as diverse as epidemiology, sociology, anthropology, population genetics, physiology, psychology and public policy, this thesis has taken a transdisciplinary approach to the study of race, racism and stress as determinants of health for indigenous peoples, with a particular focus on Indigenous Australians. Commencing with an analysis of continuing racialisation in health research, it was shown that there is no consistent evidence that oppressed ethnoracial groups, who suffer disproportionately from type 2 diabetes, are especially genetically susceptible to this disease. Continued attribution of ethnoracial differences in health to genetics highlights both the limitations inherent to health inequalities research and the manifest need for such research to be attentive to environmental *and* genetic risk factors operating within and between ethnoracial groups.

One environmental factor that has been receiving increasing attention in health research is racism. Incorporating pertinent social theory and theorising in previous health research, a comprehensive definition of racism, as an important aspect of racialisation, was explored in relation to the concept of privilege/oppression. A diagrammatic representation of the possible aetiological relationship between racism and health was presented and the range of dimensions across which perceived racism (as experienced by oppressed ethnoracial groups) can be characterised, operationalised and measured as a health risk condition was also detailed.

A review of the current body of empirical research on self-reported racism as a determinant of health revealed a relationship between self-reported racism and ill-health for oppressed ethnoracial groups after adjustment for a range of confounders. Self-reported racism was most strongly and consistently associated with negative mental health outcomes and health-related behaviours. However, the majority of research in this emerging field has been published in the past five years and is limited by a dearth of cohort studies, a lack of psychometrically validated exposure instruments, poor conceptualisation and definition of racism, debate about the aetiologically-relevant period for self-reported racism and conflation of racism with stress.

In response to debate on the relationship between racism and stress in this literature, this thesis attempted to clarify to the plethora of conceptual approaches used in the study of stress and health as a first step towards comprehending how stress interacts with both racism and health. The contemporary conceptualisation of stress was detailed and approaches to studying stressors, the stress process, stress sequelae and the distribution of stressors in society were considered. This overview identified the need to better understand the role of stressors in the interaction between pathopsychological processes, emotion-/meaning-focused coping, health-related behaviours and social characteristics.

In the review of empirical research on self-reported racism as a health risk condition, it was noted that very little research has involved indigenous populations. It was also noted that there is a dearth of synthesised information on the empirical association between stress and chronic disease for fourth world indigenous populations. This latter lacunae was addressed through a review of the evidence for a relationship between stress and chronic disease for indigenous peoples (as well as African Americans). This review found that a range of chronic diseases were associated with psychosocial stress for these populations, with a much stronger association between stress and mental rather than physical health. A small body of intervention research suggested that transcendental meditation and group-oriented stress management may be effective in reducing psychosocial stress and its effects for African Americans and fourth world indigenous peoples, respectively. Such a review highlights the importance of stress as a phenomenon associated with racism which has a similarly detrimental impact on mental health for oppressed ethnoracial groups.

Utilising the conceptual work on approaches to operationalising racism discussed above, the identified scarcity of research on racism and health in relation to indigenous peoples was addressed through the development of an instrument to measure racism and its correlates as experienced by Indigenous Australians (the Measure of Indigenous Racism Experiences (MIRE)). This instrument demonstrated good face, content, psychometric and convergent validity in a pilot study involving 312 Indigenous Australians and showed considerable potential to be used as an instrument that effectively assesses multiple facets of racism together with a diverse set of responses/ reactions to this exposure for indigenous, and other oppressed ethnoracial groups.

Of these 312 Indigenous Australians, 70% reported some experience of inter-personal racism, with racism from other Indigenous people, in employment, in public and from service providers being most commonly reported. Outer-directed reactions/responses to inter-personal racism were also more commonly reported by participants than inner-directed reactions/responses. Associations between racism, ethnoracial identity, stress and health were also examined among this group of Indigenous Australians. Strong and consistent associations were found between racism and chronic stress as well as between racism and depression (CES-D), poor/fair self-assessed health status/poor general mental health (SF-12) and a marker of CVD risk (homocysteine), respectively. The bulk of these associations were driven by inter-personal racism and responses/reactions to this racism rather than by internalised/ systemic racism or aspects of racial identity. There was also evidence that the association between inter-personal racism and poor mental health outcomes were mediated by somatic and inner-directed disempowered reactions to racism as well as by chronic stress and a range of psychosocial characteristics.

Such an identified relationship between self-reported racism and ill-health has significant implications given the high burden of ill-health experienced by Indigenous Australians. After adjusting for age differences between the Indigenous and non-Indigenous populations in Australia, Indigenous people were almost twice as likely as non-Indigenous people to report their health as fair/poor and were 1.3 times more likely than non-Indigenous people to report heart disease and/or circulatory problems (Australian Bureau of Statistics, 2006). Although there are no national data available on the prevalence of either depression or poor mental health as measured by the SF-12, Indigenous Australians are up to twice as likely to be hospitalised for mental and behavioural disorders as other Australians (Australian Bureau of Statistics & Australian Institute of Health and Welfare, 2005). The strong association between racism and conditions which disproportionately affect Indigenous Australians (and especially the high degree of variance in these outcomes explained by racism) suggests that further research on self-reported racism is warranted and that reducing exposure to racism and/or reducing its harmful effects may be of considerable health benefit.

An examination of possible approaches to addressing racism for Indigenous Australians was undertaken in the penultimate chapter of this thesis. The theoretical issues pertinent to

the empirical study of, and public policy relevant to, anti-racism were discussed along with empirical findings from social psychology on effective approaches to anti-racism at the cognitive, individual, inter-personal and societal level. Recommendations for implementing these approaches through institutional and legal policies in the domains of education and child-rearing, public service, law enforcement and media as well as monitoring racism and promoting anti-racism in civil society, were also presented. To conclude, strategies for engendering political will to combat racism in the current neo-liberal capitalist climate were briefly considered.

It is hoped that the body of research presented in this thesis has contributed to an improved understanding of the role played by race, racism and stress as determinants of health for indigenous peoples and other oppressed ethnoracial groups. Furthermore, as explored in relation to Indigenous Australians above, it is hoped that insights into the role of racism in driving population health can be utilised alongside anti-racism research to combat the divisive and insidious phenomenon of racism

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Appendix A: DRUID Study Questionnaires

A.1 DRUID Questionnaire 1

ID:

DRUID

FIGHT AGAINST DIABETES IN OUR COMMUNITY

Questionnaire 1

The following questions ask about different things to do with you, your health and your lifestyle. Every person taking part in this study will be asked the same questions. ***ALL YOUR ANSWERS TO THE QUESTIONS ARE STRICTLY CONFIDENTIAL.***

Your answers will help us understand more about diabetes and other chronic health conditions like heart disease. This information will be very useful in developing better ways of helping people with these health conditions and in stopping them from developing in those who don't have them.

For each question, please place a tick in the box for the correct answer, like this: ☑.

If you don't understand any of the questions, or you're not sure how to answer, please ask a member of the DRUID staff. If there are any questions you don't want to answer, just leave them blank.

Thank you for your help!

Section 1. Medical history

This section is about health conditions you may have experienced.

Have you ever been told by a doctor or other health professional that you had any of the following conditions?	No	Yes	Don't know
1. Heart attack	☐	☐	☐
2. Rheumatic heart disease	☐	☐	☐
3. Angina	☐	☐	☐
4. Other heart disease	☐	☐	☐
5. Stroke	☐	☐	☐
6. Kidney disease	☐	☐	☐
7. Emphysema or chronic bronchitis	☐	☐	☐
8. Asthma	☐	☐	☐
9. Cancer	☐	☐	☐
10. Eye/sight problems	☐	☐	☐
11. Have you ever been told by a doctor or other health professional that you have high blood pressure or hypertension?	☐ go to Question 13	☐	☐ go to Question 13
12. Are you currently taking tablets for high blood pressure?	☐	☐	☐
13. Cholesterol and triglycerides are fats in the blood. Have you ever been told by a doctor or other health professional that your blood cholesterol or triglycerides are high?	☐ go to Question 15	☐	☐ go to Question 15
14. Are you currently taking tablets to lower your cholesterol/ triglycerides?	☐	☐	☐

15. Have you had any infections (such as a cold or 'flu or a skin infection or boil) in the last 2 weeks?	☐	☐	☐

Section 2. Diabetes

1. Have you ever been told by a doctor or other health professional that you have diabetes or high sugar levels in your blood or urine?

- ☐ No **(⇒ go to Question 16)**
- ☐ Yes, diabetes
- ☐ Yes, high sugar levels **(⇒ go to Question 15)**
- ☐ Don't know **(⇒ go to Question 16)**

2. What type of diabetes were you told you had?

- ☐ Type 1(Insulin-dependent diabetes mellitus)
- ☐ Type 2 (Non-insulin dependent diabetes mellitus)
- ☐ Gestational diabetes (Diabetes during pregnancy)
- ☐ Diabetes – type unknown
- ☐ Other type, please specify:_____

3. Do you still have diabetes?

- ☐ No **(⇒ go to Question 15)**
- ☐ Yes
- ☐ Don't know/not sure

4. Are you currently taking any medication for your diabetes?

- ☐ No **(⇒ go to Question 7)**
- ☐ Yes

5. How often do you take your medication as instructed?

- ☐ All or almost all of the time **(⇒ go to Question 7)**
- ☐ More than half the time **(⇒ go to Question 7)**
- ☐ About half the time
- ☐ Less than half the time
- ☐ Never or rarely

6. What keeps you from taking your medication as instructed? MARK ALL THAT APPLY.

- ☐ I forget to take it or I remember at the wrong times
- ☐ I don't like the way it makes me feel
- ☐ I don't need to take it
- ☐ It's physically difficult
- ☐ Other, please specify:_____

7. Do you currently use any blood testing equipment or syringes for insulin injection?

- ☐ No **(⇒ go to Question 9)**
- ☐ Yes, blood testing equipment
- ☐ Yes, syringes for injecting insulin
- ☐ Yes, both

8. Where do you usually get your blood testing equipment or syringes?

- ☐ Diabetes Australia
- ☐ Danila Dilba
- ☐ Other clinic or doctor's office
- ☐ Pharmacy
- ☐ Other, please specify:_____

9. Do you monitor your blood sugar levels at home?

- ☐ No
- ☐ Yes, at least once a week
- ☐ Yes, every couple of months or more, but not every week
- ☐ Yes, but less often than every couple of months

10. What type of health care provider mainly looks after your diabetes?

- ☐ None **(⇒ go to Question 14)**
- ☐ General practitioner or clinic doctor
- ☐ Diabetes specialist or other medical specialist
- ☐ Nurse or health worker
- ☐ Other, please specify:_____

11. About how often do you visit this person for your diabetes?

- ☐ Never
- ☐ Once every 5 years
- ☐ Once every 2-3 years
- ☐ 1 or 2 times a year
- ☐ 3 to 4 times a year
- ☐ 5 or more times a year

12. Are there any other health care providers who help look after your diabetes?

- ☐ No **(⇒ go to Question 14)**
- ☐ Yes

13. What type(s) of other health care providers help look after your diabetes? MARK ALL THAT APPLY, BUT DO NOT INCLUDE THE PRIMARY CAREGIVER REPORTED IN QUESTION 10.

- ☐ General practitioner or clinic doctor
- ☐ Diabetes specialist or other medical specialist
- ☐ Nurse or health worker
- ☐ Other, please specify:_____

14. Within the past 12 months have you visited or been visited by any of these health professionals? MARK ALL THAT APPLY.

- ☐ Diabetes educator
- ☐ Dietician
- ☐ Podiatrist
- ☐ Diabetic foot clinic
- ☐ Community health nurse

15. At what age were you first told you had diabetes or high sugar levels?

		Years		**Don't know/not sure**	☐

⇒ Go to Section 3, Dental health

16. Have you ever been tested for diabetes?

- ☐ No
- ☐ Yes
- ☐ Don't know

Section 3. Dental health

The next questions are about your teeth and gums. Studies have shown that people with diabetes often have poorer dental health than other people.

1. Do you still have any of your own teeth?

- ☐ No **(⇒ go to Question 4)**
- ☐ Yes, most
- ☐ Yes, some

2. In the last 12 months, have you had any loose teeth?

- ☐ No
- ☐ Yes

3. In the last 12 months, did you have any teeth taken out by a dentist or other dental professional?

- ☐ No
- ☐ Yes

4. In the last 12 months, did your gums or teeth cause you any pain or discomfort?

- ☐ No
- ☐ Yes, a lot

☐ Yes, a little

5. How often have you had to avoid eating some foods because of problems with your teeth or gums during the last 12 months?

☐ Never during the last 12 months
☐ Hardly ever
☐ Sometimes
☐ Often
☐ Very often
☐ Don't know

Section 4. Health Insurance

The next questions are about health insurance. Having access to health insurance can affect the level of health care people seek or receive, and this can affect health.

1. Do you have private health insurance?

☐ No
☐ Yes, hospital cover only
☐ Yes, extras cover only
☐ Yes, both hospital and extras cover
☐ Yes, but not sure what type of cover
☐ Don't know

2. Do you have a Medicare card?

☐ No
☐ Yes **(⇒ go to Question 5)**
☐ Don't know/not sure

3. Is this because you are included on someone else's card, such as a parent or partner?

☐ No
☐ Yes **(⇒ go to Question 5)**
☐ Don't know/not sure

4. Does your doctor or health clinic have your Medicare number recorded (so you can access Medicare benefits)?

☐ No
☐ Yes
☐ Don't know/not sure

5. Do you have a Health Care Card?

☐ No
☐ Yes
☐ Don't know/not sure

The next sections are about your lifestyle such as drinking alcohol, smoking cigarettes, doing physical activity and what you eat and drink. These things have been shown to be related to health conditions like diabetes and heart disease.

Section 5. Alcohol use

1. Have you ever drunk alcohol?

☐ No **(⇒ go to Section 6, Smoking)**
☐ Yes

2. How often do you drink alcohol now?

☐ I never drink alcohol **(⇒ go to Section 6, Smoking)**
☐ I rarely drink alcohol
☐ Less than once a week
☐ 1 to 2 days a week
☐ 3-4 days a week
☐ 5-6 days a week
☐ Every day

3. On a day when you drink alcohol, how many *standard drinks* do you usually have?

☐ 1 to 2 drinks per day
☐ 3 to 4 drinks per day
☐ 5 to 8 drinks per day
☐ 9 or more drinks per day

(Note: a standard drink is equivalent to 1 handle (285 ml) of full strength beer, 1 can (375 ml) of mid-strength beer, 2 handles (285 mls each) of light beer, or a small serve (100 ml) of wine or champagne. A can of pre-mixed spirits is approximately 1.5 standard drinks, and a bottle of alcoholic soda (300 ml) is about 1.2 standard drinks.)

4. How often do you have 5 or more *standard drinks* of alcohol on one occasion?

☐ Never
☐ Less than once a month
☐ About once a month
☐ About once a week
☐ More than once a week

Section 6. Smoking

1. In terms of smoking tobacco/cigarettes, would you say you are ….?

☐ I've never smoked **(⇒ go to Question 7)**

☐ A regular smoker
☐ An occasional or social smoker
☐ An ex-smoker **(⇒ go to Question 5)**

2. At what age did you start smoking?

		Years	☐	**Don't know/not sure**

3. How often do you smoke now?

☐ Every day
☐ Some days a week
☐ Less than once a week

4. On the days that you smoke, how many cigarettes do you usually have?

☐ Less than 5 **(⇒ go to Question 7)**
☐ 5 to 14 **(⇒ go to Question 7)**
☐ 15 to 25 **(⇒ go to Question 7)**
☐ 25 or more **(⇒ go to Question 7)**

5. What was the MAIN reason you gave up smoking? PLEASE PICK ONLY ONE REASON.

☐ Cost: too expensive/couldn't afford it/wanted to save money
☐ Health reasons: bad for me
☐ Physical appearance: affected the way I looked
☐ Fitness: to be better at sport/improve fitness/couldn't do things I used to do
☐ For other people: partner/spouse/kids/baby/family members/friends
☐ Social exclusion: I felt excluded or left out/no one else was smoking any more
☐ Other, please specify: _____

6. How long has it been since you stopped smoking?

☐ Less than a month
☐ 1 to 12 months
☐ 1 to 5 years
☐ More than 5 years

7. In terms of smoking marijuana, would you say you are ….?

☐ I've never smoked
☐ A regular smoker
☐ An occasional or social smoker
☐ An ex-smoker
☐ Prefer not to answer

Section 7. Physical activity

The following questions are about four types of activity that you might have done last week:

- ***walking**, for recreation or exercise, or to get from place to place.*
- ***vigorous gardening or heavy work** around the yard.*
- ***vigorous physical activity**, things that make you breathe harder or puff and pant like aerobics, competitive sport, vigorous cycling, running, and swimming.*
- ***moderate physical activity**, like social tennis, moderate exercise classes, recreational swimming, and dancing.*

*For each type of activity, you will be asked about the **number of times** you did that type of activity in the past week, and the **total amount of time** you spent doing it. For example, suppose last week a person did one session of vigorous physical activity on Monday that lasted 40 minutes, one session of vigorous physical activity on Thursday for 30 minutes and two sessions of vigorous physical activity on Saturday, the first one for 25 minutes and the second for 35 minutes. This would mean that this person did vigorous physical activity 4 times (1+1+2) in the last week, and the total amount of time spent was 130 minutes (40 + 30 + 25 + 35), or 2 hours and 10 minutes.*

REMEMBER, IF YOU NEED ASSISTANCE ANSWERING THESE QUESTIONS, PLEASE ASK A MEMBER OF THE DRUID TEAM.

Walking

1. In the last week, *how many times* have you *walked* continuously, *for at least 10 minutes*, for recreation, exercise, or to get to or from places?

_____ times

2. What do you estimate was the *total time* that you spent *walking* in this way in the last week? (Only include times you walked for at least 10 minutes.)

hours minutes

Vigorous gardening and heavy work around the yard

3. In the last week, *how many times* did you do any *vigorous gardening or heavy work around the yard*, which made you breathe harder or puff and pant?

_____ times

4. What do you estimate was the *total time* that you spent doing *vigorous gardening or heavy work* around the yard in the last week?

hours minutes

The next questions **exclude** household chores, gardening or yard work:

Vigorous physical activity

5. In the last week, *how many times* did you do any *vigorous physical activity* which made you breathe harder or puff and pant? (e.g. jogging, cycling, aerobics, competitive tennis)

_____ times

6. What do you estimate was the *total time* that you spent doing this *vigorous physical activity* in the last week?

hours minutes

Moderate physical activity

7. In the last week, *how many times* did you do any other more *moderate physical activities* that you have not already mentioned? (e.g. gentle swimming, social tennis, golf)

_____ times

8. What do you estimate was the *total time* that you spent doing these *moderate activities* in the last week?

hours minutes

9. Was last week a typical week for you for physical activity?

- ☐ Yes
- ☐ No, I did less last week than I usually do
- ☐ No, I did more last week than I usually do

10. Which of the following best describes how physically active your job is?

- ☐ Don't have a job
- ☐ Work that involves mostly sitting or standing
- ☐ Work that involves mostly walking
- ☐ Work that involves mostly heavy labour or physically demanding work

The next two questions are about the amount of time you spend sitting down while doing things like visiting friends, driving, reading, watching television, or working at a desk or

computer. Include time spent at work and time spent outside of work. Only include the time when you are awake. Include time spent sitting while eating meals.

11. On a usual WEEKDAY, how many hours in total do you usually spend sitting down while doing things like visiting friends, driving, reading, watching television, or working at a desk or computer?

- ☐ Less than 2 hours
- ☐ 2 to less than 5 hours
- ☐ 5 to less than 8 hours
- ☐ 8 to less than 12 hours
- ☐ 12 or more hours

12. On a usual WEEKEND DAY, how many hours in total do you usually spend sitting down while doing things like visiting friends, driving, reading, watching television, or working at a desk or computer?

- ☐ Less than 2 hours
- ☐ 2 to less than 5 hours
- ☐ 5 to less than 8 hours
- ☐ 8 to less than 12 hours
- ☐ 12 or more hours

Section 8. Weight and nutrition

1. With regard to your weight, do you consider yourself to be:

- ☐ Underweight
- ☐ The right weight
- ☐ Slightly overweight
- ☐ Very overweight

2. How many serves of vegetables do you usually eat each day? (Note: a 'serve' is ½ a cup of cooked vegetables or 1 cup of salad vegetables.)

- ☐ Don't eat vegetables /none
- ☐ 1 serve or less
- ☐ 2-3 serves
- ☐ 4-5 serves
- ☐ 6 or more serves
- ☐ Don't know /not sure

3. How many serves of fruit do you usually eat each day? (Note: a 'serve' = 1 medium piece or 2 small pieces of fruit, 1 cup of diced pieces of fruit or 1 tablespoon of dried fruit)

- ☐ Don't eat fruit /none
- ☐ 1 serve or less
- ☐ 2-3 serves

☐ 4-5 serves
☐ 6 or more serves
☐ Don't know /not sure

How often do you usually have the following foods and drinks?

	Never or rarely	Less than once a week	1 time per week	2-3 times per week	4-6 times per week	1 time per day	2 times per day	3 or more times per day
4. Fish (this includes take-away fish)	☐	☐	☐	☐	☐	☐	☐	☐
5. Take away (such as fried foods, pies, hot chips, hamburgers, pizza, etc.)	☐	☐	☐	☐	☐	☐	☐	☐
6. Snacks such as packet potato chips, biscuits (sweet or savoury), chocolate, lollies, cakes and sweet pastry	☐	☐	☐	☐	☐	☐	☐	☐
7. Soft drinks, cordial or sports drinks	☐	☐	☐	☐	☐	☐	☐	☐
8. Ice coffee or other flavoured milk drinks	☐	☐	☐	☐	☐	☐	☐	☐
8a. Plain milk drinks	☐	☐	☐	☐	☐	☐	☐	☐
9. Fruit or vegetable juice	☐	☐	☐	☐	☐	☐	☐	☐
10. Coffee	☐	☐	☐	☐	☐	☐	☐	☐
11. Tea	☐	☐	☐	☐	☐	☐	☐	☐
12. Water	☐	☐	☐	☐	☐	☐	☐	☐

The next question is about meals that are prepared outside your home, regardless of where you eat them. Include food prepared at takeaways, restaurants, food stalls, canteens, cafes, sandwich shops, etc. Do not include meals prepared at someone else's home, such as a friend's or relative's.

13. Which of the following best describes how often you eat meals prepared outside the home? Remember to include breakfast, lunch and dinner.

☐ Never or less than once a week
☐ Once or twice a week
☐ Three to six times a week
☐ Once a day
☐ Twice a day
☐ Three or more times a day

14. Who does most of the cooking in your home?

☐ Self
☐ Partner/spouse
☐ Parents (yours or your partner's)
☐ Child (yours or your partner's)
☐ Other, please specify:_____

15. What is the main source of groceries in your home?

- ☐ Supermarket (e.g. Woolies, Coles, BiLo, Nightcliff, 5-Star)
- ☐ Convenience store or corner store
- ☐ Markets or food co-ops
- ☐ Other, please specify:_____

Section 9. Subjective social status (MacArthur Scale of Subjective Social Status)

Question 1.

Community comparison of social standing

Think of this ladder as representing where people stand in your community. People define community in different ways; please define it in whatever way is most meaningful to you. Imagine everyone in your community is standing somewhere on this ladder. At the TOP of the ladder are the people who have the highest standing in your community. At the bottom are the people who have the lowest standing in your community. The higher up you are on this ladder, the closer you are to those at the very top. The lower you are, the closer you are to those at the very bottom.

Where would you place yourself on this ladder, compared to others in your community? Please place a large "X" directly on the rung where you think you stand.



Top of the ladder = highest standing in your community
Bottom of the ladder = lowest standing in your community

Question 2.

Australian comparison of socio-economic status

Think of this ladder as representing where the people stand in Australia. At the TOP of the ladder are the people who are the best off—those who have the most money, the most education, and the most respected jobs. At the BOTTOM are the people who are the worst off—who have the least money, least education, and the least respected jobs or no job. The higher up you are on this ladder, the closer you are to the people at the very top. The lower you are, the closer you are to the people at the very bottom.

Where would you place yourself on this ladder, compared to all the other people in Australia? Please place a large "X" directly on the rung where you think you stand.



Top of the ladder = best off – most money, most education, most respected jobs
Bottom of the ladder = worst off – least money, least education, least respected jobs or no job

Section 10. Household

1. The place you are now living in—is it …?

- ☐ Fully owned or being purchased by you or someone in the household
- ☐ Being rented
- ☐ Being occupied rent-free
- ☐ Other, please specify:_____

2. What is your marital status?

- ☐ Never married
- ☐ Married
- ☐ Defacto
- ☐ Separated
- ☐ Divorced
- ☐ Widowed

3. Which of the following best describes your household?

- ☐ One person living on their own
- ☐ One adult living with children (include dependent, non-dependent, other people's children)
- ☐ Married or defacto couple (no children)
- ☐ Married or defacto couple with children (include dependent, non-dependent, other people's children)
- ☐ Shared/group household
- ☐ Other type of household, please specify: _____

4. How many people usually live in your household? Exclude temporary visitors.

_____ Adults (aged 15+)

\+ _____ Children under 15

= _____ Total number of people

5. How many of these people are *Aboriginal or Torres Strait Islander* people?

_____ Aboriginal and Torres Strait Islander adults (aged 15+)

\+ _____ Aboriginal and Torres Strait Islander children under 15

= _____ Total number of Aboriginal and Torres Strait Islander people

The next questions are about education, income and employment. Different health problems are associated with different lifestyle patterns. The answers to these questions will help us to understand more about patterns of health in the Darwin Indigenous community.

Section 11. Education

1. At what age did you leave school?

- ☐ Still at school **(⇒ go to Question 4)**
- ☐ Never went to school **(⇒ go to Question 3)**
- ☐ Under 14 years
- ☐ 14 Years
- ☐ 15 years
- ☐ 16 years
- ☐ 17 years
- ☐ 18 years or over

2. What is the highest year of school that you have finished?

- ☐ Year 12 or equivalent
- ☐ Year 11
- ☐ Year 10
- ☐ Year 9
- ☐ Year 8
- ☐ Year 7
- ☐ Year 6 or lower

3. Which of these describes the highest qualification you have completed?

- ☐ No formal qualifications
- ☐ Yr 10 Certificate (or equivalent)
- ☐ Higher School or Yr 12 Certificate (or equivalent)
- ☐ Trade/apprenticeship (eg Hairdresser, Chef)
- ☐ Certificate/diploma (eg Child Care, Technician)
- ☐ University degree
- ☐ Higher University degree (eg Grad Dip, Masters, PhD)

4. Are you currently studying at a TAFE, college, university or other educational institution?

- ☐ No
- ☐ Yes, as a part-time student
- ☐ Yes, as a full-time student

Section 12. Employment

1. Which of the following describes your current employment status? MARK ALL THAT APPLY.

- ☐ Working full-time (not CDEP)
- ☐ Working part-time (not CDEP)
- ☐ CDEP
- ☐ Casual work
- ☐ Home duties
- ☐ Full-time student
- ☐ Part-time student
- ☐ Retired
- ☐ Not working (but not retired)
- ☐ Permanently unable to work / ill
- ☐ Other, please specify: _____

2. What is your occupation?_____ ☐ No paid job

(Please provide as much detail as possible)

3. What is your partner's occupation?_____

(Please provide as much detail as possible)

☐ No partner ☐ Partner has no paid job ☐ Don't know/not sure

Section 13. Income

1. How would you say that you manage on the income you have available?

- ☐ It is impossible
- ☐ It is difficult all the time
- ☐ It is difficult some of the time
- ☐ It is not too bad
- ☐ It is easy

2. What is your *main* source of income? Please mark only one answer.

- ☐ Don't have an income
- ☐ Supported by parents, partner or other family
- ☐ Wages or salary
- ☐ Own business or share in partnership

☐ Any government pension or cash benefit (give examples; include ABSTUDY)
☐ Superannuation / Investment / Interest
☐ Other income, please specify: _____

3. Which of the following lines best describes your total household income *before tax* (gross income)? Note: If you are sharing a household with someone who is not a partner and you live independently then record your income only.

	Per week	Per fortnight	Per year
☐	$1,500 or more	$3000 or more	$78,000 or more
☐	$800 - $1,499	$1600-$2999	$41,600 - $77,999
☐	$600 - $799	$1200-$1599	$31,200 - $41,599
☐	$400 - $599	$800-$1199	$20,800 - $31,199
☐	$200 - $399	$400-$799	$10,400 - $20,799
☐	$80 - $199	$160-$399	$4,160 - $10,399
☐	$1 - $79	$2-$159	$52 - $4,159
☐	Don't know		

That brings us to the end of this set of questions. Thank you very much for your time!

Please let us know if you have any comments:_____

A.2 DRUID Questionnaire 2

ID:



Questionnaire 2

*These questions are about things which can affect people's health - things like stressful life events, depression, racial discrimination, social support and identification with culture. There are no right or wrong answers to any of these questions, and **ALL YOUR ANSWERS ARE CONFIDENTIAL.***

For each question, please place a tick in the box for the correct answer, like this: ☑ If you don't understand any of the questions, or you're not sure how to answer, please ask a member of the DRUID staff. If there are any questions you don't want to answer, just leave them blank.

Section 1. Culture and identity

This first section is to do with culture and identity. In the questions, the word Indigenous is used to mean Aboriginal and Torres Strait Islander people, including people who are both Aboriginal and Torres Strait Islander.

	Never	Once a year	Once a month	Once a week	Once a day	Once an hour	Constantly
1. How often do you think about being Indigenous?	☐	☐	☐	☐	☐	☐	☐
		No, hardly anybody	**No, not many people**	**Some people**	**Yes, most people**	**Yes, everyone**	**Unsure**
2. Do the people that you mix with know that you are Indigenous?		☐	☐	☐	☐	☐	☐
3. Do people you meet for the first time know that you are Indigenous?		☐	☐	☐	☐	☐	☐
		No	**Yes**				
4. Do you identify with any clan, tribal or language groups?		☐	☐ →	5. Which one(s)?_____			
		No	**Yes**				
6. Do you recognise any area(s) as your homelands or traditional country?		☐	☐ →	7. What is the name of your homelands/ traditional country? _____			
		No	**Yes**	**Don't know**			
8. Were any of your grandparents non-Indigenous?		☐	☐	☐			
		No	**Yes**	**Prefer not to answer**			
9. The next question is about the Stolen Generations. Do you consider yourself to be a part of or member of the Stolen Generations?		☐	☐	☐			

Section 2. General health and wellbeing SF-12v2™ Health Survey © 2000 by QualityMetric Incorporated - All rights reserved. SF-12v2™. Is a trademark of QualityMetric Incorporated.

This section of the questionnaire asks for your views about your health. This information will help keep track of how you feel and how well you are able to do your usual activities. For each of the following questions, please mark a √ in the one box that best describes your answer.

1. In general, would you say your health is:	☐ Excellent	☐ Very good	☐ Good	☐ Fair	☐ Poor

2. The following questions are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?	Yes, limited a lot	Yes, limited a little	No, not limited at all
2a. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf	☐	☐	☐
2b. Climbing several flights of stairs	☐	☐	☐

3. During the past 4 weeks, how much of the time have you had any of the following problems with your work or other regular daily activities as a result of your physical health?	All of the time	Most of the time	Some of the time	A little of the time	None of the time
3a. Accomplished less than you would like	☐	☐	☐	☐	☐
3b. Were limited in the kind of work or other activities	☐	☐	☐	☐	☐

4. During the past 4 weeks, how much of the time have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?	All of the time	Most of the time	Some of the time	A little of the time	None of the time
4a. Accomplished less than you would like	☐	☐	☐	☐	☐
4b. Did work or other activities less carefully than usual	☐	☐	☐	☐	☐

5. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

☐ Extremely	☐ Quite a bit	☐ Moderately	☐ A little bit	☐ Not at all

6. These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling.

How much of the time during the past 4 weeks …	All of the time	Most of the time	Some of the time	A little of the time	None of the time
6a. Have you felt calm and peaceful?	☐	☐	☐	☐	☐
6b. Did you have a lot of energy?	☐	☐	☐	☐	☐
6c. Have you felt downhearted and depressed?	☐	☐	☐	☐	☐

7. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives, etc.)?

All of the time	Most of the time	Some of the time	A little of the time	None of the time
☐	☐	☐	☐	☐

Section 3. Stress

*The following is a list of things that happen to many people on a **sudden, one-off or unexpected basis**, and that you may have experienced **in the last 12 months**. Please indicate whether these have happened and, if so, how much they affected you, by placing a tick mark in the correct box. (Note: You can include things that happened to someone else in your family or friends but which affected you personally.)*

	No	Yes, had minimal or no effect	Yes, affected me somewhat	Yes, affected me a great deal	Prefer not to answer
1. Marriage or relationship breakup	☐	☐	☐	☐	☐
2. Death of husband / wife or partner	☐	☐	☐	☐	☐
3. Death of a child, close family member or close friend	☐	☐	☐	☐	☐
4. Laid off or fired at work	☐	☐	☐	☐	☐
5. Witness to violence	☐	☐	☐	☐	☐
6. Victim of abuse or crime	☐	☐	☐	☐	☐
7. Member of family sent to jail	☐	☐	☐	☐	☐
8. Serious illness	☐	☐	☐	☐	☐
9. Serious accident or injury	☐	☐	☐	☐	☐
10. Other events, please specify:_____	☐	☐	☐	☐	☐

*The following list includes things that may have been affecting you on **a regular or continuing basis over the past 12 months**. Please indicate whether these have happened and, if so, how much they affected you, by placing a tick mark in the correct box. (Note: You can include things that happened to someone else in your family or friends but which affected you personally.)*

	No	Yes, had minimal or no effect	Yes, affected me somewhat	Yes, affected me a great deal	Prefer not to answer
11. Trouble at work, or with a boss or co-worker	☐	☐	☐	☐	☐
12. Trouble at school or with a teacher	☐	☐	☐	☐	☐
13. Trouble with relationships	☐	☐	☐	☐	☐
14. Alcohol or drug-related problems	☐	☐	☐	☐	☐
15. Overcrowding at home	☐	☐	☐	☐	☐
16. Discrimination	☐	☐	☐	☐	☐
17. Family members in prison	☐	☐	☐	☐	☐
18. Pressure to fulfill cultural responsibilities	☐	☐	☐	☐	☐
19. Pressure to fulfill family or carers' responsibilities	☐	☐	☐	☐	☐
20. Worry about family members	☐	☐	☐	☐	☐
21. Having to rely on other people for assistance with everyday needs	☐	☐	☐	☐	☐
22. Problems with access to land/country	☐	☐	☐	☐	☐
23. Gambling problems	☐	☐	☐	☐	☐
24. Money problems	☐	☐	☐	☐	☐
25. Mental or emotional problems	☐	☐	☐	☐	☐
26. Health problems	☐	☐	☐	☐	☐
27. Problems finding a job	☐	☐	☐	☐	☐
28. Problems settling into a new job	☐	☐	☐	☐	☐
29. Abuse or violence	☐	☐	☐	☐	☐
30. Problems with the police or the law	☐	☐	☐	☐	☐
31. Other troubles not listed, please describe:_____	☐	☐	☐	☐	☐

Section 4. Discrimination

The following questions are about your experiences of being Aboriginal or Torres Strait Islander. In the questions the word 'Indigenous' is used to mean Aboriginal or Torres Strait Islander people including people who are both Aboriginal and Torres Strait Islander.

Some questions ask about your experiences of 'being treated unfairly'. People can be treated unfairly for all sorts of reasons. For these questions we only want to find out about times when you were treated unfairly because you are Aboriginal or Torres Strait Islander.

Being 'treated unfairly' means being treated as if you were inferior, rudely, with disrespect, being ignored, insulted, harassed, stereotyped, discriminated against or having unfair assumptions made about you.

1. How often are you treated unfairly because you are Indigenous in each of the following situations?	**Never**	**Hardly ever**	**Sometimes**	**Often**	**Very often**	**This doesn't apply to me**
a. At work or on the job?	☐	☐	☐	☐	☐	☐
b. At home, by neighbours or at somebody else's house?	☐	☐	☐	☐	☐	☐
c. At school, university or other academic setting?	☐	☐	☐	☐	☐	☐
d. While doing sporting, recreational or leisure activities?	☐	☐	☐	☐	☐	☐
e. By the police, security personnel, lawyers or in a court of law?	☐	☐	☐	☐	☐	☐
f. By doctors, nurses or other staff at hospitals or doctors' surgeries?	☐	☐	☐	☐	☐	☐
g. By staff of government agencies like Centrelink, ATSIC, etc.?	☐	☐	☐	☐	☐	☐
h. By staff at restaurants, bars, shops, banks, motels, real estate agents, in taxis or when getting any other services?	☐	☐	☐	☐	☐	☐
i. By other people on the street, at shopping centres, sporting events, concerts, nightclubs etc?	☐	☐	☐	☐	☐	☐
j. By other Indigenous people?	☐	☐	☐	☐	☐	☐

2. When you are treated unfairly because you are Indigenous how often do you:	**Never**	**Hardly ever**	**Sometimes**	**Often**	**Very often**
a. Ignore it, forget about it or accept it as a fact of life?	☐	☐	☐	☐	☐
b. Try to avoid it in the future?	☐	☐	☐	☐	☐
c. Try to change the way you are or things that you do so that it won't happen again?	☐	☐	☐	☐	☐
d. Try to do something about the people who did it or the situation in which it happened?	☐	☐	☐	☐	☐
e. Talk to other people like family or friends about it, or write, draw, sing or paint about it?	☐	☐	☐	☐	☐
f. Keep it to yourself?	☐	☐	☐	☐	☐
g. Feel ashamed, humiliated, anxious or fearful?	☐	☐	☐	☐	☐
h. Feel angry, annoyed or frustrated?	☐	☐	☐	☐	☐
i. Feel amused, contemptuous or sorry for the person who did it?	☐	☐	☐	☐	☐
j Feel powerless, hopeless or depressed?	☐	☐	☐	☐	☐
k. Get a headache, an upset stomach, tensing of your muscles, or a pounding heart?	☐	☐	☐	☐	☐

3. Please indicate how much you agree or disagree with each of the following statements. There are no right or wrong answers.	**Strongly agree**	**Agree**	**Neither agree nor disagree**	**Disagree**	**Strongly disagree**
a. I feel accepted by other Indigenous people.	☐	☐	☐	☐	☐
b. Indigenous people have less opportunities than other Australians.	☐	☐	☐	☐	☐
c. Indigenous people should try to think and act more like other Australians.	☐	☐	☐	☐	☐
d. I feel good about being an Indigenous person.	☐	☐	☐	☐	☐
e. Other Australians think that Indigenous people are better off because they get special treatment from the government.	☐	☐	☐	☐	☐
f. Other Australians think they are better than Indigenous people.	☐	☐	☐	☐	☐
g. There is hardly ever anything good about Indigenous people in the media (TV, radio, newspapers, etc.).	☐	☐	☐	☐	☐

Section 5. Depression

This section is about ways you might have felt or behaved. These feelings can be related to your physical health or emotional wellbeing. Please indicate how often you have felt this way ***during the past week****.*

During the past week:	**Rarely or not at all (less than 1 day)**	**Some or a little of the time (1-2 days)**	**Occasionally or a moderate amount of time (3-4 days)**	**Most or all of the time (5-7 days)**	**Prefer not to answer**
1. I was bothered by things that usually don't bother me.	☐	☐	☐	☐	☐
2. I did not feel like eating; my appetite was poor.	☐	☐	☐	☐	☐
3. I felt down and even my family and friends weren't able to help me feel better.	☐	☐	☐	☐	☐
4. I felt that I was just as good as other people.	☐	☐	☐	☐	☐
5. I had trouble keeping my mind on what I was doing.	☐	☐	☐	☐	☐
6. I felt depressed.	☐	☐	☐	☐	☐
7. I felt that everything I did was an effort.	☐	☐	☐	☐	☐
8. I felt hopeful about the future.	☐	☐	☐	☐	☐
9. I thought my life had been a failure.	☐	☐	☐	☐	☐
10. I felt fearful.	☐	☐	☐	☐	☐
11. My sleep was restless.	☐	☐	☐	☐	☐
12. I was happy.	☐	☐	☐	☐	☐
13. I talked less than usual.	☐	☐	☐	☐	☐
14. I felt lonely.	☐	☐	☐	☐	☐
15. People were unfriendly.	☐	☐	☐	☐	☐
16. I enjoyed life.	☐	☐	☐	☐	☐
17. I had bouts of crying.	☐	☐	☐	☐	☐
18. I felt sad.	☐	☐	☐	☐	☐

19. I felt that people disliked me.	☐	☐	☐	☐	☐
20. I could not get "going".	☐	☐	☐	☐	☐

	Rarely or not at all	Some of the time	Often	Most of the time
21. Now think about the past year. How often have you felt depressed or sad in this past year?	☐	☐	☐	☐

Section 6. Control

During the past 4 weeks, how much of the time did you feel a lack of control with …	Never or rarely	Sometimes	Often	Very often	Not applicable
1. your life in general?	☐	☐	☐	☐	
2. your financial situation?	☐	☐	☐	☐	
3. your personal life?	☐	☐	☐	☐	
4. your job security?	☐	☐	☐	☐	☐
5. your work life (paid or voluntary)?	☐	☐	☐	☐	☐
6. your health?	☐	☐	☐	☐	

Section 7. Social support

The next questions are about relationships with family and friends.

	Never	Less than once a month	Once a month	A few times a month	A few times a week	Every day
1. How often do you talk on the phone or get together with friends or relatives who do not live with you?	☐	☐	☐	☐	☐	☐

	Not much	Some	A lot
2. How much do your friends or relatives really care about you?	☐	☐	☐
3. How much do they understand the way you feel about things?	☐	☐	☐
4. How much do they appreciate you?	☐	☐	☐
5. How much can you rely on them for help if you have a serious problem?	☐	☐	☐
6. How much can you talk to them about your worries?	☐	☐	☐
7. How much can you relax and be yourself around them?	☐	☐	☐

	Rarely/never	Sometimes	Often
8. How often do your friends or relatives make too many demands on you?	☐	☐	☐
9. How often do they argue with you?	☐	☐	☐
10. How often do they criticise you?	☐	☐	☐
11. How often do they let you down when you are counting on them?	☐	☐	☐
12. How often do they get on your nerves?	☐	☐	☐
13. How often do they drink or use drugs too much?	☐	☐	☐

Among the people you know, is there someone …	No	Yes, one person or a few people	Yes, lots of people
14. you can go with to community or social events (e.g. play cards, community meeting)?	☐	☐	☐
15. who would lend you money if you needed it in an emergency?	☐	☐	☐
16. who would lend you a car or drive you somewhere if you really needed it?	☐	☐	☐
17. you could call who would bail you out if you were arrested and put in jail?	☐	☐	☐
18. you could count on to check in on you regularly?	☐	☐	☐

	Very isolated	Somewhat isolated	Not very isolated at all
19. How isolated do you feel?	☐	☐	☐
	A lot	**Sometimes**	**Not very much at all**
20. How often do you purposefully avoid family gatherings?	☐	☐	☐
	Very likely	**Somewhat likely**	**Not at all likely**
21. Of those family gatherings you go to, how likely are you to leave early?	☐	☐	☐

Section 8. Optimism/Pessimism

These questions are about how you feel about your life in general. Please be as honest and accurate as you can throughout. Try not to let your response to one statement influence your responses to other statements. There are no right or wrong answers. Answer according to your own feelings, rather than how you think "most people" would answer.

	I disagree a lot	I disagree a little	I neither agree nor disagree	I agree a little	I agree a lot
1. In uncertain times, I usually expect the best.	☐	☐	☐	☐	☐
2. If something can go wrong for me, it will.	☐	☐	☐	☐	☐
3. I'm always optimistic about my future.	☐	☐	☐	☐	☐
4. I hardly ever expect things to go my way.	☐	☐	☐	☐	☐
5. I rarely count on good things happening to me.	☐	☐	☐	☐	☐
6. Overall, I expect more good things to happen to me than bad.	☐	☐	☐	☐	☐

That brings us to the end of the questions. Thank you very much for your time!

Please let us know if you have any comments:_____

Appendix B: Additional Chapter 9 Results

(Please refer to the end of this appendix for explanation of abbreviations and notes)

Table B.1: Percentage of missing data among study variables

Variable	Missing n (%)[a]
Predictor Variables	
Q1a	26 (8)
Q1b	15 (5)
Q1c	29 (9)
Q1d	23 (7)
Q1e	20 (6)
Q1f	18 (6)
Q1g	18 (6)
Q1h	15 (5)
Q1i	17 (5)
Q1j	14 (4)
Inter-personal racism score	38 (12)[b]
# of setting racism experienced in	52 (17)
Q2a[c]	26 (8)
Q2b[c]	26 (8)
Q2c[c]	28 (9)
Q2d[c]	27 (9)
Q2e[c]	27 (9)
Q2f[c]	28 (9)
Q2g[c]	30 (10)
Q2h[c]	31 (10)
Q2i[c]	29 (9)
Q2j[c]	31 (10)
Q2k[c]	29 (9)
Q3a	19 (6)
Q3b	18 (6)
Q3c	21 (7)
Q3d	21 (7)
Q3e	22 (7)
Q3f	22 (7)
Q3g	20 (6)
Internalised racism score	24 (8)[b]
Systemic racism score	24 (8)
Q4	10 (3)
Q5	2 (1)
Q6	28 (9)
Acute stress score/median split	20 (6)[b]
Chronic stress score/median split	16 (5)[b]
Total stress score/median split	21 (8)[b]
# of acute stressors experienced	30 (10)
# of chronic stressors experienced	49 (16)
# of total stressors experienced	61 (20)
Any experienced stressors	9 (3)

Variable	Missing n (%)[a]
Experienced stressor that affected respondent somewhat	9 (3)
Experienced stressors that affected respondent somewhat/a great deal	9 (3)
Outcome Variables	
Smoking	4 (1)
Alcohol consumption	14 (4)
Physical activity	16 (5)
High junk food consumption	10 (3)
Sufficient consumption of fruit and vegetables	4 (1)
Depression (CES-D)	33 (11)[b]
Self-assessed health status	4 (1)
SF-12 (MCS/PCS)	34 (11)
Mean waist circumference	23 (7)
Waist-to-Hip Ratio	26 (8)
Mean systolic blood pressure	4 (1)
Mean diastolic blood pressure	4 (1)
Hypertension	4 (1)
Dyslipidaemia	16 (5)
Homocysteine	12 (4)
HBA1c	14 (4)
C-reactive protein	15 (5)
Fibrinogen	17 (5)
Dental health	6 (2)
Allostatic load	48 (15)
# of chronic conditions	0 (0)
Socio-demographic variables	
Age	0 (0)
Sex	0 (0)
Household composition	0 (0)
Marital status	6 (2)
Per capita household income	0 (0)
House tenure	0 (0)[b]
Highest qualification	0 (0)[b]
Identifies with clan, tribal or language group	4 (1)
Recognises homeland/traditional country	3 (1)
Any non-Indigenous grandparents	17 (5)
Member of Stolen Generations	15 (5)
Method of administration	0 (0)
Psychosocial variables	
Optimism	0 (0)[b]
Lack of control	0 (0)[b]
Positive social capital	0 (0)[b]
Negative social capital	0 (0)[b]
Positive instrument social support	0 (0)[b]
Level of social isolation	19 (6)

a Percentage calculated with a denominator of 312. **b** as described in the text these variables were recoded to reduce/remove missing values.**c** percentage missing is reported on all answers to Q2 while analyses of Q2 include only those participants who reported some racism in Q1.

Table B.2: Significant associations between racism and a high acute stress score

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1c (sometimes)	1.49 (0.66-3.37)[a]	0.05	1.7
	0.83 (0.30-2.28)[b]	0.18	4.0
Q1c (often/very often)	**6.40 (1.35-30.33)**[a]	0.05	1.7
	3.52 (0.65-19.0)[b]	0.18	4.0
Q1d (sometimes)[c]	**2.22 (1.06-4.63)**[a]	0.06	2.8
	2.08 (0.89-4.85)[b]	0.15	1.9
Q1e (sometimes)	**3.08 (1.40-6.75)**[a]	0.07	3.6
	1.91 (0.75-4.87)[b]	0.15	1.6
Q1e (often/very often)	1.31 (0.28-6.03)[a]	0.07	3.6
	1.16 (0.21-6.32)[b]	0.15	1.6
Q1f (sometimes)	**2.68 (1.13-6.37)**[a]	0.06	1.9
	3.05 (1.09-8.53)[b]	0.15	1.8
Q1f (often/very often)	2.52 (0.22-28.6)[a]	0.06	1.9
	1.91 (0.13-29.0)[b]	0.15	1.8
Q1g (sometimes)	**2.58 (1.22-5.45)**[a]	0.06	2.3
	1.68 (0.69-4.11)[b]	0.15	1.5
Q1g (often/very often)	1.09 (0.31-3.78)[a]	0.06	2.3
	0.67 (0.16-2.83)[b]	0.15	1.5
Q1h (sometimes)	**2.96 (1.64-5.34)**[a]	0.08	4.5
	2.45 (1.21-4.97)[b]	0.16	2.6
Q1h (often/very often)	**3.13 (1.01-9.73)**[a]	0.08	4.5
	1.97 (0.53-7.29)[b]	0.16	2.6
Q1i (sometimes)	1.57 (0.86-2.84)[a]	0.06	2.2
	1.39 (0.67-2.89)[b]	0.15	1.1
Q1i (often/very often)	**4.47 (1.13-17.6)**[a]	0.06	2.2
	3.50 (0.72-17.0)[b]	0.15	1.1
Q1j (sometimes)	**2.20 (1.22-3.98)**[a]	0.08	4.3
	2.05 (0.97-4.32)[b]	0.17	3.2
Q1j (often/very often)	**4.93 (1.92-12.6)**[a]	0.08	4.3
	5.84 (1.67-20.4)[b]	0.17	3.2
Inter-personal racism score (none)	0.66 (0.35-1.22)[a]	0.09	5.6
	0.82 (0.37-1.84)[b]	0.18	4.6
Inter-personal racism score (high)	**2.82 (1.40-5.67)**[a]	0.09	5.6
	2.08 (0.91-4.77)[b]	0.18	4.6
# of setting racism experienced in (2nd quartile)	1.39 (0.69-2.82)[a]	0.07	3.4
	1.05 (0.42-2.58)[b]	0.19	5.6
# of setting racism experienced in (3rd quartile)	**2.25 (1.09-4.64)**[a]	0.07	3.4
	2.33 (0.87-6.22)[b]	0.19	5.6
# of setting racism experienced in (4th quartile)	**2.89 (1.28-6.50)**[a]	0.07	3.4
	1.97 (0.66-5.89)[b]	0.19	5.6
Q2d (never/hardly ever)	0.47 (0.24-0.94)[a]	0.05	1.1
	0.49 (0.21-1.17)[b]	0.17	3.4

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2d (often/very often)	0.71 (0.35-1.45)[a]	0.05	1.1
	0.72 (0.30-1.73)[b]	0.17	3.4
Q2e (never/hardly ever)	0.48 (0.23-1.00)[a]	0.09	5.0
	0.51 (0.19-1.34)[b]	0.19	5.6
Q2e (sometimes)	**2.20 (1.09-4.44)**[a]	0.09	5.0
	2.04 (0.84-4.94)[b]	0.19	5.6
Q2f (sometimes)	0.98 (0.49-1.97)[a]	0.03	0.0
	0.49 (0.19-1.26)[b]	0.18	4.8
Q2f (often/very often)	0.74 (0.35-1.56)[a]	0.03	0.0
	0.27 (0.09-0.84)[b]	0.18	4.8
Q2h (never/hardly ever)	**0.24 (0.10-0.58)**[a]	0.07	3.5
	0.26 (0.09-0.76)[b]	0.18	4.9
Q2h (often/very often)	0.67 (0.35-1.28)[a]	0.07	3.5
	0.50 (0.22-1.12)[b]	0.18	4.9
Q2j (sometimes)	**2.34 (1.21-4.56)**[a]	0.05	0.0
	2.46 (1.06-5.70)[b]	0.18	4.6
Q2j (often/very often)	1.20 (0.46-3.14)[a]	0.05	0.0
	0.70 (0.18-2.67)[b]	0.18	4.6
Q2k (sometimes)	**2.88 (1.42-5.86)**[a]	0.05	3.6
	3.41 (1.42-8.17)[b]	0.20	6.8
Q2k (often/very often)	0.75 (0.33-1.71)[a]	0.05	3.6
	0.68 (0.24-1.90)[b]	0.20	6.8
Q3a (neither agree nor disagree)	1.06 (0.50-2.25)[a]	0.04	0.4
	0.76 (0.28-2.09)[b]	0.16	2.9
Q3a (disagree/strongly disagree)	0.44 (0.08-2.32)[a]	0.04	0.4
	0.13 (0.02-0.92)[b]	0.16	2.9
Q4 (never/once a year)	**0.36 (0.17-0.74)**[a]	0.08	4.2
	0.55 (0.22-1.35)[b]	0.18	4.4
Q4 (once a month)	0.37 (0.13-1.04)[a]	0.08	4.2
	0.26 (0.06-1.07)[b]	0.18	4.4
Q4 (once a week)	**0.32 (0.12-0.88)**[a]	0.08	4.2
	0.31 (0.09-1.02)[b]	0.18	4.4
Q4 (once a day)	**0.33 (0.13-0.84)**[a]	0.08	4.2
	0.22 (0.07-0.74)[b]	0.18	4.4

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, highest qualification, optimism, lack of control, positive social capital, negative social capital, level of social isolation, method of administration, whether recognises a homeland/traditional country, had any non-Indigenous grandparents or is a membership in/part of the Stolen Generations.

c The base category for this variable is ‘never/hardly ever’ with the category ‘often/very often’ omitted due to a lack of variation in outcome within this category.

Table B.3: Significant associations between racism and a high # of acute stressors

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	1.70 (0.90-3.19)[a]	0.05	2.6
	0.98 (0.46-2.10)[b]	0.15	2.4
Q1a (often/very often)	**5.10 (1.07-24.4)**[a]	0.05	2.6
	3.99 (0.66-24.0)[b]	0.15	2.4
Q1d (sometimes)[c]	**2.37 (1.13-4.97)**[a]	0.05	3.0
	2.45 (1.03-5.84)[b]	0.15	2.6
Q1e (sometimes)	**3.15 (1.40-7.07)**[a]	0.06	3.5
	2.03 (0.78-5.25)[b]	0.14	1.4
Q1e (often/very often)	1.48 (0.34-6.33)[a]	0.06	3.5
	0.95 (0.18-5.01)[b]	0.14	1.4
Q1f (sometimes)	**2.90 (1.16-7.24)**[a]	0.04	2.1
	3.54 (1.19-10.5)[b]	0.14	2.0
Q1f (often/very often)	2.09 (0.18-23.7)[a]	0.04	2.1
	1.19 (0.08-17.5)[b]	0.14	2.0
Q1g (sometimes)	**3.91 (1.69-9.03)**[a]	0.06	3.9
	2.71 (1.03-7.16)[b]	0.15	2.9
Q1g (often/very often)	0.86 (0.25-2.98)[a]	0.06	3.9
	0.43 (0.10-1.84)[b]	0.15	2.9
Q1h (sometimes)	**2.79 (1.54-5.07)**[a]	0.06	4.2
	2.29 (1.12-4.67)[b]	0.15	2.1
Q1h (often/very often)	3.22 (0.98-10.6)[a]	0.06	4.2
	1.96 (0.50-7.71)[b]	0.15	2.1
Q1j (sometimes)	**1.86 (1.03-3.34)**[a]	0.05	2.7
	1.47 (0.70-3.07)[b]	0.14	1.2
Q1j (often/very often)	**3.39 (1.34-8.58)**[a]	0.05	2.7
	2.96 (0.86-10.2)[b]	0.14	1.2
Inter-personal racism score (none)	0.68 (0.38-1.24)[a]	0.09	6.9
	0.86 (0.40-1.86)[b]	0.18	5.9
Inter-personal racism score (high)	**3.85 (1.79-8.24)**[a]	0.09	6.9
	2.96 (1.22-7.17)[b]	0.18	5.9
# of setting racism experienced in (2^{nd} quartile)	1.57 (0.79-3.11)[a]	0.07	4.7
	1.26 (0.52-3.04)[b]	0.18	5.8
# of setting racism experienced in (3^{rd} quartile)	1.63 (0.80-3.32)[a]	0.07	4.7
	1.70 (0.66-4.39)[b]	0.18	5.8
# of setting racism experienced in (4^{th} quartile)	**4.18 (1.78-9.81)**[a]	0.07	4.7
	2.78 (0.92-8.38)[b]	0.18	5.8
Q2e (never/hardly ever)	**0.47 (0.23-0.96)**[a]	0.06	4.0
	0.48 (0.19-1.24)[b]	0.18	5.3
Q2e (sometimes)	1.83 (0.90-3.73)[a]	0.06	4.0
	1.78 (0.72-4.39)[b]	0.18	5.3
Q2f (sometimes)	1.12 (0.55-2.27)[a]	0.02	0.0
	0.83 (0.33-2.11)[b]	0.17	5.1
Q2f (often/very often)	0.69 (0.33-1.45)[a]	0.02	0.0
	0.25 (0.08-0.75)[b]	0.17	5.1

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2h (never/hardly ever)	**0.31 (0.13-0.69)**[a]	0.05	2.6
	0.27 (0.09-0.79)[b]	0.17	4.7
Q2h (often/very often)	0.75 (0.39-1.43)[a]	0.05	2.6
	0.51 (0.22-1.19)[b]	0.17	4.7
Q2j (sometimes)	**2.16 (1.10-4.27)**[a]	0.04	1.3
	1.91 (0.81-4.50)[b]	0.17	4.2
Q2j (often/very often)	1.10 (0.42-2.88)[a]	0.04	1.3
	0.45 (0.12-1.72)[b]	0.17	4.2
Q2k (sometimes)	**2.54 (1.24-5.20)**[a]	0.04	1.9
	3.19 (1.32-7.72)[b]	0.18	5.4
Q2k (often/very often)	1.31 (0.59-2.93)[a]	0.04	1.9
	1.13 (0.41-3.13)[b]	0.18	5.4
Q3a (neither agree nor disagree)	0.85 (0.40-1.80)[a]	0.03	0.6
	0.66 (0.25-1.79)[b]	0.16	3.5
Q3a (disagree/strongly disagree)	0.32 (0.06-1.65)[a]	0.03	0.6
	0.08 (0.01-0.55)[b]	0.16	3.5
No internalised racism	0.98 (0.38-2.54)[a]	0.04	1.4
	0.72 (0.22-2.35)[b]	0.16	3.6
High internalised racism	**0.52 (0.29-0.92)**[a]	0.04	1.4
	0.35 (0.16-0.76)[b]	0.16	3.6
Q4 (never/once a year)	**0.30 (0.15-0.61)**[a]	0.08	5.6
	0.40 (0.16-0.98)[b]	0.18	5.4
Q4 (once a month)	**0.25 (0.09-0.70)**[a]	0.08	5.6
	0.17 (0.04-0.68)[b]	0.18	5.4
Q4 (once a week)	**0.28 (0.11-0.75)**[a]	0.08	5.6
	0.23 (0.07-0.75)[b]	0.18	5.4
Q4 (once a day)	**0.36 (0.15-0.85)**[a]	0.08	5.6
	0.25 (0.09-0.75)[b]	0.18	5.4
Q5 (hardly any/not many people)	0.14 (0.02-1.18)[a]	0.04	1.8
	0.23 (0.02-2.50)[b]	0.15	2.5
Q5 (some people)	1.17 (0.32-4.32)[a]	0.04	1.8
	0.77 (0.15-3.82)[b]	0.15	2.5
Q5 (most people)	0.70 (0.41-1.20)[a]	0.04	1.8
	0.41 (0.21-0.82)[b]	0.15	2.5

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, highest qualification, optimism, lack of control, positive social capital, negative social capital, level of social isolation, method of administration, whether recognises a homeland/traditional country, had any non-Indigenous grandparents or is a membership in/part of the Stolen Generations.

c The base category for this variable is 'never/hardly ever' with the category 'often/very often' omitted due to a lack of variation in outcome within this category.

Table B.4: Significant associations between racism and a high chronic stress score

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**4.14 (2.09-8.19)**[a]	0.12	9.0
	1.80 (0.71-4.59)[b]	0.36	4.6
Q1a (often/very often)	**21.1 (2.60-170.6)**[a]	0.12	9.0
	20.5 (1.49-280.0)[b]	0.36	4.6
Q1b (sometimes)	**5.53 (1.97-15.5)**[a]	0.08	5.0
	3.69 (0.91-14.9)[b]	0.35	3.8
Q1b (often/very often)	8.39 (0.96-73.4)[a]	0.08	5.0
	37.3 (2.22-626.7)[b]	0.35	3.8
Q1c (sometimes)	**2.68 (1.18-6.09)**[a]	0.07	4.5
	2.07 (0.69-6.27)[b]	0.35	3.0
Q1c (often/very often)	**12.8 (1.60-101.5)**[a]	0.07	4.5
	12.5 (1.02-153.2)[b]	0.35	3.0
Q1d (sometimes)	**2.15 (1.02-4.55)**[a]	0.05	2.5
	2.00 (0.75-5.31)[b]	0.32	0.5
Q1d (often/very often)	3.81 (0.38-38.4)[a]	0.05	2.5
	6.03 (0.20-185.9)[b]	0.32	0.5
Q1e (sometimes)	**6.03 (2.52-14.4)**[a]	0.09	6.4
	5.83 (1.64-20.7)[b]	0.35	3.1
Q1e (often/very often)	2.65 (0.59-12.0)[a]	0.09	6.4
	5.07 (0.53-48.9)[b]	0.35	3.1
Q1f (sometimes)	**5.17 (1.86-14.4)**[a]	0.07	4.1
	8.05 (1.97-32.9)[b]	0.35	3.5
Q1f (often/very often)	2.42 (0.21-27.6)[a]	0.07	4.1
	0.67 (0.03-13.1)[b]	0.35	3.5
Q1g (sometimes)	**2.44 (1.17-5.11)**[a]	0.08	4.7
	1.66 (0.59-4.71)[b]	0.34	2.0
Q1g (often/very often)	**13.5 (1.66-110.1)**[a]	0.08	4.7
	12.5 (1.06-147.9)[b]	0.34	2.0
Q1h (sometimes)	**3.64 (1.98-6.70)**[a]	0.10	6.9
	3.10 (1.36-7.10)[b]	0.34	2.6
Q1h (often/very often)	**5.53 (1.48-20.6)**[a]	0.10	6.9
	5.24 (0.82-33.6)[b]	0.34	2.6
Q1i (sometimes)	**2.55 (1.39-4.68)**[a]	0.08	4.9
	2.33 (1.00-5.44)[b]	0.35	3.6
Q1i (often/very often)	**7.12 (1.50-33.7)**[a]	0.08	4.9
	11.2 (1.37-91.6)[b]	0.35	3.6
Q1j (sometimes)	**3.77 (2.04-6.98)**[a]	0.10	7.5
	2.81 (1.20-6.56)[b]	0.34	1.9
Q1j (often/very often)	**5.56 (2.06-15.0)**[a]	0.10	7.5
	2.54 (0.65-9.89)[b]	0.34	1.9
Inter-personal racism score (none)	**0.32 (0.16-0.63)**[a]	0.18	14.9
	0.37 (0.14-0.98)[b]	0.39	7.0
Inter-personal racism score (high)	**5.83 (2.58-13.2)**[a]	0.18	14.9
	6.17 (2.01-18.9)[b]	0.39	7.0

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
# of setting racism experienced in	**2.36 (1.12-5.02)**[a]	0.17	14.0
(2^{nd} quartile)	1.74 (0.57-5.34)[b]	0.43	10.9
# of setting racism experienced in	**6.13 (2.76-13.6)**[a]	0.17	14.0
(3^{rd} quartile)	**9.10 (2.57-32.2)**[b]	0.43	10.9
# of setting racism experienced in	**12.9 (5.10-32.8)**[a]	0.17	14.0
(4^{th} quartile)	**11.9 (2.76-51.3)**[b]	0.43	10.9
Q2a (never/hardly ever)	0.57 (0.28-1.14)[a]	0.05	2.1
	0.90 (0.32-2.57)[b]	0.35	3.4
Q2a (sometimes)	0.51 (0.24-1.08)[a]	0.05	2.1
	0.20 (0.06-0.71)[b]	0.35	3.4
Q2e (never/hardly ever)	**0.45 (0.22-0.94)**[a]	0.07	3.6
	0.36 (0.11-1.12)[b]	0.35	3.1
Q2e (sometimes)	1.32 (0.64-2.71)[a]	0.07	3.6
	1.83 (0.63-5.35)[b]	0.35	3.1
Q2g (sometimes)	**2.86 (1.36-6.02)**[a]	0.07	4.3
	4.56 (1.50-13.8)[b]	0.36	4.0
Q2g (often/very often)	1.66 (0.57-4.85)[a]	0.07	4.3
	1.23 (0.23-6.61)[b]	0.36	4.0
Q2h (never/hardly ever)	**0.38 (0.16-0.88)**[a]	0.07	4.1
	0.35 (0.10-1.19)[b]	0.33	1.6
Q2h (often/very often)	1.31 (0.67-2.57)[a]	0.07	4.1
	0.98 (0.37-2.60)[b]	0.33	1.6
Q2i (never/hardly ever)	0.51 (0.24-1.08)[a]	0.06	3.2
	0.59 (0.18-1.89)[b]	0.36	4.0
Q2i (often/very often)	**0.42 (0.21-0.85)**[a]	0.06	3.2
	0.20 (0.06-0.65)[b]	0.36	4.0
Q2j (sometimes)	**4.07 (1.93-8.60)**[a]	0.11	8.0
	12.7 (3.44-46.7)[b]	0.40	8.8
Q2j (often/very often)	**4.20 (1.42-12.4)**[a]	0.11	8.0
	3.78 (0.55-26.2)[b]	0.40	8.8
Q2k (sometimes)	**2.27 (1.09-4.70)**[a]	0.06	2.7
	3.09 (1.03-9.27)[b]	0.34	2.7
Q2k (often/very often)	1.55 (0.68-3.53)[a]	0.06	2.7
	1.03 (0.31-3.45)[b]	0.34	2.7
Q3f (neither agree nor disagree)	**2.12 (1.12-4.07)**[a]	0.05	1.7
	2.22 (0.90-5.48)[b]	0.33	1.3
Q3f (disagree/strongly disagree)	1.48 (0.66-3.30)[a]	0.05	1.7
	1.76 (0.53-5.86)[b]	0.33	1.3
Q4 (never/once a year)	**0.39 (0.19-0.81)**[a]	0.05	2.2
	0.96 (0.35-2.67)[b]	0.31	0.0
Q4 (once a month)	0.91 (0.34-2.48)[a]	0.05	2.2
	1.00 (0.22-4.57)[b]	0.31	0.0
Q4 (once a week)	0.93 (0.35-2.51)[a]	0.05	2.2
	0.92 (0.25-3.38)[b]	0.31	0.0
Q4 (once a day)	0.61 (0.25-1.47)[a]	0.05	2.2

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
	0.66 (0.18-2.41)[b]	0.31	0.0
Q6 (hardly any/not many people)	0.78 (0.34-1.76)[a]	0.05	2.5
	0.39 (0.11-1.36)[b]	0.32	0.5
Q6 (some people)	**2.20 (1.05-4.65)**[a]	0.05	2.5
	1.51 (0.50-4.54)[b]	0.32	0.5
Q6 (most people)	0.83 (0.43-1.60)[a]	0.05	2.5
	0.49 (0.19-1.31)[b]	0.32	0.5

CI: Confidence interval; **OR**: odds ratio.
a Adjusted for age and sex.
b Adjusted for age and sex, marital status, income, highest qualification, optimism, lack of control, positive social capital, negative social capital, level of social isolation, method of administration, whether recognises a homeland/traditional country, had any non-Indigenous grandparents or is a membership in/part of the Stolen Generations.

Table B.5: Significant associations between racism and high # of chronic stressors

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**3.35 (1.74-6.46)**[a]	0.08	5.5
	1.64 (0.74-3.67)[b]	0.25	2.2
Q1a (often/very often)	**7.23 (1.49-34.9)**[a]	0.08	5.5
	7.52 (1.10-51.6)[b]	0.25	2.2
Q1b (sometimes)	**5.27 (1.88-14.8)**[a]	0.07	3.9
	3.60 (1.03-12.6)[b]	0.25	2.4
Q1b (often/very often)	3.61 (0.69-18.8)[a]	0.07	3.9
	9.21 (1.18-71.9)[b]	0.25	2.4
Q1c (sometimes)	**4.50 (1.73-11.7)**[a]	0.07	3.7
	4.56 (1.44-14.5)[b]	0.25	1.1
Q1c (often/very often)	3.61 (0.94-13.8)[a]	0.07	3.7
	2.44 (0.52-11.4)[b]	0.24	1.1
Q1d (sometimes)[c]	**2.22 (1.08-4.57)**[a]	0.05	2.0
	1.72 (0.71-4.12)[b]	0.22	0.0
Q1e (sometimes)	**7.76 (3.01-20.0)**[a]	0.11	8.0
	12.9 (3.48-47.7)[b]	0.22	8.2
Q1e (often/very often)	**6.14 (1.20-31.4)**[a]	0.11	8.0
	21.9 (2.00-238.4)[b]	0.31	8.2
Q1f (sometimes)	**5.58 (2.01-15.5)**[a]	0.07	4.1
	7.77 (2.19-27.6)[b]	0.31	2.9
Q1f (often/very often)	3.04 (0.27-34.7)[a]	0.07	4.1
	1.71 (0.12-24.8)[b]	0.26	2.9
Q1g (sometimes)	**2.72 (1.28-5.77)**[a]	0.08	4.9
	2.08 (0.78-5.55)[b]	0.26	3.5
Q1g (often/very often)	**14.8 (1.82-119.8)**[a]	0.08	4.9
	16.8 (1.40-202.0)[b]	0.26	3.5
Q1h (sometimes)	**3.50 (1.92-6.37)**[a]	0.09	6.5
	3.11 (1.46-6.65)[b]	0.26	3.3
Q1h (often/very often)	**5.89 (1.58-21.9)**[a]	0.09	6.5
	7.35 (1.26-42.9)[b]	0.26	3.3
Q1i (sometimes)	**2.48 (1.37-4.50)**[a]	0.06	3.5
	1.84 (0.86-3.95)[b]	0.25	2.2
Q1i (often/very often)	3.76 (0.97-14.6)[a]	0.06	3.5
	3.61 (0.71-18.5)[b]	0.25	2.2
Q1j (sometimes)	**3.41 (1.85-6.29)**[a]	0.10	6.8
	2.99 (1.32-6.77)[b]	0.25	2.7
Q1j (often/very often)	**5.91 (2.22-15.8)**[a]	0.10	6.8
	4.43 (1.25-15.7)[b]	0.25	2.7
Inter-personal racism score (none)	**0.40 (0.21-0.76)**[a]	0.15	12.0
	0.44 (0.19-1.05)[b]	0.30	6.8
Inter-personal racism score (high)	**5.68 (2.53-12.7)**[a]	0.15	12.0
	6.92 (2.51-19.1)[b]	0.30	6.8
# of setting racism experienced in (2^{nd} quartile)	**2.48 (1.21-5.08)**[a]	0.12	9.2
	2.66 (1.03-6.87)[b]	0.27	4.4

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
# of setting racism experienced in	**3.46 (1.66-7.22)**[a]	0.12	9.2
(3^{rd} quartile)	**3.12 (1.10-8.87)**[b]	0.27	4.4
# of setting racism experienced in	**9.36 (3.81-23.0)**[a]	0.12	9.2
(4^{th} quartile)	**8.81 (2.52-30.8)**[b]	0.27	4.4
Q2a (never/hardly ever)	**0.45 (0.23-0.90)**[a]	0.05	2.4
	0.68 (0.27-1.69)[b]	0.25	2.3
Q2a (sometimes)	**0.40 (0.19-0.83)**[a]	0.05	2.4
	0.19 (0.07-0.55)[b]	0.25	2.3
Q2g (sometimes)	**2.73 (1.34-5.57)**[a]	0.06	3.0
	2.68 (1.09-6.60)[b]	0.23	0.0
Q2g (often/very often)	1.74 (0.60-5.10)[a]	0.06	3.0
	1.38 (0.31-6.08)[b]	0.23	0.0
Q2h (never/hardly ever)	**0.45 (0.20-0.99)**[a]	0.04	1.0
	0.30 (0.10-0.93)[b]	0.23	0.0
Q2h (often/very often)	0.85 (0.45-1.63)[a]	0.04	1.0
	0.52 (0.22-1.23)[b]	0.23	0.0
Q2i (never/hardly ever)	0.49 (0.24-1.01)[a]	0.05	2.3
	0.47 (0.17-1.28)[b]	0.25	2.1
Q2i (often/very often)	**0.40 (0.20-0.81)**[a]	0.05	2.3
	0.21 (0.08-0.58)[b]	0.25	2.1
Q2j (sometimes)	**3.60 (1.74-7.43)**[a]	0.07	4.4
	4.47 (1.67-12.0)[b]	0.25	2.0
Q2j (often/very often)	1.64 (0.62-4.36)[a]	0.07	4.4
	1.28 (0.30-5.44)[b]	0.25	2.0
Q3e (neither agree nor disagree)	1.46 (0.68-3.13)[a]	0.04	1.0
	1.80 (0.59-5.46)[b]	0.25	2.5
Q3e (disagree/strongly disagree)	0.73 (0.43-1.24)[a]	0.04	1.0
	0.35 (0.16-0.74)[b]	0.25	2.5
Q3f (neither agree nor disagree)	**2.16 (1.13-4.11)**[a]	0.05	1.7
	2.50 (1.11-5.63)[b]	0.23	0.7
Q3f (disagree/strongly disagree)	1.34 (0.62-2.90)[a]	0.05	1.7
	1.08 (0.36-3.24)[b]	0.23	0.7
Q4 (never/once a year)	**0.48 (0.24-0.95)**[a]	0.04	1.0
	1.00 (0.40-2.50)[b]	0.22	0.0
Q4 (once a month)	0.69 (0.26-1.82)[a]	0.04	1.0
	0.57 (0.15-2.17)[b]	0.22	0.0
Q4 (once a week)	0.91 (0.36-2.35)[a]	0.04	1.0
	1.02 (0.31-3.40)[b]	0.22	0.0
Q4 (once a day)	0.65 (0.28-1.49)[a]	0.04	1.0
	0.84 (0.28-2.51)[b]	0.22	0.0
Q6 (hardly any/not many people)	0.59 (0.27-1.32)[a]	0.06	2.6
	0.35 (0.11-1.11)[b]	0.25	2.6
Q6 (some people)	**2.26 (1.10-4.63)**[a]	0.06	2.6
	1.73 (0.64-4.64)[b]	0.25	2.6
Q6 (most people)	0.83 (0.44-1.57)[a]	0.06	2.6

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
	0.55 (0.22-1.33)[b]	0.25	2.6

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, highest qualification, optimism, lack of control, positive social capital, negative social capital, level of social isolation, method of administration, whether recognises a homeland/traditional country, had any non-Indigenous grandparents or is a membership in/part of the Stolen Generations.

c The base category for this variable is ‘never/hardly ever’ with the category ‘often/very often’ omitted due to a lack of variation in outcome within this category.

Table B.6: Significant associations between racism and having experienced stressors that affected respondent somewhat or a great deal

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1h (sometimes)[c]	**3.11 (1.17-8.28)**[a]	0.05	2.5
	2.98 (0.84-10.6)[b]	0.25	1.0
Inter-personal racism score	**0.18 (0.08-0.42)**[a]	0.13	10.2
(none)	**0.16 (0.05-0.54)**[b]	0.32	8.6
Inter-personal racism score	0.76 (0.26-2.22)[a]	0.13	10.2
(high)	0.33 (0.07-1.51)[b]	0.32	8.6
# of setting racism	**5.42 (1.98-14.9)**[a]	0.14	10.9
experienced in (2nd	3.10 (0.78-12.4)[b]	0.36	11.8
quartile)			
# of setting racism	**6.93 (2.16-22.3)**[a]	0.14	10.9
experienced in (3rd	**29.0 (3.30-254.9)**[b]	0.36	11.8
quartile)			
# of setting racism	**2.98 (1.07-8.31)**[a]	0.14	10.9
experienced in (4th quartile)	1.52 (0.29-8.04)[b]	0.36	11.8
Q2h (never/hardly ever)	0.60 (0.21-1.71)[a]	0.11	8.4
	5.89 (0.73-47.4)[b]	0.38	13.9
Q2h (often/very often)	**5.94 (1.24-28.6)**[a]	0.11	8.4
	71.8 (2.90-1778.5)[b]	0.38	13.9
Q2i (never/hardly ever)	0.36 (0.10-1.31)[a]	0.06	3.3
	0.57 (0.07-4.96)[b]	0.33	9.4
Q2i (often/very often)	0.31 (0.09-1.07)[a]	0.06	3.3
	0.07 (0.01-0.59)[b]	0.33	9.4
Moderate systemic racism	2.00 (0.58-6.86)[a]	0.05	2.5
	4.31 (0.56-33.0)[b]	0.29	4.9
High systemic racism	2.96 (0.90-9.76)[a]	0.05	2.5
	8.06 (1.22-53.1)[b]	0.29	4.9
Very high systemic racism	3.50 (0.87-14.0)[a]	0.05	2.5
	9.67 (1.14-82.4)[b]	0.29	4.9
Q4 (never/once a year)	0.81 (0.27-2.40)[a]	0.06	2.8
	0.84 (0.20-3.51)[b]	0.24	0.6
Q4 (once a month)	**0.39 (0.17-0.85)**[a]	0.06	2.8
	0.52 (0.15-1.84)[b]	0.24	0.6
Q4 (once a week)	1.35 (0.28-6.42)[a]	0.06	2.8
	0.90 (0.12-6.72)[b]	0.24	0.6
Q4 (once a day)	0.67 (0.20-2.22)[a]	0.06	2.8
	0.60 (0.13-2.74)[b]	0.24	0.6

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, highest qualification, optimism, lack of control, positive social capital, negative social capital, level of social isolation, method of administration, whether recognises a homeland/traditional country, had any non-Indigenous grandparents or is a membership in/part of the Stolen Generations.

c The base category for this variable is 'never/hardly ever' with the category 'often/very often' omitted due to a lack of variation in outcome within this category.

Table B.7: Significant associations between racism and having experienced stressors that affected respondent a great deal

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**3.91 (1.79-8.56)**[a]	0.10	6.5
	2.17 (0.84-5.57)[b]	0.27	4.0
Q1a (often/very often)	**10.1 (1.29-79.7)**[a]	0.10	6.5
	8.40 (0.66-107.6)[b]	0.27	4.0
Q1e (sometimes)	**2.56 (1.12-5.84)**[a]	0.05	2.1
	1.25 (0.44-3.59)[b]	0.24	0.3
Q1e (often/very often)	1.54 (0.42-5.74)[a]	0.05	2.1
	1.08 (0.17-7.01)[b]	0.24	0.3
Q1f (sometimes)[c]	**4.76 (1.38-16.4)**[a]	0.06	2.8
	7.48 (1.41-39.6)[b]	0.28	4.2
Q1h (sometimes)	**2.89 (1.50-5.57)**[a]	0.08	4.9
	2.66 (1.15-6.17)[b]	0.26	2.3
Q1h (often/very often)	**8.44 (1.08-66.1)**[a]	0.08	4.9
	4.66 (0.48-45.5)[b]	0.26	2.3
Inter-personal racism score (none)	0.45 (0.25-0.82)[a]	0.11	7.6
	0.49 (0.21-1.13)[b]	0.28	5.0
Inter-personal racism score (high)	**2.98 (1.32-6.75)**[a]	0.11	7.6
	1.73 (0.60-4.96)[b]	0.28	5.0
# of setting racism experienced in (2^{nd} quartile)	**2.33 (1.16-4.66)**[a]	0.09	5.9
	2.01 (0.76-5.33)[b]	0.32	8.6
# of setting racism experienced in (3^{rd} quartile)	**3.03 (1.41-6.50)**[a]	0.09	5.9
	3.06 (0.97-9.65)[b]	0.32	8.6
# of setting racism experienced in (4^{th} quartile)	**3.64 (1.56-8.49)**[a]	0.09	5.9
	2.15 (0.61-7.53)[b]	0.32	8.6
Q2b (sometimes)	1.88 (0.91-3.86)[a]	0.07	3.9
	1.43 (0.51-4.03)[b]	0.30	6.2
Q2b(often/very often)	**2.99 (1.30-6.87)**[a]	0.07	3.9
	5.05 (1.52-16.8)[b]	0.30	6.2
Q2e (never/hardly ever)	**0.45 (0.21-0.94)**[a]	0.07	3.4
	0.69 (0.25-1.90)[b]	0.25	1.6
Q2e(sometimes)	1.12 (0.51-2.48)[a]	0.07	3.4
	1.09 (0.38-3.10)[b]	0.25	1.6
Q2f (sometimes)	0.82 (0.38-1.76)[a]	0.05	1.4
	0.51 (0.17-1.49)[b]	0.27	3.2
Q2f (often/very often)	0.61 (0.28-1.33)[a]	0.05	1.4
	0.29 (0.09-0.98)[b]	0.27	3.2
Q2g (sometimes)	**2.27 (1.03-5.00)**[a]	0.06	3.1
	2.37 (0.88-6.41)[b]	0.26	2.7
Q2g (often/very often)	2.20 (0.59-8.18)[a]	0.06	3.1
	2.64 (0.25-28.2)[b]	0.26	2.7
Q2h (never/hardly ever)	**0.34 (0.15-0.77)**[a]	0.09	5.6
	0.44 (0.13-1.50)[b]	0.29	5.4

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2h(often/very often)	1.45 (0.66-2.99)[a]	0.09	5.6
	2.19 (0.78-6.13)[b]	0.29	5.4
Q2i (never/hardly ever)	0.79 (0.35-1.79)[a]	0.07	3.2
	2.27 (0.64-8.05)[b]	0.28	5.1
Q2i (often/very often)	**0.41 (0.20-0.85)**[a]	0.07	3.2
	0.34 (0.12-0.98)[b]	0.28	5.1
Q2j (sometimes)	**4.18 (1.74-10.0)**[a]	0.09	5.9
	3.44 (1.12-10.5)[b]	0.27	3.6
Q2j (often/very often)	1.96 (0.67-5.72)[a]	0.09	5.9
	2.12 (0.41-10.9)[b]	0.27	3.6
Q4 (never/once a year)	**0.48 (0.25-0.94)**[a]	0.05	1.8
	1.02 (0.37-2.85)[b]	0.25	1.6
Q4 (once a month)	0.69 (0.25-1.90)[a]	0.05	1.8
	0.87 (0.22-3.45)[b]	0.25	1.6
Q4 (once a week)	0.55 (0.22-1.39)[a]	0.05	1.8
	0.55 (0.17-1.73)[b]	0.25	1.6
Q4 (once a day)	0.51 (0.22-1.18)[a]	0.05	1.8
	0.72 (0.23-2.26)[b]	0.25	1.6

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, highest qualification, optimism, lack of control, positive social capital, negative social capital, level of social isolation, method of administration, whether recognises a homeland/traditional country, had any non-Indigenous grandparents or is a membership in/part of the Stolen Generations.

c The base category for this variable is ‘never/hardly ever’ with the category ‘often/very often’ omitted due to a lack of variation in outcome within this category.

Table B.8: Significant associations between racism/stress and current smoking

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2d (never/hardly ever)	**2.72 (1.39-5.30)**[a]	0.05	3.5
	3.03 (1.44-6.36)[b]	0.16	7.9
Q2d (often/very often)	1.61 (0.79-3.26)[a]	0.05	3.5
	1.95 (0.89-4.27)[b]	0.16	7.9
Q2j (sometimes)	**0.49 (0.25-0.94)**[a]	0.04	2.9
	0.39 (0.18-0.82)[b]	0.13	7.6
Q2j (often/very often)	1.64 (0.68-3.96)[a]	0.04	2.9
	1.43 (0.53-3.87)[b]	0.13	7.6
Q3b (neither agree nor disagree)	0.71 (0.34-1.47)[a]	0.03	1.9
	0.78 (0.35-1.73)[b]	0.12	3.7
Q3b (disagree/strongly disagree)	**2.41 (1.22-4.78)**[a]	0.03	1.9
	2.63 (1.23-5.65)[b]	0.12	3.7
# of total stressors (1st quartile)	1.44 (0.68-3.04)[a]	0.03	1.3
	1.14 (0.49-2.63)[b]	0.12	3.9
# of total stressors (3rd quartile)	0.54 (0.26-1.12)[a]	0.03	1.3
	0.43 (0.19-0.95)[b]	0.12	3.9
# of total stressors (4th quartile)	0.87 (0.44-1.72)[a]	0.03	1.3
	0.68 (0.32-1.46)[b]	0.12	3.9

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, income, household composition and highest qualification.

Table B.9: Significant associations between racism/stress and moderate/high risk alcohol consumption

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**2.34 (1.20-4.54)**[a]	0.08	1.3
	1.86 (0.88-3.96)[b]	0.17	2.1
Q1a (often/very often)	0.90 (0.30-2.72)[a]	0.08	1.3
	0.96 (0.27-3.44)[b]	0.17	2.1
Q1b (sometimes)	**2.76 (1.10-6.92)**[a]	0.08	1.2
	2.86 (1.03-7.98)[b]	0.17	2.0
Q1b (often/very often)	2.05 (0.39-10.94)[a]	0.08	1.2
	1.44 (0.24-8.57)[b]	0.17	2.0
# of setting racism experienced	1.40 (0.70-2.82)[a]	0.07	0.2
in (2^{nd} quartile)	1.18 (0.53-2.61)[b]	0.16	1.0
# of setting racism experienced	1.02 (0.49-2.11)[a]	0.07	0.2
in (3^{rd} quartile)	0.62 (0.26-1.48)[b]	0.16	1.0
# of setting racism experienced	**2.31 (1.02-5.24)**[a]	0.07	0.2
in (4^{th} quartile)	1.44 (0.55-3.75)[b]	0.16	1.0
Q2h (never/hardly ever)	1.30 (0.58-2.90)[a]	0.08	0.4
	1.09 (0.44-2.65)[b]	0.16	0.6
Q2h (often/very often)	**2.24 (1.16-4.34)**[a]	0.08	0.4
	2.16 (1.02-4.61)[b]	0.16	0.6
Q3g (strongly agree/agree)	0.91 (0.47-1.79)[a]	0.11	4.2
	0.95 (0.45-2.02)[b]	0.19	3.7
Q3g (neither agree nor	**0.24 (0.11-0.54)**[a]	0.11	4.2
disagree)	**0.27 (0.11-0.65)**[b]	0.19	3.7
No internalised racism	**3.07 (1.07-8.83)**[a]	0.07	0.3
	3.17 (1.03-9.76)[b]	0.16	1.0
High internalised racism	1.00 (0.56-1.80)[a]	0.07	0.3
	1.23 (0.63-2.39)[b]	0.16	1.0
Q4 (never/once a year)	0.80 (0.40-1.59)[a]	0.08	0.4
	1.03 (0.47-2.27)[b]	0.15	0.2
Q4 (once a month)	**0.36 (0.13-0.99)**[a]	0.08	0.4
	0.31 (0.11-0.93)[b]	0.15	0.2
Q4 (once a week)	0.67 (0.27-1.66)[a]	0.08	0.4
	0.58 (0.21-1.58)[b]	0.15	0.2
Q4 (once a day)	0.84 (0.35-2.02)[a]	0.08	0.4
	0.81 (0.32-2.06)[b]	0.15	0.2

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, highest qualification, lack of control (work settings excluded), negative social capital, method of administration and whether or not respondents had any non-Indigenous grandparents.

Table B.10: Significant associations between racism/stress and insufficient physical activity

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q4 (never/once a year)	0.99 (0.49-2.00)[a]	0.11	1.6
	1.06 (0.49-2.30)[b]	0.16	1.7
Q4 (once a month)	**0.17 (0.04-0.78)**[a]	0.11	1.6
	0.24 (0.48-1.15)[b]	0.16	1.7
Q4 (once a week)	0.74 (0.29-1.89)[a]	0.11	1.6
	0.71 (0.25-1.96)[b]	0.16	1.7
Q4 (once a day)	1.30 (0.53-3.21)[a]	0.11	1.6
	1.80 (0.65-4.98)[b]	0.16	1.7
Q5 (hardly any/not many	0.84 (0.18-3.97)[a]	0.12	2.2
people)	0.87 (0.18-4.26)[b]	0.16	1.6
Q5 (some people)	1.04 (0.29-3.65)[a]	0.12	2.2
	1.04 (0.25-4.26)[b]	0.16	1.6
Q5 (most people)	**0.47 (0.26-0.84)**[a]	0.12	2.2
	0.53 (0.28-0.99)[b]	0.16	1.6
# of acute stressors (2^{nd}	1.81 (0.82-3.97)[a]	0.12	3.0
quartile)	1.86 (0.80-4.35)[b]	0.17	2.7
# of acute stressors (3^{rd}	**2.68 (1.14-6.30)**[a]	0.12	3.0
quartile)	**2.90 (1.14-7.40)**[b]	0.17	2.7
# of acute stressors (4^{th} quartile)	1.37 (0.63-2.94)[a]	0.12	3.0
	1.67 (0.73-3.82)[b]	0.17	2.7

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, highest qualification, optimism and membership in/part of the Stolen Generations.

Table B.11: Significant associations between racism/stress and high junk food consumption

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1c (sometimes)	2.00 (0.89-4.50)[a]	0.07	1.0
	2.80 (1.02-7.68)[b]	0.22	5.3
Q1c (often/very often)	2.27 (0.68-7.58)[a]	0.07	1.0
	1.22 (0.30-4.98)[b]	0.22	5.3
Q1j (sometimes)	1.12 (0.57-2.15)[a]	0.07	1.2
	0.91 (0.42-1.94)[b]	0.18	0.8
Q1j (often/very often)	**3.12 (1.33-7.34)**[a]	0.07	1.2
	2.82 (1.02-7.83)[b]	0.18	0.8
Q2i (never/hardly ever)	**0.31 (0.13-0.76)**[a]	0.10	4.0
	0.29 (0.11-0.77)[b]	0.20	2.9
Q2i (often/very often)	1.12 (0.55-2.28)[a]	0.10	4.0
	1.05 (0.46-2.37)[b]	0.20	2.9
Q2j (sometimes)	1.67 (0.81-3.43)[a]	0.08	2.5
	1.36 (0.61-3.06)[b]	0.17	0.2
Q2j (often/very often)	**3.04 (1.17-7.91)**[a]	0.08	2.5
	1.75 (0.56-5.46)[b]	0.17	0.2
Q3b (neither agree nor disagree)	1.66 (0.77-3.57)[a]	0.08	1.1
	2.45 (1.02-5.87)[b]	0.17	1.6
Q3b (disagree/strongly disagree)	1.65 (0.79-3.45)[a]	0.07	1.1
	1.92 (0.82-4.50)[b]	0.18	1.6
Q3d (neither agree nor disagree)	4.68 (0.91-24.0)[a]	0.07	1.8
	13.45 (1.50-120.6)[b]	0.18	1.8
Q3d (disagree/strongly disagree)	0.47 (0.10-2.24)[a]	0.07	1.8
	0.43 (0.08-2.37)[b]	0.18	1.8
Total stress score	0.47 (0.10-2.29)[a]	0.07	1.0
(None)[c]	0.46 (0.08-2.62)[b]	0.17	0.1
Total stress score	**2.44 (1.06-5.62)**[a]	0.07	1.0
(Yes, affected me somewhat)[c]	1.81 (0.70-4.66)[b]	0.17	0.1
# of chronic stressors (1^{st}	0.83 (0.35-1.98)[a]	0.07	1.7
quartile)	0.96 (0.32-2.85)[b]	0.19	2.2
# of chronic stressors (2^{nd}	**0.35 (0.15-0.82)**[a]	0.07	1.7
quartile)	0.37 (0.14-1.01)[b]	0.19	2.2
# of chronic stressors (3^{rd}	0.89 (0.42-1.87)[a]	0.07	1.7
quartile)	1.18 (0.50-2.74)[b]	0.19	2.2

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, income, highest qualification, positive and negative social capital, level of social isolation and method of administration.

c The base category for this variable is 'Yes, minimal/no effect' with the category 'Yes, affected me a great deal' omitted due to a lack of variation in outcome within this category.

Table B.12: Significant associations between racism/stress and insufficient fruit & vegetable consumption

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Moderate systemic racism	**0.12 (0.03-0.49)**[a]	0.05	4.9
High systemic racism	**0.28 (0.09-0.86)**[a]	0.05	4.9
Very high systemic racism	**0.19 (0.05-0.75)**[a]	0.05	4.9
Q5 (some people)[b]	1.19 (0.14-10.2)[a]	0.03	2.8
Q5 (most people)[b]	**2.56 (1.18-5.56)**[a]	0.03	2.8
Acute stress score (Yes, minimal/no effect)	1.94 (0.54-6.95)[a]	0.05	4.5
Acute stress score (Yes, affected me somewhat)	**6.79 (1.65-27.9)**[a]	0.05	4.5
Total stress score (None)[c]	0.78 (0.10-6.24)[a]	0.02	1.8
Total stress score (Yes, affected me somewhat)[c]	**2.89 (1.05-7.90)**[a]	0.02	1.8
# of acute stressors (2nd quartile)	1.92 (0.46-8.03)[a]	0.06	5.8
# of acute stressors (3rd quartile)	0.87 (0.14-5.43)[a]	0.06	5.8
# of acute stressors (4th quartile)	**4.70 (1.29-17.2)**[a]	0.06	5.8

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b The base category for this variable is 'Yes, everyone' with the category 'hardly any/not many people' omitted due to a lack of variation in outcome within this category.

c The base category for this variable is 'Yes, minimal/no effect' with the category 'Yes, affected me a great deal' omitted due to a lack of variation in outcome within this category.

Table B.13: Significant associations between racism/stress and depressive symptoms (CES-D)

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	1.18 (0.62-2.26)[a]	0.02	1.4
	0.63 (0.26-1.51)[b]	0.27	1.1
Q1a (often/very often)	**3.00 (1.06-8.47)**[a]	0.02	1.4
	1.70 (0.42-6.80)[b]	0.27	1.1
Q1e (sometimes)	**3.74 (1.83-7.63)**[a]	0.05	4.9
	3.91 (1.57-9.73)[b]	0.30	3.9
Q1e (often/very often)	3.27 (0.88-12.1)[a]	0.05	4.9
	3.00 (0.58-15.6)[b]	0.30	3.9
Q1h (sometimes)	**1.86 (1.04-3.32)**[a]	0.03	2.3
	2.08 (0.97-4.46)[b]	0.27	1.4
Q1h (often/very often)	2.90 (0.99-8.46)[a]	0.03	2.3
	2.25 (0.57-8.94)[b]	0.27	1.4
Q1j (sometimes)	1.43 (0.78-2.60)[a]	0.03	2.8
	0.86 (0.39-1.90)[b]	0.27	1.1
Q1j (often/very often)	**3.57 (1.56-8.15)**[a]	0.03	2.8
	1.90 (0.64-5.63)[b]	0.27	1.1
Inter-personal racism score (none)	0.90 (0.47-1.72)[a]	0.02	1.6
	1.15 (0.49-2.70)[b]	0.28	1.9
Inter-personal racism score (high)	**1.91 (1.01-3.64)**[a]	0.02	1.6
	1.97 (0.84-4.59)[b]	0.28	1.9
Q2b (sometimes)	**2.10 (1.05-4.20)**[a]	0.02	1.8
	1.56 (0.63-3.88)[b]	0.28	2.3
Q2b (often/very often)	1.20 (0.57-2.49)[a]	0.02	1.8
	0.62 (0.23-1.70)[b]	0.28	2.3
Q2c (sometimes)	**2.48 (1.16-5.33)**[a]	0.03	2.1
	2.57 (0.93-7.08)[b]	0.29	3.0
Q2c (often/very often)	1.46 (0.67-3.16)[a]	0.03	2.1
	2.14 (0.75-6.18)[b]	0.29	3.0
Q2f (sometimes)	**2.52 (1.23-5.20)**[a]	0.05	4.3
	2.37 (0.92-6.13)[b]	0.31	5.1
Q2f (often/very often)	**3.14 (1.48-6.68)**[a]	0.05	4.3
	4.43 (1.43-13.7)[b]	0.31	5.1
Q2g (sometimes)	**2.82 (1.45-5.47)**[a]	0.05	4.5
	2.58 (1.08-6.13)[b]	0.29	3.4
Q2g (often/very often)	**2.95 (1.05-8.27)**[a]	0.05	4.5
	1.88 (0.42-8.47)[b]	0.29	3.4
Q2j (sometimes)	**2.63 (1.36-5.06)**[a]	0.04	3.9
	2.51 (1.04-6.04)[b]	0.30	3.4
Q2j (often/very often)	**2.70 (1.09-6.66)**[a]	0.04	3.9
	1.07 (0.29-3.94)[b]	0.30	3.4
Q3f (neither agree nor disagree)	1.76 (0.92-3.36)[a]	0.03	2.8
	1.53 (0.68-3.43)[b]	0.26	0.3

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q3f (disagree/strongly disagree)	**3.16 (1.43-6.98)**[a]	0.03	2.8
	3.01 (1.00-9.01)[b]	0.26	0.3
Acute stress score (Yes, minimal/no effect)	**2.39 (1.18-4.84)**[a]	0.02	2.0
	2.67 (1.02-7.01)[b]	0.27	1.3
Acute stress score (Yes, affected me somewhat)	2.16 (0.82-5.68)[a]	0.02	2.0
	1.69 (0.49-5.78)[b]	0.27	1.3
Chronic stress score (None)[c]	0.30 (0.07-1.35)[a]	0.04	3.7
	0.80 (0.14-4.57)[b]	0.29	2.6
Chronic stress score (Yes, affected me somewhat)[c]	**3.11 (1.44-6.70)**[a]	0.04	3.7
	3.56 (1.27-9.97)[b]	0.29	2.6
# of acute stressors (2nd quartile)	1.38 (0.59-3.21)[a]	0.05	4.7
	1.52 (0.50-4.62)[b]	0.28	2.3
# of acute stressors (3rd quartile)	1.61 (0.66-3.92)[a]	0.05	4.7
	1.91 (0.60-6.07)[b]	0.28	2.3
# of acute stressors (4th quartile)	**3.73 (1.73-8.01)**[a]	0.05	4.7
	3.91 (1.37-11.1)[b]	0.28	2.3
# of chronic stressors (1st quartile)	**0.08 (0.03-0.25)**[a]	0.11	10.2
	0.09 (0.02-0.45)[b]	0.32	6.0
# of chronic stressors (2nd quartile)	**0.20 (0.09-0.43)**[a]	0.11	10.2
	0.17 (0.06-0.49)[b]	0.32	6.0
# of chronic stressors (3rd quartile)	**0.41 (0.21-0.82)**[a]	0.11	10.2
	0.37 (0.15-0.91)[b]	0.32	6.0
# of total stressors (1st quartile)	**0.26 (0.07-0.98)**[a]	0.11	10.5
	0.26 (0.50-1.38)[b]	0.31	5.4
# of total stressors (3rd quartile)	**2.24 (1.02-4.95)**[a]	0.11	10.5
	1.98 (0.72-5.40)[b]	0.31	5.4
# of total stressors (4th quartile)	**4.20 (1.95-9.04)**[a]	0.11	10.5
	4.01 (1.49-10.79)[b]	0.31	5.4
Any experienced stressors that affected respondent somewhat/a great deal	**2.59 (1.09-6.15)**[a]	0.02	1.5
	2.59 (0.81-8.31)[b]	0.27	0.8
Any experienced stressors that affected respondent a great deal	**3.09 (1.67-5.71)**[a]	0.05	4.1
	3.10 (1.37-7.03)[b]	0.28	2.3

CI: Confidence interval; **OR**: odds ratio.
a adjusted for age and sex.
b adjusted for age and sex, household composition, income, highest qualification, optimism, lack of control, positive and negative social capital, positive instrumental social support and level of social isolation.
c the base category for this variable is 'Yes, minimal/no effect' with the category 'Yes, affected me a great deal' omitted due to a lack of variation in outcome within this category.

Table B.14: Significant associations between racism/stress and poor/fair self-assessed health status

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**2.03 (1.06-3.91)**[a]	0.05	2.7
	1.99 (0.95-4.16)[b]	0.14	0.6
Q1a (often/very often)	**4.31 (1.51-12.3)**[a]	0.05	2.7
	2.49 (0.76-8.12)[b]	0.14	0.6
Q1c (sometimes)	1.15 (0.48-2.75)[a]	0.04	2.5
	1.21 (0.46-3.19)[b]	0.14	0.7
Q1c (often/very often)	**4.31 (1.35-13.8)**[a]	0.04	2.5
	4.29 (1.18-15.6)[b]	0.14	0.7
Q1g (sometimes)	1.32 (0.63-2.78)[a]	0.03	1.4
	1.51 (0.65-3.47)[b]	0.14	0.8
Q1g (often/very often)	**3.60 (1.05-12.4)**[a]	0.03	1.4
	3.75 (0.85-16.5)[b]	0.14	0.8
Q1h (sometimes)	1.74 (0.95-3.19)[a]	0.05	2.7
	2.13 (1.05-4.32)[b]	0.15	1.5
Q1h (often/very often)	**4.50 (1.51-13.4)**[a]	0.05	2.7
	3.95 (1.13-13.8)[b]	0.15	1.5
Inter-personal racism score (none)	1.04 (0.52-2.07)[a]	0.03	1.2
	1.06 (0.48-2.30)[b]	0.14	0.4
Inter-personal racism score (high)	**2.00 (1.02-3.95)**[a]	0.03	1.2
	2.08 (0.96-4.51)[b]	0.14	0.4
Q2g (sometimes)	1.09 (0.53-2.27)[a]	0.04	2.1
	1.02 (0.44-2.35)[b]	0.18	4.4
Q2g (often/very often)	**3.25 (1.18-8.98)**[a]	0.04	2.1
	3.26 (0.97-11.0)[b]	0.18	4.4
Q2h (never/hardly ever)	0.97 (0.36-2.62)[a]	0.07	5.6
	0.72 (0.22-2.33)[b]	0.23	9.5
Q2h (often/very often)	**3.19 (1.56-6.51)**[a]	0.07	5.6
	4.01 (1.71-9.44)[b]	0.23	9.5
Q2j (sometimes)	1.67 (0.82-3.41)[a]	0.07	4.7
	1.66 (0.71-3.86)[b]	0.20	6.7
Q2j (often/very often)	**4.76 (1.87-12.1)**[a]	0.07	4.7
	4.84 (1.56-15.0)[b]	0.20	6.7
Chronic stress score (None)[c]	0.34 (0.07-1.55)[a]	0.04	2.1
	0.35 (0.06-1.85)[b]	0.16	2.5
Chronic stress score (Yes, affected me somewhat)[c]	2.11 (1.00-4.48)[a]	0.04	2.1
	2.64 (1.09-6.39)[b]	0.16	2.5
Total stress score (None)[c]	0.48 (0.10-2.27)[a]	0.04	1.7
	0.71 (0.13-4.06)[b]	0.14	0.5
Total stress score (Yes, affected me somewhat)[c]	**2.38 (1.06-5.35)**[a]	0.04	1.7
	2.24 (0.91-5.54)[b]	0.14	0.5

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
# of chronic stressors	**0.35 (0.13-0.93)**[a]	0.04	1.9
(1st quartile)	0.43 (0.13-1.40)[b]	0.14	0.2
# of chronic stressors (2nd	0.64 (0.30-1.37)[a]	0.04	1.9
quartile)	0.73 (0.30-1.81)[b]	0.14	0.2
# of chronic stressors (3rd	0.68 (0.32-1.46)[a]	0.04	1.9
quartile)	0.73 (0.31-1.70)[b]	0.14	0.2

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, household composition, highest qualification, optimism, lack of control, positive social capital and level of social isolation.

c The base category for this variable is 'Yes, minimal/no effect' with the category 'Yes, affected me a great deal' omitted due to a lack of variation in outcome within this category.

Table B.15: Significant associations between racism/stress and SF-12 mental health scores

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**-5.07 (-7.95, -2.18)**[a]	0.12	8.2
	-3.28 (-6.23,-0.33)[b]	0.31	3.3
Q1a (often/very often)	**-10.35 (-15.6, -5.06)**[a]	0.12	8.2
	-6.02 (-11.4,-0.60)[b]	0.31	3.3
Q1b (sometimes)	-3.28 (-7.30, 0.73)[a]	0.05	1.6
	-1.24 (-5.27, 2.79)[b]	0.19	0.0
Q1b (often/very often)	**-7.19 (-13.9, -0.46)**[a]	0.05	1.6
	-5.49 (-12.2, 1.17)[b]	0.19	0.0
Q1c (sometimes)	-0.04 (-3.89, 3.81)[a]	0.06	2.9
	0.83 (-2.91, 4.56)[b]	0.20	0.7
Q1c (often/very often)	**-9.53 (-15.4, -3.65)**[a]	0.06	2.9
	-7.15 (-12.8, -1.48)[b]	0.20	0.7
Q1e (sometimes)	-2.64 (-6.08, 0.80)[a]	0.05	1.7
	-1.04 (-4.37, 2.29)[b]	0.19	0.0
Q1e (often/very often)	-5.99 (-12.1, 0.15)[a]	0.05	1.7
	-6.41 (-12.6, -0.26)[b]	0.19	0.0
Q1g (sometimes)	-0.92 (-4.32, 2.49)[a]	0.05	1.2
	0.23 (-3.18, 3.64)[b]	0.20	0.3
Q1g (often/very often)	**-6.48 (-12.6, -0.35)**[a]	0.05	1.2
	-2.81 (-9.08, 3.46)[b]	0.20	0.3
Q1h (sometimes)	**-4.34 (-7.05, -1.63)**[a]	0.08	5.0
	-3.61 (-6.35, -0.87)[b]	0.22	2.7
Q1h (often/very often)	**-7.35 (-12.58, -2.13)**[a]	0.08	5.0
	-4.40 (-9.54, 0.74)[b]	0.22	2.7
Q1i (sometimes)	**-3.37 (-6.19, -0.54)**[a]	0.06	2.2
	-1.65 (-4.51, 1.20)[b]	0.21	1.0
Q1i (often/very often)	-4.29 (-9.96, 1.39)[a]	0.06	2.2
	-2.95 (-8.52, 2.62)[b]	0.21	1.0
Q1j (sometimes)	-2.42 (-5.23, 0.39)[a]	0.10	4.9
	0.63 (-2.27, 3.53)[b]	0.21	1.9
Q1j (often/very often)	**-8.40 (-12.6, -4.2)**[a]	0.10	4.9
	-4.51 (-8.72, -0.31)[b]	0.21	1.9
Inter-personal racism score (none)	0.09 (-2.95, 3.13)[a]	0.07	2.5
	-1.42 (-4.40, 1.56)[b]	0.20	0.7
Inter-personal racism score (high)	**-4.13 (-7.23, -1.03)**[a]	0.07	2.5
	-3.46 (-6.57, -0.35)[b]	0.20	0.7
Q2f (sometimes)	**-5.23 (-8.75, -1.71)**[a]	0.05	1.9
	-2.79 (-6.32, 0.74)[b]	0.22	2.0
Q2f (often/very often)	-1.08 (-4.78, 2.62)[a]	0.05	1.9
	1.18 (-2.61, 4.97)[b]	0.22	2.0
Q2g (sometimes)	**-3.95 (-7.20, -0.70)**[a]	0.07	3.3
	-2.74 (-5.96, 0.48)[b]	0.21	1.6

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2g (often/very often)	**-7.15 (-12.3, -1.97)**[a]	0.07	3.3
	-2.99 (-8.11, 2.12)[b]	0.21	1.6
Q2h (never/hardly ever)	3.93 (-0.06, 7.93)[a]	0.06	2.7
	4.59 (0.50, 8.67)[b]	0.23	3.6
Q2h (often/very often)	-2.58 (-5.74, 0.58)[a]	0.06	2.7
	-0.80 (-4.01, 2.40)[b]	0.23	3.6
Q2j (sometimes)	**-5.41 (-8.58, -2.25)**[a]	0.10	6.1
	-3.92 (-7.13, -0.72)[b]	0.23	3.7
Q2j (often/very often)	**-7.83 (-12.4, -3.23)**[a]	0.10	6.1
	-4.49 (-9.12, 0.14)[b]	0.23	3.7
Q2k (sometimes)	-3.02 (-6.39, 0.35)[a]	0.04	1.0
	-2.37 (-5.62, 0.88)[b]	0.21	1.7
Q2k (often/very often)	**-4.84 (-8.87, -0.81)**[a]	0.04	1.0
	-2.83 (-6.81, 1.15)[b]	0.21	1.7
Q3b (neither agree nor	**4.35 (0.77, 7.93)**[a]	0.06	1.6
disagree)	3.38 (-0.11, 6.86)[b]	0.21	1.9
Q3b (disagree/strongly	1.39 (-2.14, 4.92)[a]	0.06	1.6
disagree)	-0.06 (-3.51, 3.39)[b]	0.21	1.9
Q3f (neither agree nor	**-3.28 (-6.40, -0.15)**[a]	0.04	1.1
disagree)	-2.72 (-5.69, 0.25)[b]	0.21	1.8
Q3f (disagree/strongly	-0.87 (-4.76, 3.03)[a]	0.04	1.1
disagree)	-0.01 (-3.82, 3.80)[b]	0.21	1.8
Acute stress score	**-4.92 (-7.90, -1.94)**[a]	0.08	4.3
(Yes, minimal/no effect)	**-3.19 (-6.25, -0.12)**[b]	0.22	2.3
Acute stress score	**-7.33 (-11.66, -3.01)**[a]	0.08	4.3
(Yes, affected me somewhat)	**-5.45 (-9.77, -1.12)**[b]	0.22	2.3
Chronic stress score	2.89 (-2.71, 8.48)[a]	0.09	5.6
(None)	-0.55 (-5.96, 4.87)[b]	0.22	2.4
Chronic stress score	**-7.58 (-11.2, -4.02)**[a]	0.09	5.6
(Yes, affected me somewhat)	**-6.10 (-9.72, -2.47)**[b]	0.22	2.4
Chronic stress score	2.68 (-16.8, 22.1)[a]	0.09	5.6
(Yes, affected me a great deal)	-0.94 (-19.5, 17.7)[b]	0.22	2.4
Total stress score	4.78 (-1.55, 11.1)[a]	0.08	5.0
(None)	0.82 (-5.31, 6.95)[b]	0.21	1.2
Total stress score	**-7.59 (-11.5, -3.64)**[a]	0.08	5.0
(Yes, affected me somewhat)	**-5.12 (-8.98, -1.26)**[b]	0.21	1.2
Total stress score	2.86 (-16.7, 22.4)[a]	0.08	5.0
(Yes, affected me a great deal)	-0.03 (-18.7, 18.7)[b]	0.21	1.2
# of acute stressors (2^{nd}	-3.23 (-6.73, 0.28)[a]	0.08	4.4
quartile)	-1.27 (-4.79, 2.24)[b]	0.23	3.6
# of acute stressors (3^{rd}	**-5.55 (-9.42, -1.69)**[a]	0.08	4.4
quartile)	**-4.79 (-8.63, -0.95)**[b]	0.23	3.6
# of acute stressors (4^{th}	**-6.32 (-9.68, -2.96)**[a]	0.08	4.4
quartile)	**-3.83 (-7.25, -0.42)**[b]	0.23	3.6

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
# of chronic stressors (1st quartile)	**9.41 (5.42, 13.39)**[a]	0.12	8.6
	5.06 (0.54, 9.59)[b]	0.20	0.1
# of chronic stressors (2nd quartile)	**6.52 (3.19, 9.84)**[a]	0.12	8.6
	4.13 (0.65, 7.60)[b]	0.20	0.1
# of chronic stressors (3rd quartile)	**4.43 (1.13, 7.74)**[a]	0.12	8.6
	3.00 (-0.51, 6.51)[b]	0.20	0.1
# of total stressors (1st quartile)	2.97 (-0.84, 6.77)[a]	0.10	6.2
	1.51 (-2.42, 5.45)[b]	0.20	0.6
# of total stressors (3rd quartile)	-3.38 (-6.84, 0.07)[a]	0.10	6.2
	-2.20 (-5.64, 1.25)[b]	0.20	0.6
# of total stressors (4th quartile)	**-5.01 (-8.36, -1.66)**[a]	0.10	6.2
	-3.23 (-6.60, 0.13)[b]	0.20	0.6
Any experienced stressor	**-6.47 (-12.1, -0.83)**[a]	0.05	1.5
	-0.98 (-6.61, 4.66)[b]	0.19	0.0
Any experienced stressors that affected respondent somewhat/a great deal	-5.72 (-9.17, 2.27)[a]	0.07	3.4
	-2.94 (-6.43, 0.55)[b]	0.20	0.4
Any experienced stressors that affected respondent a great deal	**-5.38 (-7.91, -2.85)**[a]	0.09	5.6
	-3.07 (-5.73, -0.40)[b]	0.21	1.2

CI: Confidence interval.

a Adjusted for age and sex.

b Adjusted for age and sex, income, house tenure, optimism, lack of control, positive social capital, negative social capital, positive instrumental social support, level of social isolation and method of administration.

Table B.16: Significant associations between racism/stress and SF-12 physical health scores

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1d (sometimes)	-0.04 (-2.97, 2.89)[a]	0.05	0.0
	-0.19 (-3.06, 2.67)[b]	0.17	0.0
Q1d (often/very often)	**-8.85 (-17.4, -0.28)**[a]	0.05	0.0
	-7.87 (-16.8, 1.03)[b]	0.17	0.0
Q2j (sometimes)	-1.38 (-4.07, 1.32)[a]	0.08	2.1
	-0.12 (-2.86, 2.62)[b]	0.18	0.0
Q2j (often/very often)	**-4.37 (-8.29, -0.46)**[a]	0.08	2.1
	-3.11 (-7.09, 0.87)[b]	0.18	0.0
Q3b (neither agree nor disagree)	1.32 (-1.76, 4.39)[a]	0.08	2.1
	2.47 (-0.63, 5.57)[b]	0.22	2.8
Q3b (disagree/strongly disagree)	**-3.22 (-6.25, -0.18)**[a]	0.08	2.1
	-2.54 (-5.52, 0.44)[b]	0.22	2.8
Q3f (neither agree nor disagree)	**2.71 (0.05, 5.36)**[a]	0.08	2.6
	4.23 (1.66, 6.80)[b]	0.22	3.3
Q3f (disagree/strongly disagree)	-2.34 (-5.65, 0.97)[a]	0.08	2.6
	-0.80 (-4.15, 2.55)[b]	0.22	3.3
Q4 (never/once a year)	0.39 (-2.50, 3.28)[a]	0.08	0.8
	1.09 (-1.82, 4.00)[b]	0.20	1.4
Q4 (once a month)	-3.36 (-7.64, 0.91)[a]	0.08	0.8
	-4.71 (-9.06, -0.35)[b]	0.20	1.4
Q4 (once a week)	1.05 (-2.90, 5.00)[a]	0.08	0.8
	0.44 (-3.50, 4.38)[b]	0.20	1.4
Q4 (once a day)	2.39 (-1.17, 5.95)[a]	0.08	0.8
	1.30 (-2.25, 4.85)[b]	0.20	1.4
Q5 (hardly any/not many people)	1.69 (-4.49, 7.87)[a]	0.08	1.0
	-0.15 (-6.42, 6.11)[b]	0.20	0.9
Q5 (some people)	**-6.21 (-11.6, -0.84)**[a]	0.08	1.0
	-5.89 (-11.2, -0.63)[b]	0.20	0.9
Q5 (most people)	-0.90 (-3.19, 1.39)[a]	0.08	1.0
	-1.28 (-3.54, 0.98)[b]	0.20	0.9
Q6 (hardly any/not many people)	-1.16 (-4.60, 2.28)[a]	0.05	0.0
	-1.05 (-4.54, 2.44)[b]	0.17	0.0
Q6 (some people)	-1.73 (-4.74, 1.28)[a]	0.05	0.0
	-2.95 (-6.04, 0.13)[b]	0.17	0.0
Q6 (most people)	-1.51 (-4.34, 1.32)[a]	0.05	0.0
	-3.42 (-6.35, -0.50)[b]	0.17	0.0
Total stress score (None)	1.30 (-4.25, 6.85)[a]	0.08	2.9
	1.48 (-3.88, 6.85)[b]	0.20	0.9
Total stress score (Yes, affected me somewhat)	**-4.51 (-7.98, -1.05)**[a]	0.08	2.9
	-2.83 (-6.25, 0.60)[b]	0.20	0.9

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Total stress score	0.12 (-17.0, 17.2)[a]	0.08	2.9
(Yes, affected me a great deal)	-3.60 (-20.2, 13.0)[b]	0.20	0.9
# of total stressors (1st quartile)	2.49 (-0.91, 5.89)[a]	0.07	1.4
	4.04 (0.66, 7.43)[b]	0.21	2.4
# of total stressors (3rd quartile)	1.61 (-1.47, 4.69)[a]	0.07	1.4
	2.16 (-0.83, 5.14)[b]	0.21	2.4
# of total stressors (4th quartile)	-1.05 (-4.04, 1.93)[a]	0.07	1.4
	0.07 (-2.86, 3.00)[b]	0.21	2.4

CI: Confidence interval.
a Adjusted for age and sex.
b Adjusted for age and sex, income, highest qualification, optimism, lack of control, positive instrumental social support and level of social isolation.

Table B.17: Significant associations between racism/stress and waist circumference

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1c (sometimes)	1.11 (-4.78, 7.01)[a]	0.09	2.0
	0.97 (-5.00, 6.94)[b]	0.19	1.7
Q1c (often/very often)	**9.17 (0.26, 18.09)**[a]	0.09	2.0
	5.00 (-4.18, 14.2)[b]	0.19	1.7
Q3c (strongly agree/agree)	0.03 (-4.94, 5.01)[a]	0.16	0.5
	-1.30 (-6.71, 4.12)[b]	0.07	0.8
Q3c (neither agree nor disagree)	-3.07 (-7.63, 1.49)[a]	0.16	0.5
	-4.85 (-9.61, -0.09)[b]	0.07	0.8
Q3e (neither agree nor disagree)	**-6.06 (-11.5, -0.57)**[a]	0.08	1.5
	-4.34 (-10.3, 1.61)[b]	0.17	0.0
Q3e (disagree/strongly disagree)	-1.37 (-5.32, 2.58)[a]	0.08	1.5
	0.32 (-3.83, 4.47)[b]	0.17	0.0
Q3f (neither agree nor disagree)	-3.50 (-8.08, 1.08)[a]	0.08	1.6
	-2.61 (-7.42, 2.20)[b]	0.18	1.1
Q3f (disagree/strongly disagree)	**-6.16 (-11.9, -0.41)**[a]	0.08	1.6
	-6.28 (-12.3, -0.25)[b]	0.18	1.1
No internalised racism	2.13 (-4.89, 9.15)[a]	0.08	1.4
	5.74 (-1.42, 12.9)[b]	0.19	1.8
High internalised racism	**-4.29 (-8.49, -0.09)**[a]	0.08	1.4
	-2.73 (-7.11, 1.65)[b]	0.19	1.8
# of acute stressors (2^{nd} quartile)	**6.64 (1.43, 11.86)**[a]	0.10	2.9
	7.87 (2.49, 13.2)[b]	0.21	3.3
# of acute stressors (3^{rd} quartile)	-0.22 (-6.05, 5.62)[a]	0.10	2.9
	1.20 (-4.93, 7.32)[b]	0.21	3.3
# of acute stressors (4^{th} quartile)	3.03 (-2.04, 8.09)[a]	0.10	2.9
	2.37 (-2.84, 7.59)[b]	0.21	3.3

CI: Confidence interval.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, lack of control (work settings excluded), smoking status (smoker vs. non-smoker), junk food consumption, self-assessed health status and SF-12 physical component score.

Table B.18: Significant associations between racism/stress and waist-to-hip ratio

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Inter-personal racism score	**0.04 (0.01, 0.06)**[a]	0.17	2.3
(none)	0.02 (-0.01, 0.05)[b]	0.18	0.0
Inter-personal racism score	0.00 (-0.03, 0.03)[a]	0.17	2.3
(high)	0.01 (-0.03, 0.04)[b]	0.18	0.0
Q3b (neither agree nor disagree)	-0.01 (-0.04, 0.03)[a]	0.19	4.5
	-0.01 (-0.04, 0.03)[b]	0.22	1.9
Q3b (disagree/strongly disagree)	**0.06 (0.03, 0.09)**[a]	0.19	4.5
	0.05 (0.02, 0.09)[b]	0.22	1.9
Q4 (never/once a year)	**0.05 (0.02, 0.08)**[a]	0.19	4.3
	0.04 (0.00, 0.07)[b]	0.25	3.9
Q4 (once a month)	-0.02 (-0.06, 0.03)[a]	0.19	4.3
	-0.03 (-0.07, 0.02)[b]	0.25	3.9
Q4 (once a week)	-0.01 (-0.05, 0.03)[a]	0.19	4.3
	-0.01 (-0.05, 0.04)[b]	0.25	3.9
Q4 (once a day)	0.00 (-0.04, 0.04)[a]	0.19	4.3
	0.00 (-0.04, 0.04)[b]	0.25	3.9
Any experienced stressor	**-0.05 (-0.10, -0.01)**[a]	0.17	1.8
	-0.01 (-0.07, 0.04)[b]	0.20	0.0
Any experienced stressors that	**-0.04 (-0.07, -0.01)**[a]	0.17	2.0
affected respondent somewhat/a great deal	-0.02 (-0.06, 0.01)[b]	0.20	0.0

CI: Confidence interval.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income, lack of control (work settings excluded), smoking status (smoker vs. non-smoker) and junk food consumption, self-assessed health status and SF-12 physical component score.

Table B.19: Significant associations between racism/stress and mean systolic blood pressure

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1a (sometimes)	**-6.38 (-11.0, -1.74)**[a]	0.23	2.5
	-4.53 (-9.36, 0.31)[b]	0.20	2.2
Q1a (often/very often)	-4.78 (-12.6, 2.99)[a]	0.23	2.5
	-1.89 (-10.0, 6.23)[b]	0.20	2.2
# of setting racism experienced in	-4.38 (-9.23, 0.47)[a]	0.22	1.9
(2^{nd} quartile)	**-5.59 (-10.6, -0.60)**[b]	0.25	6.9
# of setting racism experienced in	0.06 (-5.10, 5.21)[a]	0.22	1.9
(3^{rd} quartile)	2.97 (-2.37, 8.31)[b]	0.25	6.9
# of setting racism experienced in	-4.01 (-9.67, 1.65)[a]	0.22	1.9
(4^{th} quartile)	-4.15 (-10.38, 2.08)[b]	0.25	6.9
Q2c (sometimes)	3.75 (-1.53, 9.03)[a]	0.20	0.0
	5.97 (0.11, 11.8)[b]	0.21	3.0
Q2c (often/very often)	-3.41 (-8.58, 1.76)[a]	0.20	0.0
	-0.80 (-6.32, 4.71)[b]	0.21	3.0
Q3d (neither agree nor disagree)	**-11.9 (-22.3, -1.40)**[a]	0.21	0.6
	-13.6 (-25.3, -1.84)[b]	0.20	1.9
Q3d (disagree/strongly disagree)	-4.3 (-12.4, 3.83)[a]	0.21	0.6
	-6.86 (-15.6, 1.85)[b]	0.20	1.9
No internalised racism	**-7.46 (-13.9, -1.05)**[a]	0.21	0.9
	-7.33 (-13.8, -0.88)[b]	0.20	1.6
High internalised racism	-2.96 (-6.91, 1.00)[a]	0.21	0.9
	-2.23 (-6.39, 1.93)[b]	0.20	1.6
Q4 (never/once a year)	0.60 (-4.00, 5.20)[a]	0.20	0.0
	-1.20 (-6.20, 3.79)[b]	0.21	2.6
Q4 (once a month)	4.47 (-2.34, 11.29)[a]	0.20	0.0
	8.02 (1.12, 14.9)[b]	0.21	2.6
Q4 (once a week)	-3.72 (-10.2, 2.75)[a]	0.20	0.0
	-0.81 (-7.48, 5.87)[b]	0.21	2.6
Q4 (once a day)	4.70 (-1.27, 10.7)[a]	0.20	0.0
	6.42 (0.42, 12.4)[b]	0.21	2.6
Q6 (hardly any/not many	-3.88 (-9.35, 1.60)[a]	0.23	2.8
people)	-1.39 (-7.61, 4.83)[b]	0.19	1.0
Q6 (some people)	**-5.57 (-10.4, -0.71)**[a]	0.23	2.8
	-3.08 (-8.51, 2.34)[b]	0.19	1.0
Q6 (most people)	**-5.65 (-10.3, -1.02)**[a]	0.23	2.8
	-4.69 (-9.99, 0.61)[b]	0.19	1.0
Total stress score	1.54 (-6.70, 9.78)[a]	0.20	0.0
(None)	-2.07 (-11.0, 6.88)[b]	0.21	2.6
Total stress score	-5.60 (-12.2, 1.03)[a]	0.20	0.0
(Yes, affected me somewhat)	**-7.28 (-13.9, -0.70)**[b]	0.21	2.6
Total stress score	-23.7 (-51.6, 4.13)[a]	0.20	0.0
(Yes, affected me a great deal)	-25.5 (-52.2, 1.26)[b]	0.21	2.6

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
# of chronic stressors (1st quartile)	**9.14 (3.33, 15.0)**[a]	0.23	3.1
	6.51 (-0.39, 13.4)[b]	0.21	2.5
# of chronic stressors (2nd quartile)	**8.49 (3.42, 13.6)**[a]	0.23	3.1
	5.03 (-0.48, 10.5)[b]	0.21	2.5
# of chronic stressors (3rd quartile)	2.10 (-3.11, 7.31)[a]	0.23	3.1
	3.03 (-2.45, 8.51)[b]	0.21	2.5
# of total stressors (1st quartile)	-1.80 (-7.43, 3.83)[a]	0.23	2.5
	-1.91 (-8.11, 4.28)[b]	0.21	3.1
# of total stressors (3rd quartile)	**-8.76 (-14.0, -3.56)**[a]	0.23	2.5
	-6.31 (-11.6, -1.07)[b]	0.21	3.1
# of total stressors (4th quartile)	**-6.65 (-11.8, -1.53)**[a]	0.23	2.5
	-4.15 (-9.51, 1.20)[b]	0.21	3.1
Any experienced stressors that affected respondent somewhat/a great deal	**-5.13 (-9.71, -0.54)**[a]	0.20	0.0
	-4.55 (-9.84, 0.74)[b]	0.19	0.9
Any experienced stressors that affected respondent a great deal	**-4.29 (-8.00, -0.58)**[a]	0.20	0.0
	-4.64 (-8.81, -0.47)[b]	0.20	1.7

CI: Confidence interval.

a Adjusted for age and sex.

b Adjusted for age and sex, income, lack of control, positive and negative social capital, level of social isolation, whether respondent had any non-Indigenous grandparents and alcohol consumption level.

Table B.20: Significant associations between racism/stress and mean diastolic blood pressure

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2a (never/hardly ever)	-1.04 (-4.33, 2.26)[a]	0.17	4.6
	-0.73 (-4.25, 2.78)[b]	0.16	3.8
Q2a (sometimes)	**-3.31 (-6.46, -0.16)**[a]	0.17	4.6
	-3.32 (-6.76, 0.12)[b]	0.16	3.8
Q2c (sometimes)	**3.38 (0.05, 6.71)**[a]	0.17	4.5
	3.83 (0.09, 7.58)[b]	0.16	4.4
Q2c (often/very often)	-1.94 (-5.20, 1.32)[a]	0.17	4.5
	-0.87 (-4.40, 2.66)[b]	0.16	4.4
Q3c (strongly	**-4.21 (-7.33, -1.10)**[a]	0.14	1.9
agree/agree)	**-3.61 (-7.16, -0.06)**[b]	0.13	1.6
Q3c (neither agree nor	-1.76 (-4.65, 1.12)[a]	0.14	1.9
disagree)	-2.24 (-5.31, 0.84)[b]	0.13	1.6
Q3d (neither agree nor	**-7.33 (-14.1, -0.52)**[a]	0.13	2.1
disagree)	**-9.15 (-17.04, -1.26)**[b]	0.13	0.8
Q3d (disagree/strongly	-0.69 (-5.98, 4.59)[a]	0.13	2.1
disagree)	-1.69 (-7.54, 4.16)[b]	0.13	0.8
Q6 (hardly any/not	**-4.14 (-7.70, -0.59)**[a]	0.18	5.3
many people)	-2.87 (-6.89, 1.16)[b]	0.15	3.7
Q6 (some people)	**-4.55 (-7.71, -1.39)**[a]	0.18	5.3
	-3.63 (-7.14, -0.12)[b]	0.15	3.7
Q6 (most people)	**-3.53 (-6.54, -0.52)**[a]	0.18	5.3
	-2.96 (-6.39, 0.47)[b]	0.15	3.7
# of total stressors (1st	-2.34 (-6.08, 1.40)[a]	0.14	1.8
quartile)	-2.88 (-7.14, 1.38)[b]	0.13	1.3
# of total stressors (3rd	**-5.16 (-8.61, -1.72)**[a]	0.14	1.8
quartile)	-3.38 (-6.99, 0.23)[b]	0.13	1.3
# of total stressors (4th	-2.88 (-6.27, 0.52)[a]	0.14	1.8
quartile)	-1.59 (-5.28, 2.09)[b]	0.13	1.3

CI: Confidence interval.

a Adjusted for age and sex.

b Adjusted for age and sex, income, lack of control, positive and negative social capital, level of social isolation, whether respondent had any non-Indigenous grandparents and alcohol consumption level.

Table B.21: Significant associations between racism/stress and clinically-measured hypertension

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1c (sometimes)	0.31 (0.04-2.46)[a]	0.21	5.7
	0.28 (0.02-3.47)[b]	0.31	6.0
Q1c (often/very often)	**5.41 (1.12-26.1)[a]**	0.21	5.7
	4.82 (0.48-48.0)[b]	0.31	6.0
Q6 (hardly any/not many people)	0.25 (0.06-1.05)[a]	0.20	5.4
	0.18 (0.02-1.35)[b]	0.28	2.8
Q6 (some people)	**0.31 (0.10-0.94)[a]**	0.20	5.4
	0.51 (0.14-1.81)[b]	0.28	2.8
Q6 (most people)	**0.28 (0.10-0.73)[a]**	0.20	5.4
	0.30 (0.09-0.96)[b]	0.28	2.8
# of total stressors (1st quartile)	**0.25 (0.07-0.92)[a]**	0.18	3.5
	0.23 (0.04-1.14)[b]	0.33	7.5
# of total stressors (3rd quartile)	0.44 (0.13-1.53)[a]	0.18	3.5
	0.35 (0.08-1.59)[b]	0.33	7.5
# of total stressors (4th quartile)	0.40 (0.12-1.35)[a]	0.18	3.5
	0.23 (0.04-1.21)[b]	0.33	7.5

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, lack of control, positive social capital, junk food consumption and depression (CES-D).

Table B.22: Significant associations between racism/stress and clinically-measured dyslipidaemia

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2e (never/hardly ever)	1.08 (0.54-2.17)[a]	0.04	2.5
	1.11 (0.52-2.41)[b]	0.11	5.4
Q2e (sometimes)	**2.64 (1.26-5.54)[a]**	0.04	2.5
	2.84 (1.28-6.33)[b]	0.11	5.4
Q2f (sometimes)	**2.90 (1.28-6.60)[a]**	0.05	3.0
	3.35 (1.37-8.20)[b]	0.12	6.9
Q2f (often/very often)	0.79 (0.38-1.66)[a]	0.05	3.0
	0.74 (0.34-1.63)[b]	0.12	6.9
Q2i (never/hardly ever)	**0.39 (0.19-0.80)[a]**	0.04	2.2
	0.40 (0.18-0.88)[b]	0.11	5.1
Q2i (often/very often)	0.92 (0.45-1.89)[a]	0.04	2.2
	1.00 (0.47-2.13)[b]	0.11	5.1
Q3d (neither agree nor disagree)	**0.08 (0.01-0.65)[a]**	0.05	2.9
	0.07 (0.01-0.71)[b]	0.10	4.3
Q3d (disagree/strongly disagree)	0.50 (0.16-1.58)[a]	0.05	2.9
	0.52 (0.15-1.84)[b]	0.10	4.3
Q3g (strongly agree/agree)	**0.50 (0.25-0.98)[a]**	0.03	1.5
	0.47 (0.23-0.97)[b]	0.09	3.2
Q3g (neither agree nor disagree)	0.59 (0.27-1.31)[a]	0.03	1.5
	0.56 (0.24-1.31)[b]	0.09	3.2

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, waist circumference and diabetes status

Table B.23: Significant associations between racism/stress and a high level of homocysteine

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1c (sometimes)	1.95 (0.88-4.36)[a]	0.08	1.8
	3.19 (1.13-9.00)[b]	0.23	3.0
Q1c (often/very often)	1.98 (0.62-6.31)[a]	0.08	1.8
	1.40 (0.33-5.85)[b]	0.23	3.0
Q1d (sometimes)	**2.09 (1.01-4.34)**[a]	0.08	1.3
	2.31 (1.00-5.35)[b]	0.21	0.9
Q1d (often/very often)	3.71 (0.37-37.5)[a]	0.08	1.3
	5.53 (0.39-78.5)[b]	0.21	0.9
Inter-personal racism score	0.56 (0.30-1.04)[a]	0.08	1.4
(none)	**0.32 (0.14-0.73)**[b]	0.22	2.0
Inter-personal racism score	0.94 (0.50-1.78)[a]	0.08	1.4
(high)	1.17 (0.53-2.59)[b]	0.22	2.0
# of setting racism experienced in	**2.32 (1.14-4.72)**[a]	0.09	3.0
(2^{nd} quartile)	**3.60 (1.46-8.88)**[b]	0.24	3.9
# of setting racism experienced in	1.23 (0.58-2.59)[a]	0.09	3.0
(3^{rd} quartile)	2.76 (1.00-7.62)[b]	0.24	3.9
# of setting racism experienced in	1.61 (0.73-3.53)[a]	0.09	3.0
(4^{th} quartile)	**3.59 (1.21-10.6)**[b]	0.24	3.9
Q2d (never/hardly ever)	1.12 (0.56-2.24)[a]	0.10	3.4
	1.22 (0.50-2.94)[b]	0.26	6.3
Q2d (often/very often)	1.48 (0.72-3.04)[a]	0.10	3.4
	2.96 (1.16-7.53)[b]	0.26	6.3
Q2h (never/hardly ever)	**2.55 (1.05-6.18)**[a]	0.12	6.0
	1.00 (0.31-3.17)[b]	0.26	6.3
Q2h (often/very often)	0.70 (0.36-1.35)[a]	0.12	6.0
	0.49 (0.21-1.11)[b]	0.26	6.3
Q2k (sometimes)	**2.18 (1.08-4.42)**[a]	0.11	5.0
	2.14 (0.90-5.08)[b]	0.26	5.7
Q2k (often/very often)	1.26 (0.55-2.89)[a]	0.11	5.0
	1.10 (0.38-3.20)[b]	0.26	5.7
Q3a (neither agree nor disagree)	**0.39 (0.17-0.93)**[a]	0.08	1.6
	0.27 (0.09-0.83)[b]	0.21	1.5
Q3a (disagree/strongly disagree)	0.37 (0.09-1.61)[a]	0.08	1.6
	0.34 (0.05-2.30)[b]	0.21	1.5
Q3c (strongly agree/agree)	0.89 (0.45-1.76)[a]	0.07	0.5
	0.38 (0.15-0.96)[b]	0.22	1.9
Q3c (neither agree nor disagree)	0.68 (0.36-1.27)[a]	0.07	0.5
	0.44 (0.20-0.97)[b]	0.22	1.9
Q3e (neither agree nor disagree)	0.72 (0.34-1.55)[a]	0.07	0.9
	1.20 (0.44-3.28)[b]	0.21	1.0
Q3e (disagree/strongly disagree)	1.30 (0.76-2.23)[a]	0.07	0.9
	2.04 (1.06-3.95)[b]	0.21	1.0

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
No internalised racism	1.61 (0.64-4.05)[a]	0.07	1.0
	1.60 (0.51-5.01)[b]	0.22	1.9
High internalised racism	0.64 (0.36-1.14)[a]	0.07	1.0
	0.42 (0.20-0.89)[b]	0.22	1.9
Q4 (never/once a year)	1.60 (0.82-3.15)[a]	0.09	2.2
	1.51 (0.62-3.65)[b]	0.22	2.4
Q4 (once a month)	**0.23 (0.06-0.86)**[a]	0.09	2.2
	0.20 (0.04-0.93)[b]	0.22	2.4
Q4 (once a week)	1.57 (0.63-3.86)[a]	0.09	2.2
	1.35 (0.45-4.04)[b]	0.22	2.4
Q4 (once a day)	1.34 (0.57-3.16)[a]	0.09	2.2
	1.92 (0.69-5.33)[b]	0.22	2.4
# of acute stressors (2^{nd} quartile)	0.50 (0.23-1.06)[a]	0.11	4.7
	0.56 (0.22-1.44)[b]	0.26	6.4
# of acute stressors (3^{rd} quartile)	1.34 (0.60-2.97)[a]	0.11	4.7
	2.37 (0.84-6.70)[b]	0.26	6.4
# of acute stressors (4^{th} quartile)	1.82 (0.91-3.67)[a]	0.11	4.7
	2.93 (1.15-7.44)[b]	0.26	6.4
# of total stressors (1^{st} quartile)	1.22 (0.54-2.75)[a]	0.10	3.9
	0.71 (0.24-2.11)[b]	0.30	9.6
# of total stressors (3^{rd} quartile)	**2.97 (1.36-6.46)**[a]	0.10	3.9
	6.21 (2.24-17.2)[b]	0.30	9.6
# of total stressors (4^{th} quartile)	**2.35 (1.12-4.95)**[a]	0.10	3.9
	3.53 (1.32-9.41)[b]	0.30	9.6

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, household composition, highest qualification, optimism, lack of control, positive and negative social capital, positive instrumental social support, method of administration and SF-12 physical component score.

Table B.24: Significant associations between racism/stress and HbA1c for respondents with normal blood glucose

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1i (sometimes)	**-0.17 (-0.30, -0.04)[a]**	0.17	2.9
	-0.16 (-0.29, -0.03)[b]	0.29	0.9
Q1i (often/very often)	-0.02 (-0.31, 0.27)[a]	0.17	2.9
	-0.13 (-0.43, 0.18)[b]	0.29	0.9
Inter-personal racism score	**0.22 (0.08, 0.36)[a]**	0.20	6.5
(none)	**0.23 (0.09, 0.37)[b]**	0.37	9.6
Inter-personal racism score	0.08 (-0.07, 0.23)[a]	0.20	6.5
(high)	0.09 (-0.50, 0.23)[b]	0.37	9.6
# of setting racism	**-0.19 (-0.36, -0.03)[a]**	0.19	4.8
experienced in (2nd quartile)	**-0.20 (-0.36, -0.03)[b]**	0.35	7.3
# of setting racism	**-0.19 (-0.35, -0.02)[a]**	0.19	4.8
experienced in (3rd quartile)	**-0.24 (-0.41, -0.07)[b]**	0.35	7.3
# of setting racism	-0.17 (-0.36, 0.02)[a]	0.19	4.8
experienced in (4th quartile)	-0.16 (-0.36, 0.03)[b]	0.35	7.3
Q2b (sometimes)	-0.05 (-0.21, 0.11)[a]	0.14	0.2
	0.04 (-0.12, 0.21)[b]	0.32	3.7
Q2b (often/very often)	0.03 (-0.14, 0.20)[a]	0.14	0.2
	0.18 (0.01, 0.35)[b]	0.32	3.7
Q2c (sometimes)	**0.18 (0.01, 0.36)[a]**	0.17	2.6
	0.20 (0.01, 0.39)[b]	0.33	5.0
Q2c (often/very often)	0.09 (-0.08, 0.26)[a]	0.17	2.6
	0.15 (-0.01, 0.31)[b]	0.33	5.0
Q2g (sometimes)	-0.01 (-0.16, 0.14)[a]	0.22	8.2
	0.01 (-0.14, 0.15)[b]	0.34	5.8
Q2g (often/very often)	**-0.50 (-0.77, -0.22)[a]**	0.22	8.2
	-0.36 (-0.62, -0.10)[b]	0.34	5.8
Q5 (hardly any/not many	-0.22 (-0.56, 0.12)[a]	0.16	2.4
people)	-0.31 (-0.64, 0.01)[b]	0.31	2.7
Q5 (some people)	0.19 (-0.11, 0.49)[a]	0.16	2.4
	0.03 (-0.28, 0.34)[b]	0.31	2.7
Q5 (most people)	**0.15 (0.02, 0.27)[a]**	0.16	2.4
	0.12 (-0.01, 0.25)[b]	0.31	2.7
Q6 (hardly any/not many	**-0.22 (-0.43, -0.01)[a]**	0.15	0.7
people)	**-0.24 (-0.45, -0.02)[b]**	0.29	0.7
Q6 (some people)	-0.05 (-0.23, 0.12)[a]	0.15	0.7
	-0.03 (-0.20, 0.15)[b]	0.29	0.7
Q6 (most people)	-0.08 (-0.24, 0.08)[a]	0.15	0.7
	-0.13 (-0.30, 0.04)[b]	0.29	0.7
Acute stress score	-0.15 (-0.29, 0.00)[a]	0.16	2.2
(Yes, minimal/no effect)	**-0.15 (-0.30, -0.01)[b]**	0.29	1.2
Acute stress score	-0.07 (-0.28, 0.15)[a]	0.16	2.2
(Yes, affected me somewhat)	-0.13 (-0.36, 0.10)[b]	0.29	1.2

Items	Coefficient (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Chronic stress score	**0.31 (0.08, 0.55)[a]**	0.19	5.0
(None)	**0.39 (0.09, 0.70)[b]**	0.32	3.7
Chronic stress score	-0.06 (-0.23, 0.12)[a]	0.19	5.0
(Yes, affected me somewhat)	-0.02 (-0.20, 0.16)[b]	0.32	3.7
Chronic stress score	-0.52 (-1.27, 0.24)[a]	0.19	5.0
(Yes, affected me a great deal)	-0.48 (-1.19, 0.23)[b]	0.32	3.7
Total stress score	**0.38 (0.12, 0.63)[a]**	0.21	6.6
(None)	**0.55 (0.23, 0.87)[b]**	0.34	6.1
Total stress score	-0.04 (-0.24, 0.17)[a]	0.21	6.6
(Yes, affected me somewhat)	-0.04 (-0.25, 0.17)[b]	0.34	6.1
Total stress score	-0.51 (-1.25, 0.24)[a]	0.21	6.6
(Yes, affected me a great deal)	-0.48 (-1.18, 0.22)[b]	0.34	6.1
# of acute stressors (2^{nd} quartile)	**-0.21 (-0.38, -0.03)[a]**	0.15	1.1
	-0.25 (-0.43, -0.08)[b]	0.29	1.5
# of acute stressors (3^{rd} quartile)	-0.14 (-0.33, 0.05)[a]	0.15	1.1
	-0.11 (-0.31, 0.08)[b]	0.29	1.5
# of acute stressors (4^{th} quartile)	-0.10 (-0.26, 0.06)[a]	0.15	1.1
	-0.13 (-0.29, 0.03)[b]	0.29	1.5
Any experienced stressor	**-0.37 (-0.59, -0.15)[a]**	0.20	5.7
	-0.44 (-0.70, -0.17)[b]	0.33	5.1

CI: Confidence interval.
a Adjusted for age and sex.
b Adjusted for age and sex, highest qualification, smoking status (smoker vs. non-smoker), SF-12 physical component score, waist circumference and triglycerides.

Table B.25: Significant associations between racism/stress and a high level of C-reactive protein

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q2k (sometimes)	1.90 (0.96-3.74)[a]	0.10	3.2
	1.50 (0.62-3.64)[b]	0.31	0.0
Q2k (often/very often)	**3.65 (1.53-8.72)**[a]	0.10	3.2
	2.98 (0.93-9.60)[b]	0.31	0.0
Q3c (strongly agree/agree)	0.69 (0.35-1.36)[a]	0.07	0.8
	0.31 (0.11-0.85)[b]	0.32	0.7
Q3c (neither agree nor disagree)	0.70 (0.38-1.31)[a]	0.07	0.8
	0.83 (0.36-1.92)[b]	0.32	0.7
Q3d (neither agree nor disagree)	2.17 (0.37-12.8)[a]	0.08	1.4
	8.45 (0.49-146.0)[b]	0.31	0.0
Q3d (disagree/strongly disagree)	**0.26 (0.07-0.99)**[a]	0.08	1.4
	0.54 (0.10-3.11)[b]	0.31	0.0
Q5 (hardly any/not many people)	0.25 (0.04-1.37)[a]	0.07	1.2
	0.23 (0.03-1.81)[b]	0.33	1.9
Q5 (some people)	0.43 (0.12-1.58)[a]	0.07	1.2
	0.10 (0.01-0.93)[b]	0.33	1.9
Q5 (most people)	0.90 (0.53-1.54)[a]	0.07	1.2
	0.78 (0.37-1.65)[b]	0.33	1.9
Chronic stress score (None)[c]	1.97 (0.67-5.81)[a]	0.07	0.9
	11.0 (1.52-79.3)[b]	0.35	4.0
Chronic stress score (Yes, affected me somewhat)[c]	0.84 (0.39-1.81)[a]	0.07	0.9
	0.44 (0.14-1.34)[b]	0.35	4.0
# of acute stressors (2nd quartile)	**2.11 (1.01-4.39)**[a]	0.08	1.2
	1.55 (0.58-4.13)[b]	0.32	1.3
# of acute stressors (3rd quartile)	1.31 (0.59-2.92)[a]	0.08	1.2
	1.94 (0.64-5.91)[b]	0.32	1.3
# of acute stressors (4th quartile)	1.48 (0.75-2.93)[a]	0.08	1.2
	1.21 (0.46-3.16)[b]	0.32	1.3

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, highest qualification, level of physical activity, self-assessed health status, SF-12 physical component score, waist circumference, diabetes status and triglycerides.

c The base category for this variable is 'Yes, minimal/no effect' with the category 'Yes, affected me a great deal' omitted due to a lack of variation in outcome within this category.

Table B.26: Significant associations between racism/stress and a high level of fibrinogen

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1b (sometimes)	**0.35 (0.14-0.88)**[a]	0.06	1.7
	0.06 (0.01-0.27)[b]	0.24	5.3
Q1b (often/very often)	1.77 (0.44-7.07)[a]	0.06	1.7
	0.48 (0.07-3.15)[b]	0.24	5.3
Q1i (sometimes)	**0.48 (0.26-0.88)**[a]	0.06	2.4
	0.36 (0.16-0.83)[b]	0.20	1.6
Q1i (often/very often)	2.39 (0.70-8.23)[a]	0.06	2.4
	1.29 (0.28-6.06)[b]	0.20	1.6
Q3b (neither agree nor disagree)	**0.40 (0.18-0.89)**[a]	0.05	1.2
	0.43 (0.14-1.28)[b]	0.19	0.5
Q3b (disagree/strongly disagree)	0.74 (0.37-1.47)[a]	0.05	1.2
	0.63 (0.25-1.62)[b]	0.19	0.5
Q3c (strongly agree/agree)	1.21 (0.62-2.37)[a]	0.06	1.9
	0.83 (0.30-2.28)[b]	0.20	1.6
Q3c (neither agree nor disagree)	**0.43 (0.23-0.83)**[a]	0.06	1.9
	0.38 (0.16-0.91)[b]	0.20	1.6

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, highest qualification, optimism, lack of control (work settings excluded), positive instrumental social support, level of social isolation, smoking status (never vs. ever), self-assessed health status, SF-12 physical component score, waist circumference and diabetes status.

Table B.27: Significant associations between racism/stress and a high level of allostatic load

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1b (sometimes)	**3.44 (1.34-8.83)[a]**	0.14	2.0
	3.54 (1.28-9.78)[b]	0.17	0.4
Q1b (often/very often)	1.09 (0.17-7.08)[a]	0.14	2.0
	1.22 (0.19-8.02)[b]	0.17	0.4
Q2e (never/hardly ever)	2.00 (0.85-4.68)[a]	0.16	4.7
	1.71 (0.66-4.39)[b]	0.21	4.2
Q2e (sometimes)	**2.93 (1.34-6.45)[a]**	0.16	4.7
	3.12 (1.33-7.30)[b]	0.21	4.2
Q2h (never/hardly ever)	0.98 (0.39-2.48)[a]	0.15	3.4
	1.34 (0.48-3.86)[b]	0.19	2.5
Q2h (often/very often)	**0.46 (0.22-0.96)[a]**	0.15	3.4
	0.55 (0.24-1.24)[b]	0.19	2.5
Q6 (hardly any/not many people)	0.45 (0.18-1.16)[a]	0.13	1.0
	0.51 (0.18-1.43)[b]	0.17	0.7
Q6 (some people)	**0.45 (0.21-0.99)[a]**	0.13	1.0
	0.59 (0.26-1.37)[b]	0.17	0.7
Q6 (most people)	0.86 (0.43-1.74)[a]	0.13	1.0
	1.17 (0.55-2.51)[b]	0.17	0.7

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, marital status, income as well as positive and negative social capital.

Table B.28: Significant associations between racism/stress and a high # of chronic conditions

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1c (sometimes)	2.06 (0.91-4.69)[a]	0.13	2.4
	2.35 (0.96-5.75)[b]	0.21	3.3
Q1c (often/very	**3.72 (1.03-13.4)**[a]	0.13	2.4
often)	2.46 (0.58-10.5)[b]	0.21	3.3
Q2f (sometimes)	**2.07 (1.02-4.22)**[a]	0.11	0.0
	1.58 (0.71-3.54)[b]	0.20	2.2
Q2f (often/very	1.15 (0.54-2.46)[a]	0.11	0.0
often)	1.02 (0.44-2.38)[b]	0.20	2.2

CI: Confidence interval; **OR**: odds ratio.
a Adjusted for age and sex.
b Adjusted for age and sex, lack of control (work settings excluded), self-assessed health status and SF-12 physical component score.

Table B.29: Significant associations between racism/stress and dental health

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
Q1i (sometimes)	**0.50 (0.28-0.90)[a]**	0.02	1.3
	0.40 (0.22-0.76)[b]	0.07	0.6
Q1i (often/very often)	0.92 (0.30-2.84)[a]	0.02	1.3
	0.91 (0.28-2.94)[b]	0.07	0.6
Q2e (never/hardly ever)	**0.48 (0.24-0.96)[a]**	0.03	2.3
	0.51 (0.24-1.09)[b]	0.07	0.4
Q2e (sometimes)	0.75 (0.39-1.44)[a]	0.03	2.3
	0.81 (0.41-1.61)[b]	0.07	0.4
Q2h (never/hardly ever)	**2.59 (1.18-5.71)[a]**	0.03	2.9
	4.10 (1.65-10.2)[b]	0.10	3.5
Q2h (often/very often)	0.99 (0.53-1.83)[a]	0.03	2.9
	1.09 (0.55-2.13)[b]	0.10	3.5
Q2i (never/hardly ever)	1.30 (0.66-2.57)[a]	0.03	2.5
	1.67 (0.80-3.48)[b]	0.08	1.6
Q2i (often/very often)	**2.17 (1.12-4.22)[a]**	0.03	2.5
	2.61 (1.26-5.40)[b]	0.08	1.6
Q2j (sometimes)	0.55 (0.29-1.05)[a]	0.02	1.9
	0.47 (0.23-0.94)[b]	0.07	0.8
Q2j (often/very often)	1.02 (0.42-2.44)[a]	0.02	1.9
	0.82 (0.31-2.15)[b]	0.07	0.8
Q4 (never/once a year)	0.94 (0.50-1.77)[a]	0.01	1.1
	1.03 (0.51-2.07)[b]	0.07	0.9
Q4 (once a month)	2.04 (0.78-5.36)[a]	0.01	1.1
	2.92 (1.02-8.34)[b]	0.07	0.9
Q4 (once a week)	2.18 (0.91-5.20)[a]	0.01	1.1
	2.61 (1.02-6.67)[b]	0.07	0.9
Q4 (once a day)	1.20 (0.52-2.74)[a]	0.01	1.1
	1.14 (0.46-2.80)[b]	0.07	0.9
Acute stress score	1.50 (0.83-2.71)[a]	0.02	2.0
(Yes, minimal/no effect)	1.09 (0.56-2.12)[b]	0.07	1.0
Acute stress score	**3.44 (1.43-8.29)[a]**	0.02	2.0
(Yes, affected me somewhat)	2.13 (0.81-5.62)[b]	0.07	1.0
Total stress score	0.52 (0.16-1.70)[a]	0.03	2.7
(None)[c]	0.77 (0.21-2.80)[b]	0.09	2.2
Total stress score	**3.48 (1.47-8.21)[a]**	0.03	2.7
(Yes, affected me somewhat)[c]	**3.07 (1.23-7.64)[b]**	0.09	2.2
# of acute stressors (2^{nd}	1.70 (0.85-3.41)[a]	0.01	1.1
quartile)	1.32 (0.62-2.82)[b]	0.07	0.5
# of acute stressors (3^{rd}	1.56 (0.73-3.36)[a]	0.01	1.1
quartile)	1.00 (0.44-2.31)[b]	0.07	0.5
# of acute stressors (4^{th}	**1.99 (1.02-3.89)[a]**	0.01	1.1
quartile)	1.40 (0.66-2.98)[b]	0.07	0.5
# of chronic stressors (1^{st}	**0.44 (0.20-0.95)[a]**	0.03	2.8
quartile)	0.69 (0.27-1.73)[b]	0.09	2.6

Items	OR (95% CI)	Model Adjusted R^2	Variance explained by predictor variable (%)
# of chronic stressors (2nd quartile)	**0.42 (0.21-0.84)[a]**	0.03	2.8
	0.48 (0.22-1.02)[b]	0.09	2.6
# of chronic stressors (3rd quartile)	**0.45 (0.23-0.90)[a]**	0.03	2.8
	0.48 (0.23-0.99)[b]	0.09	2.6
# of total stressors (1st quartile)	1.06 (0.49-2.27)[a]	0.03	2.6
	1.71 (0.71-4.08)[b]	0.09	2.4
# of total stressors (3rd quartile)	1.18 (0.58-2.39)[a]	0.03	2.6
	1.23 (0.58-2.62)[b]	0.09	2.4
# of total stressors (4th quartile)	**2.26 (1.14-4.49)[a]**	0.03	2.6
	2.05 (0.97-4.34)[b]	0.09	2.4

CI: Confidence interval; **OR**: odds ratio.

a Adjusted for age and sex.

b Adjusted for age and sex, optimism, lack of control, negative and positive social capital, positive instrumental social support and method of administration**.**

c The base category for this variable is 'Yes, minimal/no effect' with the category 'Yes, affected me a great deal' omitted due to a lack of variation in outcome within this category.

Table B.30: Significant associations between MIRE Q1-2 predictor variables and stress outcomes (adjusted for age and sex only)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q1a1			+	+		+
Q1a2		+	+	+		+
Q1b1			+	+		
Q1b2						
Q1c1			+	+		
Q1c2	+		+			
Q1d1	+	+	+	+		
Q1d2						
Q1e1	+	+	+	+		+
Q1e2				+		
Q1f1	+	+	+	+		+
Q1f2						
Q1g1	+	+	+	+		
Q1g2			+	+		
Q1h1	+	+	+	+	+	+
Q1h2	+		+	+		+
Q1i1			+	+		
Q1i2	+		+			
Q1j1	+	+	+	+		
Q1j2	+	+	+	+		
IPR0			-	-	-	
IPR2	+	+	+	+		+
RS2			+	+	+	+
RS3	+		+	+	+	+
RS4	+	+	+	+	+	+
Q2a0				-		
Q2a1				-		
Q2b1						
Q2b2						+
Q2c1						
Q2c2						
Q2d0						
Q2d2						
Q2e0		-	-			-
Q2e1	+					
Q2f1						
Q2f2						
Q2g1			+	+		+
Q2g2						
Q2h0	-	-	-	-		-
Q2h2					+	
Q2i0						
Q2i2			-	-		-
Q2j1	+	+	+	+		+
Q2j2			+			
Q2k1	+	+	+			
Q2k2						

Table B.31: Significant associations between MIRE Q1-2 predictor variables and stress outcomes (adjusted for potential confounders)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q1a1						
Q1a2			+	+		
Q1b1				+		
Q1b2			+	+		
Q1c1				+		
Q1c2			+			
Q1d1		+				
Q1d2						
Q1e1			+	+		
Q1e2				+		
Q1f1	+	+	+	+		+
Q1f2						
Q1g1		+		+		
Q1g2			+			
Q1h1	+	+	+	+		+
Q1h2				+		
Q1i1						
Q1i2			+			
Q1j1			+	+		
Q1j2	+			+		
IPR0			-		-	
IPR2		+	+	+		
RS2				+		
RS3			+	+	+	
RS4			+	+		
Q2a0						
Q2a1			-	-		
Q2b1						
Q2b2						+
Q2c1						
Q2c2						
Q2d0						
Q2d2						
Q2e0						
Q2e1						
Q2f1						
Q2f2	-	-				-
Q2g1			+	+		
Q2g2						
Q2h0	-	-		-		
Q2h2					+	
Q2i0						
Q2i2			-	-	-	-
Q2j1	+		+	+		+
Q2j2						
Q2k1	+	+	+			
Q2k2						

Table B.32: Significant associations between MIRE Q3-6 predictor variables and stress outcomes (adjusted for age and sex only)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q3a2						
Q3a3						
Q3b2						
Q3b3						
Q3c1						
Q3c2						
Q3d2						
Q3d3						
Q3e2						
Q3e3						
Q3f2			+	+		
Q3f3						
Q3g1						
Q3g2						
IR0						
IR2		-				
SR2						
SR3						
SR4						
Q4_0	-	-	-	-		-
Q4_1		-			-	
Q4_2	-	-				
Q4_3	-	-				
Q5_0						
Q5_1						
Q5_2						
Q6_0						
Q6_1			+	+		
Q6_2						

Table B.33: Significant associations between MIRE Q3-6 predictor variables and stress outcomes (adjusted for potential confounders)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q3a2						
Q3a3	-	-				
Q3b2						
Q3b3						
Q3c1						
Q3c2						
Q3d2						
Q3d3						
Q3e2						
Q3e3				-		
Q3f2				+		
Q3f3						
Q3g1						
Q3g2						
IR0						
IR2		-				
SR2						
SR3					+	
SR4					+	
Q4_0		-				
Q4_1		-				
Q4_2		-				
Q4_3	-	-				
Q5_0						
Q5_1						
Q5_2		-				
Q6_0						
Q6_1						
Q6_2						

Table B.34 Significant associations between MIRE Q1-2 predictor variables and health-related outcomes (adjusted for age and sex)

	Sm	Alco	PI	JF	FV	CES-D	Poor health	SF-12 MCS	SF-12 PCS
Q1a1		+					+	-	
Q1a2						+	+	-	
Q1b1		+							
Q1b2								-	
Q1c1									
Q1c2							+	-	
Q1d1									
Q1d2									-
Q1e1						+			
Q1e2									
Q1f1									
Q1f2									
Q1g1									
Q1g2							+	-	
Q1h1						+		-	
Q1h2							+	-	
Q1i1								-	
Q1i2									
Q1j1									
Q1j2				+		+		-	
IPR0									
IPR2						+	+	-	
RS2									
RS3									
RS4		+							
Q2a0									
Q2a1									
Q2b1						+			
Q2b2									
Q2c1						+			
Q2c2									
Q2d0	+								
Q2d2									
Q2e0									
Q2e1									
Q2f1						+		-	
Q2f2						+			
Q2g1						+		-	
Q2g2						+	+	-	
Q2h0									
Q2h2		+					+		
Q2i0				-					
Q2i2									
Q2j1	-					+		-	
Q2j2				+		+	+	-	-
Q2k1									
Q2k2								-	

Table B.35 Significant associations between MIRE Q1-2 predictor variables and health-related outcomes (adjusted for all potential confounders)

	Sm	Alco	PI	JF	FV	CES-D	Poor health	SF-12 MCS	SF-12 PCS
Q1a1								-	
Q1a2								-	
Q1b1		+							
Q1b2									
Q1c1				+					
Q1c2							+	-	
Q1d1									
Q1d2									
Q1e1						+			
Q1e2								-	
Q1f1									
Q1f2									
Q1g1									
Q1g2									
Q1h1							+	-	
Q1h2							+		
Q1i1									
Q1i2									
Q1j1									
Q1j2				+				-	
IPR0									
IPR2								-	
RS2									
RS3									
RS4									
Q2a0									
Q2a1									
Q2b1									
Q2b2									
Q2c1									
Q2c2									
Q2d0	+								
Q2d2									
Q2e0									
Q2e1									
Q2f1									
Q2f2						+			
Q2g1						+			
Q2g2									
Q2h0								+	
Q2h2		+					+		
Q2i0				-					
Q2i2									
Q2j1	-					+		-	
Q2j2							+		
Q2k1									
Q2k2									

Table B.36 Significant associations between MIRE Q3-6 predictor variables and health-related outcomes (adjusted for age and sex only)

	Sm	Alco	PI	JF	FV	CES-D	Poor health	SF-12 MCS	SF-12 PCS
Q3a2									
Q3a3									
Q3b2								+	
Q3b3	+								-
Q3c1									
Q3c2									
Q3d2									
Q3d3									
Q3e2									
Q3e3									
Q3f2								-	+
Q3f3						+			
Q3g1									
Q3g2		-							
IR0		+							
IR2									
SR2					-				
SR3					-				
SR4					-				
Q4_0									
Q4_1		-	-						
Q4_2									
Q4_3									
Q5_0									
Q5_1									-
Q5_2			-		+				
Q6_0									
Q6_1									
Q6_2									

Table B.37 Significant associations between MIRE Q3-6 predictor variables and health-related outcomes (adjusted for all potential confounders)

	Sm	Alco	PI	JF	FV	CES-D	Poor health	SF-12 MCS	SF-12 PCS
Q3a2									
Q3a3									
Q3b2				+					
Q3b3	+								
Q3c1									
Q3c2									
Q3d2				+					
Q3d3									
Q3e2									
Q3e3									
Q3f2									+
Q3f3									
Q3g1		-							
Q3g2									
IR0		+							
IR2									
SR2									
SR3									
SR4									
Q4_0									
Q4_1		-							-
Q4_2									
Q4_3									
Q5_0									
Q5_1									-
Q5_2			-						
Q6_0									
Q6_1									
Q6_2									-

Table B.38 Significant associations between MIRE Q1-2 predictor variables and health-related outcomes (adjusted for age and sex only)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allos	#CC
Q1a1			-										
Q1a2													
Q1b1										-		+	
Q1b2													
Q1c1													
Q1c2	+				+								+
Q1d1							+						
Q1d2													
Q1e1													
Q1e2													
Q1f1													
Q1f2													
Q1g1													
Q1g2													
Q1h1													
Q1h2													
Q1i1								-		-	-		
Q1i2													
Q1j1													
Q1j2													
IPR0		+						+					
IPR2													
RS2							+	-					
RS3								-					
RS4													
Q2a0													
Q2a1				-									
Q2b1													
Q2b2													
Q2c1				+				+					
Q2c2													
Q2d0													
Q2d2													
Q2e0											-		
Q2e1						+						+	
Q2f1						+							+
Q2f2													
Q2g1													
Q2g2								-					
Q2h0							+				+		
Q2h2												-	
Q2i0						-							
Q2i2													
Q2j1													
Q2j2													
Q2k1							+						
Q2k2									+				

Table B.39 Significant associations between MIRE Q1-2 predictor variables and health-related outcomes (adjusted for potential confounders)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allos	#CC
Q1a1													
Q1a2													
Q1b1										-			
Q1b2													
Q1c1							+						
Q1c2													
Q1d1													
Q1d2													
Q1e1													
Q1e2													
Q1f1													
Q1f2													
Q1g1													
Q1g2													
Q1h1													
Q1h2													
Q1i1								-		-	-		
Q1i2													
Q1j1													
Q1j2													
IPR0							-	+					
IPR2													
RS2			-				+	-					
RS3								-					
RS4							+						
Q2a0													
Q2a1													
Q2b1													
Q2b2								+					
Q2c1			+	+				+					
Q2c2													
Q2d0													
Q2d2							+						
Q2e0													
Q2e1						+						+	
Q2f1						+							
Q2f2													
Q2g1													
Q2g2								-					
Q2h0													
Q2h2													
Q2i0						-							
Q2i2											+		
Q2j1													
Q2j2													
Q2k1													
Q2k2													

Table B.40 Significant associations between MIRE Q3-6 predictor variables and health-related outcomes (adjusted for age and sex only)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allos	#CC
Q3a2							-						
Q3a3													
Q3b2										-			
Q3b3		+											
Q3c1				-									
Q3c2										-			
Q3d2			-	-		-							
Q3d3									-				
Q3e2	-												
Q3e3													
Q3f2													
Q3f3	-												
Q3g1						-							
Q3g2													
IR0			-										
IR2	-												
SR2													
SR3													
SR4													
Q4_0		+											
Q4_1							-						
Q4_2													
Q4_3													
Q5_0													
Q5_1													
Q5_2								+					
Q6_0				-				-					
Q6_1			-	-	-							-	
Q6_2			-	-	-								

Table B.41: Significant associations between MIRE Q3-6 predictor variables and health-related outcomes (adjusted for potential confounders)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allos	#CC
Q3a2							-						
Q3a3													
Q3b2													
Q3b3		+											
Q3c1				-			-		-				
Q3c2	-						-			-			
Q3d2			-	-		-							
Q3d3													
Q3e2													
Q3e3							+						
Q3f2													
Q3f3	-												
Q3g1						-							
Q3g2													
IR0			-										
IR2							-						
SR2													
SR3													
SR4													
Q4_0													
Q4_1			+				-				+		
Q4_2											+		
Q4_3			+										
Q5_0													
Q5_1									-				
Q5_2													
Q6_0								-					
Q6_1				-									
Q6_2					-								

Table B.42: Significant associations between stress predictor variables and health-related outcomes (adjusted for age and sex only)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DP	All	#CC
ASS1						+		-														
ASS2					+			-												+		
CSS0																	-					
CSS2						+		-														
CSS3																						
TSS0																	-					
TSS2				+	+		+	-	-											+		
TSS3																						
#AS2										+							-	+				
#AS3			+					-														
#AS4					+	+		-												+		
#CS1						-		+				+								-		
#CS2				-		-		+				+								-		
#CS3						-		+												-		
#TS1						-								-								
#TS3						+						-	-			+						
#TS4						+		-				-				+				+		
AS								-			-						-					
AGD						+					-	-										
GD						+		-				-										

Table B.43: Significant associations between stress predictor variables and health-related outcomes (adjusted for potential confounders)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DP	All	#CC
ASS1						+		-									-					
ASS2								-														
CSS0																	-	+				
CSS2						+	+	-														
CSS3																						
TSS0																	-					
TSS2								-				-								+		
TSS3																						
#AS2										+							-					
#AS3			+					-														
#AS4						+		-								+						
#CS1						-		+														
#CS2						-		+														
#CS3						-														-		
#TS1									+													
#TS3	-											-				+						
#TS4						+										+						
AS																	-					
SGD																						
GD						+		-				-										

Table B.44: Variance explained by MIRE Q1-2 predictor variables by stress outcomes (adjusted for age and sex only)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q1a1			9.0	5.5		6.5
Q1a2		2.6	9.0	5.5		6.5
Q1b1			5.0	3.9		
Q1b2						
Q1c1			4.5	3.7		
Q1c2	1.7		4.5			
Q1d1	2.8	3.0	2.5	2.0		
Q1d2						
Q1e1	3.6	3.5	6.4	8.0		2.1
Q1e2				8.0		
Q1f1	1.9	2.1	4.1	4.1		2.8
Q1f2						
Q1g1	2.3	3.9	4.7	4.9		
Q1g2			4.7	4.9		
Q1h1	4.5	4.2	6.9	6.5	2.5	4.9
Q1h2	4.5		6.9	6.5		4.9
Q1i1			4.9	3.5		
Q1i2	2.2		4.9			
Q1j1	4.3	2.7	7.5	6.8		
Q1j2	4.3	2.7	7.5	6.8		
IPR0			14.9	12.0	10.2	
IPR2	5.6	6.9	14.9	12.0		7.6
RS2			14.0	9.2	10.9	5.9
RS3	3.4		14.0	9.2	10.9	5.9
RS4	3.4	4.7	14.0	9.2	10.9	5.9
Q2a0				2.4		
Q2a1				2.4		
Q2b1						
Q2b2						3.9
Q2c1						
Q2c2						
Q2d0						
Q2d2						
Q2e0		4.0	3.6			3.4
Q2e1	5.0					
Q2f1						
Q2f2						
Q2g1			4.3	3.0		3.1
Q2g2						
Q2h0	3.5	2.6	4.1	1.0		5.6
Q2h2					8.4	
Q2i0						
Q2i2			3.2	2.3		3.2
Q2j1	0.0	1.3	8.0	4.4		5.9
Q2j2			8.0			
Q2k1	3.6	1.9	2.7			
Q2k2						

Table B.45: Variance explained by MIRE Q1-2 predictor variables by stress outcomes (adjusted for potential confounders)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q1a1						
Q1a2			4.6	2.2		
Q1b1				2.4		
Q1b2			3.8	2.4		
Q1c1				1.1		
Q1c2			3.0			
Q1d1		2.6				
Q1d2						
Q1e1			3.1	8.2		
Q1e2				8.2		
Q1f1	1.8	2.0	3.5	2.9		4.2
Q1f2						
Q1g1		2.9		3.5		
Q1g2			2.0			
Q1h1	2.6	2.1	2.6	3.3		2.3
Q1h2				3.3		
Q1i1						
Q1i2			3.6			
Q1j1			1.9	2.7		
Q1j2	3.2			2.7		
IPR0			7.0		8.6	
IPR2		5.9	7.0	6.8		
RS2				4.4		
RS3			10.9	4.4	11.8	
RS4			10.9	4.4		
Q2a0						
Q2a1			3.4	2.3		
Q2b1						
Q2b2						6.2
Q2c1						
Q2c2						
Q2d0						
Q2d2						
Q2e0						
Q2e1						
Q2f1						
Q2f2	4.8	5.1				3.2
Q2g1			4.0	0.0		
Q2g2						
Q2h0	4.9	4.7		0.0		
Q2h2					13.9	
Q2i0						
Q2i2			4.0	2.1	9.4	5.1
Q2j1	4.6		8.8	2.0		3.6
Q2j2						
Q2k1	3.6	5.4	2.7			
Q2k2						

Table B.46: Variance explained by MIRE Q3-6 predictor variables by stress outcomes (adjusted for age and sex only)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q3a2						
Q3a3						
Q3b2						
Q3b3						
Q3c1						
Q3c2						
Q3d2						
Q3d3						
Q3e2						
Q3e3						
Q3f2			1.7	1.7		
Q3f3						
Q3g1						
Q3g2						
IR0						
IR2		1.4				
SR2						
SR3						
SR4						
Q40	4.2	5.6	2.2	1.0		1.8
Q41		5.6			2.8	
Q42	4.2	5.6				
Q43	4.2	5.6				
Q50						
Q51						
Q52						
Q60						
Q61			2.5	2.6		
Q62						

Table B.47: Variance explained by MIRE Q3-6 predictor variables by stress outcomes (adjusted for potential confounders)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q3a2						
Q3a3	2.9	3.5				
Q3b2						
Q3b3						
Q3c1						
Q3c2						
Q3d2						
Q3d3						
Q3e2						
Q3e3				2.5		
Q3f2				0.7		
Q3f3						
Q3g1						
Q3g2						
IR0						
IR2		3.6				
SR2						
SR3					4.9	
SR4					4.9	
Q40		5.4				
Q41		5.4				
Q42		5.4				
Q43	4.4	5.4				
Q50						
Q51						
Q52		2.5				
Q60						
Q61						
Q62						

Table B.48 Variance explained by MIRE Q1-2 predictor variables by health-related outcomes (adjusted for age and sex only)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS
Q1a1		1.3					2.7	8.2	
Q1a2						1.4	2.7	8.2	
Q1b1		1.2							
Q1b2								1.6	
Q1c1									
Q1c2							2.5	2.9	
Q1d1									
Q1d2									0.0
Q1e1						4.9			
Q1e2									
Q1f1									
Q1f2									
Q1g1									
Q1g2							1.4	1.2	
Q1h1						2.3		5.0	
Q1h2							1.5	5.0	
Q1i1								2.2	
Q1i2									
Q1j1									
Q1j2				1.2		2.8		4.9	
IPR0									
IPR2						1.6	1.2	2.5	
RS2									
RS3									
RS4		0.2							
Q2a0									
Q2a1									
Q2b1						1.8			
Q2b2									
Q2c1						2.1			
Q2c2									
Q2d0	3.5								
Q2d2									
Q2e0									
Q2e1									
Q2f1						4.3		1.9	
Q2f2						4.3			
Q2g1						4.5		3.3	
Q2g2						4.5	2.1	3.3	
Q2h0									
Q2h2		0.4					5.6		
Q2i0				4.0					
Q2i2									
Q2j1	2.9					3.9		6.1	
Q2j2				2.5		3.9	4.7	6.1	2.1
Q2k1									
Q2k2								1.0	

Table B.49 Variance explained by MIRE Q1-2 predictor variables by health-related outcomes (adjusted for all potential confounders)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS
Q1a1								3.3	
Q1a2								3.3	
Q1b1		2.0							
Q1b2									
Q1c1				5.3					
Q1c2							0.7	0.7	
Q1d1									
Q1d2									
Q1e1						3.9			
Q1e2								0.0	
Q1f1									
Q1f2									
Q1g1									
Q1g2									
Q1h1							1.5	2.7	
Q1h2							1.5		
Q1i1									
Q1i2									
Q1j1									
Q1j2				0.8				1.9	
IPR0									
IPR2								0.7	
RS2									
RS3									
RS4									
Q2a0									
Q2a1									
Q2b1									
Q2b2									
Q2c1									
Q2c2									
Q2d0	7.9								
Q2d2									
Q2e0									
Q2e1									
Q2f1									
Q2f2						5.1			
Q2g1						3.4			
Q2g2									
Q2h0								3.6	
Q2h2		0.6					9.5		
Q2i0				2.9					
Q2i2									
Q2j1	7.6					3.4		3.7	
Q2j2							6.7		
Q2k1									
Q2k2									

Table B.50 Variance explained by MIRE Q3-6 predictor variables by health-related outcomes (adjusted for age and sex only)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS
Q3a2									
Q3a3									
Q3b2								1.6	
Q3b3	1.9								2.1
Q3c1									
Q3c2									
Q3d2									
Q3d3									
Q3e2									
Q3e3									
Q3f2								1.1	2.6
Q3f3						2.8			
Q3g1									
Q3g2		4.2							
IR0		0.3							
IR2									
SR2					4.9				
SR3					4.9				
SR4					4.9				
Q40									
Q41		0.4	1.6						
Q42									
Q43									
Q50									
Q51									1.0
Q52			2.2		2.8				
Q60									
Q61									
Q62									

Table B.51: Variance explained by MIRE Q3-6 predictor variables by health-related outcomes (adjusted for all potential confounders)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS
Q3a2									
Q3a3									
Q3b2				1.6					
Q3b3	3.7								
Q3c1									
Q3c2									
Q3d2				1.8					
Q3d3									
Q3e2									
Q3e3									
Q3f2									3.3
Q3f3									
Q3g1		3.7							
Q3g2									
IR0		1.0							
IR2									
SR2									
SR3									
SR4									
Q40									
Q41		0.2							1.4
Q42									
Q43									
Q50									
Q51									0.9
Q52			1.6						
Q60									
Q61									
Q62									0.0

Table B.52: Variance explained by MIRE Q1-2 predictor variables by health-related outcomes (adjusted for age and sex only)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allo	#CC
Q1a1			2.5										
Q1a2													
Q1b1										1.7		2.0	
Q1b2													
Q1c1													
Q1c2	2.0				5.7								2.4
Q1d1							1.3						
Q1d2													
Q1e1													
Q1e2													
Q1f1													
Q1f2													
Q1g1													
Q1g2													
Q1h1													
Q1h2													
Q1i1								2.9		2.4	1.3		
Q1i2													
Q1j1													
Q1j2													
IPR0		2.3						6.5					
IPR2													
RS2							3.0	4.8					
RS3								4.8					
RS4													
Q2a0													
Q2a1				4.6									
Q2b1													
Q2b2													
Q2c1				4.5				2.6					
Q2c2													
Q2d0													
Q2d2													
Q2e0											2.3		
Q2e1						2.5						4.7	
Q2f1						3.0							0.0
Q2f2													
Q2g1													
Q2g2								8.2					
Q2h0							6.0				2.9		
Q2h2												3.4	
Q2i0						2.2							
Q2i2											2.5		
Q2j1													
Q2j2													
Q2k1							5.0						
Q2k2									3.2				

Table B.53: Variance explained by MIRE Q1-2 predictor variables by health-related outcomes (adjusted for potential confounders)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allo	#CC
Q1a1													
Q1a2													
Q1b1										5.3			
Q1b2													
Q1c1							3.0						
Q1c2													
Q1d1													
Q1d2													
Q1e1													
Q1e2													
Q1f1													
Q1f2													
Q1g1													
Q1g2													
Q1h1													
Q1h2													
Q1i1								0.9		1.6	0.6		
Q1i2													
Q1j1													
Q1j2													
IPR0							2.0	9.6					
IPR2													
RS2			6.9				3.9	7.3					
RS3								7.3					
RS4							3.9						
Q2a0													
Q2a1													
Q2b1													
Q2b2								3.7					
Q2c1			3.0	4.4				5.0					
Q2c2													
Q2d0													
Q2d2							6.3						
Q2e0													
Q2e1						5.4						4.2	
Q2f1						6.9							
Q2f2													
Q2g1													
Q2g2								5.8					
Q2h0													
Q2h2													
Q2i0						5.1							
Q2i2											1.6		
Q2j1													
Q2j2													
Q2k1													
Q2k2													

Table B.54: Variance explained by MIRE Q3-6 predictor variables by health-related outcomes (adjusted for age and sex only)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allo	#CC
Q3a2							1.5						
Q3a3													
Q3b2										1.2			
Q3b3		4.5											
Q3c1				1.9									
Q3c2										1.9			
Q3d2			0.6	2.1		2.9							
Q3d3									1.4				
Q3e2	1.5												
Q3e3													
Q3f2													
Q3f3	1.6												
Q3g1						1.5							
Q3g2													
IR0			0.9										
IR2	1.4												
SR2													
SR3													
SR4													
Q40		4.3											
Q41							2.2						
Q42													
Q43													
Q50													
Q51													
Q52								2.4					
Q60				5.3				0.7					
Q61			2.8	5.3	5.4							1.0	
Q62			2.8	5.3	5.4								

Table B.55: Variance explained by MIRE Q3-6 predictor variables by health-related outcomes (adjusted for potential confounders)

	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DH	Allo	#CC
Q3a2							1.5						
Q3a3													
Q3b2													
Q3b3		1.9											
Q3c1				1.6			1.9		0.7				
Q3c2	0.8						1.9			1.6			
Q3d2			1.9	0.8		4.3							
Q3d3													
Q3e2													
Q3e3							1.0						
Q3f2													
Q3f3	1.1												
Q3g1						3.2							
Q3g2													
IR0			1.6										
IR2							1.9						
SR2													
SR3													
SR4													
Q40													
Q41			2.6				2.4				0.9		
Q42											0.9		
Q43			2.6										
Q50													
Q51									1.9				
Q52													
Q60								0.7					
Q61				3.7									
Q62					2.8								

Table B.56: Variance explained by MIRE Q1-2 predictor variables by stress outcomes (adjusted for age and sex only)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q1a1			9.0	5.5		6.5
Q1a2		2.6	9.0	5.5		6.5
Q1b1			5.0	3.9		
Q1b2						
Q1c1			4.5	3.7		
Q1c2	1.7		4.5			
Q1d1	2.8	3.0	2.5	2.0		
Q1d2						
Q1e1	3.6	3.5	6.4	8.0		2.1
Q1e2				8.0		
Q1f1	1.9	2.1	4.1	4.1		2.8
Q1f2						
Q1g1	2.3	3.9	4.7	4.9		
Q1g2			4.7	4.9		
Q1h1	4.5	4.2	6.9	6.5	2.5	4.9
Q1h2	4.5		6.9	6.5		4.9
Q1i1			4.9	3.5		
Q1i2	2.2		4.9			
Q1j1	4.3	2.7	7.5	6.8		
Q1j2	4.3	2.7	7.5	6.8		
IPR0			14.9	12.0	10.2	
IPR2	5.6	6.9	14.9	12.0		7.6
RS2			14.0	9.2	10.9	5.9
RS3	3.4		14.0	9.2	10.9	5.9
RS4	3.4	4.7	14.0	9.2	10.9	5.9
Q2a0				2.4		
Q2a1				2.4		
Q2b1						
Q2b2						3.9
Q2c1						
Q2c2						
Q2d0						
Q2d2						
Q2e0		4.0	3.6			3.4
Q2e1	5.0					
Q2f1						
Q2f2						
Q2g1			4.3	3.0		3.1
Q2g2						
Q2h0	3.5	2.6	4.1	1.0		5.6
Q2h2					8.4	
Q2i0						
Q2i2			3.2	2.3		3.2
Q2j1	0.0	1.3	8.0	4.4		5.9
Q2j2			8.0			
Q2k1	3.6	1.9	2.7			
Q2k2						

Table B.57: Variance explained by MIRE Q1-2 predictor variables by stress outcomes (adjusted for potential confounders)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q1a1						
Q1a2			4.6	2.2		
Q1b1				2.4		
Q1b2			3.8	2.4		
Q1c1				1.1		
Q1c2			3.0			
Q1d1		2.6				
Q1d2						
Q1e1			3.1	8.2		
Q1e2				8.2		
Q1f1	1.8	2.0	3.5	2.9		4.2
Q1f2						
Q1g1		2.9		3.5		
Q1g2			2.0			
Q1h1	2.6	2.1	2.6	3.3		2.3
Q1h2				3.3		
Q1i1						
Q1i2			3.6			
Q1j1			1.9	2.7		
Q1j2	3.2			2.7		
IPR0			7.0		8.6	
IPR2		5.9	7.0	6.8		
RS2				4.4		
RS3			10.9	4.4	11.8	
RS4			10.9	4.4		
Q2a0						
Q2a1			3.4	2.3		
Q2b1						
Q2b2						6.2
Q2c1						
Q2c2						
Q2d0						
Q2d2						
Q2e0						
Q2e1						
Q2f1						
Q2f2	4.8	5.1				3.2
Q2g1			4.0	0.0		
Q2g2						
Q2h0	4.9	4.7		0.0		
Q2h2					13.9	
Q2i0						
Q2i2			4.0	2.1	9.4	5.1
Q2j1	4.6		8.8	2.0		3.6
Q2j2						
Q2k1	3.6	5.4	2.7			
Q2k2						

Table B.58: Variance explained by MIRE Q3-6 predictor variables by stress outcomes (adjusted for age and sex only)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q3a2						
Q3a3						
Q3b2						
Q3b3						
Q3c1						
Q3c2						
Q3d2						
Q3d3						
Q3e2						
Q3e3						
Q3f2			1.7	1.7		
Q3f3						
Q3g1						
Q3g2						
IR0						
IR2		1.4				
SR2						
SR3						
SR4						
Q40	4.2	5.6	2.2	1.0		1.8
Q41		5.6			2.8	
Q42	4.2	5.6				
Q43	4.2	5.6				
Q50						
Q51						
Q52						
Q60						
Q61			2.5	2.6		
Q62						

Table B.59: Variance explained by MIRE Q3-6 predictor variables by stress outcomes (adjusted for potential confounders)

	High ASS	High #AS	High CSS	High #CS	Affected S/GD	Affected GD
Q3a2						
Q3a3	2.9	3.5				
Q3b2						
Q3b3						
Q3c1						
Q3c2						
Q3d2						
Q3d3						
Q3e2						
Q3e3				2.5		
Q3f2				0.7		
Q3f3						
Q3g1						
Q3g2						
IR0						
IR2		3.6				
SR2						
SR3					4.9	
SR4					4.9	
Q40		5.4				
Q41		5.4				
Q42		5.4				
Q43	4.4	5.4				
Q50						
Q51						
Q52		2.5				
Q60						
Q61						
Q62						

Table B.60: Variance explained by stress predictor variables by health-related outcomes (adjusted for age and sex only)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DP	Allo	#CC
ASS1						2.0		4.3														
ASS2					4.5			4.3												2.0		
CSS0																	5.0					
CSS2						3.7		5.6														
CSS3																						
TSS0																	6.6					
TSS2				1.0	1.8		1.7	5.0	2.9											2.7		
TSS3																						
#AS2										2.9							1.1	1.2				
#AS3			3.0					4.4														
#AS4					5.8	4.7		4.4												1.1		
#CS1						10.2		8.6				3.1								2.8		
#CS2				1.7		10.2		8.6				3.1								2.8		
#CS3						10.2		8.6												2.8		
#TS1						10.5								3.5								
#TS3						10.5						2.5	1.8			3.9						
#TS4						10.5		6.2				2.5				3.9				2.6		
AS								1.5			1.8						5.7					
AGD						1.5					2.0	0.0										
GD						4.1		5.6				0.0										

Table B.61: Variance explained by stress predictor variables by health-related outcomes (adjusted for potential confounders)

	Sm	Alco	PI	JF	FV	CESD	Poor HS	SF12 MCS	SF12 PCS	Waist	WHR	SBP	DBP	HT	DL	Ho	HbA1c	CRP	Fib	DP	Allo	#CC
ASS1						1.3		2.3									1.2					
ASS2								2.3												2.0		
CSS0																	3.7	4.0				
CSS2						2.6	2.5	2.4														
CSS3																						
TSS0																	6.1					
TSS2								1.2				2.6								2.7		
TSS3																						
#AS2										3.3							1.5					
#AS3			2.7					3.6														
#AS4						2.3		3.6								6.4				1.1		
#CS1						6.0		0.1												2.8		
#CS2						6.0		0.1												2.8		
#CS3						6.0														2.8		
#TS1									2.4													
#TS3	3.9											3.1				9.6						
#TS4						5.4										9.6				2.6		
AS																	5.1					
ASGD																						
GD						2.3		1.2				1.7										

Results in bold are statistically significant at $p < 0.05$.

Q: MIRE question with numbers indicating level of variable as coded in Chapter 7.3.4.2.2; **IPR**: Inter-personal racism score; **RS**: number of settings in which inter-personal racism was reported; **IR**: internalised racism; **SR**: systemic racism; **Alco**: moderate/high alcohol risk consumption level; **CES-D**: depression (as measure by the Centre for Epidemiologic Studies Depression Scale); **Poor health/ Poor HS**: poor/fair self-assessed health status; **Waist**: waist circumference; **WHR**: increased Waist-to-Hip Ratio; **SBP**: increased systolic blood pressure; **DBP**: increased diastolic blood pressure; **HT**: presence of hypertension; **DL**: presence of dyslipidaemia; **Ho**: increased homocysteine; **CRP**: increased C-reactive protein; **Fib**: increased Fibrinogen; **DH**: dental health; **Allo**: increased allostatic load; **#CC**: increased number of chronic conditions; **Sm**: smoking; **PI**: physical inactivity; **JF**: high junk food consumption; **FV**: Inadequate fruit/veg consumption **Waist**: increased waist circumference; **High (ASS)**: high (acute stress score); **High #AS**: high number of acute stressors reported; **High (CSS)**: high (chronic stress score); **High #CS**: high number of acute chronic reported; **Affected SGD**: having reported a stressor that affected respondent somewhat/a great deal; **Affected GD**: having reported a stressor that affected respondent a great deal. **AS**: Any reported stressor; **AGD**: having reported a stressor that affected respondent somewhat/a great deal; **GD**: having reported a stressor that affected respondent a great deal.

Each MIRE item was included in a regression model exclusive of other MIRE items. All levels of the same item in each table above were included in the same regression model. For example, Q1a (sometimes) and Q1a (often/very often) were included in the same model for the results reported in any particular table, and this model did not include any other MIRE items. MIRE abbreviations in Tables B.30-B.59 refer to the level of each item as detailed in 7.4.1.2.2. Stress abbreviations in Tables B.60-B.61 refer to the level of each stress item with the level for number of reported stressors being the quartile of reported experience and the level for stress scores being 0: No; 1: Yes, minimal/no effect; 2: Yes, affected me somewhat; 3: Yes, affected me a great deal.

The base comparison categories for each of the MIRE questions are as follows:

Q1a: never/hardly ever
Q1b: never/hardly ever
Q1c: never/hardly ever
Q1d: never/hardly ever
Q1e: never/hardly ever
Q1f: never/hardly ever
Q1g: never/hardly ever
Q1h: never/hardly ever
Q1i: never/hardly ever
Q1j: never/hardly ever

Inter-personal racism score: low
# of setting racism experienced in: 1st quartile

Q2a: often/very often
Q2b: never/hardly ever
Q2c: never/hardly ever
Q2d: sometimes
Q2e: often/very often
Q2f: never/hardly ever
Q2g: never/hardly ever
Q2h: sometimes
Q2i: sometimes
Q2j: never/hardly ever
Q2k: never/hardly ever
Q3a: strongly agree/agree
Q3b: strongly agree/agree
Q3c: disagree/strongly disagree
Q3d: strongly agree/agree
Q3e: strongly agree/agree
Q3f: strongly agree/agree
Q3g: disagree/strongly disagree
Internalised racism: low
Systemic racism: low
Q4: constantly
Q5: Yes, everyone
Q6: Yes, everyone
# of acute stressors: 1st quartile
# of chronic stressors: 4th quartile
# of total stressors: 2nd quartile
Acute stress score: Yes, affected me a great deal
Chronic stress score: Yes, minimal/no effect
Total stress score: Yes, minimal/no effect

Appendix C: Other relevant journal articles published during candidature

- Paradies Y. (2006). Beyond Black and White: Essentialism, Hybridity and Indigeneity *Journal of Sociology* 42(4): 355-367;
- Paradies Y. (2005). Affirmative action and equity in Aboriginal and Torres Strait Islander health. *Medical Journal of Australia* 183 (5): 269-270;
- Kowal E & Paradies Y. (2005). Ambivalent helpers and unhealthy choices: Public health practitioners' narratives of Indigenous ill-health. *Soc Sci Med* 60:1347-1357;
- Paradies Y & Stevens M. (2005). Conceptual diagrams in public health research. *J Epidemiol Community Health* 59(12): 1012-1013;
- Paradies Y. (2005). Confounding and Interaction in Epidemiology. *Australasian Epidemiologist* 12(2): 23-25.

Beyond Black and White: Essentialism, Hybridity and Indigeneity

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Abstract

Non-Indigenous conceptions of Indigeneity have historically focused on controlling the socialisation, mobility and reproduction of Indigenous people. In the Indigenous community, we have only recently begun to demarcate our own space in which to debate the nature of Indigeneity in Australia. To date, we have successfully deployed notions of Indigeneity, via the strategic essentialism of pan-Aboriginality, to create an effective political community. However such a deployment of Indigeneity also results in every Indigenous Australian being interpellated, without regard to their individuality, through stereotyped images that exist in the popular imagination. The essentialised Indigeneity thus formed coalesces around specific fantasies of exclusivity, cultural alterity, marginality, physicality and morality which leave an increasing number of Indigenous people vulnerable to accusations of inauthenticity. Only by decoupling Indigeneity from such essentialist fantasies can we acknowledge the richness of Indigenous diversity and start on the path towards true reconciliation in Australia.

Keywords: Indigenous, identity, essentialism, hybridity, reconciliation, Whiteness

Constructions of Indigenous[i] identity have been a preoccupation of both the Australian popular imagination and the Australian nation-state from the earliest days of colonisation (McCorquodale, 1997; Chesterman and Galligan, 1998; Gardiner-Garden, 2003). Historically, non-Indigenous approaches to defining and understanding Indigeneity have focused on the need to surveil and control the socialisation, mobility and biological reproduction of those with some descent from pre-colonial peoples of Australia (Dodson, 1994). In an analysis of over 700 pieces of legislation McCorquodale (1986) found 67 different definitions of Indigeneity. Much more recently Indigenous people have begun to mark out our own discursive space in which to debate the meaning of Indigeneity in contemporary Australia (Taylor, 2001).

Although some have expressed understandable distaste at the thought of non-Indigenous people defining their Indigeneity (Huggins, 2003: 60), such defining (whether official or popular) unavoidably involves both Indigenous and non-Indigenous people in "a process of dialogue, of imagination, of representation and interpretation" (Langton, 1993: 33). It is also clear that the fluid and contextual nature of Indigenous identity over time and place is not particular to either Australia or the Indigenous context. There are innumerable types of human identity which vary across many aspects of experience; including race, ethnicity, gender, sexuality, age, class, physicality, language, religion, and profession to name only a few. All these identities are complex, multi-faceted socio-historical constructs which are established through public acts of self-representation, private accountings of oneself or through the experience of being named by others (Jenkins, 1994), including by prevailing discourses. Although I will focus here on racial identities, it is important to remember that "we do not experience the world only as Indigenous or non-Indigenous" (Cowlishaw, 2004b: 70-71) but also through many other facets of our identity which we adopt and/or are ascribed to us by others.

Indigenous people in Australia have, for some decades, been engaged in debates about Indigenous identity.[ii] One element of these debates has been the implicit (or explicit) goal of creating a distinct, coherent and thus relatively homogenous pan-Indigenous social and political community. It has been suggested that only within this haven of pan-Indigeneity can Indigenous people "resist the seduction of assimilation and confidently work at rebuilding a unique identity" (Ariss, 1988:

136). If so, such a pan-Indigenous community may be a necessary element in surviving ongoing colonial imperatives by the Australian nation-state (Dodson, 1994; Werbner and Modood 1997: 249; Russell, 2001: 76). Also, instrumentality aside, such a communal shared identity is a pleasurable and empowering experience for many (Ang, 2001: 11).

However, despite assertions to the contrary (Dodson, 1994: 9), Indigenous constructions of (pan-)Indigeneity also involve elements of boundary construction/policing which seek to construct Indigenous and non-Indigenous identities as "mutually impermeable and incommensurable" (Gilroy, 1993: 65).[iii] Such policing serves to alienate past and potential future Indigenous people (O'Regan, 1999: 194-5) or force those who inhabit Indigeneity into a "prison-house" of identity (Ang, 2001: 11; Scott, 1992: 18; Oxenham et al., 1999: 70) which may isolate them "from contemporary life and full citizenship" (Brydon, 1995: 141), and hence, may even reduce the political power of the Indigenous community. The ambivalence and alienation resulting from such constructions is especially acute for many urban Indigenous people who live in increasingly anonymous communities in which the need to acquire an imprimatur of Indigeneity is a source of conflict (Cowlishaw, 2004a). Moreover, the powerful tropes of Indigeneity mobilised during this boundary construction interpellate every Indigenous person, without regard to their individual characteristics, through a plethora of stereotyped images (Morrissey, 2003b: 191) that coalesce around specific fantasies of exclusivity, cultural alterity, marginality, physicality and morality.

In order to challenge prevailing stereotypes, Indigenous people are often required to publicly confess our intimate subjectivities (Anderson, 1997: 5; Nakata, 2003: 134-5). Although this requirement may be a manifestation of the colonial gaze, such confessions serve to remind us of the situated and partial nature of knowledge (Russell, 2001: 73). As such, I will utilise autobiography as a rhetorical construction (Ang, 2001: 24) along with contemporary scholarship on identity both in Australia and abroad to explore the aforementioned fantasies of Indigeneity. In declaring the particularity of my Indigeneity, I recognise that I will inevitably be interpellated by readers in ways which either establish or undermine my authority to speak on the fraught issue of Indigenous identity.

I identify racially as an Aboriginal-Anglo-Asian Australian. Along with scholars such as Ang (2001: viii) and Russell (2001: 98), my personal history compels me to identify as more than just Indigenous and as other than exclusively White, while moving beyond this dichotomy in also recognising my Asian heritage. Like Russell (2001: 98), I refuse to "surrender my other identities" in order to be Indigenous and, as such, I also identify as 'and/or' as well as 'not/nor' Aboriginal-Anglo-Asian. Descended from both Indigenous and Euro-Australian ancestors I am both coloniser and colonised, both Black and consummately White (Lehman, 2004).

Many Indigenous Australians also share my diverse racial background, with, for instance, 22% of Indigenous people reporting 'European' ancestry in the 2001 Census (Australian Bureau of Statistics, 2003). Furthermore, about half of all Indigenous people in a marriage or de facto relationship have a non-Indigenous partner, with over 80 percent of children from these 'mixed' partnerships adopting an Indigenous identity (Ross, 1999). Yet, despite this heterogeneity in the Indigenous community, asserting a multi-racial Indigenous identity is neither common nor straightforward because racial loyalty demands that anomalous individuals choose to be either exclusively Indigenous or exclusively non-Indigenous (Boladeras, 2002: 131; Cowlishaw, 2004a: 114). Those, like me, who refuse this compulsion in the hope of creating a hybrid space of multiplicity (Russell, 2002: 141-2), instead find ourselves inhabiting "a strange in-between space" (Russell, 2002: 139) where we are frequently not accepted as Black (Cowlishaw, 2004a: 116), and where we are subject to the opprobrium directed at "the half-different and the partially familiar" (Gilroy, 2000: 106). The prospect of such derision combined with the demands of racial loyalty are evident even in otherwise excellent scholarship on Indigenous identity, which seamlessly reinforces exclusive Indigeneity by discussing (in the contributors' biographies) Indigenous heritage to the exclusion of non-Indigenous heritage (Oxenham et al., 1999: 32-50).

Due, in large part, to my grandmother being a member of the Stolen Generations, I do not speak an Aboriginal language, I do not have a connection with my ancestral lands or a unique spirituality inherited through my Indigeneity, I have little contact with my extended family, and the majority of my friends are non-Indigenous. Also due to this same history, I am a middle-class, highly educated professional working in the field of Indigenous research. As such I am frequently

interpellated as Indigenous and called upon to deploy my Indigeneity in a professional context, while at the same I am labelled by some as an inauthentic "nine-to-five black"[iv] or a "coconut"[v] who has stolen the place of a 'real Aborigine' (Paradies, 2005). Despite assertions by some Indigenous scholars that a unique spirituality (Foley, 2000) or relationship to land (Moreton-Robinson, 2003) epitomise Indigeneity, the available statistics suggest that many Indigenous Australians fail to conform to the fantasy of cultural alterity: 88% of us do not speak an Aboriginal language as the main language spoken at home; only a little over half of us identify with a particular clan, tribal or language group (Australian Bureau of Statistics, 2003); and most of us live in single parent or nuclear households rather than extended family arrangements (Zubrick et al., 2004: 38).

Moreover, there is a growing Indigenous middle class as demonstrated by the fact that between 1994 and 2002 the proportion of Indigenous people with at least a Bachelor degree increased threefold (from 1% to 3%), the proportion of Indigenous people who were employed rose from 36% to 46% and mean equivalised gross household income for Indigenous people 15 years and over rose from $345 to $387 per week (Australian Bureau of Statistics, 2002).[vi] Yet despite (or perhaps because of) the fact that, in absolute terms, our lot as Indigenous people is improving, there is a prevailing misconception that "if you're middle-class you can't be Aboriginal" (Boladeras, 2002: 135). Being educated, well-remunerated or simply enjoying material assets "can expose one to suspicion of wanting to be white" (Cowlishaw, 2004a: 113; Purdie et al., 2000; Grant, 2002). Although many Indigenous people rightly desire the privileges that, until recently, have been synonymous with Whiteness, such desire is associated with being less Indigenous (Dudgeon and Oxenham, 1989; Cowlishaw, 2004a: 116).

There is no doubt that Indigenous people have suffered a deplorable history of marginalisation, discrimination and exclusion that continues to this day (Lattas, 2001; Mellor, 2003; Dunn et. al., 2005), and that such a history has lead to a "solidarity grounded in a common experience of subordination" (Portes, 1998: 9). However, it is also evident, from international contexts, that when group cohesion is premised on the impossibility of transcending such subordination, the achievement of individual success endangers this cohesion. As a result, social norms are formed

which seek “to keep members of a downtrodden group in place and force the more ambitious to escape from it” (Portes, 1998: 9). This emphasis on marginality also risks reinforcing the danger of fatalism which pervades the minds of many Australians (Brough, 1999) who, faced with the myth of worsening Indigenous disadvantage, believe there is no solution to this intractable problem and hence no point trying to address it. Furthermore, in our individualistic Western culture it is far too easy to blame such ‘intractable’ disadvantage on Indigenous people themselves (Brough, 1999).

Arguably even more prevalent than the fantasy of marginality that adheres to Indigeneity is the pernicious fantasy of the ‘Indigenous look’. Along with many other Indigenous Australians I fail to match the stereotyped physicality of an ‘Indigene’: that is, I have white skin and relatively European features. Despite assertions to the contrary (Perkins, 2004: fn 3), it is clear that skin colour and physicality are “exceptionally important in the recognition and validation of Aboriginal identity” (Boladeras, 2002: 147; Schwab, 1994: 94; Oxenham et al., 1999: 79-82), as they are in similar international contexts (Cunningham, 1997). Fair-skinned Indigenous people experience “racism, scorn and disbelief” from other Indigenous and non-Indigenous people alike, whose perennial interrogation of their identity leads to acute anxiety (Foley, 2000; Purdie et al., 2000) as well as “ambivalence, and doubts about themselves as ‘real’ [Indigenous] people” (Boladeras, 2002: 147) . This intense questioning of authenticity, which can hit you with the force of a sledgehammer, is due to the profound disruption that white-skinned Indigenes represent for the Black-White racial dichotomy, so fervently clung to in Australia. As one person put it, “sometimes… it would be just a lot easier to say I’m not Aboriginal…” (Boladeras, 2002: 116). Yet many people choose to publicly identify as Indigenous, exposing themselves to “an uneasy pathway through life…strewn with doubt, disbelief and confrontation” (Boladeras, 2002: 153).

For me, as for many other descendants of the Stolen Generations (both fair-skinned and dark), it “was the choice of others that I be denied the shared experiences…that contribute to an [essentialised] Aboriginal identity” (Russell, 2002: 137-8). However, unlike Russell (2002: 137-8), I do not accept such an essentialised view of Indigeneity, and I refuse to recognise that among individuals who have Indigenous ancestry, there is a group of people who ‘qualify’ as Indigenous

and a group who don't. Indeed, it is disturbing that Russell is reluctant to identify as Indigenous, in part, from fear of "other peoples' reactions, especially those of Aboriginal people" (2002: 141-2). Similarly, Kathryn Hay (Tasmanian parliamentarian and former Miss Australia) is ambivalent about identifying as Aboriginal "because of what people would expect if I said I was Aboriginal" (Purcell, 2002: 234-235). Clearly, such trepidation at being 'caught out' as inauthentic arises from the deleterious notion that "there are protocols and ethics to adhere to when 'becoming Aborigines,'" which require "years of hard work, sensitivity and effort" to master (Huggins, 2003: 63). Such purported 'protocols' lead to the questioning, challenging and doubting of Indigeneity by ourselves and others and results in academics like Russell (2001: 98) stating that because she was "raised to be part of the mainstream white community I feel incapable of stating that I am Aboriginal".

A particularly troubling aspect of the 'protocols of Indigeneity' is the positioning of Indigenes as inherently morally and epistemologically superior to non-Indigenes (Kowal and Paradies, 2005). Although this phenomenon is driven by non-Indigenous attempts to mitigate their privileged position (Land and Vincent, 2005) and assuage the guilt of continuing colonisation, such a positioning is more often perpetuated than protested by Indigenous people (see Paradies, 2006 for an example). The idea that Indigeneity is synonymous with suffering (Russell, 2002: 141-2) and marginality, together with the misconception that such 'victimhood' (Palmer and Groves, 2000) bestows privileged access to social truths (Stanley and Wise, 1993: 227; Cowlishaw, 2004b: 64), leads to uncritical acceptance of the views, opinions and scholarship of Indigenous people about Indigenous issues (Langton, 1993: 27). This phenomenon is sometimes also accompanied by a corresponding rejection of non-Indigenous views which are portrayed as "tainted with racism" (Cowlishaw, 2004b: 65). Such moralistic positioning is untenable given the various and contradictory views that Indigenous people hold and, more importantly, such an approach fails to recognise that engaging in debate with Indigenous people is a sign of intellectual respect (Land and Vincent, 2005).

This instinctive urge to foist moral rectitude onto those who are oppressed and marginal (Hage, 2003: 116) also leads far too many to conclude that repugnant acts such as racism cannot, by

definition, be perpetrated by Indigenous people. Of course, this reasoning is fallacious, there being “no reason why those subjected to racism of the worst kind cannot be racist themselves” (Hage, 2003: 116-7). Similarly, the view that being Indigenous requires one to resist White hegemony (Cowlishaw, 1988: 243) or strive to alleviate the disadvantage of Indigenous people (Huggins, 2003: 64), also inappropriately portrays Indigenous people as intrinsically virtuous (Russell, 2001; Cowlishaw, 1988: 243). These moral qualifications, that some would have us espouse as prerequisites of Indigeneity, evince a profound failure to recognise that “wisdom and virtue are as unevenly distributed among Indigenous people as elsewhere” (Cowlishaw, 2004b: 71).

Taken as a whole, these fantasies of Indigeneity raise the question of “who is a ‘real indigenous’ person’” (Smith, 1999: 72) and through processes of forced inclusion, which are never completely successful (Ang, 2001: 83), protocols of cultural survival end up replacing “one form of tyranny with another” (Appiah, 1994: 163). The “border patrol is kept busy erasing and denying whatever does not or will not fit” (Ferguson, 1993: 165), repudiating the variety and complexity of contemporary Indigenous Australia (Muecke, 1992) and leaving a community fragmented into those who can authentically perform Indigeneity and those who are silenced and/or rendered outside the space of Indigeneity because they cannot, or will not, perform (Griffiths, 1995: 238; Smith, 1999: 72).

In addition, this community fragmentation cleaves to fault lines of power, with some Indigenous elites, in seeking to preserve the status quo, colluding with neo-colonial governments to coerce Indigenous people into performing an essentialised Indigeneity which compromises their “capacity to be individually – and differently – Aboriginal” (Moore, 2005: 188). This phenomenon is of dubious utility, even to these elites, as shown in the application of native title law where the neo-liberal state makes an “impossible demand for authenticity” by recognising prior sovereignty only for those Indigenous people who can prove that they have “been relatively untouched by history” (Schaap, 2005: 19). In doing so, the state denies the existence of actual Indigenous people who, by adapting and changing, have survived colonialism while unavoidably shedding their pristine primeval identity.

Towards Indigeneity as an open signifier

"*Imperialism['s]…worst and most paradoxical gift was to allow people to believe that they were only, mainly, exclusively, white, or black…*" (Said, 1993: 408)

In moving beyond the binary dualism of Indigenous vs. non-Indigenous we are acknowledging that we now live in a "thoroughly hybridized world where boundaries have become utterly porous, even though they are artificially maintained" (Ang, 2001: 87-8). The collective history of Indigenous people in Australia should leave us with grave misgivings about disenfranchising others of their chosen identities (Morrissey, 2003a: 59). It is therefore imperative that we adopt an approach to Indigeneity requiring the bare minimal of essentialising (Morrissey, 2003a: 59) which avoids imprisoning Indigeneity into a fixed, frozen category of being (Dodson, 1994; Johnson, 1993; Gilroy, 2000: 13).

The United Nations Draft Declaration on the Rights of Indigenous peoples states, in Article 8, that "Indigenous peoples have the collective and individual right…to identify themselves as indigenous and to be recognised as such" (quoted in Dodson, 1994). In Australia, the Commonwealth working definition states that an "Aboriginal or Torres Strait Islander is a person of Aboriginal or Torres Strait Islander descent who identifies as an Aboriginal or Torres Strait Islander and is accepted as such by the community in which he (she) lives" (Gardiner-Garden, 2003: 4). This Commonwealth definition, formulated in the 1980s, has since been widely accepted by various Australian governments, utilised by the High and Federal Courts (Gardiner-Garden, 2003) and predominately accepted by the Indigenous community (Langton, 1993: 29; Taylor, 2001).

Embracing the Commonwealth working definition in its most inclusive form, would require Indigenous communities to accept as Indigenous anyone with Indigenous ancestry who wishes to identify as such. Furthermore, in accordance with the even more inclusive United Nations definition, the vexing issue of indeterminable Indigenous descent could also be overcome by recognising the adoption of people into the Indigenous community who cannot prove Indigenous

descent (Fischer, 2000). This would silence the acrimonious debate that has plagued famous 'Black' Australians such as Narrogin Mudrooroo (Colin Johnson) and Roberta Sykes – both of whom embrace notions of hybridity[vii] (Griffiths, 1995: 240; Mudrooroo, 1997; Kurtzer, 2001) alongside others with more credentialed ancestry, in Australia (Groves, 1998: 79; Cowlishaw, 2000: fn 8; Grant, 2002; Lehman, 2004; Selby, 2004: 148) and abroad (Hall, 2001). In once again recognising the fluid and contextual nature of all identities, including Indigeneity, such an approach would also incorporate existing practices of adopting non-Indigenous people, who work or marry into Indigenous worlds, as Indigenous in specific contexts (Oxenham et al., 1999: 85). I want to make clear that, unlike Hage (2003: 5) and Greer (2003), I am not proposing that all Australians become Indigenous. Rather, I recognise that reconciliation depends "on founding and sustaining a space for politics within which the emergence of a common identity is an ever present possibility" (Schaap, 2005: 21).

No approach to Indigenous identity can reverse the centuries of colonialism experienced by Indigenous people in Australia; nor should this be our aim. Rather, an inclusive approach to Indigeneity allows us to celebrate the "extraordinary diversity of subjective positions, social experiences and cultural identities" (Hall, 1995: 225) that comprise Indigenous Australia (Groves, 1998), while acknowledging that we have survived as Indigenous people only as a result of our ability, and that of our ancestors, to adapt and endure (Gilroy, 2000: 13). Such an approach transcends the compulsion to unceasingly patrol identity borders which have been constructed around primitivist, romantic and colonial discourses (and which are now being co-opted for neo-colonial purposes) and starts us on the long and difficult journey of freeing ourselves from "myth-making" and the internalised racism of identity politics (Oxenham et al., 1999: 59-64).

On this journey we may increase our political power by unreservedly allowing the more privileged and less marginal, amongst others, to join our ranks. On the other hand, there is a danger that inclusivity may 'dilute' the meaning of Indigeneity in popular and official discourse and undermine some of the gains made since the advent of pan-Indigeneity. As with any social change, such a journey would be a gamble with no assured consequences. What is clear, however, is that we do not want such an inclusive approach to provide a "biological ticket…to enter the

world of ‘primitivism’” (Langton, 1993: 30) nor allow the appropriation of Indigenous cultures or the specific benefits (e.g. land rights) associated with some of these cultures (Greer, 2003: 17-20). To avoid this we, as Indigenous people and as a nation, must decouple Indigeneity from disadvantage and marginality, from cultural and physical alterity and from callow moral dichotomies. It must be recognised that Indigenous people do not require particular phenotypical traits, certain forms of cultural alterity, specific ethico-moral beliefs/actions, or a level of social disadvantage in order to be Indigenous. Although like Gilroy (2000: 6), I do not “see contact with cultural difference solely as a form of loss”, I am **not** suggesting that we refrain from reviving/preserving the diverse cultures associated with various Indigenous communities in Australia, or that we cease to engage in judicious affirmative action designed to assist those Indigenous Australians who are disadvantaged (Paradies, 2005). Rather, I am suggesting that we free Indigeneity from the prison of romanticisation and recognise that although the poor and the rich Indigene, the cultural reviver and the quintessential cosmopolitan, the fair, dark, good, bad and disinterested may have little in common, they are nonetheless all equally but variously Indigenous.

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[i] The term Indigenous is used here to refer to Aboriginal and/or Torres Strait Islander people. Although it is clear that discourses of identity do vary between and within these two broad groups of people, a discussion of such variations is beyond the scope of this paper. Furthermore the terms Aboriginal and Black (as well as White) will also be used as appropriate, especially where quoted from other sources.

[ii] For instance, almost any issue of the national Indigenous newspaper, the Koori Mail, over the past several years includes highly emotive and charged letters to the editor about Indigenous identity.

[iii] In the main, authors are cited in this paper where their arguments support those presented in the text. However, the scholarship of Foley, Huggins, Moreton-Robinson, Perkins and, in some cases, Dodson and Oxenham et al. is cited in specific examples of the discourse on Indigeneity which this paper seeks to critique. Clearly, such an illustrative use of these authors' writing neither constitutes nor implies an appraisal of their broader and more complex bodies of work.

[iv] A term used to indicate an Indigenous person who does not mix with other Indigenous people outside the work context (Oxenham et al., 1999: 112).

[v] A derogatory label that indicates a person is Black on the outside (i.e. skin colour or superficial behaviour) but White on the inside (i.e. personality, beliefs etc.) (Dudgeon and Oxenham, 1989).

[vi] In 2002, the proportion of non-Indigenous people who had at least a Bachelor degree was 17% while 63% of non-Indigenous people were employed and the equivalised gross household income for non-Indigenous people was $665 per week (Australian Bureau of Statistics, 2002). Clearly Indigenous people are still disadvantaged in comparison to non-Indigenous people. The point I am making, however, is that this disadvantage, at least in absolute terms, appears to be decreasing over time.

[vii] As noted by Anderson (1997), I recognise that the term ‘hybrid’ has historically represented transgression, sterility and liminality in the Australian Indigenous context. However, shunning this term because of such colonial connotations, as Anderson (1997) does, only serves to reinforce these connotations. Rather, my intention here is to co-opt and reclaim this term as constitutive of Indigenous people and hence destabilise the trope of the ‘authentic Indigene’ (see also Oxenham et al., 1999: 102-3).

Affirmative action and equity in Aboriginal and Torres Strait Islander health

THE SECOND OF THREE FINALISTS' ESSAYS

As Indigenous Australians, our health lags behind that of indigenous groups in other settler colonial nations such as the United States, Canada and New Zealand.[1] Similarly, we are far behind these nations in relation to Indigenous participation in the health workforce and the professions generally. A compelling illustration of this is the fact that the first indigenous doctors in North America and New Zealand graduated in 1889 and 1899, respectively, while the first Indigenous doctor in Australia graduated almost a century later, in 1984.[2,3]

It is undeniable that our poor health and our low participation in the health workforce are related. Increasing recognition of this has led to a situation in which there are now over 80 qualified Indigenous doctors and almost 100 Indigenous medical students in Australia.[4] This achievement has only been possible because we live in the era of "self-determination" that was born, as was I, during the 1970s. I am a multiracial Australian, with Aboriginal, Anglo and Asian ancestry. Because my grandmother (being my only Indigenous ancestor) was a member of the "stolen generations", and because I am fair-skinned, I started life with an ambivalent Indigenous identity which has been profoundly shaped by the policies of affirmative action (or positive discrimination) that epitomise the era of self-determination.

My first experiences with affirmative action occurred in high school, when I was showcased as a role model for other Indigenous students. I went on to gain a degree in science and start a career in health research through an Indigenous cadetship. Affirmative action also provided me with the financial means to complete a Master of Medical Statistics — the first Indigenous Australian to do so — and with preferential access to scholarships, which allowed me to complete a Master of Public Health and to undertake a PhD. In return for this assistance, I have spoken at Indigenous youth summits and school career days, tutored, taught, and donated prize money to Indigenous tertiary students, and conducted research and teaching in Indigenous health on topics of importance to Indigenous people (such as the health effects of racism).[5] In addition, I have brought an "Indigenous" perspective to a range of committees, forums, round tables, community groups, conferences, colleagues and students.

These experiences are in keeping with the goals of affirmative action, which are to create equality of opportunity and outcome for Indigenous Australians and, for the benefit of all Australians, to promote diversity through equitable representation of Indigenous people in society. Affirmative action is achieved, in practice, by assisting individual Indigenous people to become more "successful". In addition, there is often an implicit assumption that the Indigenous recipients of affirmative action will themselves strive to reduce social inequities, promote opportunities for and enhance representation of Indigenous people, and act as role models.

While medicine has made some progress towards these goals, and the role played by Aboriginal and Torres Strait Islander health workers has been crucial, other areas of health have not fared as well. There has been little government support for increasing the number of Indigenous nurses or allied health professionals,[2] with the notable exception of the recent Puggy Hunter Memorial Scholarships.[6] Health research is a particularly lonely field. I knew of no other Indigenous person studying at my institution in the same degree program as me. Also, milestones such as the first Indigenous doctor and the first Indigenous clinical psychologist to gain a PhD have only been achieved in the 21st century.

Clearly, there is still considerable scope for applying affirmative action in health and other sectors in which Indigenous people continue to suffer from disadvantage. However, I believe there are complexities inherent in both the aims and practice of affirmative action that need to be considered in order to improve social outcomes for Indigenous Australians and allow our diversity to contribute to Australia's future. In this essay, I will share with you what I have learnt about the benefits and pitfalls of affirmative action as an Indigenous health professional during the past decade.

The most obvious difficulty faced by Indigenous people who are beneficiaries of affirmative action is the self-doubt stemming from accusations that we do not merit such support. Unfortunately, it appears that many Australians still think Indigenous people get "too many benefits". In one survey, almost a third of participants believed that car loans are paid for us by the government, and almost two-thirds thought that we receive more social security benefits than non-Indigenous people.[7] In another survey, more than half of respondents believed Indigenous people were "treated over generously by the government".[8] The hostility to affirmative action programs, which is compounded by these misconceptions, can only be reduced through education that explains the benefits of diversity and the need to remedy historical injustice.[9]

There is also an urgent and profound need to tackle systemic racism in Australia, which, in its most extreme form, led to the death of Private Damien Palmer. After entering the army through an affirmative action policy, he committed suicide following racist taunts and intense ridicule from fellow soldiers and instructors.[10] Systemic racism is something that white Australians, as those who benefit most from the system of racial oppression, are in the best position to combat. Indigenous people, in contrast, face anguish and rapid "burnout" when leading the fight against this insidious form of racism.

The intense doubt of our self-worth which some Indigenous people experience as a result of affirmative action can be reduced by ensuring that Indigenous people are only placed in positions and given training or learning opportunities for which they have the appropriate commitment, skills, abilities and qualifications. If we cannot do a job or don't have the capacity to benefit from a program, this is no doubt due to a legacy of oppression and colonisation. However, this situation is not remedied by the tokenism being engaged in by far too many organisations around Australia. Not only are those caught up in tokenism unable to effectively advance the goals of affirmative action, but they are also being set up to fail or, worse, set up as ever more prominent "tokens", whose increasing visibility as such severely hampers ongoing efforts to redress Indigenous disadvantage.

It is clear that affirmative action, no matter which segment of the Indigenous population is targeted, is most helpful to those who are most advantaged to begin with.[11] It is easiest, and perhaps most appropriate, for those who have the most capacity to benefit to take up whatever opportunities are on offer. However, there is still a tendency for both Indigenous and non-Indigenous people to assume that being Indigenous is synonymous with a certain "marginality or victimage".[12,13] This misconception can leave Indigenous recipients of affirmative action not only doubting their self-worth but also being seen as threatening "tall poppies"[2] and having their Indigenous identity questioned. I have certainly been accused of taking positions that should have been given to "real blacks" who weren't as acculturated to white society as me and/or who had darker skin. These issues need to be brought into the open, so that Indigenous people who are beneficiaries of affirmative action can share these difficult experiences and learn from each other about effective coping strategies.

In implementing affirmative action policies, we need to stop simply seeing "Indigenous people" as a single entity and instead see individuals with multiple personal and professional identities, including Indigeneity. We need to believe in and respect Indigenous people as individuals who have their own goals, principles, flaws and foibles and who may or may not be interested in, capable of, or suitable for a specific affirmative action policy or program. Despite what is often assumed, some Indigenous people — just like some non-Indigenous people — are not effective role models or interested in working towards emancipatory goals. Therefore, it is vital to consider, in every case, whether, and to what degree, affirmative action is aimed at helping an individual become more successful, at increasing diversity, or at aiding recipients who will then contribute to the goals of affirmative action themselves.

These questions need to be addressed by all Australians and will require us to grapple with the historical baggage that accompanies the stereotyped Indigenous identity we have inherited as a nation.[14] All of us need to truly come to terms with the diversity of Indigenous people in Australia, rather than just paying lip service to this notion. To do this, non-Indigenous Australians must overcome their reluctance to engage in debate about Indigenous affairs,[15] and Indigenous Australians must stop acting as if dissenting views are nothing more than simplistic attacks on Indigenous people.[2]

If there is one thing that most people agree on, it is that there is no simple solution to the complex problems in Indigenous health.[2] In relation to our history as a nation, the struggle to improve the lives of Indigenous people in Australia has only just begun and has a long way to go before equity is achieved. In the field of Indigenous health research, in which I work (and, I suggest, in many other fields), we require a greater clarity of purpose and increased patience in relation to affirmative action if we are to avoid jeopardising our efforts at improving the plight of Indigenous people.

Not all health research can involve Indigenous researchers, because those with sufficient training and experience are few and far between. We are not well served by setting up Indigenous people as "researchers" when they are not, or by prioritising affirmative action to such an extent that the very research we do is compromised. Let us hope that the spectacular rise in the number of Indigenous doctors continues until equity in medicine is reached and that we can match this achievement in Indigenous health research. In working towards these goals we need to recognise that the era of self-determination, like those before it, won't last forever. What could perhaps be called the "partnership era" may already be replacing it, as exemplified by policy approaches such as shared responsibility agreements.[16] The role of affirmative action in this new era is not assured and will only be maintained by addressing the difficult issues now, so that Australia may one day become an inclusive nation where, as Indigenous people, we can maintain our unique identity while playing a full role in society and enjoying the same level of health as other Australians.

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Ambivalent helpers and unhealthy choices: public health practitioners' narratives of Indigenous ill-health

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Abstract

Public health practitioners in Australian indigenous health work in a complex political environment. Public health training is limited in providing them with conceptual tools needed to unpack the postcolonial nexus of 'fourth-world' health. A workshop was designed by the authors to facilitate critical reflection on how the concepts of race and culture are used in constructions of indigenous ill-health. It was attended by researchers, students, clinicians and bureaucrats working in public health in northern Australia.

A thematic analysis of the workshop minutes provided insight into public health practitioners' narratives of Indigenous ill-health. The major themes that emerged included tension between structure and agency and between sameness and difference, and ambivalence surrounding the 'helper' identity of public health practitioners. We suggest that these narratives can be understood as attempts to maintain the moral integrity of both Indigenous people and practitioners. This task is necessitated by the specter of cultural relativism intrinsic to contemporary liberal discourses of multiculturalism that attempt to reconcile the universal rights of the citizen with the special rights of minority groups.

We argue that the concepts of self-determination and neocolonialism mark the spaces where universal and particular discourses overlap and clash. Practitioners who seek to escape neocolonialism must inhabit only the discursive space of public health congruent with self-determination, leaving them in a bind common to many postcolonial situations. They must relieve the ill-health of indigenous people without acting upon them; change them without declaring that change is required.

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Introduction

Indigenous health in Australia is a highly politicized and contested arena. The poor state of Indigenous health is widely attributed to colonization and its ongoing manifestations (Paul, 2002; Johnston, 1991; Paul & Atkinson, 1999). The health system has been heavily implicated in the damaging, some would argue genocidal, practices of the State (Clarke, Andrews, & Austin, 2000; Human Rights and Equal Opportunities Commission, 1997; Paul, 2002). Indigenous people from Australia and elsewhere have spoken of research as "a dirty word" (Smith, 1999) and have described their communities as "researched to death" (Atkinson, Graham, Pettit, & Lewis, 2002; Chataway, 1994). They have demanded recognition of past and ongoing exploitative research practices, and fought to change the balance of power within research (Humphery, 2001; Henry, et al., 2002). Some are critical of the health

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system for not doing enough (Australian Medical Association, 2002; Ring & Brown, 2002), whilst others see governmental and non-governmental intervention as neocolonialism (Pearson, 2000) and advocate for a minimal role of non-Indigenous people in Indigenous health research (Humphery, 2001).

It is within this contested political landscape that practitioners[1] working in Indigenous public health find themselves. Public health practitioners in Australia are primarily middle-class, university-educated and White, placing them in an ambivalent and contradictory subject position vis-à-vis the postcolonial context of Indigenous public health.[2]

Practitioners working in public health do not generally participate in the academic discourse that analyses post- and neocolonialism (for important exceptions see Aviles, 2001; Inhorn & Whittle, 2001). While popular books that attempt to diagnose the 'Aboriginal problem' such as *Why Warriors Lie Down and Die* (Trudgen, 2000) are widely read, academics who critique aspects of postcolonial race relations in Australia such as Lattas (2001), Cowlishaw (2003), Povinelli (2002) and Rowse (2002) are generally not accessible to those without prior exposure to critical disciplines. Consequently, practitioners may not have the conceptual tools required to appreciate the existence (let alone the complexity) of their subject positions.

This was illustrated in a workshop held in 2001 by the Cooperative Research Centre for Aboriginal and Tropical Health (CRCATH)[3] in which Indigenous and non-Indigenous health researchers were brought together to discuss Indigenous health research. Participants were split into Indigenous and non-Indigenous groups and asked to consider what research projects had been effective in improving health for their own cultural group. Responses from the non-Indigenous group included comments such as "I've only ever researched in other cultures, I have to dig deep to look at my own", whilst others commented that "[we] are not very good at seeing our own culture," and "a lot of non-Indigenous people will say they have no culture" (Franks et al., 2002).

These findings prompted the authors to design a further workshop for practitioners in Indigenous public health focused on the key themes of race and culture. The authors wanted to encourage self-reflection through an interrogation of practitioners' own subject positions as well as the concepts of ill-health, inequality, disadvantage and Indigeneity which they employed.

This paper first outlines the theoretical tensions between the universal and the particular that are useful for interpreting public health practitioners' narratives. We then outline the aims and conduct of the workshop and approaches to analysis in the Methodology section. In the Findings we present the major themes that arose during the workshop, and in the Discussion we show how these themes can be understood as attempts to manage the relativist possibility that 'universal' rights to health are in fact inappropriate for Indigenous people. We conclude by exploring how this research relates to a broad spectrum of issues confronting liberal democracies globally, and how these methods can be used to stimulate productive debate around the complex issues practitioners face every day.

Theoretical ground: the universal and the particular

The material presented in this paper informs the ongoing debate within political theory concerning the universal rights of the citizen and the specific rights of minority groups within the nation-state. We will argue that the narratives of public health practitioners presented below are best understood as attempts to manage the tension between the universal and the particular, tensions that take specific forms in the realm of health.

The idea that health is a right can be traced to the Constituent Assembly of the French Revolution in 1791. From its origin, the obligation of the State to provide health care to its citizens was accompanied by the obligation of the citizen to maintain their own health for the benefit of the State (Porter, 1999, p. 57). In the twentieth century, the concept of health as a universal right extended to all peoples has emerged (for instance in the 1946 constitution of the World Health Organization). With the development of this concept, Western nations have directed new forms of attention to the health of minority and colonized populations.

As infectious diseases became less prevalent in many parts of the world, the twentieth century also saw a shift in the emphasis of public health. With the increasing importance of chronic disease, and the links between individual behaviors and these diseases, twentieth century public health in Western countries has focused on promoting healthy lifestyles by changing individual behaviors such as smoking, diet and exercise (Porter, 1999, p. 298). Foucauldian scholars have noted a parallel shift in the style of governing, from repressive state power, to the imperative for individuals to fashion

[1]The term 'practitioners' refers to those working within the realm of public health such as researchers, students, bureaucrats, or clinicians.

[2]'Indigenous public health' is used throughout this article to denote the sub-field of public health that is applied to Indigenous health problems. 'Indigenous' is used interchangeably with the specific Australian term 'Aboriginal'.

[3]The Cooperative Research Centre for Aboriginal and Tropical Health (now the Cooperative Research Centre for Aboriginal Health) was a partnership between 6 (now 12) organisations involved in the research and delivery of Aboriginal health in Australia.

themselves as worthy citizens through techniques of self-surveillance and bodily control (Foucault, 1979; Rose, 1999).

Inherent in this expansion of personal responsibility is the moral weight placed on accepting it. Morality and ethics are the major sites of governmental power: controlling one's diet, weight, sexuality, and addictions have become moral obligations for the citizen (Lupton, 1995; Brandt & Rozin, 1997). Thus we will contend that Indigenous people who fail or refuse to fulfill these obligations of citizenship create a *moral* problem for those who try to help them to be healthy.[4]

For minority groups such as Indigenous Australians, universal rights and obligations are complemented by special rights. Prominent multicultural theorist Will Kymlicka argues that minority groups should have the right to special political representation, to autonomy on some issues (such as education and resource development), and to special support or exemptions from laws, as long as these special rights do not encroach upon the universal rights of group members (Kymlicka 1995, p. 35–44).

Indigenous Australians achieved formal equality in the 1960s and Indigenous health received government funding only from the 1970s. The official discourse of Indigenous health as a universal right shifted in the 1980s to health as a special right, with self-determination accepted as crucial for health improvement, and the recognition of Indigenous concepts of health (National Aboriginal Health Strategy Working Party, 1989).

Critics of Kymlicka have argued that this formula of 'universal plus special' rights is problematic, because the recognition of a 'special' right reveals that the 'universal' right is itself specific: "multiculturalism deflates the absolutist pretensions of liberalism and requires it to acknowledge its contingent historical and cultural roots" (Parekh, 1999, p. 74). Applied to health, this would mean that recognizing the existence of Indigenous notions of health inevitably calls into question the 'universal' ideas of health as living a long life free from disease. Rowse (2002) provides an example of this tension when he questions whether high unemployment in remote Indigenous communities can be taken as the failure of equality (the government has failed to provide jobs in remote areas), or the success of self-determination (Indigenous people freely choose to live a traditional lifestyle supported by welfare payments).

Notwithstanding this potential incommensurability between universal and specific rights, the State is obligated to work to improve Indigenous health, and ultimately improve life expectancy.[5] Indigenous groups have long demanded a greater commitment from the government and critiques from within public health have argued that the State has a moral obligation to improve the health of the Indigenous minority (Morgan & Allen, 1998).

While important gains have been made (e.g. reduced infant mortality), adult mortality still remains high despite over 20 years of effort from the government and the Indigenous community-controlled health sector (see Australian Bureau of Statistics & Australian Institute of Health and Welfare, 2003). Although the dominant discourse attributes this failure to the lack of real commitment from the government (for example, Ring & Brown, 2002), we argue that many public health practitioners are troubled by the possibility that Indigenous rights to self-determination and to define their own notions of health, are in conflict with universal measures of and approaches to improving health. While the authors remain agnostic as to whether universal measures are always appropriate to Indigenous people, we are convinced that the *tension* produced by the possibility of this relativism is a driving force in Indigenous health.

Methodology

Several studies have suggested that workshops and courses on topics of race, racism and culture can be beneficial to participants (Kobayashi, 1999; Gamble, 1999; Locke & Kiselica, 1999; Jakubowski, 2001). Elements and approaches that have been found to work best have included valuing flexibility and diversity, avoiding an authoritarian style, addressing emotional issues (Cohen, 1995), creating a safe space of trust, support, acceptance and respect (Locke & Kiselica, 1999; Baron, 1992), using multidisciplinary, action-oriented approaches, multimedia formats and free discussion (Cohen, 1995), as well as remaining neutral and utilizing critique, analysis and self-exploration (Kobayashi, 1999; Gamble, 1999). For this workshop we adopted a self-reflexive, participant-focused approach in which participants used problem-based and experiential learning techniques to reach their own conclusions through critiquing appropriate materials and their own worldviews.

The overall aims of the workshop were two-fold: (i) to encourage practitioners to consider the ways that they approach Indigenous ill-health, and the differing uses of race and culture in these approaches and; (ii) to examine

[4]It should be noted that this moral problem is also present for Indigenous people in a helper role. In this paper we artificially separate the categories of 'Indigenous people' and 'public health practitioners'. While the experiences of Indigenous public health practitioners are important and distinct, they are outside the parameters of this discussion.

[5]The fact that Indigenous people live 20 years less than non-Indigenous people remains one of Australia's most infamous and unchanging statistics (Ring & Brown 2003).

DAY 1

Session 1: Groundwork

The concepts of 'identity', 'race', 'racism', 'Whiteness' and 'culture' were introduced along with the notion of 'constructions' of Indigenous ill-health (Robinson, 1999; McIntosh, 1990).

Session 2: Explaining health inequalities

Small groups considered the reasons used to explain various health problems of Indigenous people, which were then mapped into categories and related to existing theories of health inequalities. Existing multi-level models of Indigenous ill-health were then analyzed in relation to these theoretical perspectives.

Session 3: The cultures of public health

A guest presenter surveyed the history of Indigenous health research (Thomas, 2001) followed by a plenary session mapping exercise of the characteristics of past and present research. A second speaker presented a recent ethnography of public health bureaucrats in Northern Australia (Lea, 2002).

DAY 2

Session 4: The use of culture in public health

The constructions of culture implicit in a range of written, visual and video texts about Indigenous ill-health were analyzed in small groups (Bartlett, 1995, p. 208) and a synthesis of constructions of culture in Indigenous health was discussed.

Session 5: White identity theory

The Implicit Association Test (https://implicit.harvard.edu/implicit/demo/) and an excerpt from *Spinning Into Butter* (Gilman, 2000) were discussed. A small group exercise using the White Identity Stages (Helms, 1992; Helms, 1995) was undertaken and the associated theory was considered.

Session 6: Where to from here?

Learnings from the workshop were consolidated including recommendations, of the authors, on the use of 'race' and 'culture' in public health practice and discussion of insightful quotes, from various sources, on issues of 'race', 'culture' and 'health'.

Fig. 1. Outline of workshop program.

the implications of these different constructions of Indigenous ill-health for their practice. Sessions generally consisted of an explanation of an exercise by the facilitators followed by work in small groups and then plenary discussion. After each exercise, relevant literature from a range of social science disciplines was presented (see Fig. 1 for an overview of the content and see also Kowal, & Paradies, 2003).

The workshop was prepared over the course of a year on a part-time basis by the two authors, who also facilitated the event. It was made clear at the outset that the facilitators' role was to elicit ideas from the participants as fellow public health practitioners, and not to act as experts who could provide answers or moral judgments on the issues that arose during the workshop. The workshop created a space of honesty and trust where participants talked to and challenged each other, disclosed personal experiences and expressed strong views and opinions, which were not dominated by particular participants or viewpoints. In this sense, the workshop was similar in method to a focus group, where facilitators structure the topics that are covered but not the way they are discussed (Wilkinson, 1998; Albrecht, Johnson, & Walther, 1993).

Workshop participants were recruited through an advertisement distributed via the CRCATH e-mail distribution list, which includes several thousand recipients. Twenty-one non-Indigenous practitioners attended the workshop, made up of students, professional researchers, bureaucrats, and clinicians. During the first session participants (and facilitators) discussed various aspects of their subjective identity. All participants identified their race and gender, with most being female (14) and White (18).[6] Participants also

[6]In this paper the term 'White' is used rather than 'non-Indigenous' in order to draw attention to the racialized aspects of White identities (rather than leave 'White' as an unmarked, invisible category), and because we do not attempt to account for the experiences of non-White non-Indigenous people.

discussed their professions, hobbies and sports, ancestry, nationality, religion, where they had lived, and their significant relationships. In the findings below we are unable to do justice to the diversity of identities held and expressed by participants. The reader should be mindful of this throughout the paper, particularly as the use of paraphrasing rather than quoting (for purposes of confidentiality) may create the misimpression of a unified voice.

As workshop facilitators and as authors, our subject positions form an important aspect of the research process. The first author is a White Jewish female clinician and doctoral student in public health anthropology, and the second author is an Aboriginal-Anglo-Asian male doctoral student in social epidemiology. The Indigenous facilitation of a non-Indigenous group is a significant, although complex, feature of this workshop. Here we will only note that the second author's identity as a professional researcher was predominant in this setting and participants did not explicitly engage with the second author's Indigeneity during the workshop discussions.

The plenary discussions of the workshop were transcribed, in the form of minutes rather than verbatim, directly onto a laptop computer by a scribe who was present throughout the workshop. The minutes were read within a week of the workshop, and discussed by the authors (and scribe) with corrections made as required. Written consent was obtained from each workshop participant to have minutes used for analysis, evaluation of the workshop, and publication of the findings. A grounded theory approach to analysis was used in which a thematic landscape emerged from the participants' discourse itself rather than being defined a priori. This involved iterative cycles of reduction, analysis, and formation of typologies through coding as well as theory testing (Miles & Huberman, 1994). A draft of the workshop report (Kowal & Paradies, 2003) was e-mailed to participants and their insights and responses were incorporated into further stages of the analysis.

We acknowledge that this research is opportunistic in nature, stemming as it does from a voluntary professional development activity. Workshop participants did not form a representative sample of public health practitioners in Australia and only minimal validation of our findings in other places or over time has been undertaken to date. As a result of the institutional setting of both the authors and the workshop, this analysis privileges the concerns and experiences of researchers over other public health practitioners, and privileges a northern Australian perspective.[7]

[7]Note that the majority of Indigenous Australians live in south-eastern Australia (see ABS & AIHW, 2003).

Findings

Structure and agency

Notions of structure and agency arose frequently throughout the workshop. Although contemporary sociology offers complex accounts of how structure and agency are co-constructed (Hays, 1994, p. 61–65), in the workshop 'common-sense' understandings of these concepts were used, defining structure as that which constrains and determines choices, and agency as the ability of subjects to freely determine their actions.

In Session 2 these notions were illustrated when the participants were divided into four groups, and each of these groups was asked to consider one of four health problems that afflict Indigenous communities: diabetes and coronary heart disease; end-stage renal disease; poor housing and environmental health; and poor obstetric and infant outcomes. We asked the small groups to think of all the reasons that Indigenous Australians suffer these problems at higher rates than non-Indigenous Australians. We specifically asked people to include 'non-politically-correct'[8](non-PC) reasons, and we used examples of both conservative and liberal newspaper articles to illustrate the task. Reasons were written on large post-it notes by each group.

After generating many different reasons, the groups were asked to come together and arrange their reasons into categories of their choosing (reproduced in stylized form in Fig. 2). Examples of reasons in each category are given in Table 1. 'Non-PC' examples were identified as such by the groups that wrote them. In the table below they are marked with an asterisk.

The results of this exercise were insightful. There was a clear tendency towards structural attributions for Indigenous ill-health, including reasons grouped under *the health system*, *historical context*, *money/financial*, and *remoteness* categories. Complementary to this, there was discomfort with explanations that stressed agency, demonstrated by the fact that nearly all the reasons that were identified as politically incorrect were within the

[8]The term 'politically correct' was not explicitly defined but its implicit definition may have been: A statement which would be seen by the mainstream left/progressives/liberals as racist. While people would have agreed that the reasons they cited were politically-incorrect, it is unclear whether all participants thought those reasons were *wrong*, or were uncomfortable using them. It is likely that some participants thought the politically-incorrect reasons were right but were not comfortable using them. All participants, however, *knew* what was politically (in)correct, even if they may have disagreed with that designation.

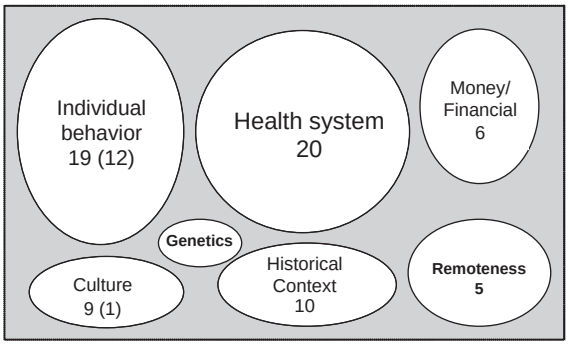


Fig. 2. Schematic representation of reasons given for excess Indigenous ill-health. Subset of non-politically correct reasons shown in brackets.

individual behavior category.[9] That is, participants were more likely to blame the system, and were reluctant to nominate Indigenous people's choices or actions as a cause of their ill-health.

More can be gleaned from the reasons within the *individual behavior* category which participants did *not* consider to be politically incorrect. Terms like "wanting to share in a non-healthful behavior identity" were necessary in order to speak of Indigenous agency in a politically correct way. This laborious phrase indicates the lengths to which practitioners' must go in order to feel comfortable with the fact that Indigenous people may freely make 'morally unsound' decisions.

This ethos of political-correctness is clearly a response to notions of *victim-blaming*, a term given to expressions of Indigenous agency that are generally seen as racist (for instance, blaming Indigenous ill-health on their cultural practices). However, as one participant put it—"in our attempt not to blame the victims we are *victimizing* them." The notion of victim-blaming assumes that people are truly victims, while the term *victimize* suggests that people's status as victims is created by external actors.[10] Both terms accentuate structure, and minimize the agency of Indigenous people.

In saying that participants were uncomfortable with agency, we do not mean to imply that they would all have agreed that the politically incorrect reasons were racist. Rather, all participants recognized that *the progressive ethos that has created these implicit definitions of political (in)correctness is a philosophy uncomfortable with agency.* This ethos appears to be dominant in contemporary Indigenous public health in Australia. It results in what we call an *overstructuration* of Indigenous ill-health, where Indigenous agency is inherently problematic and must always be deemphasized.

Sameness and difference

Despite our suggestion above that public health practitioners are uncomfortable with Indigenous agency, they do make statements about Indigenous agency all the time, and this workshop was no exception. In discussing what Indigenous people do, don't do, should or shouldn't do, the tension between sameness and difference was a recurring theme. Are Indigenous people the same as non-Indigenous people, but with different living circumstances, or are they different? Or are they the same, but in a different way?

As part of the workshop, participants viewed a video concerning the problem of petrol sniffing in Indigenous communities (Communications Branch, 1986).[11] The video excerpt featured a White nurse who worked in a remote Indigenous community. She recounted how a recent and gruesome child death from petrol sniffing in the community did not lead to a sustained reduction in the rate of sniffing because, she explained, Aboriginal people attributed the death to sorcery rather than to petrol. She describes how other Whites living on the community initially believed that the death would show the community the ill-effects of sniffing and thus lead to a reduction in this practice.

In response to the video, one participant commented that sorcery was really about social relationships. Others thought that the video showed that Indigenous and White people had different ideas of causation. White people, it was suggested, perceived petrol sniffing as the cause of death, and saw this as a means to make general statements about the dangers of petrol sniffing. In contrast, it was proposed that Aboriginal people were more interested in the particularity of the boy involved and how he came to be in that place at the time of the 'accident'. Another participant suggested that an initial reduction of petrol sniffing in the week following the death may not have been a 'normal' (if temporary) response to the death (i.e. community elders stopping children sniffing), but instead could have been due to the petrol sniffers being distracted from their sniffing by 'funeral business' (the Aboriginal English term for a funeral and its associated organizational requirements).

[9]The explanation of 'genetics' was mentioned only once and not discussed further during the workshop. This lack of discussion is related to the complex status of racialised genetics in public health, which will be explored further in subsequent work.

[10]In the context of this quote there is the suggestion of the paradoxical 'double victim': a victim of being labelled a victim. This trope has been discussed by others in Australia as 'victimhood' or 'victimology' (see Cowlishaw, 2004).

[11]Petrol sniffing is a significant problem for youth in many remote Aboriginal communities associated with injuries and lead poisoning (see ABS & AIHW, 2003; Brady, 1992).

Table 1
Reasons given for excess Indigenous ill-health by category

Category	**Number** (non-'PC' subset)	**Examples**
Individual behavior	19(12)	Having kids too young*, wanting to share in a non-healthful behavior identity (such as "drinking culture")
Culture	9(1)	Community dysfunction, different beliefs about health behavior.
Health system	20(0)	Culturally inappropriate interventions, poor choice of health services, cycle of disadvantage, lack of interpreters, institutional racism, lack of control
Historical context	10(0)	Past and present discrimination, forces changes in ways of living
Money/Financial	6(0)	Poverty, welfare dependency
Remoteness	5(0)	Expensive to provide care, problems with maintenance of health hardware

Subset of non-politically correct reasons shown in brackets.

These are complex explanations. Overall, their effect is to make Indigenous reactions understandable, to show that their actions, while superficially illogical, make sense when you appreciate their different context. In the comment about "funeral business", the speaker is saying that even when their behavior is the same (a decrease in petrol sniffing following a death), the rationale behind it is different (funeral business rather than a realization of the dangers of petrol sniffing). Thus the subsequent resumption of petrol sniffing after the initial decrease is deemed intelligible. In this process of 'making sense' of apparent difference, Indigenous people are preserved as morally sound health-seeking citizens.

Ambivalent subjectivities

In the first session of the workshop, we asked participants to think of all their different identities. For a number of people, their work in the Indigenous arena formed an important part of their identity, particularly where their social world included people who did not share their progressive ideologies. Individually and as a group, there was much difficulty in establishing a term to describe a White person who works in Indigenous health. "Indigenous helper", "altruistic", and "Southern pinko do-gooder"[12] were all put forward as possible candidates (the last one facetiously), and later in the workshop the label WHIPS—White Helpers in Indigenous Projects—was suggested (also at least partly in jest).

In Session 5 we considered the merits of the phrase, 'I help Aboriginal people to help themselves' for encompassing what practitioners in Indigenous health do. A number of people thought that 'help' was a paternalistic term, and that the phrase implied "imposing self-determination." As a group, the alternative phrase, "I support Aboriginal people to fulfill their aspirations" was suggested. However, some participants still felt dissatisfied with it, as "people aren't always in the position to identify their problems."

There was a general level of discomfort about finding a label for 'us', "whose job it is to do something," in this case, to improve Indigenous health (Ferguson, 1990). This unease was expressed in humor, as well as in the inability to come up with an adequate label. Practitioners felt disturbed by their own agency.

Many practitioners believe they are working to benefit Indigenous people, or at least recall that this was a major motivation for their entry into Indigenous health. However, some workshop participants were dubious about the likelihood of Indigenous participants benefiting from a public health project, while "ninety-nine percent of the time, practitioners are going to benefit."

This discomfort was also revealed in the language of 'victimology' discussed above. Practitioners are aware that their efforts to combat 'victim-blaming' may result in 'victimizing' Indigenous people. This double-bind has no obvious resolution, but points to the inherently problematic nature of practitioner agency in the uneven power relations of the former settler-colony.

Discussion: Maintaining moral integrities

We have shown above how 'political correctness' works to emphasize structure and downplay agency; how 'sameness' is emphasized to maintain moral similarity between practitioners and Indigenous people; and how ambivalence about the identities of practitioners reflect uncertainty about both the efficacy and the benevolence of their interventions. In this section we argue that these issues raised by practitioners as they ponder Indigenous ill-health can be understood as

[12]'Southern' refers to the south of Australia. White people who are long-term residents of the Northern Territory are stereotypically conservative, working-class, hold racist views, and look with disdain at stereotypically liberal-minded, middle-class Whites who grow up and are educated in the southern states of Australia before moving to the Northern Territory to work in the Indigenous arena.

strategies to manage the relativist possibility discussed above.

We suggested above that Indigenous public health is based on two parallel moral obligations: the moral obligation of the citizen to maximize one's health, and the moral obligation of the State to improve the health of the Indigenous minority. In postcolonial Australia, many White Australians feel a personal moral responsibility to address Indigenous disadvantage, as evidenced by the grass-roots Reconciliation movement. Many public health practitioners are among those who have assumed the State's moral obligation towards Indigenous health.

Just as the obligation of the State to provide health is linked to the obligation of the ill to seek their health improvement, the moral integrity of practitioners and Indigenous people are interlinked. The possibility that Indigenous people's ideals of health are truly different from Western ones jeopardizes the moral integrity of both Indigenous people and of practitioners. In the moment that this relativist possibility is seriously entertained (see Folds (1993), for a striking example of this), two parallel immoralities are exposed. Indigenous people who disregard public health messages are revealed as not really caring about their 'health' and thus are beyond any intervention; and practitioners are revealed as imposing their Western views on Indigenous people and thus further colonizing them. In order to maintain the moral integrity of both practitioners and Indigenous people, this relativist possibility must be resisted.

The material presented in the previous section can thus be interpreted as attempts to stave off the possibility of relativism and thus maintain the respective moral integrity of practitioners and Indigenous people.

The *overstructuration* of unhealthy Indigenous behaviors (attributing them to structure and not to agency) works to fend off the possibility that Indigenous people freely make unhealthy choices based on a non-Western model of health and life more generally. To maintain the moral integrity of both Indigenous people and practitioners, Indigenous people must always be potential public health subjects who will naturally adopt healthy behaviors once the appropriate barriers are removed.

This analytic provides insight into the controversy created by Indigenous social entrepreneur Noel Pearson's insistence that external intervention will not improve the situation of Indigenous people, and that progressive tolerance of Indigenous 'bad' behaviors is damaging (2000). Similar debate surrounds anthropologist Peter Sutton's suggestion that violence in Indigenous communities is not purely the result of colonizing influences, but was a feature of the pre-colonial era (2001). These analyses challenge the overstructuration of Indigenous choices that helps maintain the moral integrity of public health intervention.

We have noted that practitioners' discussions of sameness and difference work to make Indigenous actions understandable. When faced with an apparent difference that might allow the relativist possibility an entree, workshop participants emphasized the inherent rationality of Indigenous actions.

This approach could be described using development theory as *utilitarian*, where the different behavior of Indigenous people is explained as rational within its own context. A competing theory is the *dualist* theory, which contends that people act irrationally because of their cultural difference (Ferguson, 1990). As a further example to the petrol sniffing situation described above, let us take, for example, the observation that cars are more quickly damaged and overused on Indigenous communities.[13] A dualist explanation of this may be that Indigenous people assign little value to material goods, perhaps related to their history as semi-nomadic peoples. A utilitarian explanation may be that Indigenous people have to share resources with so many kin that when they finally get their turn, they utilize the resource to its fullest extent despite the damage that may be done.

A dualist approach would cast aspersions on both Indigenous people and practitioners, while utilitarian explanations maintain the moral integrity of both. Actions that may seem immoral to public health practitioners, like petrol-sniffing, are reframed by phrases like "want to share in a non-healthful behavior identity" as understandable—anyone would do the same in those circumstances.

Finally, the uneasiness practitioners display when defining their professional identities can also be understood as uneasiness with the relativist possibility. If the notion that Indigenous aspirations are completely different to those of practitioners is allowed to flourish, practitioner's actions will be seen as "imposed self-determination" or neocolonialism.

We can further examine the concept of self-determination as a key analytic in Australian Indigenous politics, and arguably the central 'special right' of Indigenous people. The liberal humanistic philosophy of public health practitioners (Lupton, 1995, p. 2) is aligned with the discourse of self-determination, a concept that emphasizes the celebration of moral and cultural pluralism, valorized notions of Indigenous agency awaiting release from dominance and oppression, and a prohibition against White power in the Indigenous world (Cowlishaw, 1999, p. 223–243). Since at least the first National Aboriginal Health Strategy in 1989, government rhetoric has considered self-determination a key strategy in improving Indigenous health

[13]Note that we are *not* commenting here on whether this observation is true or not, just that it exists as an observation made at times by, for example, White staff of community health centres commenting on the health center vehicles.

(National Aboriginal Health Strategy Working Party, 1989).

We can productively map the two concepts, self-determination and neocolonialism, onto the notions of universality and particularity that we began with. In effect, these concepts mark the boundaries where the universal and particular overlap and clash. 'Neocolonialism' marks the space where 'special' rights, including the right to self-determination, are infringed upon by Western universalist discourses. Thus efforts to improve health become neocolonial when it is perceived that they clash with self-determination principles.

At the same time, the possibility of *not* intervening is not open to public health practitioners who seek to accept responsibility for harm inflicted on Indigenous people by the State. The need to "propose remedies for distress, answers to social problems, and plans to improve the social world" is a "feature of the citizenship practices of socially responsible metropolitan residents" in Australia generally (Cowlishaw, 2004, p. 177).

Thus the practitioner is left in a bind: compelled to act, but always in danger of inflicting further harm. Cowlishaw takes this point further, provocatively arguing that intervention aimed at helping Indigenous people can only make them White. Aspects of 'culture' acceptable to cosmopolitan tastes are marked for preservation, with the remaining difference targeted for 'correction': "…the marginal can only ever be temporary and partial; it is always headed toward the mainstream if redemptive strategies are correctly shaped. The onus for change is inevitably one-way" (Cowlishaw, 1999, p. 248).

Whether or not Cowlishaw is right in asserting that intervention is by definition assimilationist, it is clear that the need of public health practitioners to maintain their own moral integrity leaves them trapped in an inescapable bind. They must inhabit only the rhetorical space of public health that can be made to speak the language of self-determination. Their impossible task is to relieve the ill-health of Indigenous people without acting upon them; changing them without declaring that change is required.

Conclusion

We have argued that the tension between universality and particularity animates the narratives of public health practitioners, whose moral integrity is intertwined with that of Indigenous people. This postcolonial predicament has implications for other sites where the benevolence and effectiveness of State intervention is questioned. Critiques of international development have accused development efforts of furthering the agendas of Western powers without real concern for local peoples (Escobar, 1994) and similar debates have occurred around efforts to alleviate sickle-cell anaemia for African-Americans (Wailoo, 2001). While little scholarship in these areas has addressed the binds we have discussed, the issues highlighted here are likely to be pertinent wherever States have attempted to grant minority groups special rights while simultaneously acting to improve their social conditions.

The contradictions that cultural relativism holds for universal goals such as 'health' are unlikely to be solved in the near future. However, ethnographic studies of those who are living the contradictions, such as public health practitioners in postcolonial situations, may yield important insights to this end.

The format of a professional development workshop is another approach that can effectively stimulate debate around the complex issues inherent to postcolonial intervention, particularly when the facilitators are also colleagues. While our evaluation did not attempt to objectively assess the impact of the workshop on participants' practices, their written feedback suggested that the workshop "will contribute to the quality and thoughtfulness of research in Indigenous health".

The discursive work of preserving the moral integrity of both Indigenous people and public health practitioners is clearly necessary for the practice of intervention. It is difficult to continue to work tirelessly on a task you consider fundamentally flawed, or for people whom you cannot understand in sympathetic terms or whom you consider to be immoral. However, we do not suggest that practitioners start to think of themselves or Indigenous people unsympathetically. We only suggest that they become aware of the perceptual filters and rationalities which they may employ in the project of public health.

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Conceptual diagrams in public health research

Yin Paradies, Matthew Stevens

Constructing a conceptual diagram is as much art as science

Conceptual diagrams, models, and frameworks are often used in public health literature to help illustrate relations between health exposures and outcomes. However, it is not always clear what these conceptual tools are describing, what they mean or how best to use, design or present them. In fact, the gaze of even the most critical reader tends to glaze over when suddenly faced with a conceptual diagram that is barely, if at all introduced, let alone explained in the accompanying text.

The online Oxford English Dictionary defines a diagram as a "set of lines, marks, or tracings which represent symbolically the course or results of any action or process" or a "delineation used to symbolize related abstract propositions or mental processes". Adapted from what Earp and Ennett[1] call a "conceptual model", we define a conceptual diagram as a "diagram of proposed relationships among a set of concepts, factors, or variables about a particular hypothesis, question, context, problem or topic". Conceptual diagrams are used to organise and synthesise knowledge, define concepts, provide explanations for causal and associative linkages, generate hypotheses and specific research questions, plan and target interventions, designate variables to be operationalised, and anticipate analytical approaches.[1] Conceptual diagrams delineate the scope of an inquiry and break up phenomenon into manageable pieces that map out the conceptual terrain in an area of interest.[2] These diagrams also assist in: interpreting statistical models, identifying knowledge gaps, directing future research,[3] and transferring research into policy and practice.

A cursory search through recent issues of the *JECH* uncovered a number of conceptual diagrams that we use below to illustrate key points in the use and misuse of these diagrams in public health research. Excluding mathematical/statistical diagrams and protocol/organisational flowcharts, conceptual diagrams can be categorised into two main types: causal/associative and descriptive/structural, with some diagrams including aspects of both types. Causal diagrams usually consist of polygons containing objects linked by (weighted) arrows and are designed primarily to show the aetiology of a topic that has been delineated into its constituent components. Figure 1, as reproduced from an article by Starfield, is an example of this type of conceptual diagram.[4]

Descriptive/structural diagrams are designed to clearly delineate layers of phenomena or types of objects to aid in organising and synthesising knowledge, designating variables, and so on. Figure 2, as reproduced from Barrett *et al*, is an example of this second type of conceptual diagram.[5] Causal/associative models generally provide predictions that can be tested and falsified whereas descriptive/structural diagrams provide paradigmatic ways of thinking through phenomenon.

The most common failing in the use of conceptual diagrams is their inclusion in an article devoid of sufficient description in the text. This is akin to presenting tabular or graphical data without any interpretation and, not surprisingly, severely limits the utility of these diagrams for potential users. As an exam-

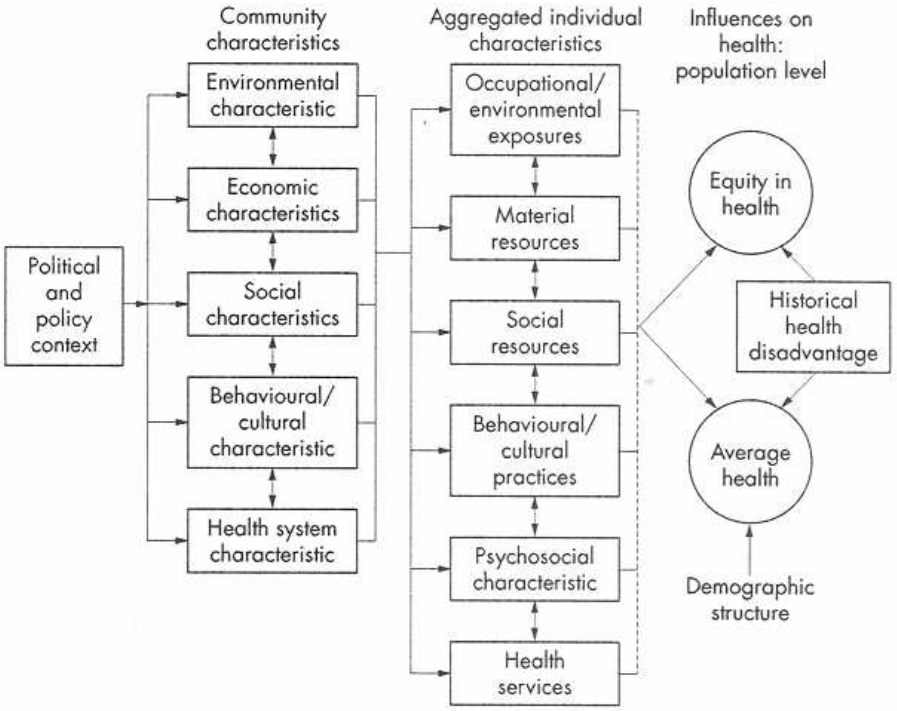


Figure 1 Example of a causal/associative conceptual diagram.[4]

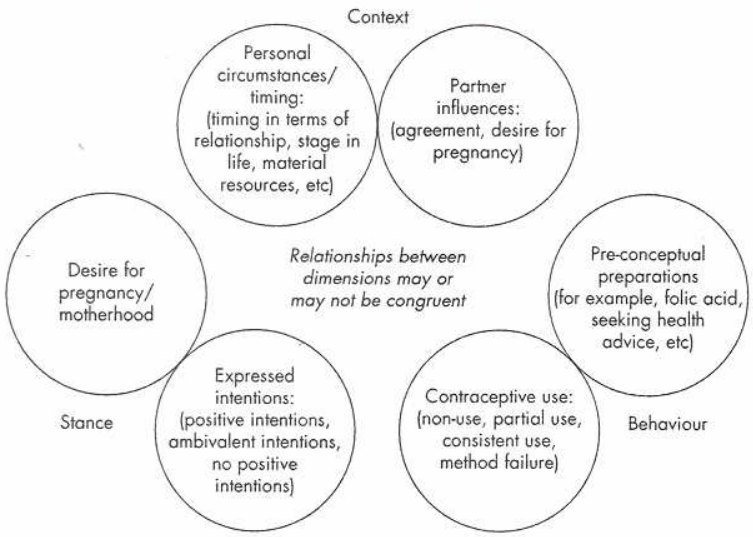


Figure 2 Example of a descriptive/structural conceptual diagram.[5]

ple, figure 4 in Moreau *et al*, which is a three dimensional conceptual diagram depicting the relation between job conditions and sick leave as mediated by a mix of positive and negative health related outcomes, is presented with no explanation whatsoever.[6] In contrast, Starfield devotes over a quarter of her editorial to describing her conceptual diagram.[4]

Before creating a conceptual diagram it is important to decide on its scope: what is the topic of interest, how many levels/types of phenomenon will be included to convey the necessary relations and what portion of the causal web or typology will be described. The best conceptual diagrams are explicit about their scope, are informed and described by theories and/or empirical evidence and "parsimoniously convey complex information, allowing the viewer to quickly visualize and grasp complicated relationships".[1] Diagrams that attempt to cover too great a scope as well as those that could just as easily be described in a single sentence should be avoided.

For instance, the figure included in Droomers *et al*[7] which consists of three boxed concepts joined by two arrows (between boxes one and two and between boxes two and three) in a single causal line, is described sufficiently in the text as: "the association between parental socioeconomic status and adolescents' alcohol consumption might be explained by...predictors of high alcohol consumption".[7] Nothing is gained by the inclusion of a conceptual diagram in this case.

Given the recognised complexity of developing meaningful conceptual diagrams, public health researchers have been slow to adopt multiple spatial and temporal scales, nested hierarchies of socioeconomic and biophysical environments and feedback loops between phenomenon,[8] as used in disciplines such as ecology. However, as conceptual diagrams incorporate ecological approaches to situating individuals in social, cultural, and physical worlds,[9] it is vital to retain a "clearly designated category for the individual"[10] so as not to fall into the trap of over-emphasising structure at the expense of individual agency.[11]

It is important to recognise that "constructing a conceptual [diagram] is as much art as science".[1] Although there are no hard and fast rules or easy checklists for their construction it is, none the less, clear that readers appreciate elegant, informative, visually edifying, and adequately described diagrams, making it all the more important that the construction and use of conceptual diagrams is taken seriously in public health research.

What this paper adds

This article seeks to focus attention on the role of conceptual diagrams in public health research by illustrating key points in relation to the use and misuse of diagrams

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Confounding and Interaction in Epidemiology

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Confounding and interaction are two of the most fundamental concepts in epidemiology. These concepts are also the topic of significant debate and create considerable confusion for students of epidemiology and epidemiologists alike. The concept of confounding has a long history in epidemiology with discussion in textbooks throughout the 20th century, whilst the concept of interaction was not distinguished from confounding until the publication of *Theoretical Epidemiology*[1] in 1985.[2] In this paper I attempt to clarify the meaning of these concepts as applicable to epidemiological practice and would welcome any further explication from readers of the AE that would shed light on these concepts.

Confounding

The term 'confounding' refers to at least four distinct concepts as used in the fields of epidemiology, psychology, statistics in general and experimental design in particular.[3] However, here I will focus only on the epidemiological concept of confounding which refers to the inter-mixed effects due to non-random distribution of risk factors within a study population.[4] The notion of statistical confounding – the non-collapsibility of the association between a factor (A) and outcome (O) over levels of a factor (B)[3] – is often conflated with epidemiological confounding. Although I don't have the biostatistical expertise to cogently describe how these two concepts differ, it has been clearly shown by others that epidemiological confounding is based on notions of causality whilst statistical confounding is a purely mathematical concept that can occur with or without epidemiological confounding and vice versa.[3]

Epidemiological confounding (hereafter 'confounding') can be defined by adopting three conditions which are both necessary and sufficient.[5] First a confounder (B) must be a risk factor – that is, it must be an actual cause of (i.e. on the causal pathway leading to) a particular outcome of interest (O).The important point here is that the association between the confounder (B) and outcome (O) must not derive *only* from a statistical correlation with another factor (A) which is a risk factor for the outcome (O) (as this is a case of a proxy risk factor as discussed below). Second, a confounder must be statistically correlated with the factor (A) which is of interest in the population in question. In combination with the first condition this effectively means that factors (A) and (B) must have a common cause or set of causes at an earlier point in the causal web. So, although (A) and (B) are by definition, not *directly* causally related to each other, there is frequently a reason why these two variables are statistically correlated (i.e. other than systematic or random error) if a different scale of analysis is considered. For example, the fact that smokers are also likely to consume alcohol and vice versa is due, at a broader scale of analysis, to certain socioeconomic factors that lead to both alcohol consumption and smoking. The third condition is that a confounder must not be directly causally related to a factor of interest (A).[6-7] This third condition was originally formulated to differentiate between a confounder and a factor which wholly or partially mediates (i.e. is on the causal pathway) between factor (A) and outcome (O). Such mediators should not be treated as confounders at the risk of distorting the true causal relationships being investigated. The more general wording of this third condition is due to the fact that other factors which are either causally affected by factor (A) (without being a mediator) or causally affect factor (A) should also *not* be treated as confounders.[3,6-7] It should be noted that some authors consider the three criteria above to be necessary but not sufficient in the trivial sense that there may be several confounders that are perfectly balanced and hence produce no *net* confounding effect.[7]

Figure 1: B is a confounder of A in relation to O[8]

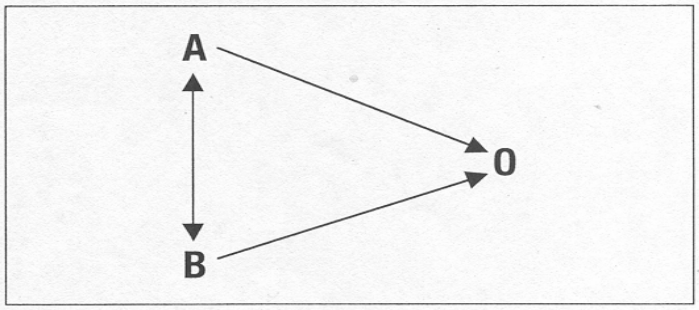
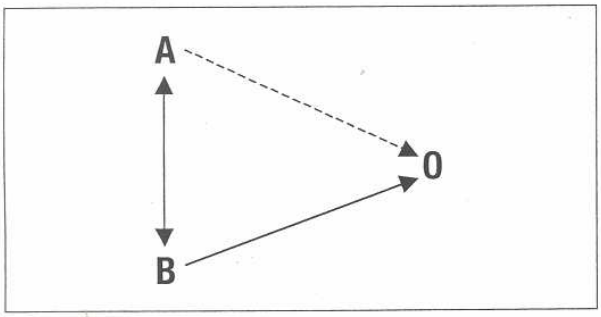


Figure 1 shows a classic three-variable example of confounding that illustrates how a risk factor (B) confounds the variable of interest (A) in relation to an outcome (O).

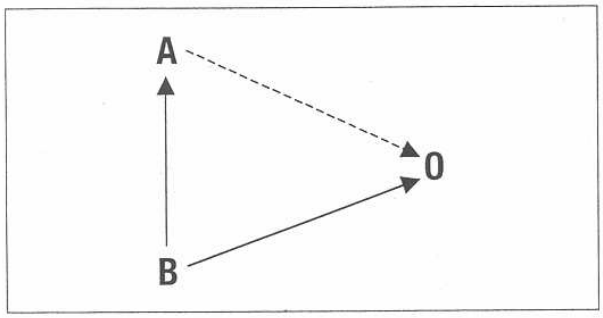
The choice of which factor (A) or (B) is the confounder of the other in the case depicted in Figure 1 is entirely dependent on which factor is of primary interest. Furthermore, it is also possible for the factor of interest (A) to be causally unrelated to the outcome (O). So, for example, there may be an interest in determining whether alcohol consumption (A) is related to oral cancer (O).[5] In this case, smoking (B) would act as a confounder of alcohol consumption (A) (in that it meets the three conditions above) which is *not* itself causally associated with oral cancer (O) (see Figure 2).

Figure 2: B is a spurious confounder of A in relation to O and A is a proxy risk factor for B in relation to O[9]



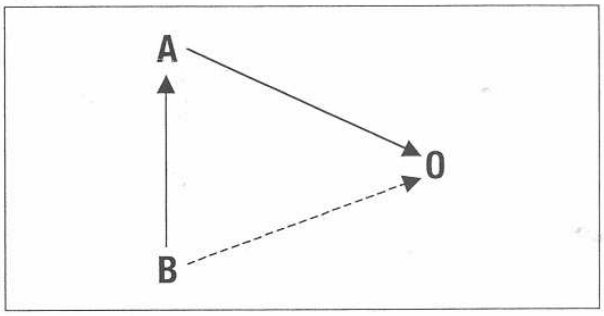
The concept of confounding can also be defined more broadly as any factor (B) that is related to a risk factor of interest (A) regardless of whether this confounding factor is causally associated with the outcome of interest (O).[3] Some authors refer to this latter class of confounders as 'spurious confounders', while confounders which meet the three conditions above are known as 'true confounders'.[2,8] The notion of 'spurious confounding' is conceptually identical to Figure 2 in which (A) would be considered a spurious confounder of (B) in relation to (O). Spurious confounders are simply instances of proxy risk factors[3,9] as shown in Figure 2 where (A) is a proxy risk factor for (B) in relation to (O) in that (A) is statistically correlated with (B) which in turn is causally associated with (O). Proxy risk factors can also occur as in Figure 3 in which (A) is causally associated, and hence also statistically correlated, with (B) which in turn is causally associated with (O).[9]

Figure 3: A is a proxy risk factor for B in relation to O[9]



For both spurious and true confounding the determination of whether, how much and what type of confounding has occurred relies upon aetiological knowledge of the factors under consideration[3,7] and, as with causation more generally, this determination cannot be made through reference to statistical evidence alone.[3] This fact can be seen by comparing the case of spurious confounding in Figure 2 with the statistically identical situation of true confounding in Figure 1. Similarly, the spurious confounding in Figure 3 is statistically identical to mediation (Figure 4) and can only be distinguished through causal reasoning.[10]

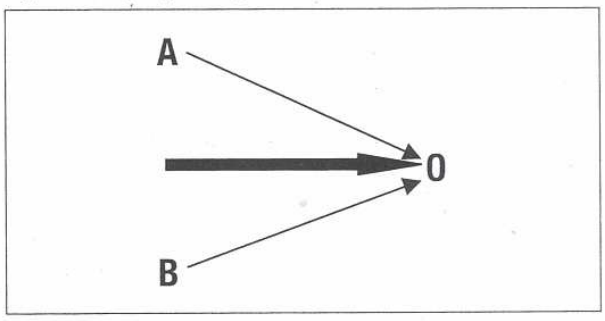
Figure 4: A mediates the effects of B on O[9]



Interaction

Interaction (also known as moderation or effect modification) occurs when two factors (A and B) are each independently causally associated with an outcome (O) and the co-occurrence of (A) and (B) modifies the *quality* or *kind* of their joint causal association with (O). If we consider the case where (B) moderates (A), then the presence of (B) affects the relationship between (A) and (O) without directly affecting the level/probability (i.e. distribution) of (A). Rather than affecting the distribution of (A), factor (B) instead produces a different causal chain between (A) and (O) based on the level/probability of (B).[9] For example, the relationship between hypertension (A) and stroke (O) is moderated by oral contraceptive use (B) in that the risk of stroke is greater in the presence of (A) and (B) then would be expected from the independent causal effects of (A) and (B) alone (see Figure 5).

Figure 5: B synergistically moderates A in relation to O

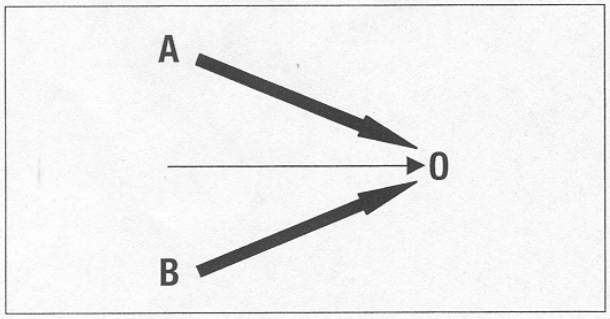


The defining feature of an interaction is that the effect of factor (A) cannot be determined without information about the presence (and level/probability) of (B).[5] As with true confounding, interactions expressed as '(B) moderates (A)' can also be expressed as '(A) moderates (B)' depending on which factor is of primary interest. In practice the distinct causal chain produced by factor (B) in the presence of

factor (A) must be sufficiently different to what would be expected from the independent effects of factor (A) *and* (B) on (O). This 'sufficient difference' is generally determined statistically through the presence of 'effect modification' (i.e. the nonadditivity of A and B). However, this differential causation is not a function of statistical interaction alone (which depends on which linear model is used[9] and power to detect difference) but rather is determined through an understanding of the aetiological processes in question.

Terms like susceptibility vs. resiliency, buffering vs. exacerbating factors as well as synergism vs. antagonism are all applied to interaction/moderating effects. Synergism occurs when the combined presence of (A) and (B) has an enhanced effect on the outcome (O) compared to their independent effects (known statistically as superadditivity, see Figure 5) while antagonism occurs when the combined presence of (A) and (B) has a reduced effect on the outcome (O) compared to the independent effects of (A) and (B) (known statistically as subadditivity, see Figure 6). When the outcome (O) is a disease or other non-desirable outcome, antagonism is synonymous with resiliency and also with buffering the effect of, say, factor (A) through (B) (i.e. B moderates A) while synergism is synonymous with the terms susceptibility and exacerbation.

Figure 6: B antagonistically moderates A in relation to O



Note: A uni-directional arrow from one factor to another indicates that the former causes the latter, a bi-directional arrow indicates statistical correlation without causation and a dashed arrow indicates a spurious association.

Confounding and interaction are two distinct concepts in epidemiology with differing characteristics as discussed above. However, it is important to note that a factor may act as both a confounder and a moderator for a particular outcome (O) in a particular population.[1] This occurs when there exists a factor (B) that is (1) causally related to an outcome of interest (O); (2) statistically correlated (but not directly causally related) to another factor (A) (i.e. true confounding); and (3) produces a different aetiological effect on an outcome of interest (O) in the presence of a factor (A) (i.e. interaction). This combination of confounding and interaction is equivalent to combining Figure 1 with Figure 5. For example, exposure to asbestos may synergistically moderate the relationship between smoking and lung cancer (as in Figure 5) at the same time as smoking and asbestos exposure are statistically correlated as in Figure 1 (perhaps through lower socioeconomic status as an underlying common cause).

It should be clear from the above discussion that differentiating between confounding, mediation, proxy risk factors and interaction/moderation relies heavily on sound epidemiological, and in particular aetiological, knowledge. The best tool for achieving this differentiation is a causal diagram which summarises available information on the relationships of interest (including confounding and interaction) in a particular study.[7] Such diagrams can be used both informally as an aid to analysis and formally using a range of statistical approaches.[11] However, given that "guidelines for determining causal pathways as commonly taught in epidemiology courses are woefully inadequate",[12] an improved treatment of causation in epidemiological teaching will be a necessary prerequisite to the appropriate construction and use of such diagrams.

Acknowledgements

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